MEDICATIONS OF PARKINSON'S DISEASE

OR

ONCE UPON A PILL:
PATIENT EXPERIENCES WITH DOPAMINE-ENHANCING DRUGS AND SUPPLEMENTS

JANICE WALTON-HADLOCK

MEDICATIONS OF PARKINSON'S DISEASE

OR

ONCE UPON A PILL:
PATIENT EXPERIENCES WITH DOPAMINE-ENHANCING DRUGS AND SUPPLEMENTS

JANICE WALTON-HADLOCK

ILLUSTRATIONS BY JOHN BATESON

Medications of Parkinson's Disease or Once Upon A Pill: Patient Experiences with Dopamine-Enhancing Drugs and Supplements

May, 2003
revised Dec 2003
© Janice Walton-Hadlock
copyright Library of Congress, USA, TX 5-787-941

The Parkinson's Recovery Project
343 Soquel Ave. #413
Santa Cruz, CA 95062
www.pdrecovery.org

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without permission in writing from the author.

DISCLAIMER

PLEASE READ THE FOLLOWING BEFORE READING THIS BOOK:

The attached writing about Parkinson's disease medications is not meant to be used by patients for the purpose of self-determining their appropriate medications or their correct doses. People with Parkinson's disease who are taking medications must, by law, work with their MDs to determine their best medications and dosages. The author of this material, Janice Walton-Hadlock, is NOT an MD (doctor). Janice Walton-Hadlock, the author, has no legal rights to prescribe medicine, nor does she pretend to any moral rights to prescribe medicine. The author, in this book and in person, cannot and will not make any recommendations about medications. Any person wishing to make modifications to dosage of prescription medications must work with his/her MD.

The author is not responsible in any way for any harm, either mental, emotional or physical, untimely death, or expenses that may be considered to have been, in full or in part, a result of this book. The word harm is understood to include any negative results of patients changing their medication regime with or without a doctor's prescription. The Parkinson's Recovery Project, a non-profit organization dedicated to educational and charitable work, the board of directors of the Parkinson's Recovery Project, and any person associated with the operations of the Parkinson's Recovery Project, including the webmaster and website provider, are not responsible in any way for any harm, either mental, emotional or physical, untimely death, or expenses that may be considered to have been, in full or in part, a result of this book. The word harm is understood to include negative results of patients changing their medication regime with or without a doctor's prescription. The author, the Parkinson's Recovery Project, and all persons associated with the Parkinson's Recovery project and its website are not responsible for any health care decisions that any person may make as a result of reading this book. The author and the Parkinson's Recovery Project are not responsible for any penalties or liabilities incurred by readers of these pages, including patients and doctors. The author is not responsible for any harm that occurs due to errors in the new theories and hypotheses that appear in this book. The reader must understand that it is the nature of new science to propose ideas that will most likely be found incomplete or even wrong by future researchers.

About the wording in this book: Despite the diligence of excellent editors, some typos and ambiguous or poor phrasings have doubtless slipped through. Please forgive these. And in case any usages of the word "will" or "will occur" have slipped past the proofreaders, please do the following: in such a case where the use of the words "will" or "will occur" appear to imply a definite, conclusive, future event or outcome, please substitute the words "may" or "may occur," respectively, in order to make it understood that the event in question might possibly, but might possibly not, occur.

Copyright: Janice Walton-Hadlock, author, has copyright on the material on this website, and no part of it is to be reproduced in any manner, including electronically, without the permission of the author.

Acknowledgements

It would be impossible for me to list by name all the people who have contributed to this book, starting with my supremely supportive parents, Anne Hadlock and Clay Hadlock, and ending with John Bateson and Paul Saxon who, respectively, volunteered the artwork and the graphs and suggestions for layout. Thanks must go to all the acupuncturists, doctors, and hopeful, heartful people with Parkinson's who have shared their experiences, thoughts, fears, and dreams. To all of you, my love and thanks.

And it would be ungrateful in the supreme if I should fail to thank Steve, my patient husband who literally fed me when I was too preoccupied to eat, who read my material when I could no longer tell what it said, and who forced me to sleep – and, as he would say, (Shakespeare-lover that he is) "perchance to dream" – when I had lost all sense of time and was incapable of knowing how many hours or even days had passed, absorbed in this collection of words. He edited, read, and proofread this book more thoroughly than any other readers and editors have done. There are not words to thank him enough. Steve, you know how much I love you.

Finally, I was uplifted throughout the difficult times, despite my patients' pain and fear, and the fear and doubt in my own heart, by the inspirational words of Paramahansa Yogananda: "God is love; His plan for creation can be rooted only in love…Every saint who has penetrated to the core of Reality has testified that a divine universal plan exists and that it is beautiful and full of joy."

In seeking to experience that Love, in delving for evidence of that universal plan that was hiding, cloaked, behind the drama of human suffering, I have been led to the findings presented in this book.

To You, my deepest gratitude,

- J

TABLE OF CONTENTS

Foreword…… i

1. Drug Dangers …… 1
2. Worrisome drug problems…….. 21
3. Three hypotheses…… 33
4. A case study: Becky……... 55
5. PD terminology…….. 73
6. Becky's journal…….…... 101
7. Dopamine distribution……... 115
8. Becky and Xanax……. 139
9. Addiction…….. 157
10. The anti-parkinson's drugs…….. 191
11. The big D: dyskinesia in depth…… 203
12. Leading up to a solution…….…. 217
13. The hidden significance of On-Off patterns…… 233
14. Cycles of change…….. 255
15. A ten percent solution…… 273
16. Strange behaviors……. 289
17. The last little bit……..301
18. Supporting our hypotheses…… 315
19. A two-year struggle……. 347
20. Too much pain…….….. 357

21. Glimmers of hope and other danger signs……. 369

22. Questions and answers…….. …. 395

23. Buzz and Hermann……...409

24. Variations in addictability……443

25. Becky and Mirapex……...……..…… 463

26. Statistics and conclusion……...……. 473

Appendix 1 - The various names of the anti-parkinson's drugs……...…..…… 485

Appendix 2 - Specifics about individual drugs…….…..…… 491

Appendix 3 - Symptoms of drug reduction/withdrawal…….. 535

Appendix 4 – Tips for surviving drug reduction……. 547

Appendix 5 – Why your doctor thinks what he does…… 555

Appendix 6 – Dopamine fallacies……...…….. 577

Appendix 7 – Alpha primate/cocaine study…….. 593

Appendix 8 – Working with your doctor…….. 603

Appendix 9 – Another Sinemet case study…….. 609

Appendix 10 – Drug-related pain in Parkinson's disease……..613

Appendix 11 – Guess work about Build-Ups……615

Appendix 12 – Alcohol……. 619

Index ……621

April 18, 2003

Foreword

This book grew out of a research project in which we used Asian medicine to treat Parkinson's disease. Our research led us to the realization that the decrease in dopamine is not the cause of Parkinson's disease but that somewhat the opposite is the case: Parkinson's disease is the cause of the decrease in brain dopamine.

Ignoring the dopamine decrease (a situation which slowly reverts to health all by itself once the PD-causative problem is removed), we work instead on the underlying trigger of the Parkinson's disease. We use a type of Asian massage to treat the energetic distortion around the unhealed, usually long-forgotten, childhood foot injury that was found, still unhealed, in every person in our Parkinson's research project. It appears, based on our patients' results, that idiopathic (unknown cause) Parkinson's disease is not only curable, but no longer idiopathic. For more information on this subject, please visit the website of the Parkinson's Recovery Project, a non-profit, charitable and educational organization, at www.PDrecovery.org.

This book about medication

At the beginning of our project, all our medicated patients were assured by their doctors that anyone who recovered from Parkinson's could simply stop taking his medication on that happy day when he woke up healed. These doctors were wrong in two ways. First, there never is an overt moment in which a person suddenly flips from Parkinson's disease to Radiant Health. Recovery, like the descent into Parkinson's, is gradual. And second, a person who has taken the drugs has probably set in motion permanent changes in his brain. He may never be able to stop taking the medication. He may die if he too abruptly decreases or stops taking his accustomed meds. He may die from side effects of his medication if he no longer has Parkinson's but continues taking anti-PD drugs.

Because there had been no effective treatment for Parkinson's disease in the past, there was no way of knowing what the medications might do to a person who used to have Parkinson's disease but who then recovered.

Most people in our program who were taking the medications continued to take them even though they could tell that their Parkinson's symptoms were easing up or changing. Based on what their doctors said, it seemed safe and reasonable for our patients to continue taking their medication even though they were showing recovery symptoms. In the early days, we all assumed, based on these breezy dismissals by the local MDs, that a recovering Parkinson's disease patient could continue taking medications with no ill effects.

Only after our recovering patients who continued with their medication quickly developed symptoms that appeared to be extreme, hideous parodies of the listed adverse effects of the antiparkinson's medications did we realize that the medications might behave utterly differently in a person who has recovered from Parkinson's disease than in a person with idiopathic Parkinson's. Many of our recovering patients were unable to make even slight decreases in their medication. The opposite was the case: their physiological need for the medication increased rapidly even as their Parkinson's symptoms ebbed; and yet the drug side effects became more ominous.

Then, several recovering patients who were still taking medication either died, developed life-threatening adverse effects requiring constant sedation, or went crazy.

At this point we decided that medicated patients should not enter our program. We posted this caveat on our website. Despite our warnings, people who were medicated for PD continued to recruit health practitioners to use those instructions for treating Parkinson's that we have posted on the Internet. More than a year after posting the warning, the most frequent emails coming into our office continue to be those pleading for help with the ghastly symptoms that can develop quickly in medicated patients after they began to exhibit symptoms of recovery.

Therefore, in order to provide a more convincing statement than "medicated patients should not enter into a recovery project," this book was written.

Who are we?

A common question from readers is, "Who is the 'we' that you refer to in this book?" "We" are members of the Parkinson's Recovery Project (PRP). A Berkeley acupuncturist colleague and I were the first two members of this group. We sat around on weekend afternoons of 1997 trying to figure out why Yin Tui Na, when applied to old, forgotten foot injuries, could alter rigid muscles in the arms and legs or an expressionless face, and how on earth dopamine levels could be altered due to a glitch in the foot. We recruited PD patients for free treatments so that we could test our theories.

In 1998 a friend volunteered to build and manage a website to distribute our theories and our request for verifications from other acupuncturists with PD patients. The "we" began to grow. Several local (Santa Cruz, California) acupuncturists volunteered to try these techniques on their own patients. A PDer from San Francisco named the loose-knit group the "Parkinson's Recovery Project" and his son-in-law wrote up the non-profit paperwork. A board of directors was recruited. This board currently includes two acupuncturists, an MD, and an editor/English professor. An acupuncturist in New York started treating patients in her hometown and gently but persistently prodded me to offer weekend classes. Five Branches Institute of Traditional Chinese Medicine in Santa Cruz offered space for a free clinic.

As more patients recovered from Parkinson's or not, notes from these patient cases were added to the free information on the website. Email came in from around the English- and French-speaking world with comments and questions about the website. The website information was translated into Portuguese, German, and French by volunteers with Parkinson's.

Several articles on our findings were published in acupuncture journals. The flood of new incoming questions and answers was added to the free website information. In 1999, John Bateson, a professional illustrator from Canada, in gratitude for his own turnaround from Parkinson's, sent a collection of illustrations to be added to the website information, which had grown to the size of a book. The illustrated edition of this book was posted on the Internet for free in June 2000. Since 1999, low-cost weekend classes have been offered in various US cities and abroad.

A group of Five Branches student interns (some of whom repeated my three-semester class on treating Parkinson's two times or more) and a group of local acupuncturists who regularly sat in on the classes and helped the interns became part of the "we." We started regular weekly meetings in Santa Cruz to compare notes on our

patients and on the bizarre medication problems that were arising. We set up a Yin Tui Na training program for visiting out-of-town health practitioners and their patients with Parkinson's. This program grew to become the PD Team of Santa Cruz (PDteam@cruzio.com).

By 2001, answering email queries was taking several hours every day. A volunteer acupuncturist now helps to answer emails, phone calls, and written missives. In 2002 a volunteer took over the job of mailing out hardcopy orders of the Internet book and answering mailed queries. One paid employee keeps track of the banking and bills. We have no fundraisers. We rely on unsolicited, tax-deductible contributions to cover the expenses of the project.

So in 2003, who are "we?" We are primarily the volunteers who keep the project afloat, the team of acupuncturists who have been working with Parkinson's patients in our own private practices, and the student interns and acupuncturists who volunteer at the free Parkinson's clinic in Santa Cruz.

Source of material for this book

Case studies referred to in this book are from my private patients and patients at the free clinic. Despite a tremendous amount of emailed case study material from health practitioners outside our local area, I have refrained from using those case studies in this book. So that I can stand firmly behind every example and case study put forth in this book, the patient case studies and statistics herein only include those of patients actually treated by me in my private practice or those patients in the free clinic, which I oversee.

What is in this book

This book includes hypotheses and brain models of dopamine metabolism that offer a way to make sense of the "unpredictable" nature of these drugs. There are examples of methods of drug reduction that were or were not successful. There are detailed descriptions of those adverse effects from drugs that can indicate overmedication and addiction. Finally, case studies are interspersed throughout, making more powerful examples than any mere list of symptoms. This is not a how-to book for drug reduction, nor is it a substitute for a well-informed neurologist.

Our hope

This book is intended to put the brakes on anyone who brazenly assumes that he can "start recovering" from Parkinson's and reduce his medications later. This book may be helpful for those doctors who are uncertain how to implement the manufacturers' vague suggestions to "decrease this drug slowly" or who have not intuited the significance of the phrase "may take up to ten weeks before effectiveness is apparent."

This book is most of all a tribute to the brave individuals who became inadvertent researchers when they began to recover from Parkinson's disease.

Janice Walton-Hadlock, Lac.
Director, Parkinson's Recovery Project
www.PDrecovery.org

"The desire to take medicine is perhaps the greatest feature which distinguishes man from animals."[1]

Sir William Osler, Regius Professor of Medicine, Oxford, co-founder of modern (western) medical methodology (1849-1919)

1. DRUG DANGERS

THE DEATH OF ROSE: THE BIRTH OF PIONEERS

People taking antiparkinson's medications that enhance dopamine should not try to recover from Parkinson's disease.[2] If they are dosed correctly (a remote contingency), the medications may be relatively benign, *if* their illness is actually idiopathic Parkinson's. However, when in the process of recovering from Parkinson's disease a person's nervous system reverts to the parasympathetic (non-injured, non-emergency) mode, the antiparkinson's drugs can rapidly become just as dangerous to body and mind as they are to any healthy person.

A person who is still taking antiparkinson's drugs when the brain shifts from addiction-resistant to full-blown addictability courts death and dementia. This shift can be abrupt, and can occur whether or not brain dopamine increase has become apparent. Although some people have managed to get off their medications in a timely fashion, or have struggled mightily against them after they started to recover and lived to tell the tale, they are the exceptions rather than the rule.

In response to the Yin Tui Na treatments that reverse the erratic electrical currents of Parkinson's disease, we noticed that medicated patients had wild and unpredictable changes.[3] We have formed several new hypotheses that apply to medicine in general, and Parkinson's in particular, based on four years of charts, graphs, and weekly, detailed reports from every one of our patients.

The hypotheses are these:

First, calculating the effective timeframe of psychoactive/psychotropic medication should be based on its short- and long-term effects in the brain, not on fleeting blood half-life levels as currently measured. Next, the brain does change in response to psychoactive and psychotropic drugs, so that the results of any given dose are affected by the preceding doses. There are both short-term (less than twenty-four hour) and long-term (possibly permanent) brain changes in response to any dose of these drugs.[4] Third, the chemical

---

[1] We now know that animals do self-medicate. However, I suspect that Sir William was referring to the desire, the mental attitude of man, not the nutritional aspect of medicinals.

[2] Antiparkinson's medications that enhance dopamine include not only L-dopa-containing drugs, but dopamine agonists, MAO inhibitors, antidepressants and anti-anxiety drugs (tricyclics, SSRIs, GABA enhancers, and **any** other drugs that directly or indirectly elevate dopamine. L-dopa-containing medications, dopamine agonists, MAO inhibitors and other dopamine-enhancing drugs and supplements will be discussed by name and function in following chapters and in the appendices. A list of dopamine-enhancing drugs (DEDs) is in appendix 1.

[3] For more information about Yin Tui Na and the Parkinson's Recovery Project, please read the forward of this book.

[4] This has since been proven by the National Institute of Drug Abuse in their research on dopamine-enhancing drugs.

basis of addictability and addiction resistance of an individual is altered by changes in emotional/social condition.[5]

Our hypotheses may be wrong. However, they provide a powerful new way to think about these drugs. Some patients who have applied these hypotheses, their conclusions, and corollaries, have been able to safely get off their medication. Other recovering patients, despite their awareness of our findings, were unable to apply them; upon their return to a parasympathetic condition, they were abruptly swept away on the tide of drug-induced insanity and addiction.[6]

Although these hypotheses may cause controversy, there is a possibility that they are helpful and even correct. Therefore, I am making public the findings of our program. This book contains the case studies that led to our conclusions, expansions and proofs for our hypotheses, warnings, and detailed descriptions of the methods used by those who successfully got off their medication.

However, the primary reason for writing this book is to make the point, as clearly as possible, that medicated patients should not attempt recovery from Parkinson's.

Naughty, unstable drugs

The antiparkinson's drugs are often described as "unpredictable" and "unstable." Our research suggests that the drugs are predictable; the traditional theories of drug metabolism are incorrect.

In treating Parkinson's patients using techniques of Asian medicine, we found that once the recovery from PD commenced, our sixty medicated patients' responded with behaviors dramatically different from those of the thirty Parkinson's patients, even those with advanced PD, who were not taking medication: most medicated patients that responded positively to our treatments rapidly developed conditions that apparently corresponded to the "adverse effects" or "overmedication" symptoms listed by their drug's manufacturer. Even if they reduced their medication in response to these changes, many of the medicated patients had wholly unexpected complications.

Trying to make sense of the patients' differences and complications, we examined over ten thousand logged hours of patient records and charts chronicling drug dosage, drug onset, duration, effects and adverse effects. Applying new hypotheses of drug metabolism to these patient records, we were able to create a construct in which these medications

---

[5] This has since been proven in a study on primates (see Appendix 7). SPECT scans of their brains showed altered addictability/receptivity to dopamine in response to adrenaline-producing conditions.

[6] Chronic immersion in the sympathetic system is often consciously sedated with methods ranging from meditation to food, or diverted into constant, unusually intense levels of focused activity by PDers. After a brief time, the sympathetic system may no longer manifest the outer symptoms of increased heart rate or bronchiodilation associated with this system. However, as most PDers will attest, inability to access the parasympathetic and a lifetime of heightened vigilance and harm avoidance suggest a chronic imbalance towards the sympathetic system. Furthermore, the PD tremor is an outward manifestation of a long-standing internal vibration, the rate of which corresponds to the theta wave – a wave that indicates stress or injury. Although the physical attributes of the sympathetic system can be trained and controlled using the terrific will power of the PDer, the limbic brain remains keenly aware that the system is in a chronic state of emergency.

Regarding theta brain waves, they "normally occur in children and in adults experiencing stress. They also occur in many disorders of the brain" (from *Principles of Anatomy*, Tortora and Anagnostakos, 5th edition, Harper and Row, 1987, p. 323). Theta waves also occur in some adults during deep meditation and during flotation therapy.

worked predictably. After we learned that these drugs were in fact performing in a logical fashion, we were able to make further hypotheses that accurately predicted how and in what time frame Parkinson's drugs affected PD symptoms and/or created drug-induced brain damage. These hypotheses were used by patients to create their own paths for drug reduction, paths that proved safer than the guesses usually recommended by physicians.

This book presents those hypotheses, with excerpts from medicated patients' case studies, a glossary of Parkinson's terminology, a small amount of brain physiology, an introduction to each of the various medications with details on their mechanisms and side effects, and references to the latest research. This book is not a how-to book, nor is it a substitute for an informed physician. However, we hope that it may be a fruitful contribution to the study of dopamine-enhancing drugs.

Warning

Despite the findings of our project, there is never any foolproof guarantee of safety when reducing antiparkinson's medications. Survival of drug reduction, even if using the formulas that we discovered, depends on the individual's age, accuracy of diagnosis, which drugs or combinations of drugs he is using and in which doses, his overall health, the availability of a stalwart, daily companion who can act as medication gatekeeper, and other factors too involved to mention in this first chapter.

We hope that this book will be used as a source of information for people who are considering taking medication for the first time, and by people whose medications are no longer working well and are considering increasing their medication despite the presence of dyskinesias, On-Offs and other symptoms of overmedication. It should occasion second thoughts in those who, despite the warnings, brazenly think that they can chance recovery even though they are taking drugs. It is especially for the latter group that I am writing this book.

The Parkinson's Recovery Project recommends that people who are taking antiparkinson's drugs should not try to recover from Parkinson's. Most medicated PDers have ignored this warning, necessitating the writing of this book. Hopefully, this book will put their brakes on: although this book shares the methods of drug reduction that were most successful for the most people, it also shares stories of tragic failure.

Background

This book is the outgrowth of a previously published book, *Recovery from Parkinson's Disease, A Practitioner's Handbook*.[7] That book offered a new theory on the cause of Parkinson's disease and described what appears to be an effective treatment. What was missing from that book was any information about the medications of Parkinson's disease. Because of a dearth of information about how to reduce these medications, the patients in our program became human experiments. Their doctors had no guidance in this area, as the pharmaceutical companies only describe how to increase these meds, not decrease them.[8]

I am not an MD and cannot give advice about any aspect of prescription medication, but I was able to work closely with these pioneers and write up their weekly reports.

---

[7] This book is available for free on the Internet at www.pdrecovery.org.

[8] The manufacturers of antiparkinson's medications usually make the vague suggestion that these drugs be reduced slowly. No specifics are included, nor is there an industry standard for the word "slowly."

Thousands of hours of interviews have been collected. Hundreds of bold patients, both in our clinic and from around the world, have contributed their experiences in person and via email. Through their patient trial and error, we were able to accumulate enough data to discern a mathematical formula for what appears to be the safest way of reducing the medications.

We also learned that there was much more to medication than the taking of pills. Often the most painful situations for recovering patients were not caused by reductions in medication but by the regrettable confrontations with doctors or family members when these reductions were attempted. The assumption of doctor infallibility on the part of concerned loved ones created much intra-family tension because patients were acting on their own initiative; they were "not following orders." The painful months of drug withdrawal were difficult for all the patients, but the clamoring of family members and resentment from some doctors added emotional pain to the physical torment. In recognition of that, this book includes information about doctor training – specifically, their lack of experience and knowledge in this field – and stresses the need for family support.

Because this book is written primarily for patients, I can make no assumptions of the reader's background in medicine. The book includes a thorough discussion of Parkinson's and drug side effect vocabularies, and the appendix contains a brief history of dopamine and how our understanding of this chemical has changed in the last five decades. Throughout, there will be case studies (brief descriptions of medical history and response to treatment) that illustrate our findings; these will include details of what patients attempted that was successful as well as what was not successful. The information in the book proper is with regard to all dopamine-enhancing drugs.[9] Specific details on each of the Parkinson's drugs are discussed, along with illustrative case studies, in the appendices. I will also briefly introduce in this chapter our findings and frustrations, and share the case study that pushed me into writing this book.

STRANGE HAPPENINGS

Our project began in 1997. We were treating both medicated and unmedicated PDers. Over 90% of the patients in the project began manifesting symptoms of recovery[10] within less than a year after starting treatment. We observed that people recovering from Parkinson's disease who weren't taking the medication had a pretty straightforward time of it. The recovery was painful, exhausting, emotional, and took a long time, but the stages of recovery were still fairly predictable. The people who were taking medication behaved, for the most part, in a completely different way. After receiving treatment, and starting to recover, as indicated by the reappearance of sensation and proprioception in the extremities, new emotions, changes in muscle behaviors and the return of healthy sleep patterns, some medicated people also had an extraordinary acceleration of adverse effects, many of which resemble Parkinson's disease symptoms. Their dyskinesias and

[9] The anticholinergics and other non-dopamine enhancing medications will also be addressed. The textual information about the brain is oriented to understanding how all the anti-PD medications work: information needed to understand the chemical-specific material in the appendices.

[10] See *Recovering From Parkinson's Disease, a Patient's Handbook*, available for free at www.pdrecovery.org, which lists the symptoms that occur during recovery.

freezings[11] rapidly worsened. Others, suddenly experiencing drug-induced euphoria, initiated rapid increases in their medications over a short period of time, sometimes taking three and four times as much medication as they had been taking just a few weeks earlier. They became radiantly illogical and either supremely paranoid or ludicrously self-confident.

This did not happen with all medicated patients. Those who slowly eased off their meds before they even recovered had a very different path from those who waited – they often became drug-free. Those who abruptly quit invariably failed to stay off the medication more than two weeks. They quickly resumed their meds – in most cases, at even higher levels than before.

We Ask the Doctors

We were certain that our patients' doctors could explain these puzzling drug changes that occurred during recovery. We were wrong. Some neurologists, when asked about medication side effects, simply humored their patients by telling them that after they had completely recovered from Parkinson's disease, they could just stop taking their medications – what could be simpler? They all laughed off the idea that anyone might ever recover.

Most doctors insisted that their increases in dyskinesia and medication side effects were coming from their Parkinson's disease, not the medication. Some doctors bristled at the idea of adverse effects, declaring that the drugs were perfectly safe, and that it was impossible that the medications caused the increasing insomnia, freezing episodes, and/or violent thrashing movements[12] – all symptoms listed in the Adverse Effects column of the medication inserts provided by the companies that made the drugs.

Impervious to Addiction

Meanwhile, we had learned from our relentless patient interviews that prior to their diagnosis with Parkinson's disease, most of our patients had proven themselves to be impervious to addiction. Many had quit smoking with ease; many had quit alcohol or strong drugs simply by deciding to do so. Not one PDer had ever had difficulty in overcoming an addiction – though many had used addictive substances. As a historical corollary, it had been recognized as early as the late 1960's that most non-PDers simply cannot tolerate L-dopa;[13] they rapidly become addicted, developing life-threatening side

---

[11] "Dyskinesias" and "freezings" refer to the excess and extreme abrupt loss, respectively, of movement. These and other side effects of the medications will be described in pitiless detail in the chapters ahead.

[12] As you will read in much greater detail later on, side effects such as violent thrashing, spasming, grimacing and writhing are NOT symptoms of Parkinson's disease, a disease characterized by increasing poverty of movement. The resting tremor and the rhythmic, exaggerated tremor that can occur in Parkinson's disease, especially during times of stress or anxiety, are the only forms of excessive movement that occur in unmedicated, idiopathic Parkinson's.

[13] There are some types of disorder, such as Restless Legs Syndrome, in which people appear to be able to tolerate dopamine-enhancing drugs without immediately experiencing the addiction symptoms. These patients evidently have some form of dopamine deficiency and elevated sympathetic condition. We have observed that these patients usually, within approximately five years if taking L-dopa, develop either Parkinson's disease or parkinsonism.

effects, even developing parkinsonism.[14] PDers can usually take L-dopa[15] for years before the strange side effects and addiction-driven changes appear. It appeared as if PDers had some sort of protection in their brain that prevented them from becoming addicted to anything, including their otherwise addictive medication.

Ex-PDers became addictable

The medications of Parkinson's disease, by the way, are far, far more addictive than cocaine, nicotine, and the various opiates.[16] And yet, when people with Parkinson's disease take these medications, they can often tolerate them very well.[17] Only when PDers in our project recovered did their brains become normal; their brains began producing dopamine, and they became susceptible to addiction and intolerant of the drugs.

Horribly, if a person recovered and continued to take the drugs at a level that was even slightly too high (as evidenced by the usual side effects of excess dopamine) for a period of more than seventy two hours, that person could become lost to an extreme form of brain change and addiction, utterly unable to ever overcome the new, physiological need

---

[14]Parkinsonism is the name for any chemical-, illness-, or injury-induced disorder that mimics some or all of the symptoms of idiopathic (idiopathic = unknown origin) Parkinson's disease. Because idiopathic Parkinson's disease cannot be replicated in lab animals, all in vivo research on idiopathic Parkinson's disease is actually performed on animals that have been inflicted with parkinsonism rather than with Parkinson's disease. It is especially ironic that most of this research is directed at finding ways to prevent the onset of parkinsonism in these induced cases of parkinsonism, even though this has clearly been shown to have nothing whatsoever to do with preventing the onset of idiopathic Parkinson's disease. The chemical and cellular features of parkinsonism and idiopathic Parkinson's disease are quite different – it is only their outer symptoms that are similar. This approach would be akin to car researchers breaking into cars in order to discover better ways to prevent vandalism so that they might ultimately improve gas mileage. There is simply no logic to it. However, millions of dollars in research money have been earmarked for Parkinson's research. The words parkinsonism (small "p") and Parkinson's disease sound a lot alike, but there is no way to create idiopathic Parkinson's disease in a lab. So do be wary when you read about "exciting new research for Parkinson's disease!" Read the fine print and see if the research was done on a "Parkinson's-like disorder" or on lab animals with parkinsonism.

[15] L-dopa is the longest studied of the dopamine-enhancing medications and will be used frequently as an example in this chapter. However, all of the dopamine-enhancing drugs, a group which includes the dopamine agonists and the MAO inhibitors, will ultimately set in motion the same regrettable brain changes, although the chemical/cellular pathways may originally be different. More on this later.

[16] The research supporting this statement will be forthcoming in later chapters. Meanwhile, if you can't wait, visit the website of the National Institute on Drug Abuse, a subdivision of the (United States) National Institute of Health, and read up on Dopamine, which is defined by them as the neurotransmitter of pleasure and addiction.

[17]In this sentence, the idea of tolerating a drug refers to the ability of the patient to take the drug without appearing to develop side effects. Another important meaning of the word "tolerance" is the one intended in many drug warnings – this meaning is "addiction." The need to make regular, sometimes rapid, increases in the dosage level of drugs because the body has learned to build up defenses against the drug or because the drug is addictive is referred to as "developing tolerance." For example, the body develops a "tolerance" to heroin, and so an ever-higher dosage is required to attain the same level of euphoria. This word play is helpful to drug manufacturers and doctors. A patient, reading in the Drug Warning insert that "a person can develop tolerance to this drug" might think to himself, "Ah, good, my body will learn a way to best abide this drug." But, in fact, when this phrase occurs in medical writing, it means only one thing: the starting dose of this drug will soon wane in effectiveness, and the patient will need to increase dosage. There may also come a time when the drug simply is no longer effective at any dosage level: the body will have developed a tolerance.

for the drug. It appeared that as soon as a person's brain went from Red Alert to All Clear (a switchover which seems to occur quite rapidly during successful treatment of Parkinson's), even though the body's muscles and coordination might not yet have resumed normal function, the medication could suddenly cause the same violent, "unpredictable," and life-threatening symptoms which had been observed in the early days of dopamine research on non-PD patients.[18] For some patients, addiction was rapid: attempts at reducing the medication after addiction had started caused supreme terror and even deadly respiratory distress.[19] Others had slow, gradual increases in addiction and adverse effects.

Misdirected rage

Finally, as a few recovering patients began having serious problems from their meds, requiring hospitalization or burial, I began a slow steam, and, looking for someone to blame, directed my silent wrath at the unsuspecting and hapless neurologists. Part of my resentment was due to the gag order placed on acupuncturists. Although in California we are primary care providers, we are forbidden to make any comment to individuals about their prescription medications: such comments might be construed as "prescriptive" and are outside our scope of practice.

When I referred my patients to their MDs for medication advice, which is the correct legal course, most MDs proffered information that contradicted the manufacturers' suggestions or reflected gravely outmoded notions of the drugs. Some researchers who knew about the dangers of the drugs told outright lies (and confessed later, when pressed).[20] When I shared updated research documents with my patients or read to them from the drug company inserts, proving that their doctors were giving wrong information, the patients were deeply uncomfortable, having to walk the fine line between working with their doctor – who alone could prescribe medication – and following the new research on their own as far as actual dosage was concerned.

In my impotent desire to blame someone, anyone, for the drug-related tragedies I was witnessing, I, like my patients, blamed the uninformed doctors. Of course, the information about dopamine and addiction was coming in so fast that no clinical doctor could be expected to keep up with it. The patients had to become researchers. They were uniquely qualified.

---

[18] Please read Oliver Sachs, *Awakenings*, HarperCollins, New York, NY.

[19] Because this may seem so implausible that the reader will be ready to hurl this book across the room, grudging the paper it is printed on, I am gratified to report that a recent study using monkeys and cocaine was able to prove that alteration in addictiveness/addictability actually does occur in response to changes parallel to those changes which occur in a person who recovers from Parkinson's disease. More on that later, but I wanted you to know that this unlikely statement will be supported later in this book.

[20] Some of my patients were seeing doctors at the Parkinson's research clinic a few counties away. One doctor there who, as a researcher, knew perfectly well that dopamine was the neurotransmitter involved in addiction, still, when asked point blank, up until 2001, if the drugs were addictive, would say that they were not. By 2001, even the Sunday supplement in the local newspaper had articles on drug addiction that referred to dopamine as the key to addiction. He then admitted that they were addictive.

Brilliant mind

People with Parkinson's disease tend towards a high level of intelligence and self-motivation.[21] Our local, senior neurologist, Dr. Rafferty,[22] was quoted as saying, "My Parkinson's patients are the most informed of any of my patients. They already know as much as I can tell them. They do their research." Members of our project began to expect less and less of their well-meaning but often uninformed doctors with regard to medication problems, and instead put their intelligence and self-motivation to good use, becoming researchers. They collected ongoing research tidbits, and more importantly, recorded their own responses to the medications. Many kept detailed accounts about their medications, logging the expected and the unexpected behaviors of their bodies before, during, and after every dosage. Each week I shared with my patients what was happening with the other patients. I joined my patients and my colleagues in the Parkinson's Recovery Project in reading books and research on psychoactive/psychotropic[23] medications.

There were a few disasters. After two patients were lost from failure to reduce meds quickly enough, a conviction plagued me that every medicated patient was racing a clock. Patients who delayed reducing medication even by a matter of a few days could defect abruptly from the group that was steadily reducing meds slowly and safely into the group of people who were essentially lost, their minds captured by the allure of the drug, their bodies writhing in either spasm or ecstasy. Patients became alarmed by what they saw happening to other members of the project. The patients began to realize two things: one, the exactly correct dosing of their meds might spell life or death for a person recovering from PD, and two, their doctors simply couldn't help them. The information that the patients needed did not exist. They began demanding information of me – information that I didn't have. They asked my advice about the medication, advice which I am legally forbidden to give even if I had it. And I followed the law.

By this point, I was seeing peculiar patterns in the drug usage and drug reduction that made me suspect why the drugs were working the way they did. By making hypotheses of the drugs' pharmacodynamics, based on projections from the known trajectories of other, more studied dopamine enhancers, such as cocaine, I was able to guess with greater accuracy than the patients or their doctors just what might happen to a given patient in a certain period of time at a certain level of the drugs. I found I could

---

[21] This bold statement, offered here without specific quotes to back it up, is based on findings that are worthy of a book in their own right. Suffice it to say that the Parkinson's Personality is a subject that has been studied since the 1930's, and as recently as 2002 an article on the subject was published in a most highly regarded journal of western science. Please see "Personality traits and brain dopaminergic function in Parkinson's disease." Valtteri Kaasinen, MD, PhD, *Proceedings of the National Academy of Sciences USA* 2001; 98:13272-7. (More about this article in a later footnote.) Although it is currently less common in western medicine to associate physiology and mental/emotional characteristics, there is much reason to suspect that these attributes are not always unlinked. In Asian medical systems, including the Chinese and Ayurvedic, thought patterns and personality are recognized as powerful forces, often accompanied by characteristic illness or health patterns.

[22] All doctor and patient names are fictitious, and genders of some have been reversed in this writing. The purpose of this book is not to point a finger at any particular doctor or patient, but to tell the story of our patients' experiments with Parkinson's disease and its medications.

[23] Psychotropic and psychoactive are words often used interchangeably to describe drugs that act primarily on the brain. Literally, the former means a drug whose chemistries are directed to the brain, and the latter means drugs whose effects occur primarily to the mental and emotional processes.

accurately guess when a patient was teetering on the edge, when only an immediate change in the medication could prevent a falling into the abyss of addiction, but, and follow me closely here, I couldn't say a word of recommendation to my patients. The drugs' effects were predictable. Legally I could not make any comment to a patient that might be construed as advice.

FIRST, DO NO HARM

I felt a moral pressure building on me to find some way to help my patients, but I was afraid. I am a licensed acupuncturist, not an MD. I cannot prescribe medication. My opinions, if shared with a patient *or a doctor*[24] constitute illegal advice about prescription medication. If I spoke up, I risked losing my acupuncture license and suffering various penalties. So, although every week I would share with my patients what I had seen in the last week as a result of other patients decreasing, increasing or changing their drugs, I could not give any individual advice. This system (or lack of system) insured that people were going to get hurt. I felt responsible for the people whose lives were being ruined by their lack of knowledge about the drugs. When a word from me might have been critically helpful, I had to make a choice between possibly losing my license or silently watching people succumb to the dangers of the drugs.

Medicated patients not suitable subjects for treatment

I decided to avoid the issue – we would no longer take on new patients who were taking antiparkinson's medications. There was a snag: my work had been published, even translated into foreign languages, and people around the world were apparently recovering from Parkinson's disease. Most of them were taking medications; without better information about the drugs, these people too would probably be lost to the pills. Already, the most common questions from health practitioners had to do with the bizarre symptoms erupting in medicated patients shortly after they started to recover.

Ignoring the warnings

We posted a warning on the website: People taking antiparkinson's medications were not suitable subjects for the recovery project. Can you imagine the response we got? People who were at their rope's end, desperate because their medications were no longer effective, ignored this warning. The PDers for whom the medications were still effective felt that they were different, that the warnings didn't apply to them.
Essentially, by publishing my findings about Parkinson's disease and how to treat it, even if I included a warning that it was not safe for medicated patients, I was writing a potential death sentence or worse for those people who tried to recover but who were taking the medications.

[24] An article in one of my professional acupuncture journals pointed out that it has been determined by some board or another that making a suggestion to an MD regarding a mutual patient's use of prescription medication constitutes prescribing without a license. Acupuncturists can lose their license for making suggestions about prescription drugs to doctors. Because almost any statement about drugs might be construed by a doctor as advice, this little bit of law pretty much amounts to a gag order in the medical world.

I tried approaching local neurologists; an MD's statement might carry more weight. Responses ranged from polite embarrassment to abject rudeness.[25] I decided that it was unrealistic to think about partnering with a neurologist.

Besides, by this point the neighborhood neuros were warning their patients not to be involved in my program, telling their patients that I was loony for reading to them publicly available, published information that indicated that the drugs might be dangerous. So, uncertain what to do, I just steeped myself in bitterness towards the town medicos. I petulantly made the case to God in my prayers that I hadn't asked to be put in this position of holding information that ran contrary to the accepted dogma. Meanwhile, my anxiety mounted for my patients who were rushing helplessly towards the cataracts of drug trauma while I watched from the sidelines, legally gagged. I wallowed in my resentment that someone wasn't taking responsibility for this problem.

Writing up our results

I started tentatively writing up a few pages of carefully worded hypotheses that suggested, based on well-proven, published information about dopamine, the antiparkinson's drugs might be more addictive than was previously suspected, together with case studies from my own practice that seemed to make the point. I figured that my license might prevent me from making specific, prescriptive advice for individuals, but as long as my First Amendment rights to free speech existed, I could write about the possible findings of our project. I did not make these early writings public, but emailed them to health practitioners when they had general questions about antiparkinson's medications. I thought that I could appease my conscience in this manner until Rose died as a result of overmedication.

Sweet Rose

Rose's case illustrates several problems that can arise during recovery. She was sixty years old. After two years in our program in Santa Cruz, she had been moved into a care facility back east because of her family's wishes. She was hideously overmedicated against her will, doctor's orders. Writhing as she stood by a counter at the care facility one morning, her arms lashed out uncontrollably, violently, as they did thousands of times per day. But this time it was different; her arm struck the wall with a random force that sent her flying backwards, head first, onto the floor. Her brain hemorrhaged. Her nightmare world of five years of furious flailing was over. After two days in a coma, she died.

Rose was taking medications for Parkinson's disease when she entered our experimental program

As Rose progressed in our program, she was getting no help from her neurologist, Dr. Leslie, regarding if or when to start decreasing her meds. She had joined the project merely hoping that the treatments might lessen her dependence on antiparkinson's

[25] Dr. Pender, a respected Parkinson's specialist, was friendly enough to me when I was introduced to him as a fellow PD researcher. We exchanged a few pleasantries, and he asked for my card. When I presented him with my business card, which says "Licensed Acupuncturist," he pulled back his hand as if I had offered him a wart on his nose. He turned and refused to speak to me afterward. If this was the sort of closed-mindedness that my patients were experiencing from their neurologists, no wonder they were so willing to go their own unadvised ways in experimenting with their drug reduction.

drugs.[26] (When she signed on as one of our first volunteers, we were not certain that recovery from Parkinson's disease was possible at all, let alone for someone with such a difficult case as hers.) Her greatest problem was in finding any sort of stability with her medications. When the meds were working well, she was blissfully beaming as her limbs writhed and thrashed. When the pills wore off, she was terrified and turned into quivering stone. There was almost no time in between these two extremes when she would have anything that might be described as normal movement.

When Rose first started taking antiparkinson's medications, they had worked well for her. Within a few years they had become problematic. By the time she started our program, she had had Parkinson's disease for eleven years. Her daily Parkinson's medication included 800 mg of L-dopa (a 25/100 Controlled Release Sinemet pill every three hours), 3 mg of Mirapex (a dopamine agonist, half a .5 tab every three hours), and Clozaril (a tricyclic derivative antipsychotic which had been prescribed as a muscle relaxant, half of a 25 mg tab, twice a day). (There will be detailed information on the mechanisms and characteristics of most of these medications in the Appendices.)

Rose spent most of her day flailing wildly, her head spasming painfully from side to side, her arms and legs thrashing. Her legs were a mass of bruises. When her pills wore off, she could barely talk or move any muscles. If she was set in a sitting position when Off, she would start to lean to one side. If no one caught her and righted her when Off, she would lean further and further until she fell to the floor. She was unable to make the slightest move to right herself. During these Off times between pills, she needed to be safely tucked into her reclining chair or propped up with pillows on all sides until her next dose of medication kicked in.

The first time she entered my office, we were unprepared for her peculiar dynamic. By the time she ricocheted into the consulting room, she had knocked over two free-standing six foot tall Japanese screens, kicked the chairs and footstools in the waiting area across the room and my framed diplomas had been knocked off the wall. I spent an hour with her. During that time her medications wore off; she became perfectly rigid, her hands and chin trembling with a quick, fluttering tremor like the dead leaves on a stark, unyielding tree in winter. Her husband and caregiver, Mike, quickly grabbed pillows from the examining table and built a pillow wall around her. She was 58 years old. She had been telling me details about her condition, but when she went Off abruptly and could no longer talk, Mike continued the intake with me. Rose had a rich life, Mike explained; she was still working part-time, visiting shut-ins, going swimming, going out to concerts and going to tribal gatherings in the summer. Her life was increasingly limited by her illness – she could not drive and she needed help in the morning to get to her first dose of pills, and she needed help with dressing and eating unless her pills were at full On, but her mind was sharp and she enjoyed working and living. Rose always saw the bright side.

About twenty minutes after she had turned into stone, the medication that she had taken forty-five minutes earlier began to show its effect, and she shimmered back to life. She explained, "I think that my illness helps me in my work. People who might otherwise not relate to a social worker can see that I have problems too. They see me needing help, and

[26] The collective name for all the various types of Parkinson's disease medications is, or used to be, "antiparkinson's drugs" with a small "p". This may be changing, and may no longer be current. Vogues in naming, as in prescribing, come and go.

see that I'm also just a person, also dealing with big problems. I think it makes me more approachable."
My own feeling was that it was her beaming smile and joy-filled heart that made her approachable. She was an angel of positive thinking and good deeds. I was grateful for her sake that during the two years she was in our program, receiving weekly treatments of Yin Tui Na (extremely gentle Asian massage) on her feet, she began to recover.

Rose's changing symptoms

Her first indication that she was recovering, after two months in our program, was that her medication suddenly seemed too strong. Her thrashing was more violent than ever. She made a small reduction in her drugs and felt better. Eight months after starting our program, feeling was restored in her long-numbed feet. The pins and needles feeling in her foot was so strong that she was certain a needle was there, and she even looked several times for a pin or splinter. She continued to slowly reduce her drugs. After that she noticed that she felt better and sometimes moved better when the medications wore off than she did while they were working. She was able to move easily during the night, when she was unmedicated. She could give herself her first pill of the morning, rather than waiting motionless in bed for Mike to administer her first pill. Over months and then years, her levels of native dopamine (dopamine made in her brain, as opposed to the dopamine from the pills) appeared to increase by a few minutes every few days. By the time she left our program, she was able to get out of bed in the morning, dress herself, brush her teeth, and take her pills before her native dopamine was used up. Just before she left, she was having enough native dopamine time to even have a bite to eat in the mornings before she needed to start her pill regimen. She was able to gradually reduce her medication down to much less than half of what she had been taking; at that point, just before she had to move, she was taking no Mirapex, only 3 pills of Sinemet, and rarely any Clozaril. Her case notes record her statement that she had more "normal" time each day, time when she was neither shaking nor kicking (her words for tremoring and thrashing). When she was kicking, however, the kicking had taken on a violence that she had never experienced before. Also, the shaking phase was more frightening than it had been. During the shaking phase, she said, "I feel as if I am incapable of being loved. I feel hopeless and stupid and scared." The intensity of the new emotional component to her Offs made her afraid to institute further drug reductions.

Increasing adverse effects

As her native dopamine increased she had new side effects of the medication. The worst was a burning sensation in her mouth. She described it as "excruciating burning pain in my mouth and nasal passages, with thick bitter foam pouring out of glands in my sinuses or somewhere, and from the upper back part of the mouth and lower salivary glands, with pins and needles in the tongue, with buzzing and burning." This pain would occur while her medication was switching Off or On.[27]

[27] An explanation for the switching pattern will be given in Chapter 21 of this book. The adverse effects of L-dopa become more powerful as a person develops intolerance for the drug, usually after about five years. Around that time, there may start to be a switching period while a dose of the meds is just beginning to work, and again as that dose wears off. This switching phase can be much worse, more painful than either the fully drugged or the non-drugged condition.

She saw three different doctors for the mouth pain, including Dr. Leslie, and even though pain in the mouth is listed as an adverse effect of Sinemet, not one of her doctors felt that her mouth pain could have anything to do with her drugs. She was prescribed pain relievers and told to drink more water, neither of which helped with the fiery pain.
And then, a health crisis came up for Mike. While he was in the hospital and then in convalescent care, Rose realized the precarious nature of her situation. She could not drive and was fearful of living alone. Rose's family, who all lived across the country, decided that the best solution would be for Rose to quit work and to move to a care facility near to her children. With misgivings, but determined to keep a positive outlook, Rose made the move. The admitting doctor at the care facility communicated with Dr. Leslie to learn the proper doses of Rose's medications.

Dr. Leslie, Rose's neurologist

Dr. Leslie had never been happy that Rose had been seeing an acupuncturist, and, despite Rose's insistence that she was now doing better with far less medication, he deceitfully conveyed to the care facility doctor that Rose was taking her medications at the same level as she had been taking them before she ever started our program. She was then required by the care facility to take her drugs at that level.[28]

Dr. Leslie's unfamiliarity with PD symptoms

Though Dr. Leslie is a neurologist, he is not a specialist in PD. He had been seeing Rose biannually for six years. After all these years of working with her, he had been shocked when, for the first time, he actually saw Rose during an Off, or rigid, phase. Rose had been in his office for her biannual checkup when the violent spasming stopped and the quivering rigidity abruptly set in.
"What's happening! What just happened?!" cried Dr. Leslie.
"Oh, this is what happens when the meds wear off," Mike explained. "This happens all day long. Don't worry. When the meds kick in, she'll be just fine."
"Well, do something! Hurry! Do something!" Dr. Leslie was clearly frightened and had never seen such a thing – a person with PD who was in between medication highs, or in other words, someone manifesting actual, advanced PD.
This scenario is not unusual. Younger neurologists, I suspect, have not seen many advanced cases of old fashioned, unmedicated Parkinson's disease; they see PDers who are medicated. They are so used to seeing people grimacing, spasming, jerking and flapping that they have come to imagine that this is Parkinson's disease. They do not remember from their years in med school that PD is an illness characterized by extreme slowness and rigidity – the absence of movement. The only extra movement in PD is the tremor, if any,[29] and a restless, shuffled pacing.

---

[28] An important thing that we learned in this project, and something which may not be generally known, is that a person in a care facility has no leeway in administration of medications. He must take the prescribed amount. Such a person cannot determine for himself what is the proper amount of medication. This is a good legal requirement, and it protects the facility from having to make decisions about a person's medications. On the other hand, it means that a person who needs daily fine-tuning adjustments in medication is not going to get them.

[29] Tremor, often thought to be the definition of Parkinson's disease, actually only appears in 60 to 85% of PD patients, the numbers varying depending on whom you read.

Poverty of movement

A brief description of tremor is needed here. The tremor of Parkinson's disease is for the most part a quivering, vibrating movement with no power behind it. The tremor in PD can become a large, exaggerated version of itself in times of high stress. This means that if, for example, the hand and arm are involved in tremor, those same parts will perform larger, jerking, back and forth motions during times when adrenaline briefly surges through the system.

Those excess movements, and those alone, are the movements that break the stillness of advanced Parkinson's disease. The writhing, body-jerking, eye-rolling movements brought on by the meds are not a part of Parkinson's. However, most neurologists in my experience in the United States associate the violent movement with the progression of the illness rather than with the medication. This is not willful disregard for the facts, but is instead the result of their seeing, primarily, medicated patients. Most people are put on medication as soon as they are diagnosed with PD. There is not much opportunity to see the unmedicated advancement of this illness.[30] Unmedicated Parkinson's disease is traditionally described as "poverty of movement."

Rose's neurologist, like most, did not know this. He thought her violent thrashing, kicking, and jerking were simply signs of increasing Parkinson's disease. The actual Parkinson's disease, that frozen-to-stone, utter immobility, accompanied by fluttering, quivering hands and chin that Rose manifested in his office, struck terror in his heart. He did not know the real face of Parkinson's disease.

Rose's response to the increased drugs

After she moved into the care facility, Rose wrote to me only twice; writing was difficult for her because of her explosive dyskinesia. She wrote that she was taking very high levels of medication and that her movements were completely out of control. At the care facility, despite her protests, including letters written by friends and by me stating that she had been performing better and less painfully on a lower medication level, she had been put back on the high medication levels that she had used before she started our program. Possibly because she had made great improvements in her condition, she could no longer tolerate the medications at these high levels. The violence of her thrashing increased to an extraordinary level.

She had no choice. She was told she must take her medication as the doctor ordered or she would not be allowed to stay in the facility. She obeyed her instructions and died, little over a year later, as the result of a drug-induced paroxysm. Her statistics joined our program's list of those whose lives or minds have been lost to medication at the insistence of well-meaning neurologists.

Guilty conscience

I had contented myself for years with nursing my bitterness towards the local doctors and complaining that "somebody" ought to be doing better research on these drugs. Rose's

[30] I have been extremely fortunate in this regard. Possibly because acupuncturists often see patients who are less trusting of western medicine, I have had the opportunity to work with many unmedicated and undermedicated PDers.

death was the push I needed to start compiling this book.[31] I wanted to blame someone for Rose's death and the finger kept coming back to me. Would she have been better off if I had broken the law? If I had told her from the start to reduce her meds, and then told her exactly which dosages to reduce and how quickly, would she still have died?
Even while she had been experimenting with her medications, I had suspected that she was not reducing in the best way. I saw her making mistakes that others had made with her drug reduction and hadn't said anything, for fear of losing my license. I did describe similar cases, telling her what patients in the exact same situation had done that had been successful and what others had done that had not. But when she asked me, as she always did, "What exactly do you think I should do?" I had to tell her that I could not answer that question. She did hear what I said, but I think that my refusal to make specific recommendations made her err towards the side of caution at the very times when quick action might have been advantageous. If I had been able to advise her, might she have gotten off her meds more quickly and recovered to the point where she would have been able to live on her own rather than being forced into a care facility?

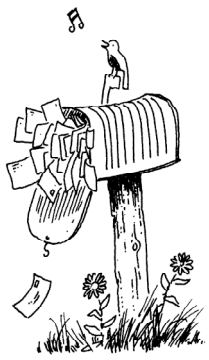
A blessing in a dreadful disguise

By the time Rose died, there had already been two others who had been lost to the drugs. I was wracked with the heart-breaking might-have-beens! And yet, as this book is coming together, I finally see that it might have been a blessing that I had not been allowed to prescribe medication. What I had considered a gag order may have been the greatest boon that our project could have had. If I had been telling my patients what to do based on my ideas from the beginning, we never could have compiled the material that we had. What we have now isn't just a mere handbook of tips on reducing medication based on my hunches; the patient records actually show a completely new way of thinking about the medication, about any dopamine-enhancing drug, and about Parkinson's disease.

GRASSROOTS RESEARCHERS

Unlike the harried MDs who spend fifteen minutes with a patient twice a year, I spent an hour every week with each patient. As the project grew, my colleagues did the same. Patients gave me a detailed verbal account of their week, or they recorded and presented to me their previous week's medication doses, time of day of dosing, side effects within half an hour of taking the meds, side effects when the meds were in full swing, what happened as the meds began to wear off, what happened if a pill was missed, what happened if the pills were taken with food, with juice, with protein, what happened if the patient had house guests, what happened if he had been playing more classical music during the week, etc. What happened the first day of a drug reduction? The second? What happened the next week? Ten weeks later?

Reports pour in



[31] I am sometimes asked why I haven't previously published m
article published in acupuncture journals every year since 1998. But no j
possibly accept an article of mine on medication. In the first place, the p
were due to people continuing meds after starting to recover – and no we
accept an article that presumes Parkinson's disease is curable. My adviso
that it would be twenty years at least before my work became accepted b

Literally tens of thousands of seemingly pointless bits of information have been faithfully recorded. Contributions of case studies have come in from other health practitioners around the world who were using our methods to treat Parkinson's disease. One amazing correspondent, Dominic,[32] has written us for over a year with every day's hourly details of his father's battle with the antiparkinson's drugs. He writes the time of day and what size doses of medication are used, responses to the meds in extreme detail, sleep patterns, functionality of the patient in terms of Activities of Daily Living, responses to exercises, exact information about diet, etc. His reports just go on and on. His father, Rufino, had, over a year, been able to reduce his medications from 1000 mg/day to 300 mg/day L-dopa.[33] He has thereby also reduced the power and violence of his dyskinesia that began when he started to recover and his medications were over the 1000 mg/day level. And while this reporting level is extreme, there are many others who sent weekly or monthly reports.

Every pioneer patient handled the challenge in a different way. Many kept charts, graphing their daily meds and daily side effects. Others kept written journals. Some tried rapid dose reductions, some tried taking smaller pills but more often, and others tried increasing the dosage while reducing the number of dosings. Others tried anticipating predictable schedules for reduction, which never seemed to work, and others planned drug reductions around family holidays, hoping for family support to help them through the hard times. A few tried stopping their drugs all at once, with disastrous results. A large number tried following their MD's suggestions for drug reduction[34] – and every one of these people had horrible results, and many ended up in hospital.

Drug company suggestions

Helpful drug company advice was non-existent. One company to which I wrote twice, asking for advice on reducing the medication, wrote back each time a note saying only: "This drug has been proven by the FDA to be safe." Other companies simply repeat the statement that the drugs should be reduced "slowly." There was never a hint as to whether "slowly" means reduce by a pill per day or by 5 milligrams a month. So the pioneers all set off on their own path to figure out what worked for them with the medications and what didn't.

As I look back over the thousands of pages, the years-worth of information that we have acquired about the medication, I see it has all come about because of this very gag order. Because my patients knew both that I could not make suggestions about the medications and that their doctors were badly misinformed, my patients realized that they were on their own.

---

[32] Thank you, Dominic!

[33] Two years later, as this book is being finished, I'm pleased to report that Rufino has been taking no medication for nearly a year. There will be more later about his case.

[34] A typical, dangerous MD suggestion was, "If you really want to stop taking your medication, then just do it." Another one was, "If you want to take less medication, come off it slowly – take a full week to get off all your medication." You will see why both suggestions were equally deadly by the time you are halfway through this book.

The revised mantra

Up to this time my colleagues and I had used a sort of chant that we repeated anytime a patient wanted advice about meds: "I am not a prescribing physician. I cannot give you prescriptive advice about the medication. I will share information with you; you can share with your doctor. You must work with your doctor." The patients learned this by heart and could recite it along with us. But after patients in our clinic and around the country started ending up in hospital because of unsound advice from their neurologists, we added this line: *"It is likely that the advice that your neurologist will give you is wrong and even harmful."*

PIONEERS, HO!

Knowing that their doctors were as likely to harm them as hurt them, the pioneers became true independents, sometimes shocking their doctors with their new knowledge. Dr. Rafferty, a warm-hearted neurologist, brooked no nonsense about acupuncture and the holistic medicine trend. He was nonplussed when the pioneers started challenging him on his drug recommendations; he was told boldly by a formerly compliant, elderly patient, after he suggested that she try a hot new PD drug that elevated acetylcholine levels, "You prescribed me a drug several years ago that was an *anti*cholinergic. At that time evidently too much acetylcholine was part of the Parkinson's problem. Now you're trying to tell me that the problem is that there's *not enough* acetylcholine? Make up your mind!"

For ourselves and our posterity

The pioneers understood that they were rarities: reluctant human subjects in an unguided research project – end result: unknown. Becky, whose story you will read over the course of several chapters, reduced too late and suffered extreme agonies of drug withdrawal for over two years. She often said, "I'm old, and what's happening with these drugs is killing me, but I figure if what I'm going through can help save even one other person from this hell, then it's worth it." Through their experiments, some deadly, some brilliantly effective, we discovered things about the medications that were utterly shocking, counterintuitive, and which answered some of the questions raised in the first great work on L-dopa, *Awakenings,* by Oliver Sacks.

Meanwhile, in other fields of research, information was beginning to come in that applied to our project. Researchers for the antianxiety/antidepression drugs were suddenly realizing that these drugs were addictive, that some of them took months to become fully effective, and that there were long- and short-term side effects from the withdrawal of these drugs. Some of these popular drugs cause brain alterations that appear to be semipermanent, even initiating tremors that resemble the tremor of Parkinson's disease. These alterations may not appear until years after the drugs have stopped being taken. And what did researchers find was causing these side effects from well-known drugs such as Prozac, Paxil, Xanax, and Zoloft? These addictive, long-term effects were apparently being caused by the impact of these drugs on brain dopamine levels.[35]

In 1999, just as I was beginning to write up the preliminary findings of the pioneers, the National (American) Institute on Drug Abuse, a subset of the National Institute of Health,

[35] J. Glenmullen, MD, *Prozac Backlash: Overcoming the Dangers of Prozac, Zoloft, Paxil, and Other Antidepressants with Safe, Effective Alternatives*, Simon & Schuster, NY

was announcing breakthrough discoveries about the role of dopamine in addiction and drug withdrawal. Cocaine, opiates, alcohol, nicotine, and methamphetamines were all proven to be addictive because of their dopamine-enhancing properties. None of these drugs were nearly as strong as the antiparkinson's drugs, of course. These street drugs merely increased dopamine briefly, imparting a fleeting sense of emotional well being or unnatural levels of strength. The Parkinson's drugs were ever so much stronger – they could literally impart sustained movement in a person who previously could not move. Mere cocaine couldn't do that! Our conclusions about dopamine were being confirmed – not by Parkinson's researchers but by researchers working with drug addicts.
In 2001, research was published proving that the Parkinson's personality is not dopamine dependent. This further supported our original theory that the personality is required to set in motion the Parkinson's, rather than being a side effect of the dopamine problem.
In 2003, as I am finishing this book, research on addiction has shown that primates with normal propensity for addiction can become immune to addiction if their social situation changes.[36] Primates who become alpha (dominant) males, a role which demands a high level of wariness, constant alertness to possible attack from subordinates and the ability to not show weakness or admit injury (any of which could lead to loss of the alpha position, if not death) show a change in dopamine receptor activity in the brain, and a concomitant increase in resistance to drug addiction. The same individuals, prior to becoming alphas, had normal susceptibility to cocaine addiction. (The relationship of cocaine to the antiparkinson's drugs will be described in later chapters.[37]) The exciting thing was this proof that addictability was not genetically predetermined; external situations and even social settings could alter dopamine receptor activity levels, and *these changes in turn altered addictability.*

Drug abuse researchers

Now, when people ask me for more information about drug reduction, I tell them that their starting point is the National Institute on Drug Abuse (NIDA). The NIDA website is a treasure of information about dopamine. Log on and do a search for "dopamine," the latest information on the role of dopamine in drug addiction will pop up.[38] Armed with this information, patients are less likely to humbly submit when their MDs assure them that their drugs are not addictive, or that their drugs are not causing their dyskinesias, paranoias and tremors. If the patient's MD insists, despite new evidence, that the drugs are benign and non-addictive, the informed patient understands that any journey through drug reduction is one that he must do by himself, most likely without any help from his intelligent, well meaning, but under-informed MD. Another patient researcher is born.

IN CLOSING

Our patients did something that is nearly impossible to do in today's world: original, intuition-based drug research on humans. Their charts proved that dopamine-

---

[36] *Nature Neuroscience* 5(2): 169-174, 2002, by Mike Nader. More about this study is written up in an appendix at the back of this book.

[37] The personality of Parkinson's disease and its parallels in the alpha monkeys are subjects worthy of a chapter of their own: see appendix 7.

[38] That is unless they've changed their search format again.

enhancing drugs are predictable, not unstable, if viewed in the context of brain accumulation and long-term drug response. The dramatic change in their drug-related symptoms when their electrical systems reverted to normal suggests a reason for addiction resistance in PDers. Their subsequent illogical passion for the drug and rapid increases in dosage despite hideous side effects prove the NIDA contention that dopamine-enhancing drugs can be highly addictive.

They discovered under which circumstances the drugs cause freezing and dyskinesia. They discovered the correct (rarely used) dosages and time frames for using, increasing, or reducing their medications. From their macedoines of drug combinations, they learned which drugs were causing which side effects. No doctor, however well-meaning or well-informed, could have legally led them through the thicket of misinformation, confusing symptoms, experimentation, and the horrible, inevitable deaths, without having his license revoked. If I had legally been able to advise my patients, I could never have come up with the dozens of experiments that occurred as desperate patients did their own exploring.

I finally see that the infuriating gag order may have been a blessing, albeit a painful one. My heart still aches from the death of our beloved Rose, and deaths and traumas of others who have been lost, but maybe there was a blessing in that too, though it eludes me. Rose clearly came to peace with the decision that she would leave our project. She told me just before she moved away, "God is calling me to ____ (the place to which she moved), for work He wants me to do there." She was already at peace. I remain in limbo.

The spirit of science

Despite Parkinson's researchers' current interest in brain cell change, dopamine loss did not originally define the syndrome of tremor, slowness of movement, rigidity, and loss of balance named Parkinson's disease. James Parkinson, in the 1800's, described this collection of physical symptoms, some of which are dopamine related and some of which are not, and named this syndrome the Shaking Palsy. Later on in the late 1800's, a French researcher, Charcot, renamed the shaking palsy "Parkinson's disease" in honor of the brilliant work of James Parkinson. Charcot felt the underlying problem in Parkinson's was electrical. Only in the 1950's did the dopamine fixation began. Science is a collection of ever-changing hypotheses.

In that spirit of honoring the research work that has gone before, in which we do not condemn the partial truths and mistakes of the past nor deny the inevitability of being ourselves proved partly or completely wrong in the future, I offer up this book as a contribution to the research on Parkinson's disease. Hoping for less bitterness at lives lost, and with gratitude for the work of the Parkinson's Pioneers, the grassroots researchers, I offer you our findings, with this solemn caveat: a person who is taking dopamine-enhancing medications should not try to recover from Parkinson's disease.

"For many men that stumble at the threshold
Are well foretold that danger lurks within."

Shakespeare's King Henry VI

2. WORRISOME DRUG PROBLEMS

OUR DRUGGED PATIENTS DIFFERED FROM THE REST

This chapter will share examples of how medicated patients veered off from the recovery path demonstrated by *un*medicated patients. I will include two detailed case studies: one of a patient who slowly became addicted, the other a patient who quickly increased her medication despite life-threatening side effects.[39]

Something afoot

With uncanny good fortune, the first four Parkinson's disease patients I ever had were unmedicated. In fact, I didn't even have a PD diagnosis for my first three Parkinson's patients. Had I known that what I was treating was Parkinson's disease, I would have probably only used the specified scalp points for PD on this trio. These points were in vogue in China ever since the advent of the western PD theories on brain cell death. But, by great good fortune, or fate, my first three PD patients had never been diagnosed.[40] When these patients came to me with baffling symptoms and no diagnosis, I spent hours exploring every inch of their bodies looking for some clue as to what had gone awry.[41] I found a curious pattern of energy confusion, similar in each patient, which was traced to the foot. This pattern of Rebellious Qi is one that was mentioned in the ancient literature and referred to briefly in lectures while I was a student, but it was a pattern for which I had learned no practical application.

Shortly after recognizing this energetic pattern in these patients and successfully treating them for their ailments,[42] I had my first "officially diagnosed" Parkinson's patient. Her

---

[39] The reason for the individual variances in restoration of addictability is discussed in chapter 24.

[40] All three of these patients, two recently from Russia, one from Korea, had little faith or even interest in western medicine. The first was, in fact, terrified of doctors. This preference for Asian medicine over western allowed me to work with people who, in retrospect, had classic Parkinson's, without the blinders I must necessarily have worn had they been given an official diagnosis of Parkinson's disease.

[41] I had only graduated from Asian medical school a few years earlier and had a slowly-building practice. I was sometimes able to spend long hours, at no extra charge to a patient, just exploring diagnostically, if I was curious about a health problem. I had the time and inclination to devote whole days to a single patient without charging a fee if I so chose.

[42] This occurred much to my amazement. I was still extremely dubious about Asian medicine, and I had digested my course material by interpreting its precepts through a protective screen of western science and biology.

No one could have been more startled than I to discover that Backwards Running Qi (Rebellious Qi) was actually just that – electrical currents that were running in the reverse direction. Always keenly sensitive to electric charge or static in people's skin (I am blessed with extremely poor eyesight and an overdeveloped sense of touch is my compensation.), I could actually feel with my hands the difference between the direction of electrical current flow in my patients… (Footnote continued on next page.)
(Footnote continued from previous page.) with PD-like symptoms compared to the flow patterns of normal

energetic patterns were similar to my previous Rebellious Qi patients. Her symptoms, too, responded to treating the foot injury that appeared to be the source of the irregular energetics in her body. More importantly, her responses and apparent recovery followed the strange course that I had seen in my mystery patients. It was only then that I reviewed the symptoms of Parkinson's disease and began to suspect that my first three patients with shuffling footsteps, tremoring hands, and masked facial expression might all have had Parkinson's disease.

After that, I embarked on a small research project to see whether or not this particular energetic anomaly was present in other people with and without Parkinson's. Something I still can't explain inspired me to visit the local Parkinson's support group. I stammered out my preliminary findings and recruited patients for my small study by offering two months of free acupuncture and foot massage in trade for letting me examine their feet. I got twelve recruits over the next two months. They had all been diagnosed with Parkinson's disease and they all had detectable Rebellious Qi in their legs. It appeared that, in all subjects, the source of the rebellious pattern was a usually unremembered, and most importantly, unhealed, childhood foot injury. This pattern of Rebellious Qi was not present in twelve people picked at random from the rest of my patient base.[43]

After working with them for several months, most of them began having changes similar to the changes that my first three patients had had. At this time I was also blessed with two officially diagnosed PD patients who were not medicated. Of course, my very first Parkinson's patients, who had never been diagnosed with anything, were not taking antiparkinson's medication.

Based on the recovery symptoms of these unmedicated patients, I had already compiled data that we could use as signposts in determining whether or not a person was recovering and how far along they might be in recovery. This enabled us to recognize that medicated patients were behaving very differently from unmedicated patients. The medicated people in this small project responded favorably to the same treatments I was now using on all the Parkinson's patients, but only to a point. Within a few months of the appearance of apparent recovery symptoms in medicated PDers, an inexplicable divergence became apparent between them and the unmedicated patients, in both their recovery rate and their symptoms of recovery. Worse, a few medicated patients who appeared to be making rapid recovery from Parkinson's abruptly developed ghastly, medication-related symptoms.

Forebodings

It was at about this point that I began to fear that I had stumbled across the cause of Parkinson's disease. I had grown up in the western sciences. I had learned decades

---

people. It was only when I realized that some people had variant patterns of electricity under their skin that I suspected that this objective (to me, anyway) phenomenon might be related to the Qi that I had spent four years studying in school. Suddenly, Qi went from being a theoretical concept to a tangible, measurable quality. There was no name for this discernable energy flow in western medicine or even in the entire field of biology. However, based on how it moved, changed, and was affected by other electrical patterns, it seemed related to the principles of electricity and magnetism I had learned more than thirty years earlier, in physics class.

[43] J. L. Walton-Hadlock, Primary Parkinson's Disease: The Use of Tui Na and Acupuncture in Accord With an Evolving Hypothesis of Its Cause from the Perspective of Chinese Traditional Medicine, *American Journal of Acupuncture*, Vol. 26, no 2/3, 1998, p. 163-177.

earlier, in 1968, that Parkinson's disease was incurable, and the incurability of it had been hammered home in every physiology class, both western and eastern, that I had ever attended. I knew that, just as the sun came up in the east, Parkinson's disease was incurable. I feared the direction that my thoughts were headed. I did not want to be the person to find a possible cause for this illness.

I was, at that time, an extremely private person. Finding the cause of idiopathic Parkinson's disease carried a moral responsibility to publish the information. If the medications posed a special problem, I also had a responsibility to uncover just what that problem was and write it up.

What a monstrous position to be in! Looking ahead, I could see where writing up what I suspected about the drugs and knew about Parkinson's disease would be an invitation to hostility, mockery, and contentious debate from patients, doctors, and possibly even the hugely wealthy and powerful medication industry. I wanted no part of any of it. I was peeved by what fate seemed to be ladling out for me. And yet, and yet...how could I not follow these leads and see where they took me?

THE MEDICATION MYSTERY BEGINS

The two officially diagnosed patients who were unmedicated started having the same curious recovery symptoms that the earliest trio had manifested, but the rest, the medicated patients, started having very, very different symptoms, such as Rose's horrible burning mouth pain, increasing muscle spasms, or hallucinations. It did appear as if the medications were actually causing problems of their own.

Most patients who were medicated planned to reduce their medications and found themselves unable to do so. It wasn't so much that they didn't want to; many were starting to feel overmedicated, and their side effects from the drugs were worsening. They told me week after week that they were intending to reduce their meds any day now. When the next week's visit rolled around, they hadn't started yet on the med reduction.

At this naïve time, they all believed that since they obviously were going through powerful changes in their bodies, most of which corresponded to recovery symptoms seen in other patients, they would of course eventually recover from Parkinson's and could easily stop their medication at that time. One day they would wake up, spring out of bed, and never take a pill again. Even though their PD had developed slowly and they were warned that the recovery symptoms occurred gradually, they fully expected that their deliverance from the medication would be abrupt. The expectation of a Day When I Suddenly No Longer Need the Drugs was standard issue. That day never came.

Olli

Olli was one of the first pioneers. He was 63 years old when he joined our project. He had been diagnosed three years earlier. When he joined our study in February of 1998, he was taking Eldepryl[44] (5 mg, two times a day) and 600 mg of levodopa per day (two 25/100 Sinemet and two 50/200 Sinemet CR). When he started working with me, he had

[44] Almost all of the pills go by an assortment of names. Please see the drug list in the first appendix to find the various names by which your familiar drugs are also known.

symptoms of slowness, no arm swing, loss of voice, tremor on the left side, and slow speech. Through the masking effect of his medications, those were the only symptoms I could be certain of. Of course, there was no way of knowing what he would have been like without his meds.

After several months of receiving treatments, he was feeling less rigidity, had a deeper voice and his face was becoming more expressive. More importantly, he felt that he was changing, somehow, deep inside. After one strange rolling sensation through his head, after which his tremor dropped almost to nothing, he felt that he was different inside and associated this difference with recovery from Parkinson's. Since he felt better, his rigidity was going away, his voice was deeper and his face more expressive, he just decided not to take his pills one morning.

On the day that Olli didn't take his pills, he felt great all morning and all afternoon. Then, at five o'clock in the wintery evening, crossing a parking lot in the dark, he suddenly couldn't take a normal size step. He couldn't move his feet, and then he fell, crashing to the ground. After that day, he resumed his medications and refused to ever consider reducing his levodopa even though he started developing dyskinesia from the drugs.

It was rash and dangerous of him to stop taking these powerful medications all at once. The drug insert warnings say not to quit the drugs abruptly. In a worst-case scenario, a person can go into drug withdrawal and experience fatal respiratory distress, which means, in case I'm not being clear, death.

Afraid to reduce his levodopa medication ever again, he rapidly became overmedicated, as indicated by the increase in dyskinesia. I wondered what Olli's doctor would say. At his next check up, Dr. Grumb ignored Olli's concerns about the dyskinesia and observed brightly that it did appear that Olli once again had a bounce in his step, and that his voice and facial expression had returned. The doctor pronounced these changes to be due to a delayed (several years) reaction on the part of the medication. He encouraged Olli to increase his medication levels and plied him with a sheaf of brochures from the makers of the latest dopamine agonist drug. Since Olli had finally accommodated so well to the drugs, as evidenced by so many physical improvements, Dr. Grumb suggested that it only stood to reason that even more medication would make him feel better still. Since it clearly was taking a long time for the drugs to become effective – years, in Olli's case – Olli might be wise to increase his medication now in anticipation of advancing Parkinson's. Olli declined the offer of extra medication, but as he was leaving, the MD reminded him roguishly, "Don't forget, if you're going to a party on a Friday night, go ahead and double your dose!"[45]

Olli decided to keep working with me, since he was feeling so much better. He felt certain that he no longer needed the medications, but decided to keep taking the drugs as a sort of supplement. Over the next few months, he felt better and better. He went through the usual symptoms of recovery: extreme fatigue, the characteristic back pain, relearning to walk. Aside from a slight residual tremor, it did appear as if all of his symptoms were gone.

But following these changes, he started getting a different set of symptoms. His blood pressure became very hard to regulate. His facial grimacing worsened. After several months, his tremor returned, but it was jerkier, more staccato, and more insistent than it

[45] Doubling a dose is specifically forbidden in the drug instructions from Sinemet's manufacturer.

had been. It was at its worst an hour after taking each dose, and then ebbed when the dose's benefits wore off. He became more sensitive to the timing of the medication. His voice started disappearing, worsening an hour after taking a pill. The hoarseness lasted longer with each dose over the course of the day. An hour after his evening dose, his voice would utterly disappear and stay gone until bedtime. His balance suddenly became unsteady, after having been steadily improving for so many months. It was as if he was regressing into the Parkinson's, and yet, he often appeared overmedicated at the same time. He stopped the Eldepryl during this time and noticed an improvement in temperament; he burst into tears much less frequently. Two months after stopping the Eldepryl, his voice lost a tremulous quality that he had attributed to the Parkinson's disease. But he simply could not bring himself to ever again decrease the L-dopa by any amount.

Olli had been assuring me since the beginning of his treatments that it would be easy for him to quit his medication when the time came; he was a mental health doctor and he was completely aware of the difficulties of reducing psychotropic drugs. He assured me of that almost weekly. In the beginning, I had been relieved that I had a patient in the program who could negotiate through the reduction of his own drugs. But as time wore on, he would say at every session, "I don't know what's going on…Every day I decide that I am going to reduce one pill by just a little bit, but at the end of the day, I find that I haven't done it yet. I just don't understand." This went on for over a year. Every week, as he noticed more symptoms of overmedication, he told me that he was planning to reduce his pills. Every week, he forgot to do it.

When his tremor suddenly became much more powerful, he was almost relieved, and declared that he could not reduce the meds until his tremor got better. So I read to him from the insert that comes with the L-dopa pills (Sinemet) the Adverse Effects section in which tremor, bradykinesia and freezing are listed as side effects of the medication. His eyes bugged out. His language, always colorful, became fluorescent. Sparks flew. When he cooled off enough to use full sentences, he blurted, "The drugs I'm taking *cause* tremor? But I'm taking them to control tremor!"

It was at this point that he realized that he was in trouble with his drugs. He never was able to reduce his medication, even for a brief trial of just a small amount, just to see what would happen. As his symptoms began to worsen, Olli recognized many of his symptoms as being similar to the problems of his drug-addicted patients. He suspected that he was addicted to a powerful stimulant. His adverse effects and On-Offs continued to worsen, and he increased his medication for the temporary relief that it gave. He was adamant that he wanted to reduce his medication. He swore that he hated taking the pills and was eager to get off of them. And yet, for some reason, he never did. He added a dopamine agonist to his drug regimen. He noticed that his friends who were also in the study who were slowly, steadily reducing their drugs seemed to be regaining voice and movement while he was rapidly getting worse. He was chagrined that he hadn't decreased his medication sooner.

When his ticcing and facial grimacing become constant and he needed a walker to maintain his balance, he stopped coming in.

COMPARING PATIENTS

Within a year of starting the research, we suspected a physiological difference between medicated patients and unmedicated patients. We tried to explain it away with math: we proposed that the medicated patients were simply more advanced than the unmedicated. This specious logic did not hold up; patients who had been diagnosed less than one year and were taking medication had more difficulties in recovery than patients who had been diagnosed three or more years earlier but had refused the drugs.

I had one patient in his seventies who had had PD for over six years and, due to fear of the drugs (his wife was an MD and had done her research), had been stumbling along on sub-therapeutic levels of the drugs. He progressed towards recovery much faster and had fewer drug-related side effects than the ones on full dosage.

We looked for other reasons: differences in age, attitude, and symptoms. It appeared that the only similarity in all those patients who started recovering but then developed the rapid, even bizarre worsening of symptoms was this: they were taking antiparkinson's drugs.

Altered minds

Medicated patients even thought differently than unmedicated patients. Their thought processes and memory weren't as keen, although they appeared bright eyed and clever enough and often felt that they were smarter than most. One mental characteristic of most drugged patients was incapability in making objective observations about their own condition. For example, when they were writhing, they often imagined that they were moving lightly and with great subtlety. Their mental differences, together with the physical adverse effects, led us to suspect that we were dealing with two separate illnesses: the PD and the drug effects.

Crawling skin, jerking bodies

There was another thing we'd noticed: we could tell by the feel of their skin whether or not patients were medicated. Our hands could detect an unpleasant feeling in medicated patients, as if there were snakes crawling under their skin. Their skin gave off an erratic electrical discharge as well. Those of us who had a keen sense of touch found it unsettling to do massage – the massage that is used in the treatment of the old foot injury – on these medicated patients. Also, the subtle movements of energy and anatomy were difficult to perceive in drugged patients – the medication causes drugged patients to respond with jerking or vibrating that mimics the anatomical movements of recovery, but these are actually superficial, drug-related twitches.

Replications from abroad

Emails started coming in from other practitioners describing scenarios in which their medicated patients had worsening adverse effects from the medications shortly after they began to demonstrate recovery symptoms. The people who were taking meds had different problems in recovery than the patients who merely had Parkinson's disease.

No two the same

Any PD specialist will tell you that each PDer is unique. No two have the exact same symptoms. And yet there is a consistent logic to the disease, its progression, its treatment,

and the symptoms of recovery. The unmedicated patients were recovering in the same way. But medicated patients each went off on their own tangents. We started looking forward to the unmedicated patients. While their cases were challenging, they were still comparatively straightforward. The medicated people were quagmires; their recovery symptoms and medication-related symptoms quickly became baffling and unpredictable.

Masked Symptoms

There was another problem in working with medicated patients, as far as research was concerned: we had no way of knowing just exactly what symptoms a medicated person actually did have. A very advanced PD patient might appear perfectly normal during his best On times from the meds, or he might appear exaggeratedly nonfunctional if he was having an Off time. It all depended on when in the drug cycle we chanced to see him.
I've had many a hearty laugh with patients who recall such doctor visits: "I went to my neurologist and it happened that my medication was working well for the first time in a week. He told me I was at the perfect level with my medication after he watched me stroll down the hallway and touch my nose. I told him that I had hardly been able to move all week, and he just shook his head and told me I was exactly at the right levels. An hour after we left his office, I couldn't move again."
I also heard the reverse: "I was at my annual checkup with my neurologist and my medication was Off right then. He went into a panic and told me to grind up a pill and take it right away. My wife tried to explain that I would be On in half an hour, but he just couldn't believe how bad I looked. He told me to double my medication."

(By the way, please forgive the lengthy asides. There will be more. I would love to present all the information I've gathered in some sort of straight-line progression, but in truth, there was information coming in from all sides at once.)

The gist is this: under those layers of medication, the doctor and even the patient may not be aware of what his PD symptoms actually are.

Mistaking side effects for symptoms

The patients who have been taking the drugs for years and have side effects from their medications think they know what their symptoms are. They are often wrong.
Most patients assume their On/Offs or their dyskinesias (excess, uncontrolled movements) are their most troublesome symptoms. As for rigidity, poverty of movement, balance trouble or tremor – the actual symptoms of Parkinson's – they may not be so concerned about these. These problems may be masked by their medications.
But returning to the main point, when a person is taking drugs, it is impossible to know what his symptoms are, so it is impossible to know just how much progress he has made during recovery, or how much recovery work there is still to be done. Just imagine: we were trying to treat people who weren't sure what their actual symptoms were.

Better off with Parkinson's?

After several patients became wildly overmedicated to the point of pain, we began to wonder if some medicated patients were better off never recovering; since the medication appeared to be more powerful and more addictive after people recovered than it was while they still had PD, and since many recovering patients apparently did not have the ability to willingly decrease their medication, we had to ask ourselves if possibly these

patients would have been better off if they had never entered the program. Certainly, Zoe would have been better off.

Zoe

During this time, we'd been working with Zoe, a delightful woman in her early 60's. She had been diagnosed six years earlier but was still trying to keep up with her weekly yoga class. She was taking Sinemet CR 50/200 six times a day, a pill every four hours. (She woke up in the night to take her midnight dose.)

She did well in our program. She regained her ability to sweep the sidewalk and wash her own hair. Her walk had a lilt again and her tremor was gone. She was shaping up to be one of our greatest success stories. But then she hit that phase of recovery where it is just impossible to get up in the morning. (There is a stage during recovery where people feel heavily sedated for two hours, usually from about seven until about nine in the morning.[46]) When Zoe got to this stage, she grew alarmed. She interpreted her inability to rouse herself promptly in the morning as a sign that she needed more medication. She tried doubling her 6:00 a.m. dose. Still she was tired and sleepy from seven until nine o'clock. So she tripled her 6:00 a.m. dose. She was still groggy. This threw her into a panic. Even though I told her repeatedly that all the other patients at this stage of recovery weren't able to move from 7 to 9 either, she refused to believe me.

Even though she was able to move better than ever during the rest of the day, even twitching and grimacing (new symptoms that indicate overmedication), she was terrified of the helpless, anesthetized feeling she experienced for two hours every morning. By now she was taking 8 pills a day instead of 6. A few weeks later, she came beaming into my office telling me how well she was doing. She was up to 10 pills a day and felt just great. She was no longer groggy in the mornings. Evidently, she said, what she had needed was more medication. She felt so good that she increased her medication again the next week, up to 12 pills a day. Now she was taking a total of 600/2400 (carbidopa/levodopa), far more than the manufacturer's suggested daily amount, and she looked as if she could waltz through the ceiling.

She no longer had any symptoms of Parkinson's, but she was strangely euphoric. She laughed, sang, threw her arms around with abandon, and acted very suspiciously like some of the subjects in Oliver Sack's early study using L-dopa on non-Parkinson's patients.

Then the diaphragm trouble started. She noticed that if she was more than twenty minutes late with her pills, she became unable to breathe. Her diaphragm would seize up and she would gasp for air as if someone was choking her. She went to the hospital during one of these choking fits and the doctors were puzzled; her blood oxygen levels were within normal limits. She was evidently getting enough oxygen although she was bent double, terrified, and gasping for air as if she was being choked. Her breath would come in great staggering rushes, and then pause in between while she turned a bit purple in the face,

[46] There are certain physiological processes that take place at specific times of the day. Stomach channel healing is especially strong between 7 and 9 in the morning. Many PD recoverers go through a phase when they cannot move, when they are as if heavily anesthetized during these hours. This can last for a few days or several months, depending on the speed of healing in the individual.

and then there would be another huge gasp. It was horrible to watch. So she increased her medication. The breathing problem got rapidly worse. She increased again.

Zoe saw Dr. Pender every six months. On her first visit to him after she started in our program, he noted that she was doing very well. He even wrote on her chart that she might be overmedicated. Zoe brought me copies of her doctor's notes. At her next visit with Dr. Pender, he wrote in her chart that possibly she had been misdiagnosed.[47]

But the next report was even more important. Zoe showed me a photocopy of the notes Dr. Pender had written up: he had noted that he did not know why this patient had pretended to have Parkinson's disease for six years.

I was excited. Zoe's doctor was a big shot at an important clinic that specialized in PD. I could understand his feelings; if he had misdiagnosed her, he was in a bit of a spot. Therefore it must be that the patient had pretended to have PD for some crazy reason of her own.

Having decided that she had only been pretending to have Parkinson's, he told her that, based on her flailing dyskinesia and breathing problems, she was dangerously and unnecessarily drugged. She should stop taking her medication immediately. She should reduce it slowly, but promptly. He did not define "slowly."

Shortly after that, she increased her medication again.

I asked Zoe to keep a chart of her medication, the time she took it, and certain symptoms of overmedication, including her gasping. She tried many times to take half a pill less, two or three times during the day. What we noticed from her detailed charts was that for two days at the slightly lowered dosage she had fewer episodes of the labored breathing. But within three days or four days at the most, she would feel helplessly panicked, even though the breathing was less strained, and she would go back to the higher drug levels. This back and forth behavior went on for months.

She couldn't explain the panic. She was taking 14 pills a day at this point, 2800 mg of levodopa. She would reduce by about 100 mg/day, have a very slight improvement in the breathing, maybe not have as lengthy a struggle with the breathing, and then, within four days, she would be back at the higher level. This went on for month after month. The breathing pattern was getting more powerful. It overwhelmed her when the breathing attacks hit.

Finally, Zoe's husband got involved. I had been telling him for some time that I was concerned about his wife's drug levels and her breathing. He vigorously asserted that she was an adult, and that her pills were her own responsibility, and that he didn't want to get involved. I replied that from what I was seeing in my office, there was no way in the

---

[47] Patients with Parkinson's disease can't recover: Parkinson's is defined as incurable. Therefore, the official proof of our success is when the MD declares that a patient was misdiagnosed.

I was so pleased with Zoe's possible change of diagnosis that I wrote to her clinic with a signed release form and asked for copies of all her records. Dr. Pender was highly antagonistic to our program. So I wasn't surprised but I was disappointed when the clinic sent me only a partial file – they had left out her two most recent visits. I called the receptionist and asked why the two most recent reports had been omitted. She was baffled. She remembered putting all the records on the doctor's desk for his approval before sending them to me. She couldn't imagine what had happened to the two missing ones. Dr. Pender was out of town for two weeks and couldn't be asked about the missing paperwork, so she, on her own initiative, sent me the two that I was missing.

world she would be able to reduce her medication without some kind of support, but he pooh-poohed the whole thing.

It was only after Dr. Pender told Zoe to reduce her meds and she increased them instead that her husband started getting concerned. He asked me what he should do. I explained that I couldn't give any advice, but that he should visit the MD again and get specific advice about how to deal with the medication. Meanwhile, every time it neared the hour for her next dose, his wife would have the feeling of being suffocated, the horrible, loud gasping sounds, and the grasping for breath, her eyes bugging out in terror. Only another pill could bring relief.

Dr. Pender suggested that Zoe should cut the pills in half, and take them a little closer together for better coverage. Zoe ended up taking her pills closer together, and she inadvertently increased her total dosage as well. She was up to 16 pills, 3200 mg/day of levodopa. This time her husband called me on the phone and demanded that I tell him what to do. I said that they should see the doctor. Now the husband raised his voice with me. He screamed, "What good would that do! She won't obey!"

Zoe wasn't able to follow her doctor's advice – his suggestions seemed to make the symptoms worse. Zoe and her husband showed him their detailed, daily charts of the timing of the pill doses and her symptoms, but he wasn't interested. At this point, after all, he had concluded that she was only pretending to have Parkinson's so that she could get her hands on the drugs. He refused to see her. She went to another neurologist who, seeing from her charts that she had Parkinson's, renewed her prescription.

The breathing problem became so severe that they decided to have Zoe hospitalized. The hospital, not realizing that it takes nearly ten weeks[48] for any decrease in this medication to be stabilized, treated her with strong sleeping drugs for three days while decreasing her medication, and then sent her home with her L-dopa at half the level it had been. Within a week of being home, her drug levels were back up, and from there, they increased further, to 18 pills, 3600 mg of levodopa a day.

I was no longer treating Zoe. Her condition was out of control. She went back east to stay with family because her husband was afraid to leave her home alone. She wasn't making much sense; she was increasingly illogical and emotionally overwrought. Her arms and torso were in constant movement. I did not see her for over a year. When I last saw her, on a visit to her home, she was taking 20 pills of CR 50/200 carbidopa/levodopa a day – 4000 mg a day of levodopa, more than double the highest recommended amount. She was in constant motion, and her eyes were dilated like those of a cat. Her pupils were so large and round that the light reflecting off her retina glowed like the eyes of a lynx at midnight. She was alternately euphoric or sobbing. It was terrifying.

Through the grapevine I have heard that she is back east again, and that her husband is planning to have her admitted to yet another hospital. They will try once again to stabilize her over several days. I hope for the best. She remains in my prayers.

---

[48] This number, ten weeks, also known as "two to three months," is coming up with increasing frequency in books on addictive drugs. It was first recognized in the cocaine addiction pattern and is now noted in drug books in reference to many of the psychoactive drugs, such as Paxil. However, most doctors, including most neurologists that prescribe L-dopa, are not familiar with this time frame. Most of them still imagine that it takes a few days, maybe a week, to stabilize a drug at a particular level despite the drug inserts that note that drugs such as Levodopa may take months before the drug shows its effect. Most MDs evidently do not read the drug instructions; in our experience, most of them expect a patient to have results within a few days and prescribe accordingly.

TREATING TWO ILLNESSES, NOT ONE

People who were taking drugs for their Parkinson's disease apparently had two problems, not one: they had Parkinson's disease, and they also had drug-related problems. If they ever, even briefly, had been overmedicated, manifesting adverse effects, they seemed to have permanent brain damage from their medication. Even if most of their PD symptoms vanished and they stopped the drugs, they might have residual, long-term, drug-induced symptoms such as tardive dyskinesia. These manifestations of the brain damage are similar to the symptoms of Parkinson's disease; in fact, they are called "drug-induced parkinsonism" in medical literature. Despite a similar name, there is a significant difference between the two conditions: Parkinson's, based on our findings, appears to be treatable – brain damage from excessive antiparkinson's medications may not be, at least not by any method known at this time.[49]

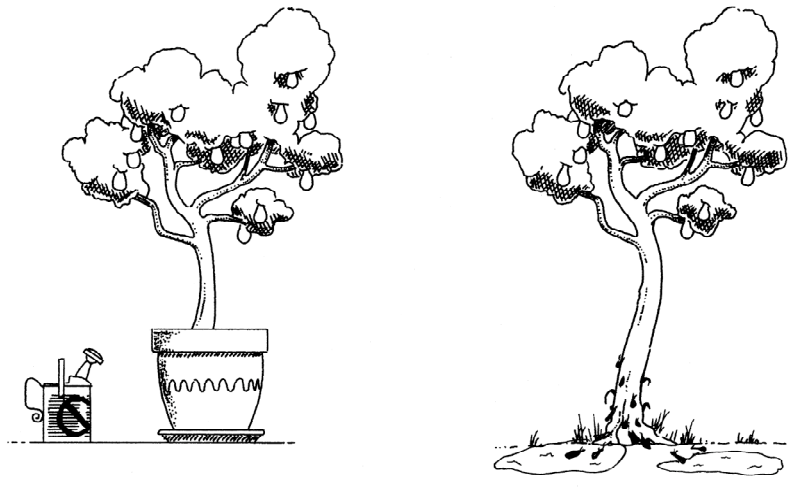


Fig. 2.1 Different root causes

Both plants have clumps of dying leaves hanging down; the plants look identical. The plant on the right is doing poorly because bugs have invaded the roots and are sapping the trees nutrients: a remediable condition. The plant on the left has been fed poison: the roots have all died. Although the dead leaves of both trees look similar at this early stage of the process, the plant on the left cannot be restored to health; the plant on the right can recover if the bugs are removed. (Explanation for Fig. 2.1 is continued on the next page.)

In the case of drug- or toxin- induced parkinsonism, brain damage from drugs and toxins may well be permanent; all current research indicates that this is the case. The root problem in idiopathic Parkinson's, an electrical disarray, appears to be treatable. PDers who are taking medications have *both* problems: a treatable, backward-flowing electrical system and permanent damage from their drugs.

[49] In Parkinson's disease, "although dopamine is depleted, the cells in the striatum are preserved. This is unlike the PD-like disorders (drug- and toxin-induced parkinsonisms) where, in the striatum, the dopamine content is decreased and the cells are lost." A. Lieberman, MD, "Curing Parkinson's Disease in our Lifetime," *Parkinson's Report*, National Parkinson's Foundation, Fall 2000, p. 10.

Summary

Wrong doses, ignored warnings

The examples of Olli, Zoe, and the well-meaning but unhelpful, even obstructive reactions of their neurologists, may give you a sense of our bafflement and helplessness during these early times in our study. A large part of the medication problem appeared to be ignorance on the part of doctors as to how these medications worked and the correct dosing. This ignorance contributed to conditions such as Rose's writhing and thrashing and Zoe's inability to reduce her medication.

We realized, to our dismay, that most doctors were not only failing to prescribe according to the manufacturers' recommendations, they were blithely ignoring the published drug warnings. But most curiously, it appeared as if the drugs did not work in the ways that they were purported to work, especially with regard to drug half-life and brain adaptations.

The next chapter will share hypotheses that we developed that, unlike the existing "facts" about dopamine-enhancing drugs, actually seem to correspond to what happens with people who take antiparkinson's medications over an extended period of time.

"The golden guess is morning-star to the full round of truth."

Alfred, Lord Tennyson's "Columbus"

3. THREE HYPOTHESES

OUR RESEARCH BORE US FRUITS: THESE THREE IDEAS

A new model for dopamine-enhancing drugs

Some patients, even before starting treatment, had a history of problems with their "unpredictable, unreliable" drugs. Some *recovering* patients' drug problems were, although similar, highly exaggerated, almost a caricature of the normal drug problems. As we charted years of the latter's extreme drug responses during drug decreases, increases, and unchanged doses, unexpected patterns emerged. Certain patterns could only be explained using a model with the following characteristics: the drugs have a much longer effective period than the published half-lives, the motor threshold (dopamine level required for movement) rises and drops over the course of twenty four hours in response to excess or insufficient dopamine, and the baseline level (native, brain-produced dopamine) of dopamine activity declines over several months in response to dopamine excess.

This new model, unlike the drug theories in the textbooks, predicted a motor (movement) response to dopamine-enhancing drugs that actually matched what was happening with the medicated patients, whether their problems were mild or cataclysmic. This model transformed "problems with drugs" from mysterious to predictable.

As a corollary subject that will be discussed later, a completely new model for neuron (brain nerve) dopamine-processing rates also needed to be built. Motor function, frontal lobe (thinking and emotion) function, and limbic (primal, non-thinking) function each seemed to be traumatized or restored to health at different rates during our patients' ten-week drug reduction or increase cycles. To account for the apparent differences, we constructed a model in which the various types of neurons from the various areas (motor, frontal, and limbic) had different refresh rates. (Refresh rates are the pause after a nerve impulse, during which the nerve cannot fire off again.) The refresh rate in the motor area appears to be very fast, the frontal lobe somewhat slower. The limbic area of the brain appears to adjust to dopamine changes much more slowly than the motor area – possibly over months. It also appears that excess dopamine can slowly accumulate in the vesicles (dopamine storage tanks) that feed the limbic-function neurons, but not in the motor area. Motor function can, in the short term, respond more quickly to a quick flash of dopamine drug than can the limbic area. With regard to longer-term dopamine distribution or accumulation, the motor area is subordinate to the limbic zone.

Using this corollary, we propose that the production rate of native dopamine could slowly decline in response to an accumulation of externally supplied drug-based limbic dopamine, and also the reverse: it could slowly – within the constraints of Parkinson's disease – increase in response to limbic insufficiency. Supported by this model of various brain areas having different dopamine-processing rates, we can defend more fully the first model in which dopamine levels go up and down based on half-life, motor thresholds quickly go up and down in a short-term response to changes in dopamine

levels, and native baseline dopamine levels slowly go up and down in a long-term response to slowly accumulating or dispersing limbic dopamine levels. We could suddenly predict not only how the drugs would behave over the course of a day, but also how they would change in effectiveness over the long term, whether increasing or decreasing the medication.
The new discovery that excess dopamine is at the root of drug addiction dovetails neatly with these models and will be connected in later chapters. An ominous corollary to this proven discovery is this: all dopamine-enhancing drugs (DEDs) are addictive.
Finally, another realization emerged: the brain's addiction response to DEDs is different in a PDer than in a non-PDer.[1]

Condensing the models

We lumped the details of the first two models together, and created the two basic hypotheses that were described in the first chapter: dosing of psychoactive/psychotropic drugs must take into consideration both the full span of the drugs' effective period (as opposed to the venerated blood half-life), *and* the brain's threshold and baseline can change in a predictable manner in response to the drugs. Our third hypothesis only applies to people with Parkinson's, and holds that the unique attributes of PDers render them somewhat resistant to addiction until such time as they recover. This chapter will expand on these hypotheses. Our suspicion that different types of neurons respond to dopamine at various tempos will be discussed in chapter seven.
Superficially, many exceptions to these hypotheses seem to exist. Over the course of this book the seeming exceptions and contradictory experiences of some antiparkinson's drug users will be explained after all the factors are put on the table.
This chapter will expand on these basic ideas and provide graphed examples, but first, before jumping into the meat of the hypotheses, here are some preliminary details.

PRELIMINARY DETAILS

The graphs cannot be true to scale; the minute amounts of dopamine involved in the exquisite precision of brain chemistry compared to the gross dumping of pharmaceutical dopamine precursors or agonists into the brain cannot be adequately portrayed on a chart. Also, PDers assess drug response based on motor function, not molecular-level changes. Therefore, the charts in this book that graph functionality per pill will show the motor effects, for the most part.
When reference is made to "dopamine activity" or the "dopamine system," it refers to the entire constellation of molecules and receptor sites that influence dopamine in the brain.[2]

---

[1] Since this book was first web published, a report has been published in the journal *Neurology* Nov 25, 2003, Drs. Steiner and Wirguin, (2003;61;1451) confirming this. Four patients who had been misdiagnosed with Parkinson's disease and given L-dopa had behaved as if the L-dopa was highly addictive: "agitation, palpitations, fear, and sweating accompanied attempts to discontinue the medication, despite lack of efficacy and gradual tapering of the dose over a two week period." The report also mentioned unauthorized, very large, rapid increases in dosage in non-PDers who were mistakenly prescribed L-dopa. This rapid desire for dose increase does not occur with people who actually have Parkinson's disease who are taking their medication at the correct level, nor do their drug reduction symptoms correspond so closely to addiction symptoms if the drugs are decreased correctly.

[2] A significant amount, possibly more than 90%, of a body's neurotransmitters are produced and utilized in the non-brain portion of the body. In fact, some researchers suspect that most of the body's

Dopamine levels in the brain cannot actually be measured. Also, one cannot measure dopamine activity by simply looking at the quantity of dopamine being delivered by a drug dose; one must also consider the level of native dopamine production, the number and accessibility of dopamine receptors, the quantity of dopamine transport, reuptake, or breakdown enzymes, and whether or not these enzymes are being stymied.

Vocabulary for interpreting charts

These charts have axes representing thresholds, baselines, and motor excess.
The threshold is the amount of dopamine activity necessary to elicit a motor response.
The baseline is the amount of dopamine activity produced by the brain, also called native dopamine, as opposed to drug-derived dopamine. Even though baseline amounts can change, they change slowly, over weeks, not hours. Therefore, the baselines on the daily charts will appear to be straight lines. In a healthy person, the baseline and the threshold are the same line. In a PDer, the baseline is lower than the threshold.
The changing threshold will show the short-term changes that occur in response to dopamine-enhancing drugs, as evidenced by motor function.
The changing baseline will show the long-term changes that occur in response to dopamine-enhancing drugs.
For our purposes, whether or not the change occurs in the production of dopamine itself, the accessibility of dopamine receptors, or the quantity of dopamine transport, reuptake, or breakdown enzymes, we will graph these changes as either changing threshold (short term) or changing baseline (long term) depending on the time frame of the change, and not on which specific molecular process in the dopamine system is modified.
The excess line is the line above which obvious motor pathologies erupt: dyskinesia, On-Offs, ticcing, shaking, or freezing, to name a few.
When in response to the day's drugs, the threshold begins to rise, the graph will show both the original threshold and the new, changing motor threshold.
The dopamine line shows the changing levels of dopamine-enhancing drugs. This line may go up and down several times during the day in response to dosing.
The dopamine excess line is the line above which addiction can occur. This line is located a hair's breadth above the original threshold line and is not drawn in nor labeled. It does not move. Any amount that goes over the original threshold by even a few molecules may violate the excess line. Even if the motor threshold rises during the day, the dopamine excess line remains in the same place, a hair above the original threshold.
As you observe these graphs then, be aware that the line not drawn, the line that should signify Too Much and which represents the amount that triggers addiction, is the merest breath above the original threshold line. The Off to On (Not Moving to Moving) line in a healthy person is at the original threshold. This line changes considerably and quickly in response to daily drug accumulation; the baseline changes slowly in response to Parkinson's disease or drugs. So bear in mind as you read these graphs that almost all motor function visibly in the On zone due to the use of drugs is also in the dopamine excess (addiction) zone.

---

neurotransmitters, including dopamine, serotonin, norepinephrine and others, are produced in the digestive tract. However, in this book, I am referring specifically to those neurotransmitters produced and employed inside the brain. A barrier membrane surrounding the brain prevents most inner-cerebral and extra-cerebral neurotransmitters from intermingling.

More about addiction

Excess dopamine is the trigger for drug addiction. While some people will be shocked to learn this, and most of the neurologists of my experience will be dumbfounded, it is a cornerstone of the medical establishment's current paradigm that dopamine-enhancing drugs are addictive. Although far too many out-of-date neurologists may consider this to be shocking information and new, you can read about it in the health section of your newspaper's Sunday supplement. Later in this book, there will be references and instructions on how to satisfy yourself as to the validity of this statement using current research tools, just in case you are working with a neurologist who has not kept up. But, as I say, this is old news, and is not a part of our new hypotheses. I only mention it here because it factors in to the charts of this chapter that explain some previously inexplicable drug behaviors.

THREE NEW HYPOTHESES – MORE DETAILS

1. BRAIN EFFECTS VS. MEASURED BLOOD AMOUNTS

We hypothesize that the amount of drug inside the brain's protective membrane (the blood-brain barrier), the drug's life span in the brain area, and duration of all brain-related drug effects are the main numbers to be considered when determining correct dosing. A major error in existing drug theory is the traditional idea that the amount of psychoactive drug that is floating in the *bloodstream* determines the effectiveness time frame of the drug.

In determining correct dosage levels, manufacturers and doctors often look for the fastest possible (though safe) way to obtain a *visible,* motor function result, seeming to ignore the recognized ten week build-up needed for these drugs to attain full effectiveness. They also base their recommended doses on the half-life of the drugs. I have repeatedly heard lecturers, drug company reps, and doctors explain their method of dosing based on the proven half-life of these antiparkinson's, psychoactive drugs. These half-lives are determined using blood tests.

The effectiveness of a psychoactive drug should be determined by how much of the drug is in the brain, not how much is in the blood. The blood-brain barrier selectively pulls certain chemicals inside the brain while refusing admittance to other chemicals. This means that concentrations of various items such as sugar, vitamins, hormones, neurotransmitters, bacteria, etc., can exist at different concentrations in the brain than they do in the rest of the blood stream. Brain amounts of drugs may be considerably different from bloodstream supply.

This is old science and has been around for more than thirty years. Why this knowledge has not been applied to the understanding of brain drugs is most likely due to the fact that we have no way of measuring what is going on in the brain, and therefore, *blood* measurements of brain-altering drugs are rigorously studied as if they were of supreme consequence.

Also, the mechanisms for dismantling chemicals in the brain (enzymatic, primarily, and based on supply and demand) are different from the mechanisms used in the rest of the body (digestive, liver and kidney, primarily). Thus, due to brain selectivity and variation in breakdown method, both the amount of a drug in the brain and its duration in the brain may be considerably different from the amount and its lifespan in the bloodstream.

While it may seem obvious to you, the reader, that the effectiveness of psychoactive drugs depends on brain levels rather than blood level, this principle is not usually used in drug research. This may be because there is no process for determining how much drug passes into the brain region, and, conversely, it is easy to measure (using blood tests) just how much drug is floating around in the bloodstream. Therefore, this relatively misleading number, the blood drug level, is used in making guesses about drug duration. Based on tracking the patterns of pill dosing, mobility, and dopamine baseline/threshold changes over tens of thousands of patient hours, we empirically derived actual rates for

certain half-lives of drugs in the brain. These rates, as opposed to the theoretical rates, match what happens in real life.[3]

Two hour half-life of drugs in the blood: the current theory

This chart shows the rate at which levodopa is used up in the body, *according to the current thinking*. Carbidopa/levodopa has a stated half-life of 1 to 2 hours. That means that within two hours of taking a pill, carbidopa/levodopa in the bloodstream will have already surged up to its peak level, and then, due to breakdown mechanisms, will have decreased down to a quantity of one half of the peak amount. The theory continues that, within another two hours, the amount in the blood will have again reduced by half. According to this theory, a person taking carbidopa/levodopa every four hours will have a response to the drug as predicted by the following chart.

The current model

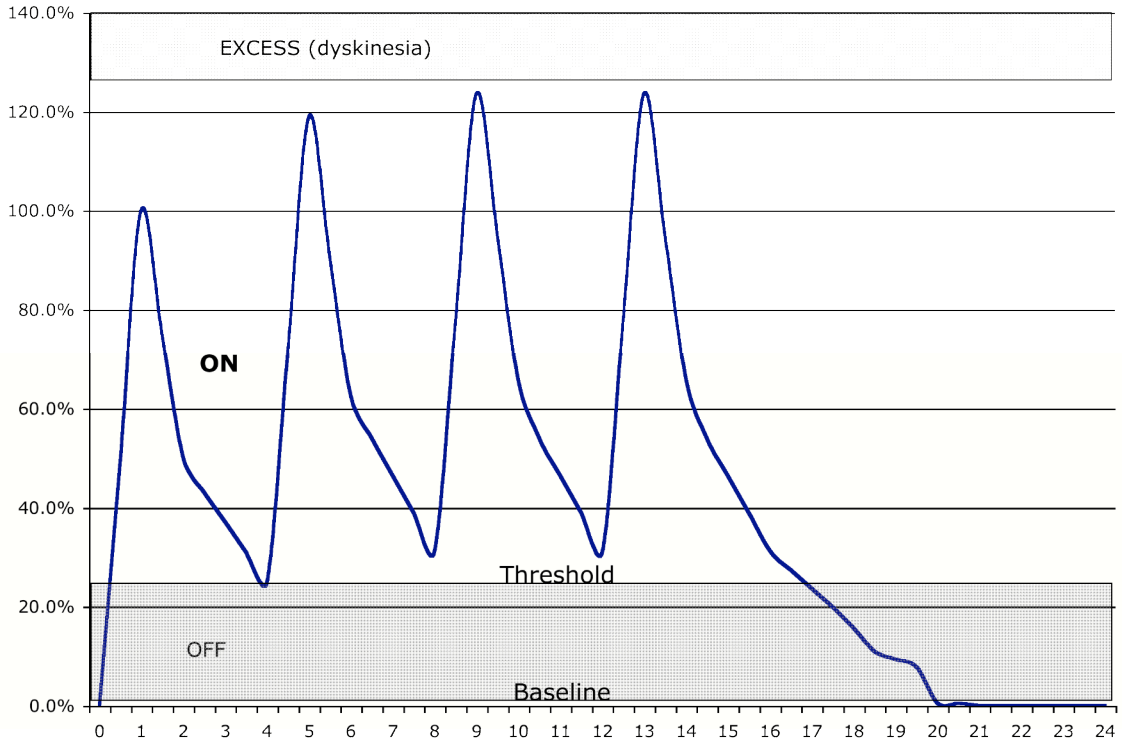


fig. 3.1

In the above graph, 100% refers to the amount of drug from one dose. The up and down line is the dopamine line.

The baseline shows the pre-pill dopamine level in the brain. The threshold is the amount of dopamine required to initiate movement. The EXCESS area of the graph shows levels of dopamine that are so high that the brain rebels via dyskinesia, freezing, and other visible or obvious forms of excess, including the other known adverse effects. Addiction is not noted on this graph – it occurs, in susceptible people, at any point above the

[3] Details on brain rates for most of the dopamine-enhancing anti-PD drugs are in the appendices.

threshold. The On area of the graph indicates the amount of dopamine above the threshold, but below the EXCESS line.
As far as brain health is concerned, dopamine levels in the above graph (fig 3.1) are excessive throughout most of the On area. Unfortunately, only when overt dyskinesia or other adverse effects appear can we see that the brain is grievously overmedicated. Again, anything that is more than a hair above the perfect amount is actually excessive. In these graphs, however, the Excess zone only refers to anything *obviously* excessive to the naked eye.

According to the half-life-in-the-bloodstream theory, by taking a dose every four hours, a PDer should be able to have the following motor function: he will be On for most of the day and never dip down into the Off zone or go up into the EXCESS zone.
Note that in the above graph (fig. 3.1), the second pill of the day does not start down at a baseline of zero – there is still enough medication left in the blood from the first pill of the day that the second pill of the day piggybacks onto the first pill's remains. In theory, this combined effect – the residue of the first pill together with the second pill – allows a person to be comfortably On all day, with no apparent Off times, nor any dyskinesia.

The new model

A 4- to 6-hour half-life inside the brain

However, this idealized condition does not actually occur. After a person has been taking the medication for long enough (two to five years, and in rare cases ten years) to be experiencing "instability" of the drugs, it becomes visibly obvious (due to long-term, addiction-related changes in the threshold and baseline) that what has been happening right along was a pattern of drug build up over the course of each day in which the peak amount from each pill is noticeably higher than the previous peak.
Eventually, as dosage levels increase, the increase in peak amounts over the course of each day accumulates all the way up into the obvious, clearly visible Excess zones. What is going on with the drugs, whether or not we can actually perceive the build up at small doses, is less like the first graph (fig 3.1), in which every dose after the first one attains the same height, and more like this:

Build up of pharmaceutical-derived dopamine during the day

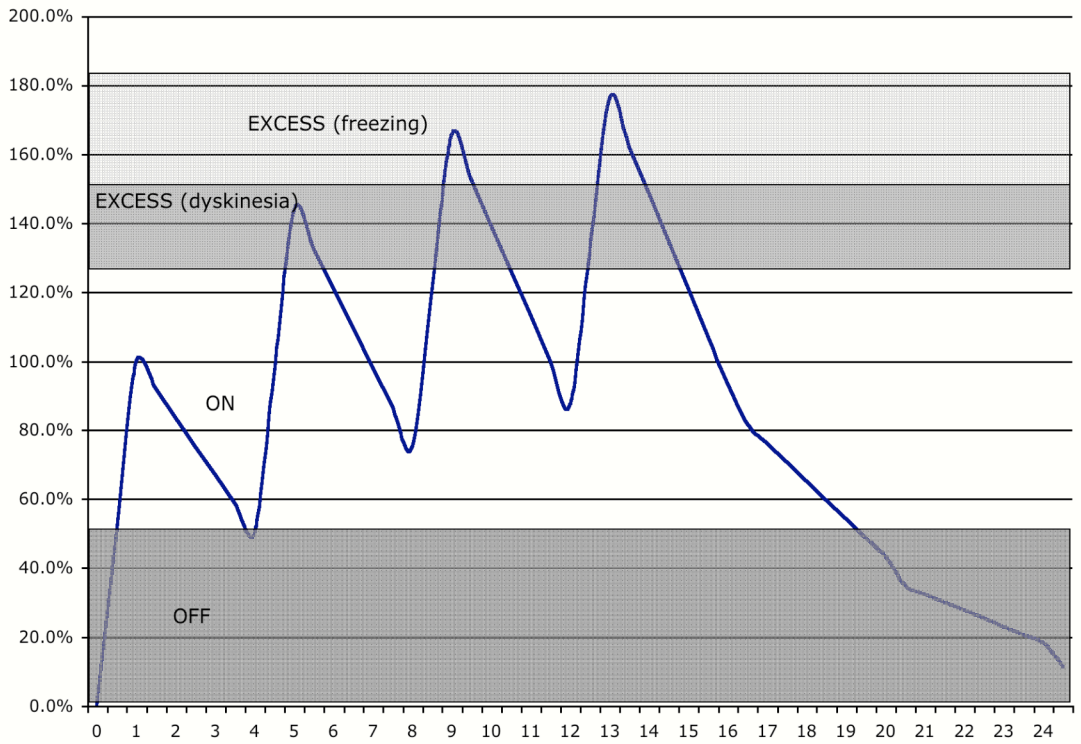


fig. 3.2 Due to accumulation, each peak is higher than the one before.

Notice that in this graph (fig. 3.2), there are two EXCESS zones: dyskinesia and freezing. The freezing zone is a severe brain response to the medication during which a person

may be unable to move and may, to all intents and purposes, look as if they had no medication in their system whatsoever. [4]

The freezing zone

Freezing in the four hours after taking a pill, during times when the drugs *should* be at levels of highest saturation, is usually due to a build up of dopamine. Accumulating over the course of the day, the excessive amount of drug eventually bulges up into the freezing zone. Most patients who had unpredictability of pill coverage found that afternoon and evening doses were the most problematic.[5]

Accumulation of dopamine corresponds to a longer half-life

When we charted the times of On, Off, and Freezing of these patients, we noticed that this increase in pill failures across the course of a day could be explained by a ***4- to 6****-hour* half-life but could not be explained by the *2-hour* half-life of drugs in the blood theory.

Example of half-life in another anti-PD drug, Mirapex

Although the above example is for carbidopa/levodopa, which has been shown to have a 2-hour half-life in the blood, this example will also hold up for all the other antiparkinson's medications – all of their dosing recommendations have been made based on blood levels. To make the same demonstration using another drug, let's look at Mirapex, a dopamine agonist. This drug is deemed to have an 8- to 12-hour half-life, with a presumed steady state reached within two days of dosing.[6] Again, this is based on blood levels – brain levels are not measured. The implication is that, despite all the selectivity of the blood-brain barrier, brain levels should somehow be at equilibrium with blood levels.

The charts of our pioneers suggest that, once inside the brain, Mirapex has an effective life of closer to three days – nearly six to nine times as long as the blood life. It also appears as if ten <u>*weeks*</u> at a given dose, not two days, are required to attain a steady state in limbic function – one in which the blood levels are at equilibrium with the brain's limbic levels. This ten week number was determined by patients starting to take Mirapex who, rather than increasing their dose every week or two – the manufacturer's recommended rate – chose to take the drug at the same, very low starting level for several months, while recording their subtle daily changes or the lack of change. Then, only after all changes that might have been drug related were in a relatively steady zone, they again

---

[4] Please note that, in order to make a meaningful graph, one that approaches the dyskinesia line, I am, in fig. 3.2, assuming a dosage level that is higher than the usual beginner's dose that was charted in fig. 3.1. Fig. 3.2 assumes a dosage level at which the side effects begin to become a problem. For most PDers, this level is not their starting dose, but may be the dose they are using after one or two drug increases. A person taking a small dose may not notice that the medications are invisibly building up over the course of the day, because even with the build up, the highest amount of the day will still be less than the dyskinesia level. Eventually, due to gradual, addiction-based changes in the baseline, the patient must increase the dose. At a high enough dose, the pattern in fig. 3.2 will appear.

[5] Grossly overmedicated patients, especially those who take drugs during the night, will not conform to this model. Their dopamine never drops back down to a starting level, even during the night. That problem will be discussed later.

[6] *Physician's Drug Handbook*, p. 849.

made an increase. Again, they stayed at that new level for months – until their symptoms stopped changing – and so on. In every case, changes in response to the drug were best seen over a period of ten weeks, not several days, which indicated that the drug was slowly accumulating in the brain in amounts much greater than would be expected based on the proposed "steady state" achieved in the blood after a mere two days.

Suggested dosage levels may be higher than needed

The implication of this build up in the brain far beyond that anticipated by measuring blood levels is that the drugs, taken over months and years, are present at much higher levels than needed, even though they may not show overt signs of overmedication in the short term. Considering that all of these drugs are highly addictive and cause brain changes – including parkinsonism – it would seem reasonable to prescribe the drugs at dosage levels much lower than the ones currently used, and explain to patients that the best results will be obtained over an extended period of time.

Quick relief vs. safety

Drug manufacturers and doctors are dealing with patients' desires to have as good a result as possible, as soon as possible. Most patients – or so the assumption goes – would not be interested in a gradual result from a drug, even if the gradual result was much safer in the long run. Drug sales are competitive. A patient who doesn't get a rapid result will likely switch to another drug. With billions of dollars at stake, manufacturers might be more concerned with attaining a quick, satisfying result than with any other aspect of drug action. However, our model suggests that smaller doses and better patient understanding of the time frames required for drug accumulation are crucial in avoiding "unpredictability" and adverse effects.

2. BRAIN CHANGES, BOTH SHORT-TERM AND LONG-TERM

The second hypothesis is that the brain itself changes in response to the presence of dopamine-enhancing drugs. Actually, this is no longer a hypothesis; as I write this in 2003, this hypothesis has been proven, confirmed by researchers in the field of drug addiction. However, this was one of our unproved hypotheses when we started, so I will include it here as one of our formative ideas. This important concept of brain changeability is ignored almost entirely by the drug-prescribing, clinical (working with patients) neurology community but is currently fascinating those doctors who are researching addiction, as well as the theoretical biochemists such as Candace Pert.[7]

---

[7]C. Pert, *Molecules of Emotion: The science of mind-body medicine,* Simon and Schuster, 1999.

Before this book went into print, one of my proofreaders gently complained to me that I had not included the letters "PhD" after Candace Pert's name. "All of the men get referred to by the title of 'doctor,' but the only important woman in your book doesn't get any recognition of status," she pointed out.

Therefore, I would like to make clear that although all the clinical doctors described in this book have been assigned fictional names and are treated as male, not all of them are actually men. I would also like to paraphrase the head editor of the *American Journal of Acupuncture*, B.G. Grace, when she declined to use my educational titles on articles: "Either ideas are valid or they are not. No quantity of initials after the writer's name should sway the reader; let the reader determine the value of a person's work on the merit of the ideas, and not on the alphabet soup that follows the writer's name."

Modern researchers in the field of brain chemistry and or brain function tend to look for the very small details, such as the specific receptor affected by a particular type of drug, the shape of a neurotransmitter transporter, or the duration of nerve response, and then publish papers on this very specific, microcosmic view of the brain; the big picture is not often considered. We did not go the micro route. Looking at the long-term, empirical results of our patients' drug practices, we made generalities about the grand scheme of brain changes that occur in response to dopamine-enhancing drugs. These changes can be immediate, short-term (immediate to 24 hour), long-term (approximately ten weeks) and/or somewhat permanent.

Short-term changes

"Short-term changes," in this book, refers to changes which occur over a span of hours, or a few days. Long-term changes take place over weeks, months, or years.

Examples of short-term changes in the threshold

An example of a short-term change in threshold is demonstrated by a morning dose that works just fine for four hours, but when the same dose is again taken in the afternoon, it only works for two hours.

Another example of a short-term change is the "crash" that sometimes follows the effective period of a dose. Patients often crashed – they were worse after a pill wore off than they were before taking the pill. Very often, the crashes would worsen in severity over the course of the day. The crash appears to be due to a rapid shut down of some aspect of the dopamine system in response to excess dopamine.

These changes imply a reduction in underlying native dopamine activity after the wearing off of a dose. In graphs, these reductions corresponded to increased and more severe Offs (dopamine line falling below the threshold) and a changing threshold over the course of the day. What brain mechanisms might cause this reduction?

Two examples of mechanisms that reduce dopamine activity

1. Levodopa: One confirmed addiction mechanism (shut down) in the dopamine system is the decline in dopamine transporters located in the nigrostriatal nerve terminals in response to L-dopa. This result was seen using SPECT scans in a large study (142 patients) of similar, early stage PDers in which some received placebos and others received various amounts of L-dopa (up to 600 mg/day) for 40 weeks. The transporters were measured at the beginning of the study and again at 42 weeks, leaving twelve days for the levodopa to "wash out" (their term) of the system. After twelve days with no drugs the patients who had taken the most L-dopa had a 7.2 % decrease in transports compared with a 1% decrease in the placebo groups.[8] This suggests that shutting down

[8] Completely ignoring the fact that levodopa's effects build up or ebb over ten weeks, the researchers cleverly chose to assess motor function after less than two weeks of drug withdrawal and proclaimed that people using L-dopa were still moving better than the placebo group even with "no L-dopa" in their system, thus reaffirming for gullible readers L-dopa's prominence on the Parkinson's stage.

This report was made at the American Academy of Neurology (AAN) 55th annual meeting. (AAN 55th Annual Meeting: Abstract S09.003. Presented April 1, 2003.)

As I have mentioned several times already in this book, most uninformed doctors imagine that three days is the time frame for "slowly" getting a person off L-dopa. But this study, known as the Elldopa study and sponsored by L-Dopa, was conducted by a high ranking, (Footnote continued on next page)

dopamine transport may be one of the mechanisms for the rising threshold.
2: Nicotine, a dopamine agonist (dopamine mimic) can latch onto a dopamine receptor as if it was dopamine.[9] The nicotine stimulates a given nerve to fire off once, after which the nerve remains unusable for twelve hours. Following stimulation by nicotine, the nerve behaves as if it has been turned off or temporarily taken out of service for half a day. Application of a large amount of nicotine, such as is delivered by one cigarette, will take a significant number of dopamine-sensitive nerves out of service for twelve hours. The decrease in accessible dopamine-using nerves following exposure to nicotine means that, for the next twelve hours, it will be much harder for the next dose of incoming nicotine to trigger as large a response as was obtained with the first cigarette. Another way of looking at this is to say that a lot more nicotine will need to be used the second time, saturating the system more fully, to get a response similar to that first, easy response when all the nerves were wide open. It is as if, in response to exposure to an EXCESS of this dopamine-enhancing drug, the threshold – the amount of dopamine needed to elicit a response – rises.
Both the nicotine-based nerve turn off and the levodopa-induced decrease in dopamine transport lead to a decrease in the effectiveness of a subsequent dose of DED (dopamine-enhancing drug). This decreased response is called a rising threshold. The threshold goes up; it requires increased dopamine to elicit a subsequent response. Graphing a rising threshold will show the decreased effectiveness of each consecutive dose and a crash (Off) during the lows that follow that dose.

(Footnote continued from previous page.) big name researcher, Dr. Fahn, the H. Houston Merritt professor of neurology and director of the Center for Parkinson's Disease and Other Movement Disorders at Columbia University in New York City.

John G. Nutt, MD, a nationally renowned professor of neurology at Oregon Health Sciences Center in Portland and recipient of a Parkinson's disease grant from the National Institute of Health, dismissed the imaging results (which showed more brain damage in the subjects with higher L-dopa levels) despite the fact that they resembled the results of an earlier study done by Mirapex, and played up the fact that the patients with L-dopa were still moving better than the placebo people after more than a week. They conveniently did not measure motor function again after ten *weeks* (the true wash-out duration) of "no drugs."

Regarding the 1% decrease in the placebo group, no mention was made of whether or not patients were screened for Parkinson's vs. parkinsonism, and whether or not any patients took antianxiety or antidepressant medications, both known to cause a decrease in dopamine levels – an effect widely ignored in the PD research community.

In a different study comparing levodopa and Mirapex over a longer period it was found that L-dopa users had a 24% decrease in dopamine transporters, compared to Mirapex users who had only a 16% decrease. No placebo patients were used in this latter study, a study designed to prove the superiority of Mirapex, and which was sponsored by the makers of Mirapex. By suggesting that the L-dopa group's damage only reflected the normal degeneration of Parkinson's disease (although L-dopa has been suspected for over thirty years to accelerate the progress of Parkinson's), the Mirapex makers went on to ludicrously suggest that, by virtue of causing less damage than L-dopa – which according to this study causes no damage (an unsubstantiated, self-serving statement) – Mirapex might have *actively slowed the decline of Parkinson's* and might even be a brake on Parkinson's disease degeneration! As noted above, no placebo patients (patients using no drugs) were used in this study. Had they been, the placebo people – with little or no decrease – would have proven that Mirapex was, in fact, causing damage, not preventing it. As you can see, it is important to find out who is sponsoring these studies and interpreting the results!

[9] I will use the nicotine example repeatedly. It is a good choice: everyone has heard of it, it is well known to be addictive, it is a dopamine agonist, thoroughly researched, and the damaging brain changes from nicotine (addiction) occur without dyskinesia taking place.

A rising threshold

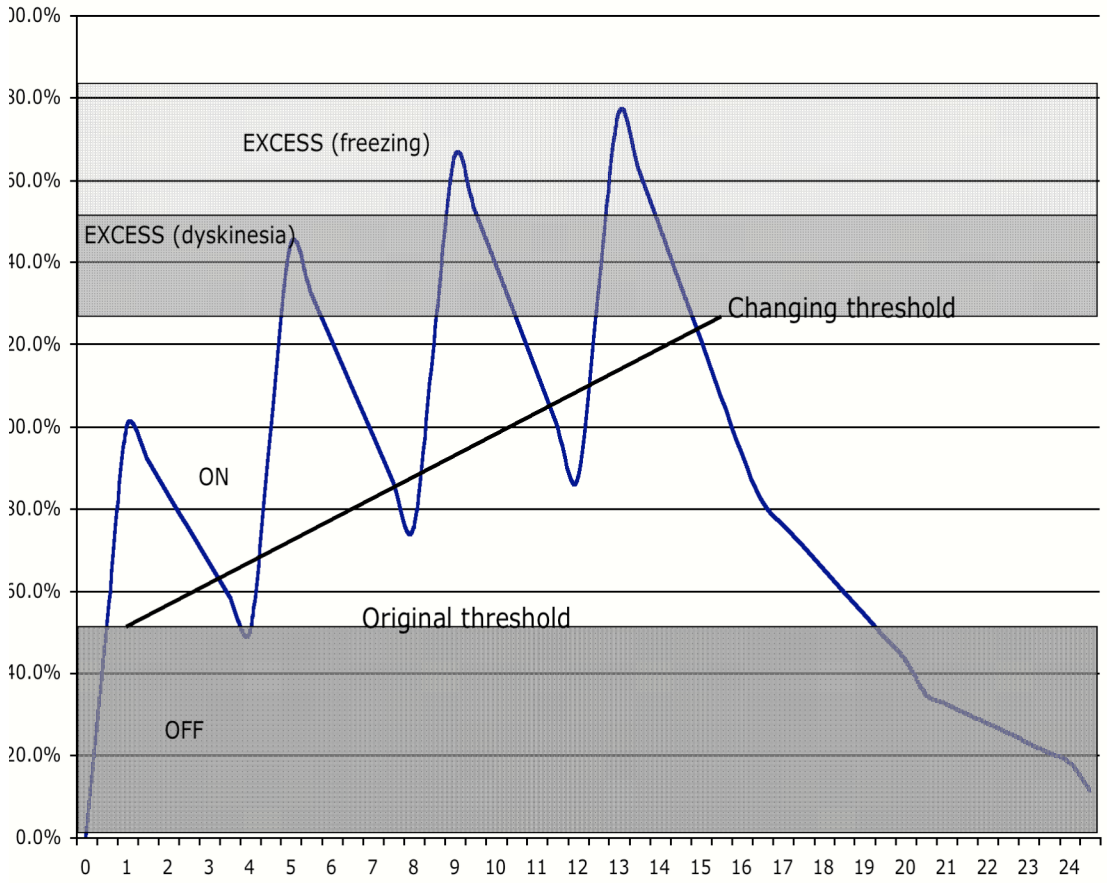


fig. 3.3

Explanation of the graph

The *original* threshold line is the line across the top of the Off zone, where the Off transitions into On. The *changing* threshold line is the thick line that moves diagonally up from left to right, starting at the original threshold line and rising up into the EXCESS zone by the end of the day. Remember, if dopamine levels are below the threshold, dopamine activity is too low to initiate movement. In the above graph, as the changing threshold rises, the lowest part of the dopamine line (the line that goes up and down with each dose) is often below the new threshold, even though the total amount of dopamine in the brain is steadily increasing over the course of the day.

Changing threshold can reset quickly

Most processes involved in this changing threshold appear to reset themselves back to their original position after twelve to twenty-four hours. These processes are the "short-term" changes. The brain's ability to set itself back to its pre-drug condition following short-term changes may be responsible for the fact that patients who do not take the medication during the night have fewer side effects and are able to take the medication

for more years before the drastic problems appear: by not taking the medication at night, they are allowing their threshold levels to reset all the way back to the original level overnight. When their thresholds are allowed to drop back to the normal level, the daily threshold-level patterns will repeat, more or less, in the same way from one day to the next.

If the drugs are taken at night and the threshold never drops all the way down, one day's thresholds may be completely different from those of the day before, and may never get an opportunity to drop back down to the original level. The strain on the brain may be the cause of increased adverse effects in these patients.

Since the threshold is affected by the amount of incoming drug, rising in response to excess dopamine levels, it is clearly advantageous to always take medications at the *lowest possible* On-producing levels, thus keeping the threshold as low as possible.

Long-term changes: affecting the baseline

Most addictive drugs can also set in motion long-term changes in dopamine activity. One such change is the decline in baseline levels of dopamine activity. Some drugs also create emotional or memory-linked changes.

Baseline of zero

In our graphs, it looks as if the baseline amount is zero. That is simply for visual convenience. Your brain baseline amount of dopamine is never actually at zero; if it were at zero, you would be dead. In fact, even if you have Parkinson's disease, your baseline amount, your naturally occurring dopamine level, is not at zero.[10]

However, the baseline (the amount of dopamine activity produced by the brain) can change due to the influence of dopamine-enhancing drugs. The baseline can slowly go up or down so that the *combined* amount of the native dopamine (dopamine grown by the brain) and native dopamine activity, plus the external supply (drugs or supplements), equals the "normal," or customary, amount of dopamine activity. Here is an equation that demonstrates this:

Drug amount
+ Native amount (changeable)
==========
Normal dopamine activity (constant)

"Native amount" refers to dopamine activity generated by the brain, an amount that can change as the brain sees fit. "Normal amount" is the same as the original baseline. In Parkinson's disease, a healthy normal amount no longer exists. With PD, the native amount, the normal amount, for reasons of its own, is lower than the healthy amount.

[10] If you take dopamine-enhancing drugs long enough at high enough doses, you can force your baseline down to a very low level, effectively approaching zero. This may be why people who abruptly discontinue their medication after having taken it for many years may risk sudden death. As the drugs slowly are cleaned out of the brain, the limbic baseline of nearly zero becomes exposed. After one to three weeks, while the pharmaceutical limbic accumulation is used up and is not replaced by the accustomed drugs, the limbic system is revealed as being dangerously low. The body may go into shock; death may occur. It takes about ten weeks for the limbic zone to accommodate to a change. More on this later.

However, in PD, the body seems to desire this decreased amount of dopamine. Any attempts to increase the amount of dopamine activity by using drugs will be met with a further decrease in brain-produced dopamine activity.

Drug amount
+Native amount (changeable)
==========
Parkinson's level of dopamine activity or, what is normal for the PDer

The point of the equation is this: your brain will gradually make less overall dopamine if it is receiving a supply of dopamine from an external source. On the other hand, if the baseline has been lowered by drug use, then when the external supply of dopamine is stopped, the baseline (native amount) can slowly increase again, over approximately ten weeks, until your brain is making *almost* as much as it was before. If you are healthy, the brain will slowly come up *almost* to the healthy level. (The degree of *almost* depends on which drugs were used, in what quantities, and for how long.) If you have Parkinson's, the brain will slowly come up *almost* to its pre-drug Parkinson's level.

You can't go home again

Unfortunately, I must use the modifier "almost" in the preceding sentences. It does appear that if a person has ever been addicted (the brain has initiated either short-term changes, long-term changes, or both), the baseline may never go completely back up to normal and will always hover near, but just below, the previous normal level (either optimally healthy or PDish).

The optimal healthy baseline of dopamine activity has a bit of breadth to it, to accommodate for daily fluctuations in everyday life. But the recovered addict rarely has a complete restoration to this level – his baseline will always be a bit on the low side of optimal. By never going back up to quite the normal level, this person's dopamine activity will always be extremely susceptible to conditions that deplete dopamine: vagaries of weather, mood, and illness, to name a few. During these types of dopamine-depleting events, a person with a previous history of addiction may actually slide too far below the healthy baseline level and may not have enough dopamine to function correctly in terms of mood or even motor function. This is why stresses of a dopamine-depleting nature can make a former addict need drugs again – his system cannot actually create enough baseline level dopamine to compensate for conditions that are in the least stressful.

Changing baseline decreases medication effectiveness.

In the original graph (fig. 3.1), notice that the baseline is at zero. Zero does NOT mean an absence of dopamine; zero on these charts means the original amount of dopamine activity when a person first started taking dopamine-enhancing drugs. Due to these drugs, the baseline will slowly go down.

For an example, I'll use an unrealistic, easy-to-look-at number to represent the amount of drug-induced baseline change: 50%. We will assume, for purposes of this example, that, on average, the drugs have added 50% to the baseline levels of dopamine. The brain will, over about ten weeks, decrease the amount of native dopamine activity by about 50%, by

way of compensation, to bring the total average dopamine back to an approximation of the pre-drug level. The new brain-produced portion of the total dopamine, the Native baseline, is now at minus (negative) 50%.
The daily amount of drug is exactly the same in the chart below as it was in chart 3.2. The accumulation over time is exactly the same and the duration of effect is the same. However, because the baseline has dropped lower, the same amount of drug does not even go into the On zone at all during the first dose. The subsequent doses also drop below the motor threshold during the beginnings and ends of each dose. This time below the threshold is Off time.
When the baseline of dopamine goes lower, the same amount of medication that previously kept a person in the On zone all day no longer provides consistent Ons, but, instead, alternates between On and Off.

Diminished baseline

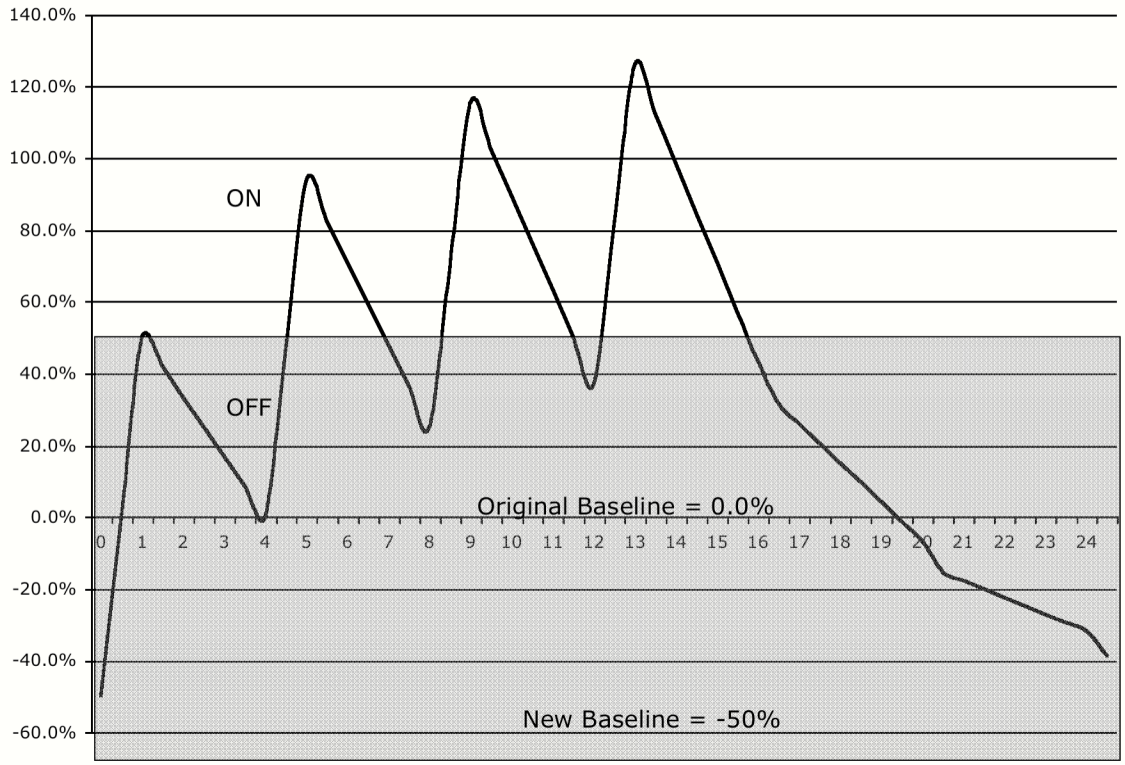


fig. 3.4

Compare this graph with the one labeled fig. 3.2.
Gradual lowering of the dopamine baseline is a normal consequence of using any dopamine-enhancing drug, including the drugs used in treating Parkinson's disease.

Variations between drugs

The rate at which the threshold drops back nearly to its original level varies from drug to drug. This in turn may affect rate of baseline change. The threshold's potential return to its original level overnight appears to be based on whether or not the brain is able to get rid of the drug between the last dose of the evening and the first dose of the morning. For example, the dopamine agonists, some of which have blood half-lives of days rather than hours, may never allow the threshold to drop back down to its original level even if taken in small doses.

These differences in drug-effect duration and the accompanying change in threshold may help explain some of the differences in withdrawal symptoms from various PD drugs. For example, although short-term drug withdrawal symptoms from the agonist drugs are usually less violent than drug withdrawal from levodopa, the post-withdrawal depression and emotional desire for the drug last longer after quitting agonist drugs than after quitting levodopa. This may be due to agonists' longer half-life, which never allows the threshold to revert back to the starting level overnight.[11] It may be that long-term baseline changes, which seem to be more permanent, take place more vigorously if the threshold-adjusting mechanisms are not adequate to reduce quickly a dopamine excess. Longer half-life drugs may fit into this more severe baseline-altering pattern.

Blaming the Parkinson's

Most neurologists in our experience do not know about changing baselines or thresholds; they attribute all decreases in drug effectiveness to the advancement of Parkinson's disease.[12] Blaming the increasing amount of Off time on the worsening Parkinson's, they assume that there is no choice but to increase the medication.

However, during the pioneers' experiments in reducing their meds during their apparent recovery (or misdiagnosis) from Parkinson's, they found that this assumption of worsening Parkinson's must have been inaccurate: in response to their extremely small reductions in medication, their baseline levels rose back up over approximately ten

---

[11] A person who successfully gets off the agonist drugs, while not undergoing the full terrors of drug withdrawal, may yet have a years-long longing for the drugs and succumb to the temptation to resume, for emotional reasons, these drugs many months or years after they thought they were recovered. On the other hand, people who experience the agonies of withdrawal from levodopa may find that, once they are six months down the road, they feel their desire for this particular drug mingled with a dread and loathing. Agonist quitters are much more likely to have only positive associations with their drugs.

The emotional attachments, physiological addictiveness and withdrawal symptoms all vary from drug to drug and are probably related to the various brain areas affected and the half-lives of the drugs. The longer half-lives of the agonist drugs may account for their preponderance of long-term changes. As you can imagine, long-term changes, all of which are aimed at reducing dopamine levels or dopamine effectiveness, are to be avoided as much as possible.

On the other hand, the agonist drugs have clear advantages over the other drugs in some respects; the relative receptor-selectivity of dopamine agonists compared to the non-receptor-selective drugs such as levodopa and Eldepryl prevents the agonists from triggering quite so many brain alterations so quickly, as the latter drugs appear to do.

[12] As did the respected Parkinson's researchers in the Elldopa study, footnoted earlier, who claimed to be "perplexed" by the (already known) finding that L-dopa caused a decrease in dopamine transport.

weeks, so that previously ineffective levels of medication gradually became effective again.

No evidence of problems in the first few years

During the first few years on the medication, a PDer invariably, though unwittingly, exceeds the subtle limit above which the dopamine-inhibiting (addiction) processes occur. This addiction limit is much lower than the line above which dyskinesia occurs. Therefore, a person can be constantly exceeding the limit without receiving any obvious physical cues, such as ticcing and grimacing, or noticing drug-related, post-dose crashes.

Damage can invisibly occur without dyskinesia

This should not surprise anyone: cocaine, cigarettes, alcohol, and heroin all cause hangovers or addiction without imposing dopamine levels that cause spasming. You would think, considering that researchers have known for over thirty years that L-dopa becomes "unpredictable" within two to five years, even at low levels that do not cause dyskinesia, they would suspect that something invisible is going on, and that they would have abandoned the very common "visible dyskinesia is *the only* indication of excess" theory.

3. Addiction and the Parasympathetic System

Finally, we hypothesize that it is their lifetime of living in a condition of emergency that provides for the relative unaddictability of people with Parkinson's.

People with Parkinson's appear to be relatively addiction-resistant compared to the general public, and they become just as addictable as anyone else when they recover from Parkinson's. It appears as if PDers live in a state of constant "sympathetic" response. (The terms *sympathetic system* and *parasympathetic system* refer to those nerve responses that accompany *emergency* and *awake relaxation*, respectively.) As PDers recover, their sympathetic system response drops almost to zero. Due to the cessation of the emergency, their entire dopamine system is reinstated immediately even as adrenaline drops back to normal (i.e., low or none) and the brain's pounding theta wave (in those with tremor) ceases.

Components of the dopamine-regulating system

We propose that during the sympathetic state, when heart rate, lung capacity, gastrointestinal function, and sensory nerves all become altered, left-right brain hemisphere integration is diminished. In its place, adrenaline pathways in the brain are triggered, pathways which go more efficiently straight through the brain to the frontal lobe, where increased focus and concentration allow for accelerated decision making. During this time, when dopamine ceases to be the neurotransmitter of choice, the entire Dopamine Package of vesicles, transport molecules, dopamine break down systems (including MAO enzymes), dopamine regulators, and *the addiction process* is suspended or greatly reduced.

We propose here that addiction is a part of the dopamine system. Because a PDer has a pathological increase in the sympathetic (adrenaline) system and a simultaneous decrease in the entire dopamine system, he is not particularly subject to addiction. As long as the dopamine system is turned off, its concomitant attributes, such as addiction mechanisms

and dopamine-related enzymes, are also turned off or functioning at a greatly reduced level.
When the sympathetic (emergency) system is finally turned off subsequent to healing the injuries that triggered Parkinson's disease, the parasympathetic system automatically starts up. Once this parasympathetic system is running again, not only is the dopamine-producing capability restored, but also all aspects of the dopamine system spring back to life, including sensitivity to dopamine – a sensitivity known as addiction.

The pain example

It has been long recognized that a person who is in tremendous pain can take large amounts of morphine, enough to "knock out a horse," as the saying goes, without becoming sleepy, "high," or addicted. We propose that this seeming immunity to addiction is due to suppression of dopamine activity during the sympathetic (emergency) condition of extreme pain.

PDers, during the course of apparent recovery, have changes in sensory, mental, and emotional processing, as well as in changes in motor function, which we attribute to increase in the dopamine system. (Return of motor function alone is not a good measure: this will be apparent only if their muscles and brain-motor connections are still sufficient to manifest the motor change. If they are not, there may be a very long period of muscle regrowth and brain retraining before the motor improvement becomes apparent and sustainable, even if their other Parkinson's-related symptoms begin to diminish.) Meanwhile, as changes indicative of increasing dopamine appear, the symptoms of lifelong reliance on the sympathetic system begin to disappear. The driven behavior and extremes of interpersonal relations cease, and personality is no longer marked by ferocious self-control or aggression, or extreme submission and passivity. The PD insomnia pattern reverts back to a healthy sleep pattern. The PDer becomes able to enjoy musing thought and slow, pensive integrative brain processes instead of the nearly instantaneous brain processing of their lifetime on adrenaline. Most wonderful of all, the PDer begins to feel a vibratory resonance with other humans instead of the almost physical pain that may have occurred in proximity to others, a pain that normally worsens through the years of advancing PD (and which is felt more often in those who tremor than in those who don't).

Addiction-prone

When PDers make the shift from a sympathetic system to a parasympathetic system, whether or not they have rebuilt enough dopamine, muscle tone, and brain-to-muscle wiring to manifest dopamine-based, healthy motor function, and whether or not their dopamine levels are still at a very low level, they become subject to addiction. We have repeatedly seen that some patients who begin to behave as if they are reverting from a state of emergency (sympathetic) back to a state of normalcy (parasympathetic) may also begin showing symptoms of violent addiction to dopamine-enhancing medication within 72 hours of the reversion. These people may not appear, physiologically, to have significant restoration of motor function, and their baseline levels of dopamine may not yet be fully restored.

Return of native dopamine does not manifest in instant motor function

However, it does appear that as soon as the sympathetic system shuts down, the entire Dopamine Package of dopamine production, dopamine transporters, dopamine inhibitors, regulators, and addiction processes all start trying to operate at full force. While it may take several years to rebuild the dopamine-producing structures in the brain so that they can provide adequate dopamine, the process of signaling the cells of the substantia nigra to resume their long-ignored tasks appears to happen almost immediately. The addiction process swings into full alert mode. This means that during the fleeting moments when the drugs pass above the original threshold the drugs suddenly are hideously dangerous and addictive, even though in the past, they were not. In the non-PD, non-emergency state, baseline and threshold changes may occur much more rapidly than they did when a person had Parkinson's.

However, even though a PDer's brain is protected from rapid, normal addiction, it is still mildly subject to addiction. We can be certain that there is, despite their protective illness, a mild level of addictiveness by looking at their slowly changing baseline (need to increase dosage over time) – a baseline that can slowly rise back up towards drug effectiveness at lower dosage when the medication is slightly reduced.

Improved baseline results from medication decrease

Again, this rising baseline of native dopamine that is seen in PDers who make a small reduction in their medication is evidenced, as described above, by a resumption of drug-effectiveness at lowered drug levels after about ten weeks at the lowered dose – whether or not they are recovering from PD. Therefore, because of the decrease in drug need that is seen in PDers who spend ten weeks at a lowered drug dosage, we conclude that much of the decrease in dopamine baseline levels is due to medication and not to advancing Parkinson's disease.[13] This would suggest that, although PDers are less addictable than normal people, they are still somewhat mildly addictable. Even with their relative protection from addiction, a PDer taking drugs at levels that, however invisibly, exceed the Safety Limit (the addiction point) will eventually have some addiction-driven decreases in his baseline dopamine levels.

This may be why the symptoms of PD appear to increase faster in people who take antiparkinson's medications than they do in those PDers who refrain from using the medications or keep their medications at levels that provide a modicum of ease but never take enough to feel truly "good." After all, many (though not all) of the symptoms of Parkinson's disease are related to dopamine levels: when dopamine levels drop, whether in response to the drugs (addiction) or from advancing Parkinson's, the apparent effect is the same – it appears as if the Parkinson's is worsening. However, when Parkinson-like symptoms worsen because of drug-related changes in the baseline, some of these changes (the long-lasting ones) are actually drug-induced parkinsonism, not idiopathic

[13] In the 1970's, before it became too costly and too dangerous from a liability perspective, patients whose drugs had become ineffective were routinely sent to hospital for a "drug holiday," in recognition of the addictability of these drugs. After spending more than ten days with little or no medication, their drugs would slowly resume a slightly better level of effectiveness. Too few doctors are still practicing medicine that remember the "drug holidays." Now, an increased need for drugs is nearly always assumed to be the result of worsening Parkinson's.

Parkinson's disease. Therefore, a medicated patient may eventually develop parkinsonism, in addition to Parkinson's.

PDers are addiction-resistant, not addiction-proof

The average person, a person who is not in pain, emergency, or experiencing Parkinson's disease, can become addicted to dopamine-enhancing drugs within a matter of a few uses of these drugs, over the course of a few days, or even an afternoon. After just a few uses of dopamine-enhancing drugs, an addict may find that he craves the drugs and needs to use more than before to replicate his original high. The PDer, on the other hand, usually is able to stay at the same drug level for three to six months before needing to make an increase. The decreasing baseline, though gradual, still may require gradual increases in medication over the months and years to maintain drug effectiveness.

These increases, rarely given in amounts exact enough to compensate for the subtle changes in baseline, instead soar with increasing frequency into the Excess zone, where they cause an increase in the appearance of side effects. Meanwhile, the increased doses also accelerate the daily rising of the threshold and the further decline in the baseline.

Slow increases in medication ➔ increasing adverse effects

Over the years, as the drug invisibly exceeds the addiction amount, these factors – the rising threshold and the decreasing baseline – begin to combine and overlap. At some point the baseline becomes so low that a drug increase must be instituted. At the same time, the threshold change in response to this increased surge of drugs may become so high that any On time will only occur during the Excess zone.

Drug side effect (freezing) mimics Parkinson's disease

As the baseline continues to lower, such very high amounts of medication will eventually be needed to rise up into the effective zone that any subtlety in achieving a desired effect will be impossible; the huge pill amounts, at peak times, may surge far beyond the Excess zone into a sort of Super-Excess zone where movement is impossible. This immobility area, above and beyond the dyskinesia zone, might be named the Frozen Limit. At this point, when the drug excess is causing freezing (immobility) and slowness of movement – symptoms that are practically indistinguishable from Parkinson's disease – further *increases* in medication are usually suggested. Within a few months these increases cause *worsening* of symptoms (increased adverse effects) rather than improvement in motor function. The PDer is usually told at this point that he needs more drugs because his PD is now more advanced. We propose the opposite – over time, at dosage levels that have been incorrect from the start, these dangerous, highly addictive drugs have, despite the addiction protection provided by PD, become a greater problem than the Parkinson's.[14]

[14] Again, this idea is not really new to us – the doctors who used drug holidays recognized that these drugs decrease in effectiveness over time, which is the same thing as being addictive. However, this knowledge appears to have been most inconveniently lost. Therefore, we are bringing up the subject again, with better evidence than before, based on SPECT scans, the nicotine study, and the evidence of our own patients. We are only re-proposing an idea that fell out of favor due to the expense and legal liability of drug holidays and the easy, over-familiarity of young, new doctors with these powerful meds. Possibly information about dopamine and addiction was not crucial to PDers in the past. Now however, as PDers try to recover while taking highly addictive drugs, the issue of brain damage and addiction must come again to the fore.

Summary

These three hypotheses: a larger amount and longer duration of psychotropic/psychoactive drugs in the brain than in the blood, a changing threshold of dopamine effectiveness, and the low addictability of PDers and the extreme addictability of recovering PDers – come together to paint a very different picture of antiparkinson's drugs than the models that are currently being used to determine drug dosages. In the past, these drugs were considered to be unstable and unpredictable because any given pill might have a very different effect from any other pill. However, when the three concepts in this chapter are quantified and added together, suddenly the drugs switch from being unpredictable to highly predictable. Not only that, but the daily and hourly effects of the drugs, if they are dosed with the necessary subtlety (unlikely if one is working with most doctors' instructions), are so precise that they can be used to determine whether or not a person is worsening or is hovering on the wings of a recovery from Parkinson's disease.

Whither next?

Over the course of this book, I will share specific examples of the cases from which we derived the above hypotheses. There will also be specific information about drug reduction and drug management programs based on these hypotheses that, unlike the proposals given by most MDs in our experience, appear to be safe. In chapter 18 you will be shown actual patient graphs and examples that support these new ideas.
I will also help you in another dimension of your explorations with the drugs of Parkinson's disease: to help you best work with your MD, I have included an Appendix with a brief history of the now-outmoded ideas of dopamine from the last five decades, so that you can understand why doctors in general have no agreement as to how these drugs work and why your doctor in particular, depending on when he graduated college, may be espousing a theory about dopamine that is no longer in sync with the latest paradigms.
Finally, if you detest graphs and charts, and if your eyes glaze over when you see an intersecting X axis and Y axis, be of good cheer – the rest of this book is nearly free of this sort of thing. Not only that, it is not necessary that you understand this chapter in order to follow the logic and the methods of safe drug increase or reduction hypothesized in the rest of the book.

Never lose sight of the fact that these ideas are only hypotheses. The whole truth about these drugs and the brain's constantly adapting chemistry may not be known for centuries. For now, these golden guesses are only a morning star, a hint at the knowledge that awaits.

"If I can ease one life the aching,
Or cool one pain…
…I shall not live in vain."

Emily Dickinson

4. A CASE STUDY: BECKY

A STUDY OF A PATIENT AND HER DOC

While western science reveres group statistics, the single-case study is increasingly recognized as a valuable research tool, especially in the fields of medicine and psychology. Humans are not uniform entities; predicting medical outcomes via statistical models is often meaningless at best, misleading at worst. A PDer with a PhD in medical statistics who observed our program for two weeks determined that, because of the ranges of age, medications, and symptoms in our patients – in our first 80 patients there were no two that were sufficiently similar for meaningful comparison – a minimum of a thousand patients might be necessary before useful statistics could be compiled.

Not having a thousand patients, but certain that we have something valuable to share, we have used over a hundred single-case studies of non-similar patients to create our hypotheses. Of those that are written up in this book, many are brief: a paragraph or two; some are several pages. Becky's case, which demonstrates nearly all of the principles of this book, is introduced in this chapter and extends over four chapters, interspersed throughout the book.

The case studies also serve to squelch the idea that drug reduction is easy. Charts and graphs can make the prospect of drug reduction look neat and straightforward, almost antiseptic. The truth is bleaker: although no one in our project died while reducing drugs slowly and safely, the process is unpleasant. These case studies provide examples of the risks of drug addiction, drug reduction, and the dangers of recovering from Parkinson's disease while taking antiparkinson's medications.

Becky

A quick summary of Becky's story

Becky was seventy years old when she started treatment in our program in April of 1998, and she was completely off the medication and symptom-free by October of the same year. Her only offspring, a sailor in the US Navy whom she saw once a year, came to visit in November. She wanted to show him how well she was doing and, in an inspired moment, thought of resuming her drugs for a few days to impress him. She realized, after taking the drugs for the four-day period of her son's visit, that she suddenly enjoyed them in a new way. She tried to stop taking them for nearly a year and was unable to do so. She finally stopped cold turkey and went through hell. Over the next four years she was caught in a vicious cycle of taking various antianxiety, anti-insomnia, and antiparkinson's drugs to allay the symptoms of drug withdrawal from the preceding drugs. It seemed that every time she was finally getting over the withdrawal symptoms, and looking haggard

and exhausted, her son would show up, drag her, against her protests, to the doctor, and the doctor and the son would determine a new course of medications for her.
Becky feared her doctor, but she feared her son more. She and her son had never gotten along well. The son was always asking about her finances, claiming that he should be in charge of her money and could make better use of it. He wanted her to move in with him and sign her assets over to him. When Becky started our program he was pleased; he could use her involvement in an Asian medicine research program as proof of dementia, have her declared incompetent by the courts and have her assets turned over to him. He told her since she was not following her doctor's orders with regard to taking her drugs, he would have an open and shut case.
Becky was careful to see her neurologist, Dr. Leslie, regularly, to prove to her son that she was competent. Dr. Leslie, in turn, was extremely hostile to our program, to Asian and alternative medicine in general, and most of all, to the idea that any FDA-approved drug might have side effects when prescribed for the appropriate illness.[15]
In a Dickensian coincidence, Dr. Leslie was the same doctor that had treated Rose, as related in the first chapter of this book.
Becky's struggles with antiparkinson's drugs, her son, and her doctor, are ongoing as of this writing. Throughout the years of these struggles, Becky never lost sight of her goal, which was to provide information that might someday be helpful to others. While some of my other patients were whining that they had been robbed of their due fun by the evil of Parkinson's disease, Becky kept hoping that her pain and anguish might someday help someone else. Her joyful spirit kept me going through the darkest years of this project.

Background on Becky

I had known Becky, a gutsy *bon vivant*, for more than twenty years. She was always positive, always doing for others. She loved to travel, perform in local theater groups, hike with the Sierra club, and bring good cheer to shut-ins. Strong-willed and brilliant, everyone who knows her loves her. She had been diagnosed four years earlier and was taking 400 mg/day of L-dopa when she started with us.
She started getting better within a few months of starting our program. Her tremor went away, her walk got better, and her facial expression returned – all the usual things. She was shocked when she could smell her jasmine tea again, for the first time in decades. She was amused by the tingling sensations in her face and feet as she regained sensitivity in those areas. She also scowled, good-humoredly, about the strain of the recovery back pain, the inexplicable sleeping in the morning from seven to nine ("I'm turning into a lazy bum!"), the feelings of sadness ("I'm crying at Oprah, for God's sake!"), the profound lassitude and weakness in the skeletal muscles ("I don't want to go anywhere or

[15] To be perfectly fair, this same doctor did amend his patients' drug prescriptions after I sent him a heavily referenced note pointing out that his frequent prescriptions combining MAO inhibitors with levodopa drugs were contraindicated by the manufacturer of Sinemet (L-dopa). I found this out when several of my patients, during their next visit to me, mentioned the surprising phone call from this doctor to tell them that they probably didn't need both of their antiparkinson's drugs and could stop taking either one or the other.

In an oversight, he failed to send me a thank-you note, but he did remember to tell Becky and several other mutual patients that he wished he could find a way to have me jailed for practicing medicine without a license.

do anything. I plan my days around doing as little as possible!"), and the experience of all the puzzling problems that were occurring to the other pioneers.
She took a very unusual approach with her drugs. Unlike all the other drugged pioneers, she started reducing pills as soon as she started receiving treatment. At first she tried not taking one of her two daily pills. Within a week she felt very rigid and uneasy. She tried this a few times and then decided to reduce more slowly. Every day she would just nick one fourth or one third off of one of her daily pills. After several weeks of this,[16] she started breaking a corner off both her daily pills. A few weeks after this, she started cutting one pill per day in half and only taking the half of that pill, saving the remaining half pill for the next day.
She stayed at each level of decreased meds for a week or two, or even three, until she felt that the short-term effects of the reduction – primarily, feelings of general malaise and lack of focus – had passed, and she was nearly back to where she had been. But she never went exactly back to who she had been before, before the Parkinson's had ever been diagnosed. Something was starting to change in Becky, and in all the other recovering pioneers, but we didn't know quite what it was or why it was happening.

Becky starts to change

Many of these changing symptoms made her feel as if her very personality was being altered. For example, all her life she had been a high-speed worker, very productive. As her Parkinson's disease had been worsening in the previous two years, she had felt the need to strive harder to be able to maintain her old level of performance. What was happening to her now was that she felt that she was changing, relaxing, and that she didn't mind so much if she didn't get all her chores or social obligations done. She was moving more slowly but minding it less. We know now that this is normal during recovery from Parkinson's disease, and that it signifies an apparent decrease in adrenaline and an increase in dopamine levels. At this time, however, in the earliest days of this project, we did not know if her new lassitude and laissez-faire were coming from her med reduction or from her recovery, if any. We were all riding blind.
So not knowing what was causing what, she continued reducing her meds by a little chip off a pill here, a half a pill there, from her starting date in our program, in May 1988, to October of the same year. There was never any sense of urgency in the need to reduce her pills; she was merely motivated by a desire to be drug-free. We had no knowledge at that time of the dangers of the meds, and the malaise, heaviness, and weariness that followed each new, very small, drug reduction seemed to be only mild inconveniences that lasted a few weeks and then eased up.
By October, 1998, she was completely off her medication, her tremor was gone, she was moving fine, her posture was good, her arms swung when she walked, she had no foot dragging, she was sleeping a lot, suffering from the usual back pain, and feeling limp – soft, not rigid – in the legs. She had no bradykinesia and no balance problems. But she

[16] I have all the notes on all these cases. If anyone wants the exact details, the number of weeks, the amount of each pill cut, I have these records. This person's file is more than four inches thick and includes her daily diary of her drugs and symptoms from May, 1998, until the present time, and it is still growing. I am hoping that after this book is done, I will be able to put together a case study book, with nothing but the daily reports of some of the more interesting cases. It will be thousands of pages long.

was amazed at how much she was sleeping. She was even taking naps, something she had never allowed herself before.

Becky's son

Her son was going to come for a visit. She wanted to show him how good she was, so, on a whim, she decided to take just a couple of her L-dopa pills that weekend to give herself a blast of energy. It really worked. She took two half pills (200 mg) the first day, and she was going like a ball of fire. So the next day, she took two half pills again. And then, when the weekend was over, she decided it wouldn't hurt to keep taking them. At our next session, five days later, I asked why she was taking them. She assured me that she hated taking pills and that she was going to quit the next day. I left it at that. The next week she had increased her pills to 3 half pills a day. I asked her why she had increased to 3 and she assured me that she had been taking 3 halves the week before. She admitted that she had not decreased her pills, but she certainly hadn't increased.

Becky keeps a daily journal on her computer. She never goes back and reads past entries; she only types in how she feels in the moment, and she also enters her medication regime and symptoms. Once a week she prints it off and brings it to me for her file. When she insisted that she had been taking three half pills the previous week, I showed her the printout from the last week. She was puzzled when she read that she had only been taking two half pills a day the previous week, not three, as she thought, but assumed that she had made a mistake the previous week. I was a little nervous, because at this point Zoe also was starting to increase her pills without realizing she was increasing. Two weeks later, Becky was taking 4 half pills (400 mg) a day. She assured me that her previous journal entries had been in error. Not only that, she was going to start reducing her medication. For the next year, she told me once a week that she was going to start reducing her medication. She tried lots of tricks. She cut the pills and played with time of day and dosage level, but her total daily med levels remained the same.

Within six weeks of going back on the pills, she started having a nervous tic in her hand. It was a violent, jerky motion of the hand moving from her waist over to her stomach. It was extremely rapid; it did not have the rhythm or look of her old tremor, which had been an index finger-thumb pill-rolling tremor. It looked suspiciously like the violent ticcing motion described by Oliver Sacks in one of his L-dopa experimental (non-Parkinson's) patients.

She started to panic when a year had gone by and she hadn't reduced the dosage. Meanwhile, the ticcing was getting extremely violent. She referred to it as "El Twitcho," and she used to playfully swat at her hand and arm when she was in a good mood, telling it to stop. Sometimes it would go crazy, and her hand ticcing would shake her whole body. I had another patient who had advanced PD and who had whole body tremor when her meds would wear off, and it did not look anything like El Twitcho. What was happening to Becky when her meds kicked in looked truly terrifying: her arm especially would move so quickly I could not count the movements per minute.[17] Her bicep muscles would be burning from the rapid-fire frantic spasming of her arm as it ticced

[17] A pill-rolling index finger-thumb tremor from Parkinson's disease tremors at a rate of 4 to 8 Hz, (Hz = beats per second) which is the rate of the brain's theta wave – a signal generated by the brain when trauma is present. Most people can suppress a physical response to the theta wave. In Parkinson's, the parts of the body that are no longer consciously connected to the brain will vibrate in time with this wave pattern.

uncontrollably. She understood this to mean that her PD was getting worse, and she finally decided that she should increase her meds beyond what had been initially prescribed.

I asked her to read a chapter in *Awakenings* that described a person with the exact same symptoms as she had, to see if it seemed similar. That person in the book had been taken off the L-dopa, and the spasming had gone away. Becky was a voracious reader; she had read *Awakenings* many years ago. Oddly enough, she found that she could not bring herself to read even one chapter of the book a second time.

This was a first for me. This coyness, this inability to read or hear words of negative import regarding the drug of choice, turns out to be a not unusual feature of people who have an addictive relationship with a dangerous drug. Since this first time with Becky, I have seen this blind spot repeatedly. A patient who is addicted to L-dopa cannot hear the words if you speak harshly about the drug. A patient who is addicted to dopamine-enhancing drugs cannot bring himself to read negativity about the drug. It is like trying to warn a love-besotted woman that her Romeo has a history of murdering his girlfriends. Deaf and blind to all logic, the Juliet will just gaze at you demurely and assure you that he is wonderful and that no one has ever understood him but her.

The analogy of dopamine-enhancing drugs and the demon lover is one that has held up through the years. Years later, when Becky was off the L-dopa for good, she asked me why I hadn't told her about the addictiveness of the medication. I assured her that we had discussed it every week. She looked at me for a long time and then countered, "You only said words to me. I never heard you until today."

But that is jumping ahead.

One week, her report managed to surprise me: she had reduced her pills! Her report for the week showed that she was taking about 50 mg/day less for a whole week. She confidently assured me that she had had no ill effects from the reduction and that she would reduce by another 50 mg/day the next week. Later that week, one night in her sleep, at home in her apartment, she opened her eyes and found that she was staring at herself in the bathroom mirror, the pill bottle in her hand, and she had just swallowed a whole pill. In all her years with the medication, she had been a daytime user only. She had no precedent for taking the pills at night. To the best of her knowledge, she had never taken a pill at night. She counted her pills. She had evidently been sleepwalking, taking the pills in her sleep during the two weeks when she thought she had been reducing.

I assumed that the shock of this would get through to her, that she would begin to understand that she needed some help to get off these pills. I was naïve. She assumed that if she was walking in her sleep for the drugs, it was her body's way of telling her that she needed the medication.

Then came The Attack of the Breath Monster!

El Twitcho had become huge. He was always there; he would wrack her body and he would wake her at night. She didn't mind El Twitcho. In fact, she snickered, "El Twitcho is my friend…Because of him, I always get a seat to myself on the bus!" But then something happened that got through to her drug-fogged brain. She started having the same breathing problems as Zoe.

Becky and Zoe had become good friends since they had met at the local Parkinson's support group. Becky didn't drive, so Zoe drove her, and they had acupuncture and Tui Na treatments at the same time. They shared notes on their symptoms and drugs with each other and attended social events together. But when Zoe had started the breathing problems, Becky had been horrified. Becky had repeatedly told Zoe to reduce her meds. It was clear as day to Becky that Zoe's breathing horrors were coming from the meds, but Zoe of course wouldn't hear it.
Becky didn't mind El Twitcho and refused to consider that he was medication-related, but she understood that the breathing spasms were related to the meds. Nearly twelve months after going back on the medication after having gotten completely off of it and having been free of all PD symptoms, Becky started having the breathing problem. It was the exact same diaphragm spasm as Zoe had. Becky named it the Breath Monster. It attacked her right around her middle. It felt like she was being gripped around the ribs and having the breath sucked out of her. This wasn't happening, of course. In the times that I got to watch the gasping and struggling for breath, it never ceased to amaze me that Becky was actually able to keep breathing. There was never any sign of hypoxia. There was no light-headedness, no turning blue. It almost seemed to me that Becky was *in terror* of not being able to breathe, and going through the motions as if suffocating, but that, in fact, the body was functioning somewhat normally, in terms of oxygen exchange and lung function.

NOTE – Do not think that because some patients in my limited experience have been able to breathe even though they were feeling as if they were suffocating and gasping that this means the problem is only psychological. Respiratory distress from overly quick withdrawal from L-dopa can cause death. Anytime a person feels that they are suffocating, it is a concern. Whether they can actually, physically, go through the motions of breathing, if they *think* that they cannot, they might be in danger. Do not underestimate the danger of these meds. And now, back to our story.

When Becky started having visitations from the Breath Monster, she knew she was in trouble. She really wanted to reduce her meds. She also realized, based on her sleepwalking and taking the pills in her sleep, that she could not do it alone. I reread to her her own typed notes, along with my weekly notes for our hour-long sessions. She saw the clear pattern of determination and failure. She made a reckless decision. She gave her pills to a friend and the two of them flew off to Hawaii for eight days. She told her friend not to give her any pills unless she was dying. For a few days she felt fine, proud of herself for making this bold step. Then she started feeling a bit depressed, but she figured she was through with the worst of it. On the eighth day, while flying home in the plane, "all hell broke loose." All the way home she was pacing the airplane, unable to stay in her seat, hunched over, gasping for breath, shaking violently, and feeling as if she would vomit. In fact, she was in the exact condition of another patient I had seen just once for opiate withdrawal.
(I don't usually see patients with illegal drug problems. A concerned mother brought her college-age son into my office when he was going cold turkey from illegal opiates. He shuffled around the room, hunched over, tremoring and gasping for breath. I was fascinated. He looked just like some of my Parkinson's patients who had tried to reduce

their meds overfast. At this point I was still not clear on the relationship between dopamine and addiction, but I was getting more ideas every day…)

All the way home in the airplane, Becky felt worse than she had ever felt in her life. She hoped it would end in a few hours. She hoped it was food poisoning. She kept telling herself she could take it by focusing on getting through one hour at a time. She was never so wrong. It was not going to be a matter of a few hours.

By that night, home in her apartment where she lived alone, she had started shaking. It was at the same rate as El Twitcho, but now it was both arms and both legs that were ticcing. The right arm was still the worst; it was flexing and straightening, jerking back and forth so that she was slamming herself in the chest with her fist. It was impossibly fast. She was panicked but figured that she was tense from the long flight. The spasming was exhausting. It was powerful and muscular, not like the mild tapping of her finger tremor from so long ago. It was the same sort of twitching that was set in motion by the meds, only now it had fear – terror – associated with it, and it seemed, if anything, more violent. This spasming was using every bit of energy in her body. She managed to get herself into bed and fell into a brief sleep.

The next morning she woke and immediately began the violent spasming. She called me on the phone. I asked her if she had read the chapter in *Awakenings* about the woman with the violent, rapid arm spasms,[18] which I had asked her to read a long time ago.

"I tried to read it several times," she responded, "but I just can't bring myself to. It's strange. It's as if my mind doesn't want to know what's in that book. I can read anything else, but I cannot read about L-dopa."

I thought that was so strange at the time, but now I know that it is normal. Some people who become addicted to L-dopa are able to read about it, but either cannot remember what they read or cannot see that the descriptions of symptoms have anything to do with them. They feel that their own case is different from anyone else's. It's as if they cannot even hear the words you are saying. People who have become addicted to L-dopa or any of the dopamine-enhancing drugs consistently have this same inability to listen to logic about their drugs or see their drug situation realistically. Some patients, while thrashing violently under the influence of their drugs, have assured me that they are looking particularly good at the moment, and they point in proof to the fact their arms are just floating along, feeling as light as a feather. If I say they are thrashing abnormally, not floating gracefully, they just smile sweetly and say, "You don't understand."

Becky's drug withdrawal — ten days to ten weeks

She continued ticcing and gasping for breath all day, and pacing the floor. I put some acupuncture needles in her ears and forehead to calm her down, and that did seem to reduce the thrashing by just a bit. She assured me she would be OK soon and that it would just be a few days, but I wasn't so sure. I had already had some experience with other patients making reductions in their L-dopa, and I had seen that they often felt fine for a week to ten days, and then slowly, over several days, the results of the drug decrease began to be apparent. At first I had not understood this pattern, but while doing research on dopamine, which led me to study up on cocaine, I had read that the most likely time

[18] This chapter was about a woman who did not have PD but who developed the rapid, powerful arm ticcing after having taken L-dopa during the drug's earliest research stage.

for a recovering cocaine user to find himself using again was ten days to *ten weeks* after quitting.

I had seen this pattern over a dozen times by now with my patients who were reducing their medication by just a small percentage, so I had reason to fear what was going to happen to Becky from her abrupt stoppage of her drugs. Over the next few days, I shared with her as much information as I had uncovered about drug withdrawal in general and L-dopa in particular, but it was too late; she was unhearing ears, uncomprehending eyes. As she pleaded with me to do something, she never heard a word I said.

Over the next few days she started having the full-blown Breath Monster. He would vary in intensity throughout the day, but he was usually worse at night. The sensation was that someone was grabbing her just above the waist, drawing her lower chest in hard so that she could not inhale. She never turned blue or passed out, and, in fact, she was breathing the entire time the Breath Monster was present. What made it so monstrous was her internal voice which told her that that she was dying, that she would never breathe again. In fact, she was never sure which would attack first: the fear or the spasm.

When it hit, she would be filled with wild paranoias about suffocation and pace madly around the room, hunched over, with her arms wrapped around her waist, breathing loudly, in gasping, huge breaths. That was exactly the way Zoe had been: breathing in huge, gasping breaths, as if she had been underwater for two minutes and had lungs about to burst. Based on facial color, there appeared to be sufficient air going in and out of Becky, but the diaphragmatic spasms were coupled with wide-eyed terror and screams of "I'm dying! I can't breathe. It's got me, it won't let go!" This usually only lasted for twenty minutes at a time during the day, after which she would get up to ten minutes of rest. At night it could go for hours.

Within a week Becky started having the full-blown symptoms of drug withdrawal. She had constant nausea; she had to force herself to choke down a slight amount of food. She would buy her favorite foods, or go to her usual deli, but she couldn't bring herself to eat. The violent shaking had become constant and filled her with terror. She would often grab at her right arm with her other arm and hold it until the frantic flexing tore itself out from her grip. She would sit on her arms and her whole body would tic. She would lie down on her arms, and her legs would twitch at the same incredibly fast rate. The Breath Monster came at irregular intervals during the day. Paranoid about being home alone, she was also afraid of being around other people. The news on TV scared her. Drawn and pale from lack of sleep, she was also losing weight rapidly.

The Breath Monster

Around this time I told her to call me anytime, day or night. I warned her that it would be about eight more weeks until she had passed the ten week period, and, somehow, she seemed to hear that through her panicked haze, and she assured me that she was going to go the distance. I wondered if this was a good idea. I was very fearful for her, but she was determined to keep it up. As she pointed out in a rare moment of lucidity, she had the twitch when she took the drugs, and she and her friend had the breathing problem even when they took the drugs, so you were damned if you do, damned if you don't. She was certain of impending death, but she hoped to save something from the wreck by keeping a journal to let others know of her experience. She could no longer type, so a friend bought her a small tape recorder so she could continue her journal verbally. I transcribed it once a week.

Around this time, she found a new source of strength that mightily amused her. She had never used drugs or known people who did. She had grown up in rural Pennsylvania, among upright religious folk, and she was still very oriented towards the moral superiority of self-control. Her colleagues from her work as a librarian and as a leader of hiking trips pointed to her as an example of old-fashioned, clean living. One day, as she staggered from her apartment, the groundskeeper who was raking nearby walked up to her and put a hand on her shoulder.

"Hang in there," he condoled. "It's a hard stage, but you'll get through this withdrawal. Be strong. I'm rooting for you."

Becky laughed out loud as she recounted it. "He thought I was a junkie!" she howled. "He thought I was going through drug withdrawal!" She was greatly amused, that she, a woman from a good family, who had never taken any drugs except those ordered by a doctor, and who never drank anything stronger than beer or wine, had been thought of as a sister by someone who clearly knew the signs of drug detox.

As a joke, she started referring to her symptoms as detox. She could not have known that this choice of vocabulary would cause trouble down the road. It turns out that Dr. Leslie is particularly hostile to the idea that Parkinson's drugs are addictive. He is even more rabidly opposed to the idea of detoxification from perfectly safe, *FDA-approved* drugs. Many weeks later, when Becky told Dr. Leslie that she was going through detox, he hit the roof. Dr. Leslie decided that I, the acupuncturist, must be a crazy, pure-food fanatic. Based on Becky's use of the term "detox," Dr. Leslie decided that I was a dangerous person, brainwashing the patient into doing a fringe medicine "cleansing." This set the stage for some real trouble, I can assure you.

The big, broad outlook

Meanwhile, and incredibly, Becky maintained almost two personalities. One aspect, her physical reality, would come to my office every week, gasping, flailing about the office, ticcing and begging for help. Her alter ego, the other part, was an outside observer, making jokes about her behavior and complementing herself on small, daily victories. It was as if, as time passed, the drugs that had been clouding her brain were gone, and she was able, now and then, to see herself clearly. Bless her heart; the whole thing bemused her on some deeper level. She continued her daily journal, recording her attempts at sleep, how many hours were spent pacing, whether or not she had been able to eat anything, and where she went everyday and whom she visited.

The biggest change, aside from the physical symptoms, seemed to be that she was increasingly homebound, after having always been outgoing.

After four weeks, she thought she had it down to a routine. The shaking was violent, the nausea was constant, and the Breath Monster was relentless: "When he's not actually grabbing me, he's always there, watching me, looking over my shoulder, just waiting to pounce. He never goes away completely." The insomnia was shaping up as the worst problem, only allowing her a few hours of sleep each night. Typical of her brilliant attitude, she was able to laugh even at this, and she often included entries in her journal such as "I have always slept like a baby. Sleep was always easy for me. I used to think that people with insomnia had something wrong with them, (and) I never felt sorry for them. So I'm learning from all this. Gotta keep learning (in order) to stay young!"

It had said in a book on cocaine addiction that the most difficult stage of withdrawal might last "…ten days to ten weeks," and she was entering the fifth week. It seemed impossible that her symptoms could get any worse. I hoped that the "ten week phase" meant that after five weeks she would begin to get better. But what the book said was that from ten days to ten weeks is when a person is most likely to give up and start taking the drugs again. I came to learn that this meant it was sometimes up to ten weeks before the person began to see that there was hope, that there was light at the end of the tunnel. It didn't mean that in ten weeks you were better. It only meant that after ten weeks, you could begin to see that maybe, just maybe, things might be better, someday. Becky's condition continued to deteriorate.

During the sixth week, when she started the hallucinations, she did not appear to have any Parkinson's disease symptoms at all. She moved quickly all day long, pacing, always pacing. But there was no bradykinesia, no slowness of speech, and no rigidity in her limbs. She had full facial expression, no balance problems, and no resting tremor. When she wasn't grabbing at her waist during breathing spasms, she had good posture. She was quick, but she was a mess. She looked exactly like a person going through the most intense drug withdrawal I had ever seen. Her face was haggard and she couldn't eat or sleep. She was often hunched over when she was gasping, and the violent twitch was ever present. She was in a near constant state of paranoia, convinced that she was dying, that she was about to fall down and die, that she would starve or that she would suffocate. She screamed that she was terrified of death but that she would be better off dead. Her journal during this time makes many references to the bleakness of encroaching winter, the early

darkness, and the rain. The long hours of darkness seemed to have new menace to her, though in the past she had thrived on our coastal chill and fog and loved to hike around in it, always preferring cool weather to hot. Now the cold and dark seemed to hold threats and lurking evil. Almost daily she despaired in her notes from the paranoia and lack of hope crushing in on her from the rain, the cold, and the dark. True Parkinson's disease is usually characterized by a lack of movement, but she would never have been regarded as having PD. Her voice was fast, full, and expressive. And she was starting to hallucinate. She called me one evening just before I got off work. The recorded message was "Please help me! You've got to come over, this is getting bad." She had never sounded so scared. It was Thanksgiving weekend, late November. She had stopped the drugs on October 8th. When I found her apartment, it took her several minutes to unlock the door to let me in. Her shaking was so severe that it seemed that the shaking rather than the diaphragm spasms was preventing her from breathing. But she told me the shaking wasn't bad, not nearly as bad as the monsters. "When I was a little girl, I went to church," she told me, "and they told me about devils and angels, and I didn't believe in either one. Now, I've seen both." There were devils, angels, voices, and monsters in her apartment. Her bed had become a thing of terror, and she couldn't bring herself to lie down on it. She had been pacing the floor nonstop for the last two nights, unable to sleep. She had dragged her living room futon-sofa mattress onto the middle of the living room floor and was trying to sleep on it. Also, she found that when she lay flat out, spread-eagled on the cold kitchen floor, it seemed to calm her down for a bit. She could not go into her bedroom; something evil was lurking there.

I inserted ear needles for calming, plus needles in her forehead, top of the head, and wrists. (There is a specific drug detox protocol using ear acupuncture that is used in many clinics in Canada and the United States. I used those acupuncture points, just like I had for the last few weeks.) In drug rehab clinics, these points are needled every single day, but my practice is not set up for me to do that, and I was still only seeing her once a week up until this time. About ten minutes after I needled her, she began to breathe more easily and the violent shaking slowed to a pace that enabled her to use her hands. I heated up some food from the fridge and she was able to eat a bit and drink several glasses of water. We discussed the situation, and she agreed that she needed more help than she was getting. The hallucinations were the last straw. She needed to start getting some sleep. She had been trying everything to get to sleep: hot spiced milk, hot baths, milk of magnesia, classical music, and Benedryl tablets. (Benedryl is an antihistamine that can cause drowsiness.) I was able to convince her that she might be able to get a stronger sleep-inducing medication, maybe even some good advice about what else to expect, from an MD who was experienced with L-dopa. She agreed and made an appointment to see Dr. Leslie the following Monday.

I am simply not competent to describe what Becky was going through. The word "hallucination" is not adequate. Please allow me to include here an excerpt from Oliver Sacks' description of one of his first experimental L-dopa patients, after her reactions to the drug became unstable and she had to stop taking it. The following was based on his discussions with her and also from a "remarkable diary" which she kept and shared with him during the time that both her L-dopa and her withdrawal from it became torturous.

> Certain feelings haunted her…These were feelings of astonishment, rage, and terror that *such things could happen to her*, and feelings of impotent outrage that she, Miss D, could do nothing about these things. But deeper and still more threatening feelings were involved: some of the 'things' which gripped her under the influence of L-dopa – in particular, her gnawing and biting compulsions, certain violent appetites and passions, and certain obsessive ideas and images – could not be dismissed by her as 'purely physical' or completely 'alien' to her 'real self,' but, on the contrary, were felt to be in some sense *releases* of *exposures* or *disclosures* or *confessions* of very deep and ancient parts of herself, monstrous creatures from her unconscious and from unimaginable physiological depths below the unconscious, pre-historic and perhaps pre-human landscapes whose features were at once utterly strange to her, yet mysteriously familiar, in the manner of certain dreams. And she could not look upon these suddenly exposed parts of herself with detachment; they called to her with siren voices, they enticed her, they thrilled her, they terrified her, they filled her with feelings of guilt and punishment, they possessed her with the consuming, ravishing power of nightmare.[19]

In Sacks' footnote to this paragraph he writes that

> …gnawing and biting compulsions, along with gnashing and grinding of the teeth, and a great variety of other abnormal or abnormally perseverative movements are among the commonest 'side effects' of L-dopa. Such movements may be quite irresistible, of great violence, and liable to inflict considerable damage upon the gums, tongue, and teeth.[20]

The rest of Thanksgiving weekend I was at her apartment every evening. The needles seemed to calm her down so that she could eat a bigger meal. The meal in turn seemed to relax her so that she was able to fall asleep. She would fall into a deep sleep at around 10:30 at night and wake up between 1:00 and 2:00, spending the rest of the night ticcing and spasming, pacing the apartment hunched over and grabbing at her waist while gasping, lying on the cold floor spread-eagled, using the bathroom ("every hour on the hour"), or laying on the futon on the living room floor with her arms tucked under her back to restrain them. She desperately needed sleep.
She agreed to see Dr. Leslie on the condition that I accompany her. She didn't tell me why she was afraid to go alone. I was utterly unprepared for the vehemence, even hostility, that was in store for me.

We visit the neurologist

(The following dialogue is in quotes for readability, but it is paraphrased, as the exact words were not written down at the time.)

Dr. Leslie took one look at Becky and snapped, "Your Parkinson's disease is much worse."
"No," countered Becky, "this isn't Parkinson's disease…I'm going through detox from the meds, and I need something to help me sleep."

---

[19] O. Sacks, *Awakenings*, p. 55 (italic emphasis in the original).

[20] The above selection provides a description of what the drug insert blandly describes as "grimacing." While most PDers in my experience do not have the more violent facial movements until several years on L-dopa, still, grimacing is often one of their first dyskinesias and often heralds the beginning of overt overmedication and looming drug unpredictability.

This is when he stiffened. Becky should never have used the word detox. It seemed to ignite his internal powder keg. “There is no such thing as detox from legal drugs! Where have you heard of such a thing! Your Parkinson’s is much worse!”
“I don’t think the PD is worse, the problem is the detox.”
At this point, I cautiously interrupted. “Excuse me…Detox may be a poor word choice; she’s having withdrawal symptoms from the L-dopa.”
“Why isn’t she taking her Sinemet? There’s no such thing as withdrawal symptoms from L-dopa! What she’s having is violent symptoms of Parkinson's disease.”
“I’ve been reading up on this drug,” I quavered, nervously shifting my weight from one foot to the other, “and her symptoms are textbook for L-dopa withdrawal.”
“There is no such thing! Look at her gasping for breath!” He fixed me with a look from the Inquisition. Torquemada had nothing on Dr. Leslie. I attempted a retort.
“She was gasping for breath before she stopped her drugs. That’s why she stopped them. Respiratory problems can be a side effect of L-dopa.”
”That’s not true,” he roared. “Respiratory problems are caused by Parkinson's disease. L-dopa does not cause respiratory problems!”
“The tightness in the chest from Parkinson’s doesn’t look like that,” I stammered, fighting tears. “But drugs, not Parkinson’s, *can* cause this exact type of breathing pattern. The breathing constriction pattern in PD looks like this…” (I demonstrated rigid chest muscles forcing the body to breathe with the short, shallow breathing that can occur in PD.) “Becky’s diaphragm spasms are a not unusual form of the dyskinesia associated both with too much medication and with dopamine withdrawal.”
“You don’t know anything!” shrieked Dr. Leslie. “I am a doctor! She needs L-dopa; she had Parkinson's disease – look at her face. It’s a rigid mask!”
“Well, actually,” I said, my voice husky, “her face looks pretty expressive right now. She’s not smiling, but she doesn’t have an expressionless face. What she has right now is a look of terror.”
At this moment, possibly due to Dr. Leslie’s iridescent hostility plus the tension in the room, Becky’s diaphragm went into extreme spasm.
I continued hurriedly, “Her face is registering terror, which is actually pretty expressive. In your waiting room just now, she was laughing and smiling with me, just before she came in.”
”No she wasn’t!” screamed the neurologist. “I haven’t seen her smile, and now she has a mask, and that’s a symptom of Parkinson's disease.”
“Could you test her right now for PD with the usual physical tests?” I asked, hoping for the persuasive power of physical evidence.
In rapid order, Becky did the balance test, the cogwheeling wrist test and the finger tap test.
My assurance somewhat restored by her excellent responses and Dr. Leslie’s gape of surprise, I remarked, “She did all those tests pretty well, and that finger tap test was excellent. It didn’t used to be.”
“Yes, it was fast,” he sneered, “but that doesn’t matter. I don’t care! She still has Parkinson's disease.”
“Let’s have her walk down the hall and see how she does, OK?”
“Yes! Go walk down the hall!”

Just then another spasm hit, and Becky started gasping for air, bent over, clutching at her waist. Becky staggered down the hall just the same. On her way back up the hall, her clenched body lightened up its grasp, and she straightened and walked much better.
"See! She still has Parkinson's disease!" he snorted.

"Walking hunched over during a diaphragm spasm does not yield a diagnosis of Parkinson's disease," I tried to point out. "A diagnosis of Parkinson's disease should be based on the four categories groups of symptoms that include…"
"I don't care! I diagnosed her with Parkinson's disease and *I'm not wrong*!"
At this point Dr. Leslie was shaking, his nostrils were flaring and he was stamping his foot. "I diagnosed her! I'm right! If I say she has Parkinson's disease, then she has Parkinson's disease!"
We left the hallway and returned to the consulting room. The curious receptionists jumped back in their chairs and pretended they hadn't been watching.
I persisted. "The reason we came is that Becky was wondering if there was any medication that you might be able give her to help her sleep. That's the real problem that brought her here today."
"I will not give her anything to help her sleep! She has Parkinson's disease and she has to do what I tell her. I'm the doctor! She has to do what I say!"
Now my voice rose in turn. This false idea that MDs have the power of a judge to command is so encouraged by many MDs that far too many patients actually believe that the word of an MD is the word of law. I was not about to let this pass. My voice may have quavered, but I got the words out.
"Well, actually, she doesn't have to do what you say. As long as she is a competent adult, she can do what she wants, and that includes not taking medication as long as there is no risk to her or others. So since you refuse to help, I guess we'll be going."
We trundled off, and neither of us looked back. I was shaking, but Becky brought me back to reality when she turned to me and twinkled, "I didn't know doctors could make flames come out their nostrils! Do you think they learn it in medical school?"
I grabbed Becky by the shoulders and looked her in the eye. "Look at you! Why didn't you smile like that when we were in his office?"
"Are you kidding? Smile? I was scared to death in there! That man is a terror."

A ray of hope

The seventh week brought a hint of relief for Becky. She was taking Tylenol PM or Benedryl each night to help with sleep and they seemed to be working. I made sure she ate a large hot meal at bedtime, and she was able to get a bit of sleep every night. It wasn't a lot of sleep, but from either the sleep or the inevitable improvement in the drug withdrawal, the hallucinations went away. Although she continued to have withdrawal symptoms day and night, she thought that it was possible the symptoms were lessening slightly in intensity. And then, she had one day in which she felt a genuine ray of hope. The twitch was less violent, and she also had an appetite. She ate a huge sandwich from the deli and took an afternoon nap. She called to tell me that she was done with withdrawal. She was very hopeful. The next day was back to the usual withdrawal symptoms, and "the Breath Monster was a little worse. I think he wanted revenge because I'd had a good day. It's hopeless. I'm going to be like this for the rest of my life." Then,

three days later, she had another good day. Then the pattern of a good day followed by two slightly worse than usual days was repeated.
In the eighth week, she had good days alternating with bad days. She became convinced that she would never have a good day without being what she called "punished" by having a worse day to follow. She suspected at around this time that she had started praying. She wasn't sure what praying really was, but she just found herself talking out loud, making deals with the universe. She wrote in her journal, "I would give anything to have two good days in a row. Just once, if just once I could have two good days in a row, I would be happy. I'm asking for help now. I don't know who I'm asking, but if that's prayer, I guess I'm doing it. Is anybody listening? What do I need to do to have two good days in a row?"
In the tenth week, she had a shocking event: two good days in a row, then three bad days. Her journal reflects the hopelessness of this time: "There is no sense to it, no logic, the drug withdrawal demons are playing with me." She had never been a particularly overtly religious person, and she greatly enjoyed word play and classical references. She was really enjoying the use of references to good and evil, demons and angels. The people on the bus who helped her to her seat, her wonderful friends, these were now angels. She used these terms laughingly, mocking herself, as one will who has grown up in the USA in the cynical nineteen forties and fifties, when intelligent people with university degrees kept a cool distance from this sort of language.

Cranking and Jonesing

She also had mastered a new drug withdrawal vocabulary, learned from the gardener. She started describing herself as "cranking" when the paranoia would appear and the ticcing and pacing was at its worst. "Cranking," she informed me with pride, "is what your body does to you when you are going through drug withdrawal. That's what the junkies call it. My body was really cranking at me on Monday! Also, 'Jonesing.'" She paused. "I believe that is the correct usage. I may have it wrong, of course. I'm not really sure what the difference is between Jonesing and cranking." And so with devils and demons and angels and cranking and Jonesing all enriching her vocabulary, she continued her self-deprecating and honest journal and her weekly visits with me.
She had been clinging to the idea that at the end of ten weeks some sort of miracle would occur. That was the number in the book about cocaine. There were no books for PDers, not one book about withdrawal from antiparkinson's medications. It was assumed that PDers never reduced their drugs. Since PD was considered incurable, back in the days when it was thought that nerve cells could not regrow and that the brain was unchanging and irreparable, no one ever considered studying how to safely reduce L-dopa levels. She was a pioneer in a chartless land, and the medical books about cocaine, heroin, methamphetamine and alcohol were our only guides. But it was nearly week ten, and she was still having two good days alternating with three bad days. What was hard for her to take was that none of the good days seemed as gloriously good as the very first "good" day had been. But the bad days were not as bad either.

Becky's story continues, later in this book, but before going further, you will need to be sure of our terminology. The symptoms of Parkinson's and reactions to the PD drugs have led to the creation of a specialized vocabulary. Words such as on, off, tremor and dyskinesia all have special meanings when used in the Parkinson's context. In the

next chapter, before we share any more case studies, I will bring you into the rarified PD Communication Zone with the Parkinson's community's definitions of these words.

"You say po-tay-to, I say pa-tah-to, You say tom-ay-to, I say to-mah-to…Let's call the whole thing off!"

Cole Porter

5. PD TERMINOLOGY

THE NEED FOR UNIFORMITY OF TERMS

Back at the research project, we were recognizing yet another kind of problem: everyone in the program had different words for describing their symptoms. Some pioneers were referring to their tremors as dyskinesia, and others were saying that their dyskinesia was tremor. Rose used the word "kicking" to describe her arm thrashing and head jerking and "shaking" to describe her periods of stone-like rigidity.

Many had invented a vocabulary. Sometimes it took me weeks just to figure out what patients had been talking about specifically when their "power went out," or their "sinking times" were worse. "Sinking" meant foot-sticking-to-the-floor, and a "power failure" was an inhibiting emotional insecurity related to anticipation of drug failure rather than a problem with immobility, as it turns out.

As for the meaning of the word "Off," the definition varied from one person to the next. For example, one patient used the word Off to mean those times when he could scarcely move. Except during the times when his four daily pills were at the height of their fever, he was immobile, his voice was a whisper, and he could not control his drooling. He would therefore complain that he was Off most of the day. Another patient who complained that he'd been Off all day due to drug failure pointed to his day's lousy golf score, a whopping 140 for 18 holes, as proof that he'd been Off. The drastic difference between the meanings of Off in these two PDers was typical of the chimerical way in which PD vocabulary was applied. While this spontaneity of meaning and the individualized PD patois lent an air of mystery, even poetry, to our verbal communications, it was not practical.

Every drug user had his own slant for the official, medically-approved PD terms, and some had word inventions not found in any book. The misinterpretations that PDers made when applying their unique translations to the official PD literature sometimes led to serious, potentially deadly, consequences. Here follow some selections from case studies that help to illustrate the terminology problem.

Rufino

Rufino was still taking his 1000 mg/day of L-dopa even after his FSR practitioner thought Qi had resumed normal flow in his legs. Soon after the Qi flow change he started experiencing violent ticcing. The ticcing, which he called tremor, would ebb for up to an hour, about twenty minutes after he took his drugs, but then get even worse in the second hour of the pills' effectiveness, which is when the drug levels in the brain were at their highest. Also, his violent shaking would calm down about six hours after a dose, when the immediate hit from the drugs wore off. In the morning, the shaking was not as violent as it was in the evening. By the end of the day, as the meds built up in his body, his shaking was so terrible and powerful that his torso and legs would become stone rigid,

unable to move, while his arm went slamming up and down making a huge movement, faster than the eye could follow. He would be gasping for air, his eyes bugging out in fear, while a force beyond all knowledge seemed to take over his arm and wrench it up and down. His athletic adult sons were unable to restrain it in the slightest. They insisted that it was tremor, however, because it would stop for a brief while, up to an hour sometimes, about twenty minutes to half an hour after taking his pills. One son, a physical therapist who felt strongly that this movement was PD tremor, argued that the father needed more drugs. The other son, a sports coach, argued that the drugs appeared to be part of the problem.

Here is a description of the symptoms, written up by the second son.

> Dad had taken his Sinemet two hours earlier. It's like this almost every night: Dad awake at 7:52 p.m., tremor immediate. I had to help him up off the couch. As he sat in his chair at about 8:15, he called to me to help him. He had a very pained, panicked look on his face. "This tremor is going crazy right now! It won't let go of my arm!" He was holding his left arm with his right hand and begged me to do something. I grabbed his left hand and forearm. The forearm was ROCK HARD from the spasming of the tremor. I pulled his hand backwards toward his wrist and raised his arm to relieve the spasm, but then his leg did it. It just seemed to lock in there for about 30 seconds. Then it relieved itself a bit. Then it started again.

Eventually, after this patient reduced his medication slowly and carefully over more than a year, the violent ticcing stopped and his original tremor – a small, fluttering, weak movement – reappeared, amazing him and his family. For the first time, the family members were willing to admit that possibly the violent ticcing was a drug-related twitching – not tremor, a normal symptom of Parkinson's disease. The violent ticcing showed no signs of slowing until he got down to 300 mg/day (from his high of 1000 mg/day) and stayed at this lowered level for three months.

Another patient had violent twitching throughout her body until she decreased from a high of 400 mg/day down to 50 mg/day.[1] When she had been at 50 mg/day for nearly two months, the violent shaking abruptly ceased, and she asked me why she was doing "this really annoying, weird, little flutter thing." When I told her that that was her Parkinson's disease tremor, which was the reason she had started taking the L-dopa, she was amazed. "But the jerky twitchy stuff was much worse than this! Who would ever take drugs that cause that violent stuff just to temporarily mask this stupid tremoring?" I had to point out to her that in point of fact, she had done just that.

"But I thought the twitching was the tremor!" she bleated. "That's why I kept increasing my drugs!"

"But I told you that what you had was drug-induced ticcing. I said every week that what you had wasn't tremor, and you didn't believe me. I pointed out that muscle twitching was listed right here in the list of adverse effects of the drugs."

---

[1] A person with moderate Parkinson's disease can usually tolerate a dose of 400 mg/day of carbidopa/levodopa without having extreme side effects. However, this patient had apparently recovered from PD. This is why she was grossly overmedicated even at a "mere" 400 mg/day.

"I don't know what twitching is. I assumed that any extra movement was tremor. What the heck is tremor then, anyway? This stupid, annoying little shaking – is this really the tremor that they talk about?" Why don't they describe it better in the books?

We needed a standardized vocabulary, and some good, explicit descriptions.

Standardized Terminology

Based on everything we could read on the subject, we have compiled the following descriptions of the basic Parkinson's terminologies. If you're going to go any further with us, you need to be using the same words for the same conditions that we are. So before we get into the really juicy stuff, let's review some basic terminology that is used and misused by PDers, their friends, and their doctors. These terms are: On, Off, On!, freezing, dystonia, tremor, and dyskinesia.

Ons and Offs

When a person first starts taking antiparkinson's medications, he can often take two or so pills a day and get even coverage. Even coverage means that there is never an obvious time when the medication starts working or wears off. Instead, a newcomer to the medication may notice that after a few days or months, sleep comes more easily, fluidity of motion returns to some limbs, life just seems a little more worth living, and voice and balance might have some gradual improvement. It may not matter, in these early stages, if the medication is taken in a timely way. Even if an entire dose is forgotten, there may be no apparent lapse in coverage.

Often, a naïve PDer who is at this stage of medication will assure his friends that he's one of the lucky ones. His attitude might be expressed in this way: "Some of those people have a really bad time with their meds, but they work just fine for me! I'm not going to develop problems with the drugs. I don't really notice any effect from the drugs; I just feel a little better all day long." This patient does not yet have an Off/On scenario.

Invariably, after taking PD meds for a few months or maybe a few years, a person will begin to notice that at some times of the day he feels better than at others. His drugs still work most of the time, but at times his movements are nearly as stiff and

uncomfortable as they were before he started taking the drugs. He may notice a real change in functionality if he forgets to take a dose. At this point, the MD will usually prescribe an increase in the medication.[1] Soon, within about two years of starting PD medication, a person will detect noticeable improvement about one to two hours after taking a pill. When the visible (motor) portion of the pill effect wears off, somewhere between two to six hours after the dose, he feels noticeably stiff, depressed, or weak. This is still not On/Off.

Eventually, the drug will start to become "unstable," to use a biology term. This means that the results of any given dose are not exactly the same as every other dose. This is when a person will start having strange symptoms that are different from the symptoms of unmedicated PD.

Various patterns can indicate that the drugs have become unstable: the onset of medication effectiveness might now start or end with a jolt. The abrupt onset and ending times may be preceded and followed by short periods of feeling even worse than the normal PD feeling. Instead of having a good capacity for movement throughout the day if dosing at regular intervals, this drug user may lurch into movement at certain times. At other times, the pills won't work at all, as evidenced by lack of mobility for several hours after taking a dose. Or a person may have, several doses into the day, a gradual decrease in mobility, until he simply can't move anymore despite taking more pills. Or he may abruptly cease all movement after having been moving just fine. This is the beginning of Offs and Ons. When the Ons and Offs make their first appearances, they may be intermittent, occurring only once a month or so. Over time they may become a regular feature of the drug use. Eventually, these On and Off periods may manifest with no seeming relationship to the timing of the doses. These On and Off episodes are a side effect of long-term use of antiparkinson's medications.

On: *An On is a period during which a person has some modest fluidity of movement. This is in contrast to other periods during which the person has little or no mobility. Not all PD symptoms will disappear during an On. Some PD symptoms may still be apparent even though the person is able to move somewhat. For example, if movement initiation is possible at a somewhat conventional pace, the person is On, even though the person may be drooling or have no facial expression.*

---

[1] The implication is that the Parkinson's disease is getting worse, and the dopamine levels in the brain have decreased. Our research suggests that this is possibly not the case. It appears increasingly likely that when the meds become "unpredictable," it is due to overmedication. It seems, based on our observations, that any amount of buffered L-dopa above 400 mg/day exceeds the amount needed by *any* brain, even in a person with highly advanced Parkinson's disease. ("Buffered levodopa" refers to those medications which combine levodopa with an anti-digestive agent, such as carbidopa.)

Regardless of dosage or stage of Parkinson's (mild to highly advanced), drug withdrawal symptoms – including an apparent increase in PD symptoms – in all our patients who were taking more than 400 mg/day only lasted up to ten weeks, after which the medications became more effective again even at the lower level. The withdrawal symptoms or amplified PD symptoms subsided after the drug reduction phase was over. Our understanding of this is that the medication must have been too high rather than the PD being too advanced. In people taking less than 400 mg/day, this was not always the case – which might mean that these lower doses were actually treating the Parkinson's rather than compensating for addiction. It appears that any amount of buffered levodopa over 400 mg/day is being used to counter the addiction effects rather than the advancing Parkinson's.

Off: *An Off is a period of severely limited mobility or immobility. This Off period is in contrast to those periods when the person can initiate movement and perform the usual activities of daily living at a somewhat conventional pace.*

Again, an On period is not necessarily one in which a person has no symptoms of Parkinson's disease. The On period is a period in which a moderate degree of movement is possible, *as compared to the Off.*

The Off time is not simply any time when a person feels lousy or is showing some symptoms of Parkinson's. Off time refers to those times when he is partially immobilized (motor function comes in fits and starts) or fully immobilized, or partially or fully manifesting his other symptoms related to motor initiation, such as lack of speech or inability to swallow.

Not On, Not Off

Some people have periods during which they can move but not as well as they would like. This condition is the most difficult to describe with a simple one word term, as it can range all the way from "moving slower than usual" to "not at the top of my form." This in-between phase is best described as "Somewhat On." A patient will do well to make a scale from 1 to 10 to describe what he means by "somewhat On" and monitor himself against this scale. It is important to describe this phase as "somewhat On" rather than "somewhat Off." The Off terminology must be reserved for times of partial (comes and goes) or full immobilization and cessation of motor initiation functions.

It is difficult to be precise here, because many people who cannot initiate a normal stride can still shuffle along. If a person can initiate strides under the influence of the drugs and cannot initiate a stride when the drugs wear off, but can shuffle, he can consider himself to be Off when shuffling. The use of the phrase "initiate movement" refers to the ability to initiate somewhat normal movement. It may still be possible for a person, though Off, to be tremoring, shuffling in slow motion, or falling forward, all forms of abnormal movement. Therefore, there may still be movement of a sort during these times of "immobility."

There are several categories of Off as well, depending on whether or not they are being caused by Parkinson's, inadequate medication, or excess medication. In general, as long as there is the capacity to initiate somewhat normal movements, the person is "somewhat On."

Here is an example to help clarify what is meant by initiation of movement. If a person needs help to get up off the sofa, but once standing can say "Thank you," and then walk across the room, this person is On. If, upon being pulled up from the sofa and supported by the arm, this person shuffles precariously towards the opposite wall, head, eyes and body turning neither to the left nor the right, speaks in a whisper or drools, and the upper body is leaning increasingly forward with relation to the shuffling feet, in danger of falling forward, this person is Off. In the former case, there is some capability for initiation of some normal movement. In the latter case, although there is motion occurring, the PDer cannot initiate any normal movements.

One standard that many patients use is this: if they can move well enough to take their next pill, they are somewhat On. If they are so immobilized that they cannot take a pill and must wait for someone to help them get the next pill in their mouth (fully

immobilized), even if they might be able to yell for help by working themselves, over several minutes, into a mental frenzy, they are Off. This is a good standard. Also, this standard will enable many PDers who complain about their Offs affecting their aim in a game of horseshoes to realize that they were, in fact, never Off during that horseshoe tournament.

These On and Off periods can be gradual or abrupt. They can last several hours or several minutes. They are distinct spans of time, rather than a moment of hesitation in choosing a word or the temporary pause prior to initiating movement. Some patients have long-lasting and predictable Ons or Offs; maybe they are On for most of the day, but then a predictable Off of forty-five minutes duration occurs just after dinner, after which they can move normally again until bedtime.

ON!

Most patients correctly use the term "On" to refer to any time when they feel even somewhat able to move. However, a few PDers have a slightly different meaning for the phrase "On time." These patients have learned to use the medications to create a mild sense of euphoria. These patients refer to their bursts of drug-induced confidence and power as their On! (The use of the exclamation point is mine, but it helps make the point and differentiate between the two meanings of On and On! time.) This small group considers ordinary movement, which to the outside observer looks perfectly good, to be merely an in-between stage. Their On! time is those periods when they feel an exalted condition.

A person who is using the drugs to create an On! time of power and confidence will respond with derision to any suggestion that these highs are unnatural and constitute an abuse of the drug. People who have become accustomed to feeling high from their L-dopa cannot understand that there is anything unnatural about their overly bright eyes, their fast speech, and their illogic. They invariably assure one and all that they can be certain that they are not drugged, that they are normal. They point out that, if they were drugged, they would be able to tell. Instead, when they are On!, they feel more normal than they have ever felt.[1]

During their On! time, they often have feelings of supreme confidence. For example, I have had several patients who became compulsive gamblers, helped in part by the invincible, even omniscient feeling created during these On! times.[2] These patients

---

[1] This brings the inevitable comparison with Freud's early research on cocaine, in which he extolled the drug, stating that it was not like a drug, in the way that morphine and opiates were drugs, because the feelings that it produced were so utterly natural. He felt that cocaine was an enhancement of the natural joy that already existed but which was being stifled by complexes. The cocaine merely helped get rid of the complex and bring a person's natural radiance to the surface. Freud described cocaine as inducing a natural euphoria "which in no way differs from the normal euphoria of the healthy person…"

It was many years later, as he saw the tragic consequences in his friends who were using cocaine, that he began retracting his original statements, and, to the end of his days, he published strongly worded apologies and regrets for his widely published articles on the naturalness and harmlessness of cocaine. In his later writings, Freud called cocaine "the 'third scourge' of humanity, after alcohol and heroin." From R. Julien's, *A Primer of Drug Action*, Henry Holt and Co, NY, p. 118.

[2] They are, alas, no more successful at gambling than any other John, but they seem incapable of noticing that they are losing more than they are winning.

assure all comers that the drugs do not affect them and that the drugs are merely helping them to express their true selves.

Maurice

Maurice became a complete megalomaniac during his On! times. He could see individual atoms, and they spoke to him. I thought at the time that this was a unique pattern and wasn't even sure if it was drug related. Then during a revisit to Oliver Sacks' book, *Awakenings*, I read that one of his patients also saw atoms under the influence of L-dopa. When I shared this tidbit with my atom-viewing patient, he got very hostile and said that his drugs were not showing him the atoms. Instead, intelligent particles in his mind were imparting the ability to see the atoms. The PD drugs had nothing to do with it. His conversation was filled with unintelligible ramblings about his relationships with the particles – particles that only he could see. Particles were not atoms, by the way. Other people knew about atoms, but only he knew about particles.

This man was driving around town in his speedy car, golfing, gambling, and living an outwardly normal, albeit livelier than normal, life. He told me that he was a very fast driver but that he was safe because the particles told him how to drive. He refused to believe that his drugs were giving him his special particle-viewing powers, but he also felt strongly that PD drugs were designed to be taken to impart an On! feeling, not mere movement. He made no sense most of the time. He often complained to me that he had been Off all day, even though he had been driving around town, golfing, and shopping at the store. I told him that since he was moving, he was not Off, he was On, but he looked at me with gentle pity and told me that I didn't understand.

His wife complained that he twitched and kicked violently all night in his sleep, but he insisted that she was imagining things. She begged him to reduce his medication but he refused. She told me nearly every week that he had become stark raving crazy. He always laughed it off.[1]

So there is a range of understanding of what it means to be On and Off. But in general, and for the purpose of this book, On refers to a condition in which one can initiate some amount of movement, and Off refers to a condition in which one cannot initiate normal movement, can move only slowly in fits and starts with fits outnumbering starts, or cannot move at all. The Off can be either the shuffling slowness and rigidity typical of Parkinson's disease (which may be visible first thing in the morning, before any medication has been taken that day), the exaggerated, crashing Off that occurs shortly after a person's medications wear off, *or the powerful rigidity of overmedication.*

It would be better if there were separate terms for each of these three different states of immobility or slowness, the former caused by PD and the latter two caused by medication, but this is the current convention. But even though we will conform to these standard terms, you will need to learn how to discriminate between PD Offs, Offs that occur after the drug is used up, and Offs from excess medication. More information about

[1] My student interns nicknamed him Particle Man, a reference to a popular song with that title. He was actually a helpful member of the group: many patients who thought that their drugs should impart an On! decided to reduce their medication after spending a brief time with Particle Man.

the Offs that occur when the dose is expended (Crashes) will be provided in chapter thirteen.

Freezing

A condition often confused with Off is called Freezing. Freezing is a (usually) short-term, temporary paralysis. The term freezing can cover events such as one's feet suddenly sticking to the floor, or rising up on one's toes when trying to walk forward. Freezing can also include the phenomenon of festinating gait, when the legs sort of freeze up to the extent that they take smaller and smaller steps until they stop altogether, while the torso continues to be propelled forward from its forward momentum. The festinating gait often ends in a crashing halt against a wall or stationary object, or falling to the ground. In addition to the foot freezings just described, freezing can occur in the arms, face, or any other part of the body. It is most noticeable in the feet of course because sudden freezing of the legs or feet sticking to the floor can have the largest motor effect: it stops you dead or dying in your tracks. Freezing does not refer to the "clumsy leg" or perpetually draggy leg that may occur during Parkinson's. This clumsy or draggy leg is usually related to a dystonia, about which you will read later.

The onset of freezing can be quick, like in the western movies where the man with the gun shouts "Everybody freeze!" Freezing does not necessarily extend over a long period of time, although it can. It may go away by itself, or it may require the touch or voice of another to break the spell and get moving again. Freezing is not necessarily a consequence of drug use. It occurs in medicated and unmedicated patients alike, although as a consequence of drug use, incidence of freezing may increase.[1]

For an example of a freezing scenario, one minute the PDer might be walking across a room, and then, abruptly, while crossing through a doorway, become paralyzed, utterly immobile. A moment later, after someone helps him through the doorway, he can move in his normal fashion again. This temporary paralysis in the doorway was not an Off, it was a freezing episode.

Freezing can have very abrupt onset, or it may become apparent after a period of low activity. For example, a person may be reading quietly on the sofa for an hour or two before bedtime. When the time comes to get up, he finds that he cannot move: he is frozen to the sofa. This abrupt moment of freezing may disappear in a few moments if he either puts his mind to it or is given a gentle help up. Sometimes just the mere hand contact from another person is enough to break the spell and allow the freezer to initiate movement. Even auditory cues, such as danceable music, might be able to restore movement initiation in such a case.

This freezing can occur in early stage Parkinson's as well as in advanced PD. Many people, even before they are ever diagnosed, notice that sometimes they have trouble getting moving after having been still for a while. If they are given a supporting hand, or rouse themselves by mental invigoration, they are suddenly able to move perfectly normally.

Research in the 1960's showed that music, human touch, and, in the case of inability to pass through a doorway, visual distraction, were all methods that could break up a freeze. For example, turning to the left or right can be difficult for a PDer. Because

[1] Nearly all antiparkinson's medications list freezing or bradykinesia (extreme slowness of movement) as one of the adverse effects of the medication.

the anterior-lateral (front-sides) leg muscles are the leg muscles most affected in PD, it is difficult to initiate movement toward the side. The difficulty in turning to the side can cause freezing. By turning in a wide arc rather than trying to make an abrupt turn one can sometimes avoid this type of freezing while turning.

For an example of unmedicated freezing, one of our local PDers, unmedicated-by-choice, manifested this classic form of freezing in his advanced stage of PD: he would shuffle across the room until he came to the wall. Then he would wait until someone came and helped him turn around. Then he would shuffle back to the opposite wall and wait again until help came. Constant moving eased his restlessness, but he was unable to initiate a turn. This form of situational immobility is a type of "freezing."

A very different type of immobility, also unfortunately named freezing, has developed in the wake of the PD drugs. That type of freezing, which occurs as a response to the medications, will be described in the chapter on dyskinesia.

DYSTONIA

Dystonia (literally wrong muscle tone) is a condition in which a specific body part (usually a limb or the neck) is pulled into an unnatural pose by an overly tight or unresponsive muscle or a pinched nerve. In the mild form, this can look like a clumsy, twisted, or draggy limb, or a tilted head. In its more extreme form, it can look like partial paralysis. It does not look like excess movement, such as tremoring or shaking. Dystonia has a kind of stillness, an inertness or rigidity to it. Because dystonias often appear as paralysis of a body part, they are nearly always wrongly assumed to be neurological in origin and therefore incurable.[1]

Common dystonias

The distorted body part is being pulled out of its correct place by an overly tight muscle, but the origin of the problem is actually an overly weak muscle, in most cases. Most skeletal muscles (muscles attached to bones, as opposed to internal organ muscles such as heart or stomach muscles) work in paired, opposing sets. When one muscle in the paired set is damaged, its opposing muscle, having no counter balance, will pull uncontested, causing a distortion or twisting in the body part. While a rare form of dystonia can be caused by damage to brain and/or nerve tissues, most dystonias are caused by structural displacements and/or muscle damage. A slightly displaced shoulder from a softball game, a vertebra that has been tweaked to one side during a high school wrestling match, can, over time (sometimes decades), lead to a nearby muscle that is weak, no longer functional, or only partially functional. Its paired, opposing muscle, still able to function, will appear to be, in the absence of its counter muscle, overly tense.[2] The

---

[1] Again, up until the last few years of the 20th century, anything related to nerves was considered immutable. It was a fact that nerves could not heal or regenerate. After any problem was announced to be a nerve problem, the doctor could wash his hands of it.

[2] Western medicine has until recently stated that all dystonias are due to nerve damage and are therefore incurable. However, osteopaths, chiropractors, and students from many schools of alternative medicine, including Tui Na practitioners, are trained to restore structural components and re-stimulate numbed muscles, thereby curing dystonias. In my own practice, those patients who have been told that they have "permanent brain damage" causing their dystonias have been understandably miffed when their "permanent" condition responds predictably to Tui Na massage.

still-functional half of the muscle pair, no longer being opposed, can progress into a state of excess tonicity, or muscle tone, which is very different from that wooden, even steely, death-like rigidity of Parkinson's disease that can be easily felt in, for example, the anteriolateral muscles of the leg.

Drug-induced dystonias

Dystonias can also be caused by the PD medications. Because the medications can trigger muscle spasms, they can cause either dystonias or twitches. If the medications set in motion a sustained muscle tension rather than the more common, rapid-fire muscle tensions, dystonias will result instead of ticcing or twitching. In this case, the dystonia is not being caused by one muscle in a pair being weak; the drugs are causing one muscle to tense in an attempt to blow off some of the excess dopamine. These drug-induced dystonias are not related to muscle pair dystonias.

Parkinson's muscle-death dystonias

Dystonias can also be present in unmedicated PDers. These dystonias, unlike the drug-induced ones, are usually caused by musculoskeletal aberrations. They are usually not neural or chemical imbalances, but instead may be structural or related to the rigor mortis-like muscles that occur in Parkinson's. This extreme rigidity occurs in those muscles that have received incorrect electrical signals for decades. As these muscles become rigid, they no longer pull correctly on their paired muscle. The imbalance between the two paired muscles can cause a distortion, or dystonia.

In Parkinson's disease, these dystonias may exist in addition to the classic PD rigidity of the muscles along the mammary line and lateral leg. Learning to differentiate between medication-induced dystonias, musculoskeletal (injury- or illness-induced) dystonias, and the classic immobility of Parkinson's disease is difficult but important for understanding what the role of the medication is in any given person.

TREMOR AND DYSKINESIA

Dyskinesia and tremor are probably the two most misunderstood words in the lexicon of medicated Parkinson's disease. And yet, determining whether a person is having dyskinesia as opposed to tremor can be critical to understanding whether a person is overmedicated or not, and if so, by how much and when. Unfortunately, nearly all of the antiparkinson's disease medications can cause a form of tremor. This drug-induced tremor is slightly different from the PD tremor, but most doctors do not bother to discriminate between them, which further adds to the confusion.

In anticipation of resistance to the idea that PD meds cause tremor, I will point out right here that "tremor" is clearly listed in the Adverse Effects section of the medication

---

If a dystonia does respond to therapy, the neurologist will usually dismiss the recovery, stating that the problem was evidently just a pinched nerve in the neck and not a real dystonia. I had a patient who, when she began to recover from Parkinson's, was told by her neurologist that she had been misdiagnosed and that her Parkinson's symptoms, which had included tremor, facial mask, lack of arm swing, foot dragging, and festinating gait, had evidently all been caused by a pinched nerve in the neck. Depth in this subject is beyond the scope of this book.

inserts for nearly all of the antiparkinson's drugs. Ask your druggist, or read your drug insert. The word tremor will be listed near the top of the list of adverse effects.

Also, for the same reason, namely that most people are highly resistant to the idea that dyskinesia is *not* a part of PD, I will shove in a couple quotes here from a national PD journal to support the statement that dyskinesia comes from the medication: "Within two to five years on levodopa, one-half of people who use L-dopa develop dyskinesias. The movements…(are) not seen in PD patients before levodopa…"

And from the same article, "…Some patients respond minimally to levodopa...(or) …may doubt their improvement and will stop levodopa. These patients rarely develop dyskinesias."[1]

If alcohol cures depression, then PD drugs cure tremor…

Because their tremor may subside briefly shortly after taking the medication, many people assume that this is proof that a new tremor or a worsening tremor is not a side effect of the drugs. This is not correct.

In the following example, I will use alcohol, a dopamine enhancer, to represent your PD drug. I am using depression to represent your tremor. Please study the following example carefully, until you understand it deep in your bones: alcohol, in the short term, can ease depression and nervous shaking. Alcohol can, over time, cause increased depression and amplified nervous shakes. Alcohol is an effective treatment for depression in the short term (one to two hours). Alcohol causes worsening depression over the long term. (Six to twelve hours later, there is the likelihood of a hangover, and after extended use, addiction-related changes.) Therefore, the alcoholic will tell you that his shaking is not caused by alcohol. He will prove this to be true by explaining that, shortly after he has a drink, he feels just fine, and his shaking stops for a while. Therefore, contends the alcoholic, alcohol cures shaking and depression. But you and I know that the temporary illusion provided by the alcohol is just that – an illusion. The alcohol does, in fact, cause shaking to worsen over the long term. If the alcoholic had a tendency to shake even before taking up drinking, the alcohol will, over time, exacerbate the shaking.

The alcohol comparison with PD drugs holds up. PD drugs may decrease tremor in the short term, but they can be worsening your tremor in the long term. Therefore, if your tremor improves for a short while shortly after you take medication, this does *not* prove that the tremor is a PD- rather than a medication-induced movement. If your tremor improves for a short while after you take your medication, this does not prove that your medication is not causing or worsening your tremor.

The alcohol comparison is such a great example because almost everyone knows about the two faces of alcohol. In the short term, alcohol can temporarily "cure" depression because alcohol is a stimulant and a disinhibitor of mental or emotional repression. However, in the long term, alcohol is a depressant. Following a drinking

---

[1] A.N. Lieberman, MD, Curing Parkinson's disease in our Lifetime: Part 3, Parkinson Report, Fall 2000, Vol. XI, 3, National Parkinson Foundation, Miami, FL, p. 11. Please note the use of the word "rarely." The word "rarely" does not mean that dyskinesia does occur in a small percent of unmedicated PD patients. The use of words such as "rarely" is a convention observed in science writing to denote a remote possibility rather than using the word "never," because "never" is impossible to prove. Also, because other medications, such as the antianxiety drugs and antidepressants, are able to set in motion (tardive) dyskinesias, PDers who do not take antiparkinson's medications but who do take other dopamine-enhancing drugs *may* develop dyskinesias from these drugs, although not as quickly.

binge that was initiated to treat depression, a person will actually be more depressed than he was to begin with. This is well known.

Dopamine-enhancing drugs work in exactly the same way as alcohol; they provide a temporary high, followed by both short-term and long-term increase in those very symptoms that they are intended to treat. Yes, antiparkinson's drugs can cause tremor.

So, with that issue cleared up, let's begin our study of two of the most misunderstood terminologies of PD, dyskinesia and tremor. First, I will explain the literal meaning of the word dyskinesia. Then I will briefly describe both tremor and dyskinesia, emphasizing the differences between them. Then I will go into greater detail on both, giving examples. The explanation will include some physiology of the affected limbs, as well as descriptions of anatomical coping mechanisms that arise in Parkinson's.

Literal definition

For all you dictionary buffs, the word "dyskinesia" literally means "wrong movement" and refers to any movement which is not correct. This means that, literally speaking, a tremor, being an incorrect type of movement, is a form of dyskinesia.

But in the specialized argot of Parkinson's disease, dyskinesia has a more specific meaning. The "wrong movements" of Parkinson's are divided into two distinct types: tremor, which can be a normal part of Parkinson's, and dyskinesia, which, in the refined language of Parkinson's, refers to those incorrect movements caused by the medication. Again, because this is very important, *tremor can be a normal symptom of Parkinson's disease, and dyskinesia is not.*[1]

The next section of this chapter will be devoted to these two concepts: tremor and dyskinesia. I hope I do not bore you as I pound these two terms into a paste, but I have found that distinguishing between tremor and dyskinesia is one of the most difficult, and most important, discriminations that you can learn. Your doctor may not be able to help you. As mentioned earlier, some doctors in these modern times, accustomed to seeing only medicated patients, actually think that dyskinesias are a symptom of advancing Parkinson's disease; many refer to it, incorrectly, as "worsening tremor."

A brief description of tremor

Tremor is vibratory, powerless movement that occurs when the body is relaxed. The classic Parkinson's tremor is called a "resting tremor." A resting tremor is one that occurs when the body is at rest. It may cease while the body is moving. For example, a resting tremor in the hand may stop when the hand is being used in an activity. Even a mere waving of the hand, the briefest of gestures, may cause the tremor to stop temporarily. But after a few moments of stillness of the hand, such as resting the hand in the lap, the resting tremor will recommence. The resting tremor of Parkinson's most often occurs in the fingers, hands, feet, legs, and/or neck and chin.

During times of stress, the tremor can describe a wider arc and become amplified, forming a very large and exaggerated version of the original tremor. This larger movement may include muscles that do not ordinarily participate in the tremor. For example, a patient who normally has tremor in the hand may find that under the influence

[1] Please review the footnote on page 82.

of social stresses, the muscles of the upper arm also become engaged in a tremor-rate movement. This larger form of tremor is an adrenaline-induced movement that has more power than the usual resting tremor. This is not a form of dyskinesia but it is an adrenaline-enhanced tremor.[1] I will discuss this adrenaline-enhanced tremor in greater detail later in this chapter.

A very brief description of dyskinesia

Dyskinesia is muscular, spasming movement. As hinted at heavily in the chapter introduction, dyskinesia is caused not by Parkinson's disease but by the antiparkinson's medications. Dyskinesia is involuntary, medication-induced, excessive movement in the muscles of the neck, face, arms, legs, hands, feet, back, torso, diaphragm, intestines and/or heart.

Dyskinesia movements can be steady and rhythmic or irregular and unpredictable. The movements can be jerky, smooth or writhing. Some of the dyskinesias are simple muscle spasms involving a single muscle, such as the twitch of a facial muscle that lifts the eyebrow just a bit. Other dyskinesias involve groups of muscles, such as the ones that cause grimacing, complete with cheek, mouth, and chin contortions.

Some muscle spasms can be fairly bizarre, such as one eye rolling up, heavenward, and getting stuck in that position for a while. Some are not so much spasms as they are rapid, repeated movements, such as repeated tongue thrusting or toe curling. Still other movements, such as a quick, involuntary swat through the air as if batting at an imaginary fly, can appear almost normal. These can sometimes be explained away by the mildly embarrassed PDer saying, "I thought I saw a gnat…" These movements are involuntary movements, not under conscious control. They can occur in both the so-called "voluntary muscles," such as the arms and legs, and also in the "involuntary muscles," such as the heart, the diaphragm, and the muscles that line the digestive tract. Dyskinesia looks and feels like muscular activity and has some power behind it.

Just to forewarn you, there is a powerful type of dyskinetic movement that is repetitive, like tremor, and which can occur in nearly the same parts of the body as the tremor. It is a hideous parody of the tremor. This dyskinesia appears to amplify an existing tremor pattern, while also involving muscles that are not normally used in tremor. This vigorous shaking is powerful, violent, and often painful.

I've touched only briefly on dyskinesia in this chapter, merely defining it, because after you've read in chapter nine just how and why the brain responds to excess dopamine, there will be an entire chapter dedicated to dyskinesia, the whole dyskinesia, and nothing but the dyskinesia. We realized at some point in our journey that the dyskinesias can, in most patients, be the key to medication evaluation. Dyskinesias may yield the best clues as to whether or not a PDer is undermedicated, dangerously overmedicated or even addicted. By tracking the dyskinesias, we could often calculate

[1] In the first few editions of my book, *Recovery from Parkinson's Disease*, I referred to the adrenaline-enhanced tremors as "adrenaline dyskinesia." Technically, this was correct, because dyskinesia is any form of wrong movement. But because of the confusion generated by the use of this term, I am now referring to this as adrenaline tremor. This is probably better, as this change of nomenclature enables us to separate the movements into tremors, which are a normal part of PD, and dyskinesia, which is caused by the medication. The most important thing is being able to differentiate between what is being caused by the drugs and what is not.

just where the drug levels were in a person's brain. Tracking them was a lot trickier than we'd hoped: there were layers upon layers of information hidden in the onset delay, duration, and amplitude of dyskinesias when these patterns were tracked over several weeks. So in this chapter, we will simply define most of the terms. In chapter eleven, you will learn more about dyskinesia than you ever wanted to know.

TREMOR: MORE DETAILS

Now, let's dig deeper into the tremor thing. Because distinguishing between tremor and dyskinesia is so crucial, here is an expansion on the above descriptions: the resting tremor of Parkinson's disease is a rhythmic, vibrating motion in specific body parts. It occurs when the PDer is awake. Tremor usually stops when the person is deeply relaxed, such as when watching TV, having a massage, or dropping off to sleep. It stops during sleep, though sometimes a faint tremoring can occur during dreaming. The resting tremor of PD most often appears in the hand, arm, leg, foot or chin. What does the tremor look like in these areas?

The Shaking Palsy

James Parkinson, who first described this illness in such supreme detail that no one has since improved upon his description, chose, with great care, the word "tremor" to describe the movements of his patients. According to the dictionary, tremor is "a trembling, shaking, or shivering; a vibratory or quivering motion."[1]

The name that James Parkinson gave to the condition that he studied was "the shaking palsy." (Palsy comes from the same root as paralysis and refers to the immobility part of the disorder.) Parkinson's disease, if unmedicated, is a syndrome of lack of movement, sometimes accompanied by a frail, vibrating quiver. Parkinson's disease does *not* include the violent, repetitive muscle spasms that can afflict those who are taking the medication. It will confound you to refer to the powerful, dyskinetic muscle movements that are caused by the medication as "tremor," even those that mimic the movement in your tremor areas. So from here on out, if you have been referring to powerful, rapid repetitive twitching, ticcing, facial grimacing, exaggerated tremoring, arm twisting, and leg thrashing as "tremor," please stop.

I cannot tell you how many people, while flailing in every direction, look at me in astonishment when I muse that their medications seem to be causing some excess movement. They indignantly retort, "You must be crazy! Don't you see how I'm tremoring? I need more medication, if anything!"

I recall Sonny protesting my suggestion that he was moving excessively by saying, "Look at how bad my tremor is!" as his clenched fist slammed repeatedly into his lower back and his spine arched painfully backward, pulling the back of his head down in the direction of his low back. His wife shook her head and rolled her eyes as if they had this argument twenty times a day. "That's not tremor, that's dyskinesia," she sighed.

So what does a tremor look like? How can I describe it in words? Tremor looks like the fluttering of the leaves on a quaking aspen tree when there is the slightest wisp of wind. Tremor is *not* the groaning and twisting of the limbs of the mighty oak tree when the hurricane hits. Tremor is not muscular. It is not caused by "involuntary muscle

[1] *Webster's New World Dictionary*. World Publishing Co., NY, 1970.

tension," despite what it says in the 1960's medical books. People with PD can tell you that tremor (in the early days of their PD, before the medication) never really felt like muscles clenching and relaxing too quickly. Tremor is not caused by muscle strength but by muscle weakness, vibrating in time with the internal theta wave in the brain.

Hand tremor

In the hand, tremor takes many forms. When it occurs between the thumb and index finger, it is called "pill rolling." Less common are the fourth (ring) finger tremor and the five-finger tremor in which all the straightened fingers make fluttery, quick vibrating movements towards and away from the palm. Sometimes there is tremoring in the arm, but because of the weight of the arm, these movements are harder to see. Very often, tremoring in the arm is a feeling more than a visible motion, although, in some cases, tremor can cause the entire arm, neck, and chin to vibrate.

Sometimes the tremor is obvious to all observers, and sometimes it is not. For example, a tremor in an upper arm muscle, such as the bicep, may not cause the arm to bounce as long as the arm is held closely up against the torso. But an activity that relies on the bicep, such as holding an open choir book, will reveal the tremoring as the book bounces up and down.

Foot tremor

In the foot, tremoring is a steady, quick, small vibration. It is more common for the foot to move as if the toes are bouncing up and down off the floor than for the tremoring to be side-to-side, although the side-to-side does sometimes occur. Because the foot and leg are so much bigger than the hand and arm, these motions will tend to be a little larger in the lower extremities, but they are still fairly small, very rhythmic, and vibratory.

Foot tremor often occurs when one leg is crossed over the other. The leg that is crossing over, suspended over the opposite knee and hanging down unsupported, might make quick, vibrating motions of the lower leg or foot. Tremor of the foot may also occur almost any time that weight is off the foot. Leg tremoring is also a quick, vibrating motion. When standing, a leg tremor can cause the leg to bounce up and down off the floor just a bit, resembling the foot tapping of an impatient person.

Chin tremor

Tremoring can also occur in the chin and neck. It is a small, vibratory, trembling motion. Chin tremor can resemble the little trembling of the lower lip that occurs when a person is on the verge of crying but trying not to show it. Neck tremor can sometimes cause the entire head to vibrate.

Tremor's cause: weakness and atrophy

Examine closely the hand of a person with a pill-rolling tremor in the thumb and index finger. Notice that the muscle that is supposed to be alongside the 2nd metacarpal (the bone of your hand that connects the wrist to the index finger) is greatly atrophied. (This is the muscle that should pop up, bulging a little, when you press the thumb up tightly to the side of the hand, as you do when making a salute with all five fingers.) The tremor occurs because this muscle is atrophied, not because it is working too hard. If tremor were caused by muscle power, one would expect there to be strong muscle development in the areas of tremor. What we find is just the opposite: tremor occurs in areas of atrophy.

In Parkinson's disease, muscles in specific areas become slowly atrophied, and the communication of these areas with the corresponding motor area of the brain becomes increasingly weak, even dormant. In the case of the pill rolling tremor, when the deterioration in the hand muscle gets to the point that brain control over this part of the hand is gone, *and* if one has the internal tremoring in the brain which has been making him feel edgy inside for years, if not decades, this internal shaking of the brain will be, in the beginning, relieved when the muscles of the hands so deteriorate that the hand begins to exhibit a bounding pattern that moves in the same time and rhythm as the internal shaking.[1]

When the tremor first appears, its movement actually relieves some of the mental tension caused by the internal shaking. But the internal shaking continues to worsen over time, and eventually, the physical vibrating, together with the internal vibrating, can become traumatic. Meanwhile, as the PD progresses, the parts of the body that were damaged in the arm and leg along the lines in figures 5.1 and 5.2 become increasingly atrophied or rigid, respectively.

[1] Not everyone with PD has tremor. This internal schism in the brain is usually only present in those who have a history of foot injury on only one side of the body. Those PDers who received similar injuries on both feet tend not to have tremor. They still have the shut down of the dopamine-producing system and atrophy along the arms and legs, but it is symmetrical and does not create the internal schism that so rattles the brain of the tremoring PDer.

The Large Intestine channel

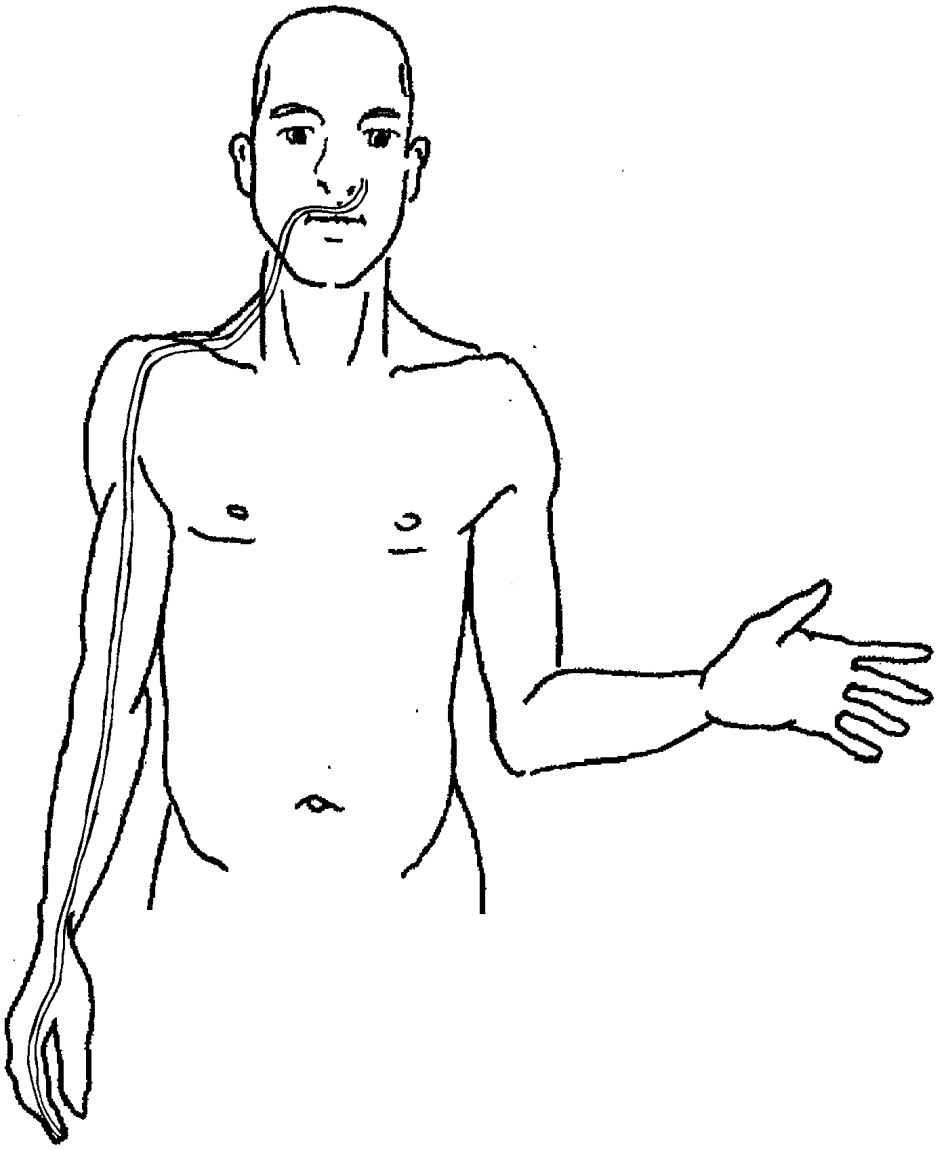


fig 5.1

The obvious atrophy in the muscle on the hand by the 2nd metacarpal continues in a line up those muscles that run up the arm to the face. In primary Parkinson's disease, the hand and arm tremors occur primarily along the line seen in fig 5.1.[1]

[1] In compound PD, other injuries are present in addition to the classic foot injury that is present in all PDers. In this form of PD, the location of the additional injuries and insults will determine which areas become atrophied and which become rigid. The almost infinite combinations possible account for the uniqueness of the symptoms of compound PDers; no two are alike.

As these areas up the arm weaken and brain contact further declines, they too are unable to resist moving in time with the driving electrical drumbeat of internal tremor in the brain. These atrophied areas shake in time with the injury-induced theta wave pattern because they cannot resist it – they are without control, having no strength and no connection to the brain's motor area.

If there is weakness along other leg channels (most often in the Gall Bladder channel), these damaged areas also cannot resist the compelling vibrations of the internal tremor, especially as the Stomach channel is not able to provide stabilization. Obvious quivering in the leg can manifest.

These quivering movements are called tremor. They are neither powerful nor muscular: they are the helpless movements that occur in abandoned body parts. During moments of movement, these useless areas are swept up in the movement of adjacent, still-functional muscles. At these moments, the tremor stops. When the supporting movement of the adjacent muscles ceases, the atrophied areas once more pick up the rhythm of the internal tremor.

There can sometimes be pain or spasm along the edges of these atrophied muscle lines. The pain and tension come from the muscles that border this corridor of degeneration. The body uses those muscles that are alongside the enfeebled ones in an attempt to still the tremor, but these muscles get exhausted from trying to control the errant groups that are tremoring away. Hours of tremoring in fingers or arms can cause painful tenderness or soreness in those muscles of the arm that can still work. So even though the tremor itself is not muscular, the efforts of the body to control the tremor are muscular, and these can cause tightness, tension and pain in the muscles.

Also, just because the movements of tremor are described here as fluttering or vibratory, this does not mean that they are insignificant or harmless. Incessant tremors can aggravate attempts at eating, speaking, dressing, writing, and nearly all the basic functions of life.

The relentless, ineffectual tremor can cause tremendous aggravation. Tremor can be a terrible symptom. Just because tremor is feeble does not mean that it doesn't drive one to desperation.

The Stomach channel

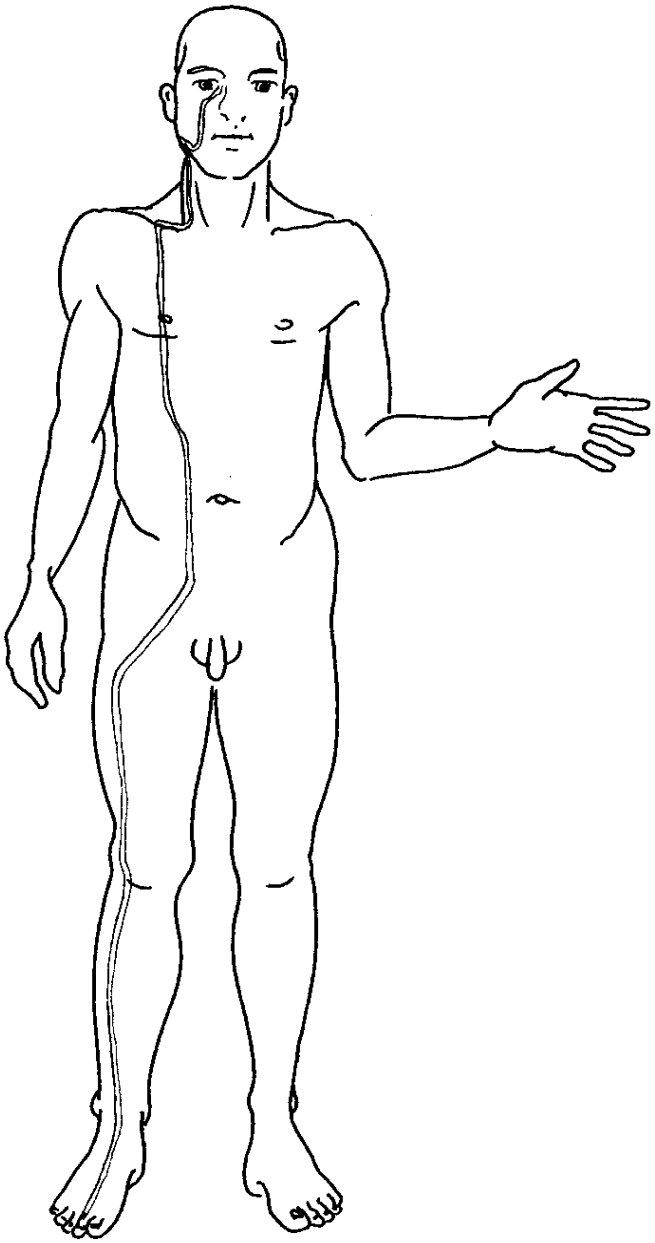


fig 5.2

The neck, chin, and muscles of the torso and leg become steely and unresponsive to brain command (as during rigor mortis[1]) along the lines of the Stomach channel.

[1] Rigor mortis is the Latin term for the extreme rigidity of skeletal muscle that occurs a few hours after a person dies; the muscles become hard as wood and inflexible.

Adrenaline Tremor

There is a form of Parkinson's movement that looks like a gross exaggeration of the tremor.[1] This adrenaline-driven, large version of tremor occurs naturally in PD and is not related to the medication. This type of tremor is produced by situations that create a sense of extreme anxiety or fear. It was hypothesized in my first writings that it is powered by adrenaline, and this hypothesis seems to be holding up.

In the book *The Shaking Palsy* by James Parkinson (dated 1817), he notes that even a person with advanced Parkinson's disease can move quickly and smoothly in times of emergency. This is most likely due to the release of adrenaline during an emergency. Adrenaline is sometimes referred to as the "fight or flight" molecule, the chemical of emergency.

Even in the early stages of Parkinson's a patient may experience adrenaline tremor. In a stressful, adrenaline-producing situation, adrenaline goes to all muscles, *even the damaged ones*. Adrenaline goes to the brain and also goes directly to the muscles. In an emergency, under the influence of adrenaline, the normal, dopamine-using system of motor function is overridden by the emergency mode.[2] Although they are very different (opposites, really), if you consider dopamine to be a movement/thought initiator during moments of calm, you might think of adrenaline as a form of super-dopamine: it initiates super-fast movement or thought in times of emergency. A person with Parkinson's might not have much movement-initiating dopamine, but he will still have the ability to manufacture movement-initiating adrenaline during dire emergencies. Under the influence of adrenaline he may demonstrate brain and motor function that resemble normal movement.

Adrenaline can cause amplification of tremor in unmedicated PD. How? How does adrenaline affect the damaged areas, those areas that tremor, since those areas are not in contact with the brain?

Ordinarily, the nerves of the tissues that are on the line of degradation no longer get switched on or off because, in addition to these particular muscles being physically degenerated, their nerves have been turned off in the brain. Even if dopamine is present in the brain it cannot get through to these damaged areas, these areas that flutter in helpless tremor. But when adrenaline takes over, the situation is altered. Adrenaline floods the body, not just the brain, and can go directly to the muscles, *whether they are damaged or not*. Adrenaline allows a person with a broken leg to run away from danger, even though under normal circumstances he would not be able to walk. In a person with Parkinson's disease, a sudden rush of adrenaline gives to those damaged areas along the arm and leg a jolt of "action" chemistry. These areas, ordinarily turned "off" at all times

---

[1] This material is from *Recovery from Parkinson's Disease*. It is included again here because so many people taking medication cannot tell the difference between drug-induced dyskinesia, tremor, and adrenaline tremor. If you know this material already, please skip this part. It is detailed, even redundant.

[2] Actually, Parkinson's disease begins to manifest when life-long super-high adrenaline levels start to ebb. However, in an emergency, the adrenal glands can still pump out an emergency dose of adrenaline. When this occurs, a PDer may appear to have normal large motor function again, until the emergency passes. Then, when the adrenaline level recedes back to the wearied, diminished level, the Parkinson's is once again evident.

due to damage, will, under the influence of adrenaline, receive the same "on" signal as all the other muscles in the body.

(Eventually, in cases of very late stage Parkinson's, the body shuts down the movement mechanisms so completely that even adrenaline cannot get through. But this occurs slowly, gradually, over years, and is only apparent in very, very late stage Parkinson's disease.)

Adrenaline in the healthy muscles

The majority of the muscles in the body of a Parkinson's disease sufferer are in pretty good shape until the Parkinson's disease becomes advanced, and they have the capability for conscious control.[1] These healthy muscles are in contact with the brain and take orders from up top. The brain can control these healthy muscles, even in the presence of adrenaline, and conscious decisions can be made as to whether or not to use these adrenaline-enhanced muscles. That means that if there is an emergency and the body gets flooded with adrenaline, imparting super-human strength to the muscles, these muscles are poised, at full attention, waiting for the brain to give the signal on how to behave in this particular emergency.

Adrenaline in uncontrolled muscles

However, the weak muscles along the lines of degradation also get charged with adrenaline, but they are no longer under brain control. They no longer connect to the central command. They are no longer coordinated, and the nerves that used to govern them consciously are dormant. So when these damaged areas receive a jolt of adrenaline, they flex and pump, back and forth, back and forth, in a pattern that looks like a magnification of the tremor. They are not healthy tissues, but they can still move a bit when hit with the jackhammer of adrenaline. But there is no motor control coming from Central Command. The brain can't communicate with these areas; they are renegades under the influence of adrenaline. These damaged areas, with their dormant nerve connections to the brain, cannot be controlled consciously like the other healthy muscles.

When these muscles are flooded with adrenaline, they pump back and forth in a very predictable set of motions, namely the motions determined by the function of the specific muscles along the line of degradation. The adrenaline tremor occurs at times when the rest of the body receives a surge of adrenaline, so that powerful, controlled movement is possible in the healthy (*non-tremoring*) areas. Meanwhile, the tremor (damaged) areas explode into uncontrolled movement. These adrenaline-triggered motions in the arm look like variations on a salute and in the leg look like someone tapping his toes violently. They are caused by the frantic flexing and pumping of those muscles that underlie the lines of degradation shown in figure 5.1 or 5.2.

These adrenaline-induced motions are different from the super-fast, inhumanly powerful ticcings of the drug-induced tremor enhancement that can even paralyze the rest of the body, as if Off, while simultaneously hammering away in one or two specific (usually tremor-affected) areas.

---

[1] Just a reminder – a decrease in dopamine does not cause Parkinson's disease; Parkinson's disease causes a decrease in dopamine.

Splinting

In non-emergency situations, when there is not too much adrenaline sloshing around, the healthy muscles will compensate for the ones that have been weakened by incorrect Qi flow. This is called splinting, when healthy muscles do their own job plus the jobs of other nearby muscles that are out of action, for whatever reason. The net effect is that the body can move somewhat smoothly even though not all muscles are working exactly up to snuff.

Emergency movement

In cases of emergency, even a fairly advanced PDer can move somewhat normally. The tired adrenals produce a blast of adrenaline. Adrenaline is always an alternative neurotransmitter (alternative to dopamine) for initiating motor function. However, adrenaline is most often associated with emergencies in which a person must respond with physical movement. For example, in the case of a pre-civilization dire emergency, a caveman PDer could move perfectly well during the course of the emergency; his surge of adrenaline could initiate all necessary movements of running and fighting. During such active times, his resting tremor would not manifest – because his body was not at rest. The healthy muscles adjacent to his damaged ones were so powered by the surge of adrenaline that they controlled the tremor motions.

Modern man: immobile during "civilized" emergencies

However, in our modern world, the exact opposite situation can occur in an emergency. In our co-called civilized[1] world, stress is more often found in situations where frantic running or staging a violent attack is unsuitable. Stress can occur while driving the car or while addressing a room full of hostile board members. In tense situations such as these, adrenaline levels surge. The heartbeat increases accordingly, blood flow to the muscles picks up, and the muscles are primed to respond to the slightest command. But the modern person, sitting in a car or speaking from a dais, can't run or throw his weight about when the adrenaline hits. No, Civilized Person orders his or her muscles to be perfectly still, in spite of the stress. The body may be primed for a primitive fight or flight response but the modern social situations that cause stress must be met with the modern social conventions; these most often call for absence of movement, or polite, restrained movement. Instead of the body firing off a muscle response, the modern person must hold that energy inside, even appearing to be at rest. In an adrenaline-producing situation, either on the freeway or at the in-laws, the healthy, adrenaline-saturated muscles of the modern man are poised and alert, not moving, waiting for cues from the brain.

But what happens to those weak and damaged areas, with their damaged nerves and poor brain/motor coordination, which are no longer under conscious control? The adrenaline hits those areas too. Those muscles start firing off. Back and forth, up and down, those muscles contract and relax as fast as they can with all of their limited strength. The other nearby muscles tense up in a hopeless effort to control the adrenaline-charged, spasming muscles. But those "lost" muscles are not under conscious control, and

[1] Mahatma Gandhi, when asked during a visit to London what he thought of western civilization, replied politely that he thought it would be a good idea.

they jerk and spasm with all the rude, animal strength they can muster. This *animal* strength is much more powerful than the *conscious* strength of a healthy muscle, and thus powerful “adrenaline tremor” occurs. The arm movements typically take one of two forms. The most common motion of adrenaline tremor is "the salute.” The arm goes up and down as if the index finger is going from chest level up to the outer edge of the eyebrow. The next most common form is the "pledge of allegiance,” in which the arm flails back and forth from the side of the body to across the chest. Adrenaline tremor usually lasts for no more than twenty minutes at a stretch, and while it is tiring and can even cause muscle soreness or severe pain, it is not excruciatingly terrifying in the way that super-human drug-induced ticcing can be.

Adrenaline tremor commonly occurs during public speaking or any situation where the body is trying to be relatively calm and controlled but stress is also present. In situations like eating or public speaking, self-consciousness about the tremor itself can amplify into adrenaline tremoring. In such a case, there may be quite a bit of uncontrolled movement going on anywhere along the lines of degradation as well as some rebounding occurring from the rest of the body. Most of this movement will cease, however, when the stress is reduced. Then the body can revert back to the relatively calm, resting tremor.

Tremor starters

Self-Awareness

Adrenaline tremor occurs during moments of high stress and anxiety and also during moments of return to self-awareness. An example of a "return to self-awareness" is the snap back to reality that occurs after watching an enthralling movie or play. A PDer watching a captivating play might utterly cease to tremor for as long as he is more aware of the characters of the play than he is of himself. But when his consciousness suddenly reverts back to himself, he may have a short burst of adrenaline tremor. The tremor may stop during the play because the concentration is so focused that the internal tremor, which is a signal of self-injury, is bypassed when the attention is focused on others. But once the concern comes back to self, the self remembers that it is injured, restarts its screaming for attention, and the internal tremor is up and running again. For the same reason, the tremor suddenly kicks up during public speaking or any other event that makes a person self-conscious. A sure way to get the tremor started up is to realize that the tremoring has stopped. This bit of self-examination goes straight to the self-awareness zone and triggers the internal source of fear, setting off the internal tremor. The physical tremor follows shortly. The beginning of the tremor at these moments can be almost as large as adrenaline tremor, but after a few minutes, the short fizz of self-consciousness adrenaline wears off and the tremor subsides into the usual fluttering motion.

Wake up tremor

Many patients experience a burst of adrenaline tremor when they make the transition from sleep to wakefulness. The startling realization, "I'm here! I'm me, and I'm alive," can often elicit a tremor response even in a person who has almost no tremor during the rest of the day. It can occur in those who take antiparkinson's drugs and in those who have never taken the medication. It resembles the startle reflex in newborn babies, a reflex that sometimes initiates cute (in babies), infantile tremoring in the chin or along the lines of the Large Intestine and Stomach channels.[1]

---

[1] My respected teacher, Jeffery Pang, descended from a long lineage of Asian doctors, taught us that the Large Intestine and Stomach channels are the last to become operational in the human. All the other channels work in utero, but these two do not start until birth. The reason should be obvious: movement of the GI tract in utero would move meconium into the amniotic fluid whence it would flow into the lungs. Also, stomach activity is not needed, since all nutrients are coming from the mother. This is why infant muscle growth and coordination along these channels is retarded relative to other muscle function and coordination.

At the time of birth, the Du channel, which starts at the anus and is most closely related to the spine and midbrain, drops down from its distribution center just between the eyebrows and connects to the upper lip. This triggers the flow of Qi skirting the lips, Qi that is the beginning and end, respectively, of the Stomach and Large Intestine channels. The descent of Qi from the point between the eyebrows to the center of the upper lip also signifies, in some cultures, the descent of the consciousness from the third eye (center of superconciousness) to the orifice of the mouth (ego awareness). These two channels are the last to develop in the infant and they are the first to weaken with the aging process. This is why Parkinson's, which accelerates the degeneration of these two channels, so closely resembles those symptoms of aging that, in turn, resemble symptoms of infantilism.

This bout of strong morning tremor may seem as if it lasts an hour but usually it only lasts about five minutes. Many recovering patients have found that this wake up tremor is one of the last symptoms to disappear. It lingers for a long time, as a sort of reminder that the Parkinson's really did exist, that it wasn't just a bad dream or a misdiagnosis.

Tremoring with meals

A person who has Parkinson's disease and is not yet taking medication will sometimes have adrenaline tremor at meals. The mild, resting tremor can make it difficult to perform the simple actions of eating and drinking. A filled water glass lifted up by a tremoring hand can splash and spill. A tremoring hand trying to hold a fork loaded with rice will likely send rice flying everywhere. This causes tension and stress, the stress causes adrenaline to kick in, and this causes adrenaline tremor, the larger, bouncing movements. It is hard to tell sometimes when the resting tremor leaves off and the adrenaline tremor sets in during a situation like this.

Because the tremoring at meals is such a common symptom, it may be that it is not only anxiety that is triggering the adrenaline but also something deeper. People with Parkinson's all have a history of foot injury in the electrical channel that is named the Stomach Channel. This channel runs from the head to the toes and is involved in the functioning of the gastrointestinal tract. It may be that the stimulation to the Stomach Channel from the anticipation or process of eating plays a part in the mealtime tremor scenario.

An infant learning to use the index finger or legs will often have large, gleeful tremoring along the same lines as that of most Parkinson's patients. Consider a baby in a high chair who has just been handed a spoon. His first clumsy attempts to hold the spoon might initiate repetitive ticcings in his arm, from the index finger up to the shoulder, with the result that the spoon, held with a death grip, bangs up and down ceaselessly on the tray. If he is enjoying himself, his legs might join in the game. His little legs, like his arm with the spoon, will bounce at the same rate as a Parkinson's tremor. As pureed food flies through the air, his little legs jerk back and forth as his arm jerks up and down, and the spoon bangs noisily and rhythmically on the tray. The mother laughs and the baby laughs. The baby makes no attempt to stop this rhythmic up and down movement, which means that the muscles alongside of the immature, spasming ones are not tense or painful. Eventually, as the muscles along these lines develop a relationship with the motor area in the brain, the baby learns to control a movement that could technically be called, in Parkinson's, "excitement-type," or adrenaline-type, tremor.

Summary of incorrect use of the word "tremor"

Patients often tell me that their medication-induced clenched fists and facial grimaces are "tremors." There is a trend to refer to any excessive or uncontrolled movement as tremor. When medicated patients start having little twitches in their toes or little facial tics, they rarely appreciate that it is caused by their medication. They assume it is a symptom of Parkinson's disease. As these movements grow into powerful muscle spasms in their gut, face, arms and legs, most people conclude that these new movements must be yet another manifestation of tremor. Sometimes, patients and even some doctors will deem these new movements to indicate a worsening of the PD, indicating the need for an increase in medication. They are wrong.

Poverty of movement

Why this confusion? Maybe people forget that Parkinson's disease is a syndrome of immobility and rigidity. In addition to forgetting this, maybe they extrapolate from the idea that the only uncontrolled movement in PD is tremor. Then, when their medications cause all sorts of new movement, they just assume that tremor is the word to describe it all. So as a reminder, once again: in unmedicated PD, as the disease advances, movement decreases. The only exception to this immobility and slowness of movement is the small, vibrating tremor and its companion, short-term, adrenaline-triggered tremor.

Still more symptoms

The terminology in this chapter only applies to symptoms of Parkinson's disease, with or without medication. What about descriptive words for the symptoms of drug withdrawal? The drug reduction symptoms that we were seeing in cases like Becky's didn't necessarily fit the descriptions of any of the basic Parkinson's symptoms, whether drugged or not drugged. The next chapter is selections from Becky's journal, picking up where we left off at the end of the previous chapter. From working with Becky and other patients who were reducing their medication, we had to suspect that there was something going on with drug reduction that wasn't simply the reemergence of PD symptoms. These new symptoms will be named and described in chapter thirteen.

Summary

Due to the use of incorrect nomenclature regarding the symptoms of Parkinson's disease, there is often much confusion generated between patient and doctor, and between patients and otherwise helpful literature. Hopefully, this chapter has provided some clues to why these symptoms occur and the correct nomenclature for each.

When you can differentiate between On, Off, and On!, and if you can distinguish between freezing and dystonia, and between tremor and dyskinesia, then you will be able to construct an accurate mental image of my patients in the upcoming case studies. From this, you may be able to determine parallels or differences with your own case. Also, by mastering the standard vocabulary, you will able to communicate clearly with your doctor and other PDers with regard to your symptoms. I recall sitting in PD support group meetings where one person after another would discuss recent problems and experiences using inventive references to symptoms, and sympathetic heads would nod in support, but the bafflement in all eyes was clear: no one but the speaker knew which physical symptoms were being discussed.

By being more exact, honing our babble into precise communication – reversing the trend of the ages – we can be more effective in helping ourselves and sharing answers.

"O Sleep, O gentle sleep, nature's soft nurse!
How have I frighted thee,
That thou no more wilt weigh my eyelids down…"

Shakespeare's King Henry IV

6. BECKY'S JOURNAL

HER WORDS, HER DRUGS, HER SLEEPLESS HELL, HER HOPE

Now that we have a shared vocabulary, we can continue with Becky's travails from chapter 4. The following is Becky's journal notes, interspersed with my notes.

December, 1999[1]

12-12-99

Took a total of 4 TPM (Tylenol PM) last night and they finally knocked me out around 4 a.m. I also had some hot milk. Woke up at 7:30. The Breath thing is still on the edges but it did not trouble me this a.m. so far. Just writing or talking about it seems to bring it on. The twitch comes and goes, never entirely disappearing. I thought of going to yoga but…no way! I am tired of feeling sick! I am actually reluctant to go to bed at night because I am so fearful of the Breath thing. During this whole ordeal I have become aware of death being close by. Philosophically good but unpleasant to live with. Called X for a little verbal sympathy. AB called and we went shopping and to tea and then on to her house for dinner. The unpleasant symptoms faded and I felt nearly normal. I think this is a lot psychological. Later had a phone conversation with X which boosted morale a lot.

12-13-99

Good night's sleep even w/cat on the bed! This a.m. I have the usual twitch and to a certain extent, the breath thing, but it is cut down to size. I am no longer panic-stricken by it. Zoe called. She is having a much worse time than I have had so far. Zoe is now up to 20 L-dopa pills a day. Don't know what to tell her. 2:30 p.m. breath thing is trying to seize control (and) Twitch is active. I am bored just doing odds and ends. Not one of the better days. A cup of chamomile tea seems in order. That, and the heating pad helped.

Note that, at this point, nine weeks after stopping her drugs, she is having a pretty good day, with no pacing or violent breathing problems, and the twitch is coming and going. She is even settling down for a cheering cup of tea. Yet, in her discussions with me at this time, she cannot remember that it was much worse just a few weeks before.

[1] These journal entries appear as Becky wrote them, except for occasional additions (in parentheses) for the sake of clarity. Names, as in all the case studies, have been changed.

She has no sense of improvement, no idea that anything has changed: she is morose. This is typical. An absolute loss of perspective occurred during her withdrawal, and when I would read her notes back to her from the previous week or even the previous day, she was always amazed that her condition had been changing. After the ten-week period passed, she experienced a clearing of the mental mists and she began to have some perspective.

> *12-14-99*
>
> Woke up around 5, got up at 6. Dare I say it? I seem to have gotten a handle on the Breath Monster. My diaphragm muscles don't seem so flaccid. Now I must work on the twitch! It makes my hands uncontrollable, my legs quiver and my teeth chatter. Yesterday at dinnertime I got really hungry. I went to the store and got a salmon steak, broccoli, red potatoes, carrots and ice cream. I dined in luxury! I am sick to death of frozen dinners. Today is Pilates[1] day. Forgot to go last week.[2] Talked to Zoe on the phone again. She is having a very difficult time. She is unable to do without the meds, yet they don't help. This is a hideous disease! The Breath monster is back. I am trying to ignore it, hoping that the brisk walk to Pilates will defeat it. I think I ate too much lunch.[3]
>
> *12-15-99*
>
> The Pilates and the walk made me very hungry and I kept snacking even after dinner. Bed at 11 and slept until 2:15 without TPM.[4] Twitch kicked in vigorously and only after 2 TPM and a cup of warm milk did I manage to go to sleep again, comforted by the cat. Woke up at 7:30. The breath thing was happening. Ignored it as much as I could and had breakfast. It is still prowling around the edges. But I have things to do. Yesterday was a fairly good day. Today has not started as well. The twitch is active. I still weight [sic] the same in spite of eating more. However, I am feeling much more normal nearing the end of the 10 week torture of withdrawal from Sinemet. Where do we go from here? (later, 4:50 p.m.) It turned out to be a bad day. The Breath Monster took over completely. All I could do was sedentary things. But at dinner things improved briefly. Now it is back.

And so, as if a cloud of terror was lifting, she started to feel better. December 15 was nine weeks and five days after stopping her L-dopa, and her weekly report was starting to show that she was living again, getting out, eating, and talking about the Breath Monster as a not unconquerable adversary.

---

[1] Pilates is a form of exercise that works on developing muscle symmetry. She regularly went to yoga or Pilates or various programs if she felt good enough to get out.

[2] She had been in no condition to go.

[3] This report excited me: she was eating well, she was going to exercise, and most importantly, she was thinking in terms of defeating the Breath Monster, whereas in the past she lived in dread of it. This signified a major attitude and perspective change.

[4] This was the first time since she started using sleeping aids that she had fallen asleep at bedtime without some pill. She still needed a sleeping aid after waking at 2:15 a.m..

Over the years, I have learned that ten days to ten weeks isn't a strict boundary of the bad symptoms; it appears to be more the beginning and end of the period of bleakest despair. It is the time when a person will say, "What the hell! What does it matter if I die a junkie, what does it matter if I take just one pill?" This is the time when the brain is arguing with itself, whispering words of hopelessness into the ear. The end of ten weeks doesn't mean that the problems are gone; it appears to be the time frame in which the positive voice of reason starts being able to stand up now and then to the voice of despair. The battle is still difficult, but after ten weeks, he may be able to remember with a bit of clarity why it was he started the fight. He starts to get his brain back.

Around this time, we started tracking the ratio of good days to bad. Slowly, over weeks, Becky went from two good and three bad days to two good and two bad. And then, she went from two good and two bad days to three good and four bad, and then two good and one bad. We shook hands and exchanged hugs when the ratio of good to bad finally became reversed so that the good days were outnumbering the bad. She had daily struggles with her new outspoken negativity, something that she had never experienced in her life prior to her recovery from Parkinson's disease. She could never shake the feeling, on her good days, that she was going to be punished for them sooner or later with a bad day. And the bad days were pretty bad. On a good day she would visit her shut-in friends, take in a play, a community event, walk along the beach, go out to lunch with some friends, or dinner and a movie. On bad days she would have difficulty figuring out how to eat breakfast; she would pace and gasp, twitching and grabbing at her waist; she would have no appetite and no interest in the outside world, feeling too scattered to read and hating the television; and she was afraid to go out for fear of a vague something that she could not identify. The insomnia continued. She usually took two Tylenol PM at night and two again at 2:00 in the morning, for a total of four a night. The package of TPM says to not take it for more than two weeks. She had been taking it for nearly two months. I was concerned about the Tylenol PM, but she was at least getting some sleep.

January, 2000

1-7-00

Nothing unusual…(The) twitch is annoyingly active but got chores done. Left message with neurologist telling him that I have not taken the Sinemet and a follow up app't is not necessary. He will probably be mad.

1-8-00

Last night I went to a play. It was opening night and a wine buffet was served. Was very hungry and ate a lot. I got a teeny bit tight and felt very mellow (did not make a fool of myself to my knowledge). Twitch was noticeable but subdued. Breath monster was completely dormant. I did not try to hide or excuse my twitch. Relied on friends to fetch and carry for me (very uncharacteristic!) Had a great time! Home to bed w/o pills. Woke up late with violent twitch. Took 2 TPM and slept thru the night. Woke up at 7:30. Noticed times when there WAS NO TWITCH. As soon as I realized it the twitch came back violently. Very slow and clumsy this a.m. Checked bank statement and realized that I don't do this

sort of thing well. No patience for figure work. Said to hell with it. Uncharacteristic! It is noon and I had lunch. Eating helps. Good days seem to alternate with bad ones. I am very tired but lying down is no good either. Too much twitch. Will take a walk later. Can't concentrate. Brain has turned to mush! Feel depressed. Not a good day.[1]

In late January her son called and pressured her to move in with him. Becky suspected that his motive was less than altruistic: he had figured out that it would benefit him financially, somehow, to have her as a dependent. This sent her into a spin of horrible days, with twitching and restless legs at night, plus insomnia that didn't respond to anything. She had terrible muscle spasms in her arms and legs, shaking her "like a terrier shakes a rat." She walked for miles that next day, to wear herself out and calm herself down, but it didn't help. She had two terrible days, but then she wrote the following entry.

1-27-00

(I) feel fairly OK. Re-read my journal for the week, JW-H has nailed the symptoms right on (I had read to her some material on drug withdrawal.): breath monster, spasms and all. Yesterday and the day before were very bad. Today should be OK if the pattern holds. Must get as much done as possible while I feel fairly good.

Halleluiah! This was a new attitude! It was the first time that she had been able to review her previous days and realize that there was a pattern, and that she could expect a perfectly good day during which she would be functional. It was an exciting time because it meant that the brain haze from L-dopa and from withdrawal was lifting. Once she was able to see the big picture again, we hoped she would be more emotionally stable. She was working with a hypnotist to help her with her insomnia, and she was practicing visualizing herself being calm and relaxed.

February, 2000

In February she started to realize that she could not sleep without Tylenol PM. She was taking up to 5 per night. On 2-3-00, she wrote:

I have changed pills. TPM taken in frequent doses is hard on the liver I hear.[2]

She stopped taking the Tylenol PM. On her own initiative, after consulting with someone at a nutrition store, she switched to an herbal product, Calm-R-rest. Four days

[1] Two months earlier, she would have been grateful for such an uneventful, ordinary day.

[2] Acetaminophen, the active ingredient in Tylenol, can cause liver damage and death if taken outside prescribed guidelines. In an FDA study of 300 cases of acute liver failure, 38% of these failures were associated with excessive acetaminophen use. Most over-the-counter guidelines suggest a limit of two weeks of Tylenol. I.K. Smith, MD, "The Tylenol Scare," *Time*, April 9, 2001. (The page number for this citation is not on my photocopy. Sorry. The article is in the "Personal Time: Your Health" section of the journal.)

after stopping the TPM, she began suffering terribly, possibly from the abrupt change in her sleeping aid. This was her first experience with drug withdrawal shock from a non-prescription, over-the-counter drug, and one that was not, in theory, a dopamine-enhancing drug. Note that her symptoms of drug withdrawal are the same as the symptoms for dopamine abuse. One possible explanation is that, because these drugs ease pain, a role normally played by dopamine, her extended use of this drug was allowing dopamine to be redistributed in the limbic area. Then, when she abruptly stopped this drug, the limbic system was overexposed, leading to those drug withdrawal symptoms that are normally associated with dopamine-enhancing drugs. In her case, the withdrawal from Tylenol PM most closely resembled the withdrawal symptoms from heroin, another pain sedative. With heroin withdrawal, insomnia and hypersensitivity are the dominant problems.

Again, please note that during this time her voice was vibrant, and her speed of movement was normal, maybe even exaggerated. Her posture was fine, except when she was attacked by the diaphragmatic spasms of the Breath Monster. Her symptoms did not resemble those of Parkinson's in any way except for the violent ticcing, which bears a superficial resemblance to the tremor of PD. Based solely on this twitch, her neurologist was always certain that Becky's Parkinson's was worsening.

> *2-1-00*
>
> It is 2:55 a.m. and the twitch is so bad I cannot sleep since I woke at 1:30. I have taken hot milk, aspirin, and herbal relaxant to no avail. I have prayed, I have paced. The Breath Monster has taken hold after a day's respite. I can't go on like this. I can't visualize. I am afraid to take sleeping pills. HELP!
>
> (9:05 a.m.)
>
> 0h well, I took the sleeping pills and they worked eventually. I got about 1.5 hours more sleep. I dread bedtime. Why do the shakes torment me at night worse than in daytime?

And so it went. During withdrawal from Tylenol PM, the insomnia was the biggest problem. She started experimenting with sleeping aids, NADH, L-phenylalanine, L-glutamine, vitamin C, progreens, vitamin E, multiple vitamins, and Octacosanol. The rigidity and bradykinesia of PD were not an issue, and she had a strong voice and good muscle control when the shaking stopped. She was clearly not a vibrantly healthy person – she was exhausted and twitching. But she did not fit the picture of unmedicated Parkinson's disease. And at last, the ratio of good days to bad days was slowly, over weeks and months, beginning to improve.

At the end of February, she noted that she was beginning to feel a bit more normal and she was panicking less. Her shaking and twitching were milder on 2-24-00 than they had been since September of 1999, just before she had realized that the L-dopa was causing the twitch and she decided to quit the drugs. It had been five months. The five months number struck a chord with me: one of the patients in Oliver Sacks' study on L-dopa had developed hallucinations and was therefore taken off the drugs. Though this patient's hallucinations decreased in intensity after stopping the L-dopa, they had continued for five months. I wondered if the five-month number was significant. Since

then we have seen a few instances when a person appears to go through a sea change after five months, in addition to the ten-week changes.

March, 2000

In March, Becky had some nights that she recorded as "almost normal." One night she slept until 5:30 in the morning. She noticed that while watching movies on television, she twitched during the high emotion moments, and she also noticed that she was becoming better at controlling the twitch for short periods of time via relaxation techniques. But there were still many bad days, especially if it was rainy and cold. She kept daily notes, but I am selecting bits and snatches that capture the tone. (Writing up all her notes would create a 300-page book.)

3-15-00

Foggy (weather-wise and mentally!). Slept fairly well (10:00 to 4:45 when the twitch became active). Got a supply of Walgreen's sleeping pills (generic Benedryl)[1] and will try using them as a tranquilizer. It seemed to work on Mon. No I won't depend on them. I got my lesson through Sinemet![2] Since last week I really think something changed for the better. After the last acupuncture treatment is was like a curtain going up.

3-23-00

A good week!

April, 2000

4-3-00

The good news is that for about 3-4 days I have had no breath problem. Also, I am not so tired. My appetite is better. What remains to be dealt with is the shakes, weakness of the muscles, and energy level.

4-5-00

The shaking now sometimes retreats to just my right hand or left foot.[3]

4-6-00

During visit to acupuncture office 50% of the time the right foot and left hand were NOT moving, only the R hand L foot was going.

4-11-00

I take back that earlier observation that I have turned a corner to recovery. This last week has been discouraging. But I can only continue

[1] The active ingredient in Benedryl is Diphenhydramine Hydrochloride. In this book I will refer to this drug as Benedryl, a patented name, but one that, through usage, is nearly synonymous in the US with Diphenhydramine.

[2] These are prophetic words. She did in fact become addicted to the Walgreen's sleeping pills.

[3] It had been whole body shakes most of the time, ever since the second week of withdrawal.

on this path because any other is a dead end. (Ha, ha, we are all indeed dead in the end!)[1]

4-12-00

Slept soundly from 10 to 6:30 with only 1 trip to the toilet. Shakes were much improved. WHAT DID WE DO RIGHT?

Now she was able to predict the specific times or events when the twitching would be bad: when talking on the phone, waiting for the bus, or upon first awaking at night. The breath monster was long gone. Two times during this week, the twitch just stopped, but when she realized it and marveled at it, it resumed.

By late April the twitch was stopping more often. If she used her arms or hands, the twitch would stop, and she could pick up a teacup without spilling. It had almost become a resting tremor. This tremor was slightly larger than her tremor of a year ago, as should be expected: dopamine-enhancing drugs can cause or worsen tremor, permanently. A few times a day, most notably upon waking, adrenaline tremor was very strong, nearly violent. It took over her whole body. It seemed to last about half an hour, but by watching the clock, she learned that the strong shaking only lasted a few minutes. The twitch stopped completely now and then during the day. If the breath thing ever did appear, it would last for one breath or two, a moment of slight tension in the diaphragm, and then disappear.

In late April she also started noticing an increased fuzziness in her head. We were to learn that this was coming from the Benedryl, but we didn't figure it out for some time. She was feeling more depressed. Also, when she felt agitated from insomnia or because the mental fuzziness was bad, a twitch would start up. The ticcing twitch seemed to have become her body's answer to any problem.

4-25-00

Tired, cranky, and unwell. I want energy and optimism! I had a horrible night. I felt like Galvani's frog and still do. Muscles ache from spasms. None of the usual remedies work. Don't know how I will get through the day. It feels like the Bad Old Detox days

May, 2000

5-3-00

More of same! How long, O Lord…? I have gotten to a point during this week where I cannot function and it seems to be getting worse. I can't read write or move comfortably. Help! I dread tonight!

On 5-4-00 she read the insert on the Walgreen's sleeping pills (a generic type of Benedryl) and realized that they were probably causing her increasing drowsiness and fuzziness. By the way, neither drowsiness nor fuzziness of the head are symptoms of PD. PDers tend to be restless more than drowsy, and intelligent rather than dull. She decided

[1] Reading the preceding days of the week, she had actually been doing very well and had only started feeling depressed the evening before, following getting chilled at water aerobics. Getting chilled is very hard on the dopamine balance. The next day she was doing well again.

to stop taking the sleeping pills. Because they were sold over-the-counter, she assumed that there would be no problem with reducing them all at once. She was wrong. It was pure hell.

Without Benedryl she was unable to sleep for more than a few minutes. The sleep deprivation led to panicked, nearly delirious exhaustion. For five days she slid towards withdrawal symptoms, worsening every day. She went from groggy to hyper-alert, and all of her Pavlovian (learned) symptoms for drug withdrawal reappeared.

> *5-9-00*
>
> I don't know what to do next! I even considered going back to Sinemet. There would be moments of "normalcy" at least. But "I have set my life upon a cast, and I will stand the hazard of the die!" (*Richard III*). Pilates this afternoon. It is a losing battle.
>
> *5-10-00*
>
> In spite of St. John's Wart tea I feel very depressed (and) I am tempted to go back on pills in spite of the horrors. I feel that it can't be that long to my end. Why suffer? If I were younger and had long life ahead things might be different. I don't see well either. How can I get relief between treatments? How can I evade the demons? Constipation might be a problem, having just had a bout with it. I have become fearful of mainstream doctors who only adhere to the pharmaceutical companies' party line. So I talk about taking their pills but probably won't. I notice occasional quiet moments before dozing off, but then the demons jolt me awake. Life was so great before this rotten disease! Yeah, that's what they all say! At night I gravitate among my bed, the floor, the chair, and pacing. During the day I have tasks to distract me.
>
> *5-16-00*
>
> WHAT CAN I DO? Faint nausea. Sleepless night. Violent twitch. I feel awful. Can't do or think anything. Nothing helps. Is death near?
>
> *5-17-00*
>
> Went to bathroom several times at night but otherwise slept fairly well. Twitch still violent in arms and legs which exhausts me day and night. Wine and food help. I could become an alcoholic easily! On the whole I do not feel well; what can we do?
>
> *5-18-00*
>
> Sometimes I can swing arms, but then, suddenly biceps seize up. Have to take a deep breath, relax, then it's OK again for a few minutes. Also, severe dry mouth wakes me up. Mouth is so dry it crackles. It's tight like shrink wrap. A sip of water dissolves it, and then I'm fine.

My own notes for that day say that her eyes were less terrified, and she was making more jokes.

5-20-00
Slept OK in spite of forgetting to take my calcium at bedtime!

She began having alternating bad and good nights of sleep around this time.

5-24-00
Slept erratically, feel very bad today. Violent shakes, breath demon, slowness, faint nausea. No relief in sight. How long will this go on? Will I even be alive next year? I need to sleep but fear bedtime.

She decided that she was suffering more from the withdrawal-induced insomnia and the terror than she had been from the drowsiness and mental confusion of the Benedryl. The sleeping pills were a lesser evil. She gave up the fight after three weeks of withdrawal from Walgreen's sleeping pills.

5-29-00
The last two nights I have gotten a whole night's sleep with the help of the sleeping pills[1] which may or may not be harmful. At this point, I do not care. The message from the medical establishment is "Life's a bitch and then you die." Thanks, doc!

5-31-00
Asked the pharmacist about over-the-counter sleep pills.[2] [His answer was,] "Highly addictive!" More despair! Massage this afternoon. Maybe that will help. The worst part of all this is that I can't get beyond myself and my illness. Later the miasma lifted after visiting "Blanche Dubois" in the nursing home. At massage I began to feel better. In fact, after TS finished my head, the twitch was GONE for at least 5 minutes. It is subdued even now, and I am actually hungry. I am having a glass of Merlot. If I'm going to be an addict, I will be a wino! It is now 7:15 p.m. and I still feel OK. See if it lasts.

After talking to the pharmacist, she decided to stop taking the Benedryl again. Her five days of resumed pills had succeeded in tempering the withdrawal symptoms, but within two days of quitting the pills yet again, she had slid into withdrawal once more. She continued to decline into the familiar, and yet ever-new, abyss. This next journal entry was written on her first optimistic day of no pills before the withdrawal boom lowered.

---

[1] This was the first time since October 8, 1999, that she had slept through the night - red-letter days!

[2] When she asked the pharmacist, she accepted his word as gospel. She had not credited the written warning on the package. She did not believe any written warnings about the medication, or my expressed concerns. Although she had long since written off her neurologist as uninformed and unreliable, she still did not believe negative information about her pills until it came from a man in a white coat. Although she could laugh about this foible, it was still very real to her.

June, 2000

6-1-00

Thank God for yesterday afternoon and evening and sleeping through the nite without pills! I felt nearly normal. But today I was shaken awake by violent vengeful constant shaking. I expect to pay for the peace of yesterday.

Her sense that she would pay for her peace was correct. For the next two nights and days, she had no sleep, and when she stretched out on the floor, she felt that she was losing consciousness. She panicked and thought she was going to die. The next night, she slept through the night again. She had a few more bad nights and started taking the sleeping pills again.

Her son called and told her that she must come to Hawaii, and that he was coming to California to see her condition in July. If she was feeling bad, he said she must sell her condo and move to Hawaii to live with him. He hadn't seen her in a year. He announced that, since she had Parkinson's, he would be able to get a court order to force her to live with him in Hawaii where he could keep an eye on her. She was in a panic, and decided that sleep was more important than mental clarity. She would accept the mental fogginess and the confusion as a permanent condition. Even with the sleeping pills, however, her anxiety about her son's intentions catapulted her twitching into high gear. She was able to sleep again with the pills, but her sleep was anxious and restless. She also felt that she was not looking good, and she needed a quick fix.

On June 16, 2000, she went to the doctor to get some drugs for her anxiety in anticipation of her son's impending visit. She was prescribed Alprazolam, which she promptly named Ali-Kazaam. It is most commonly known as Xanax. Her doctor also demanded that she start taking the Sinemet again. Faced with certain removal from her home by her son if she failed to obey the doctor, she complied.

6-20-00

Slept well after an "Ali-Kazaam" and the usual sleeping pills. Feel as near to normal as I have felt in months after 2 Sinemet CR (25-100) taken at 7:45. I AM NOT TWITCHING. That settles it. I cannot do without medication however faulty it may be. I can think more clearly. I have to force myself to eat, but that may be my boredom with food. Sinemet may be a sinister drug but it is all we have and so must bear with it. Cold turkey didn't work. It is noon and I just took 2 more Sinemets. The basic dose of 2/day is not strong enough[1]…The maximum written on the label is 2 pills 4 times/day. I suppose that then we go to a stronger pill.

I am lucky that this strength pill works at all in any quantity. It has been over a year of no meds and you can't say I did not try. I am too old to be a hero. I am at a crossroad now. I am resuming meds and considering

[1] Note this carefully, where she says, "…the dose isn't strong enough." Probably Becky's neurologist fully expected all of her symptoms of anxiety and twitching to be completely gone within 24 hours. Anything less than complete cessation of symptoms was an indication to increase the dose. Even though Becky knew from my regular readings of the drug inserts that these drugs often took months to be effective, she was once again swayed by the power of the white coat and the promise of immediate health.

> letting my son be my caretaker, which can be dangerous to my self-esteem and independence… The twitch resumed but it is not all-consuming.[1]

Here she is incorrect; it had been only 8 months, not a year, since quitting her drugs. She had completely forgotten that there was once a time when she was taking no medications and had apparently recovered from Parkinson's. She had forgotten, following her terrible first bout with drug reduction, that she had been doing pretty well until she decided to abruptly stop taking the Tylenol. In general, she was very confused about her own history on the drugs and her withdrawal experiences. She was determined to keep looking forward. She pointed out that it was my job to keep track of the past; she was going to keep looking towards the future.

Also, she was feeling great after just one day of pills, which should have been a matter of concern. The drug instructions for physicians say that L-dopa should be started at very low levels; the effect from the medication might not be visible for several months. But following her doctor's orders, her symptoms were obliterated and her cares were gone, all within 24 hours! She was obviously surfeited with L-dopa. According to the drug company's instruction, this was gross overmedication.

Becky – a review of seven months

After restarting L-dopa in October of 1998 to "impress" her son, and becoming addicted within several days, she had developed a permanent tic in her arm and was beginning to suffer respiratory distress after eleven months. At that time she was taking two pills (400 mg levodopa) per day. Panicked by the breathing problem, she had stopped abruptly in October of 1999. By February of 2000, she was doing well; her withdrawal symptoms were gone, her tic was minimal, and she was exhibiting no symptoms of Parkinson's disease. She had started taking Tylenol PM to help with insomnia during the drug withdrawal months. She was pleased and confident. When she learned that Tylenol PM might be a liver toxin, she quit taking it abruptly. The resultant agonies suggest that she had gotten addicted to her over-the-counter sleeping pills. To help her deal with the insomnia portion of the withdrawal, she started taking nightly double doses of Benedryl (or the generic form of Benedryl). Because of cloudy thinking and feeling groggy all day, she had stopped taking the high doses of Benedryl. This abrupt change had triggered all her brain's repertoire of withdrawal symptoms: twitching, nausea, free-floating terror, and utter insomnia.

We were to learn over the next few years that all patients develop a personal set of drug withdrawal patterns, and usually, when faced with withdrawal or any form of stress, the brain will fall back on these familiar patterns as a way of expressing its trauma. Now, at this stage in her journal, she was starting a new regimen of prescription drugs. Due to the impending visit of her son and his resolution to determine whether or not she was fit to live alone, her anxiety levels soared. She had never been prone to anxiety in the past, but the last seven months of insomnia and drug withdrawal had sapped her mental and physical stamina to the point of decimating her previously iron will. She was willing to

[1] Note that the twitch disappeared for a day or two. In chapter 12, in the section on super-dosing, you will see why this massive hit of the drug stopped the twitch for two days. Of course, as her body readjusted to the new dopamine supply, the twitch resumed, a semipermanent part of her repertoire ever since her first addiction event.

do anything, even taking the drugs again, so that she could continue her independence. As you will learn in later chapters, her relationship with her son was a difficult one. Every time she resumed taking drugs, it was due to a threat from the son, her only heir, a man who, she feared, was primarily concerned with his own personal financial perspective.[1]

Now she was taking Sinemet again, at higher doses than she had ever taken before. Also, she was taking Xanax. A new, even more bizarre nightmare was about to begin.

After starting the Xanax plus L-dopa, life was good for a while. Becky reverted to her old ways, which included catching up on her reading. El Twitcho resumed, but in the glow of L-dopa and Xanax it didn't bother her a bit. Then one day at the library, her favorite haunt, she decided to do some studying up on Xanax. A book reviewer had suggested a new book, *Prozac Backlash*, by Dr. Glenmullen, a Harvard MD, that was a sort of exposé of side effects of the antidepressant and antianxiety drugs, plus the industry cover-up and use of misleading research used to get many of these drugs approved. Xanax was one of the drugs included in the book's scope. The book was extensively supported with published research and technical work.[2] It is an excellent book from a researcher's standpoint. In *Prozac Backlash* Dr. Glenmullen makes the point that the real damage from the tricyclics and SSRIs may not be their short-term effect on serotonin and/or norepinephrine,[3] but their inadvertent effect on dopamine, that neurotransmitter that does seem to keep coming up in *this* book.[4] He described in detail how research proved that use of antianxiety or antidepressant drugs will, over time, cause a (possibly permanent) reduction in dopamine, the same as any of the illegal addictive drugs.[5] They were especially likely to set in motion tardive dyskinesias, also known as hand twitching: the Xanax that Becky was using could probably cause damage to her brain and, ironically, reinforce the hand twitching that she already had from the L-dopa. Her Xanax, according to the most recent research, was going to *reduce*, within a few months, her dopamine levels, and cause unpredictable physical symptoms, which would most likely include hand twitching. It was also addictive and it caused parkinsonism: in

---

[1] She finally got out from under her son's threats. In 2002, at the suggestion of a friend, she told her son that she had altered her will: if she was moved out of her home against her expressed wishes, all of her estate would go to the local chapter of the Animal Protection Society. Her son immediately ceased his demands that she come and live with him.

[2] Unlike this one!

[3] *Physician's Drug Handbook*, 9th edition, Springhouse, Springhouse, Pennsylvania, p. 23.

[4] Chapter nine has further information about the relationship between dopamine and serotonin.

[5] In the short term, these drugs cause an increase in dopamine levels. But as you know, any drug that sets in motion a dopamine increase will soon lead to a dopamine decrease. The brain will reduce native dopamine levels to compensate for the incoming dopamine. Thus, these dopamine-enhancing serotonin and norepinephrine stimulators eventually cause a drop in dopamine levels.

In the 1980s, it was guessed that serotonin works in "the opposite way" as dopamine (whatever that means). I suspect that this idea grew out of the old 1950's theory that all neurotransmitters acted in pairs. In the 1950's, acetylcholine and dopamine (DA) were deemed "opposites." In the '80's it was changed to serotonin and dopamine. The Opposites Theory is now falling apart, but old ideas die hard. I suspect that increased serotonin, far from being an opposite, may enhance DA levels. If such is the case, serotonin-increasing drugs will elevate dopamine in the short term, and therefore be addictive. The rebound effect from long-term exposure to serotonin-enhancing drops would be that native DA levels *drop*. With long-term exposure to serotonin-enhancing drugs, native DA levels will eventually adjust downward, just as they drop in response to excess dopamine. This is exactly what is seen in the lab in lab animals.

the words of the insert that accompanies the drug, possible adverse reactions for Xanax included "confusion, tremor, insomnia, nervousness, muscle rigidity, (and) weight gain or loss."

It appeared that Dr. Leslie had demanded that Becky take a drug that could cause, over the long term, tremor, which she supposedly had, insomnia, which she had, nervousness, which she had, plus muscle rigidity, which is a symptom of Parkinson's but which Becky no longer had at this time. Dr. Glenmullen's book explained very clearly how the family of drugs which included Xanax could cause brain changes which create drug-induced parkinsonism, a syndrome which resembled Parkinson's disease but which is brought about by addictive drugs.

Remember, parkinsonism is different from Parkinson's disease. Although the external symptoms of drug-induced parkinsonism are similar to Parkinson's disease, the actual mechanism in the brain is different. The two syndromes share the symptoms of tremor, rigidity, slowness of movement, balance problems, and dopamine deficiency. They both respond favorably, in the short term, to L-dopa. But the difference is that parkinsonism is caused by damage to dopamine producing cells. This damage (at this time, 2003) appears to be irreparable. Parkinson's disease, on the other hand, includes dopamine decrease among its symptoms, but it appears that the dopamine decrease is a correct function of a brain that needs to suppress dopamine production due to other events in the body. Many symptoms of Parkinson's disease are *not* dopamine related. In Parkinson's disease, the brain cells that produce dopamine are dormant. Once the injury-based, emergency, red-alert signal in the brain is turned off, the brain can once again produce dopamine. In parkinsonism, the brain cannot reverse itself. Becky's doctor was now prescribing for her a drug that was known to cause parkinsonism.[1]

Now let us draw a temporary, Act II curtain over the Becky saga. As Becky was becoming addicted to yet another drug, we were growing more successful at predicting the pace and symptoms of drug reduction in our other patients.

---

[1] This chapter raises questions about more than just medication. Why was Dr. Leslie, an intelligent man who had doubtless done well in his school exams, and who had finished med school in the 1980's, seemingly unaware of the side effects of these drugs? Why were most of the doctors fresh out of school encouraging their patients to stay off the drugs as long as possible? Why was Dr. Rafferty, almost twenty years older than Dr. Leslie, more open to new ideas about the medications than Dr. Leslie, with whom he shared an office? Why, from among more than sixty patients with the same diagnosis, did we have only *two* that were taking the same prescription? We found that the single most important factor in determining how a doctor might prescribe these drugs was the year that he graduated from medical school.

This subject, being only tangentially related to the medication issue, is addressed in appendices 5 and 6.

"...And the loud laugh that spoke the vacant mind..."

"The deserted village," Oliver Goldsmith (1728-1774)

7. DOPAMINE DISTRIBUTION

THE TIME FRAMES AND THE ROLES OF DOPAMINE

Our growing success in calculating how the body would respond to drug change over the long term, whether increasing or reducing the drugs, was due to a model in which neurons for various functions have distinct reset rates.

Various brain regions have distinct dopamine properties

When we started this project, we were conforming to the standard ideas about dopamine circa late-1980's. All neurotransmitters (NTs) were assumed to work in the same fashion: one NT storage tank per receptive nerve; and in approximately the same time frames: all nerve/neurotransmitter relationships were quick. Neurotransmitters attached on quickly, were plucked off quickly, and then, if desired, could be used quickly again.

What our patients experienced did not conform to this model. It appeared as if specific drug withdrawal symptoms, those related to motor function, mental function, or fear, each of which is known to occur in a specific brain region, each occurred at different response rates.

Our charts indicated quick motor responses to dosage change, as expected, showing surges and terminations of dopamine-related motor activity over a period of up to six hours, which could be attributed to specific doses. But other symptoms that occurred in response to drug doses and dose changes, such as permanent adoption of On-Offs, worsening of freezing, loss of self-confidence, fear, hypersensitivity, or nausea, seemed to occur during twelve-hour, twenty four-hour, ten-day or ten-week periods of increased or decreased dosage. These longer periods were far too long to correspond to any given dose, even if we were figuring a much longer half-life than the manufacturers.

The short-term rates, up to twelve hours, appeared to be primarily affecting motor function. The longer periods (ten-day and ten-week patterns) appeared to have more of an influence on the symptoms of hypersensitivity, insomnia, and raw emotion. However, once the emotional and gut level (long-term) symptoms went out of control, the motor function also seemed to go haywire, but in a different manner: several weeks after a drug decrease, panicked pacing and violent, whole-body ticcing and shaking might appear, and even increase in intensity for two months before subsiding.

To make sense of what we were seeing, we had to hypothesize a radical departure from conventional understanding in which all brain cells operated at the same tempo, with each having its own private cellular stock of dopamine. Only then did our patients' symptoms begin to make sense. We worked with this hypothesis regardless of lack of evidence from western researchers.

We nervously hypothesized that nerves might have drastically different reset rates, ranging from nanoseconds to *weeks*, a spread much greater than the mild range being suggested at the time. Based on empirical evidence, we proposed that, subsequent to

stimulation from dopamine, each kind of nerve would refresh (be ready to fire off again) at a rate specific to that nerve type or brain area.

We were comforted, as you can imagine, when we started perusing the large amount of information suggesting an eight to twelve-week period of adjustment for drugs ranging from cocaine to Prozac. However, there was still no hard proof of a reset mechanism for that unheard of span.

Imagine our gratification when, just two years into our study, it was discovered that dopamine receptors, in response to the dopamine agonist nicotine, actually have a delayed refresh mechanism that lasts for an astonishing twelve hours![1] Once the receptors had been filled with nicotine and the one brief nerve response was completed, the receptors would not accept another nicotine for twelve hours. This was a far cry from the quick on, quick off, quick on again scenario that had been assumed for all brain nerves, based on nothing in particular, ever since the discovery of acetylcholine.

Three more hypotheses

Buoyed by this finding, and suspecting that the other delayed refresh rates will also be discovered, we created a hypothetical model for dopamine in which:

Dopamine allocation is prioritized by brain zones: over the long term, limbic function is the highest priority, motor is the last.

Different brain zones process (attach and detach) dopamine at different tempos: motor is very quick, frontal lobe function is quick, and limbic can be very slow.

Limbic dopamine can accumulate, but motor area dopamine does not.

In a healthy person, motor function employs the tip of the limbic's iceberg of accumulated dopamine. When dopamine levels are temporarily altered by a flush of dopamine-altering drugs, the motor area may respond to the temporary drug surge, regardless of limbic levels. The long-term prioritizing of dopamine (abbreviated DA) will result in DA being shunted to the limbic area for accumulation.The allocation of dopamine to the various brain zones may superficially (visibly) appear to favor the motor area, but in fact, the prioritizing system will eventually direct all excess dopamine into the limbic zone.

The many hats of dopamine

Health

Dopamine is more than just a movement inducer. Dopamine is involved in the brain circuitry of pleasure, of paying attention, and of processing sight and sound. Dopamine levels influence the immune system, the perception of time, and body temperature regulation. Dopamine is involved in movement initiation and falling asleep. Dopamine's work assignment depends on what part of the brain it finds itself in, and the brain is able to shift dopamine around as needed.

[1] Nicotine, a dopamine agonist, was long considered to be a neurotransmitter that attached to acetylcholine receptors only. As recently as 1990, all of nicotine's effects were deemed cholinergic. Now, in 2003, the focus of nicotine research is on nicotine's dopaminergic properties, its effect on dopamine receptors.

Sickness

An imbalance of dopamine is related to many diseases, not just Parkinson's disease. For example, schizophrenia is caused by an excess of dopamine in the deepest, central part of the brain, paired with decreased activity in the decision-making, frontal part of the brain. It will come as no surprise to the PDers who suffer hallucinations because of their dopamine-enhancing medication that the voices and visions suffered by schizophrenics are set in motion by elevated dopamine levels.[1] Going the opposite way, Thorazine, a dopamine-suppressing drug used for treating schizophrenia in the latter half of the twentieth century, often caused a tremor and a shuffling gait just like the shuffle of Parkinson's disease. Dopamine excess and deficiency are suspected in the manic and depressive phases, respectively, of bipolar disorder.

Next, since what dopamine does depends on what part of the brain it's in, we need to learn a little bit about three main subdivisions of the think box.

OUR FRIEND, THE BRAIN

In this section you will be learning a bit about three main aspects of brain function and the role of dopamine in each part. Most of the material here conforms to the current (2003) understanding and is subject to change. Our hypotheses about the various rates of reset and dopamine processing and the prioritization of dopamine allotments based on our new model will be included at the end of each section on the specific brain function/region being discussed.

This will be an oversimplified lesson in brain topography. All of you would-be brain surgeons reading this, remember: this is extremely oversimplified. How oversimplified? To start, just because it is much easier to think of different neurons residing in departments, I am going to speak of the movement, frontal lobe, and limbic functions as if they each emanate from distinct, compartmentalized areas. You brain specialists know that all the brain functions are integrated, and even frontal, parietal (side), and rear zones of the brain have contact with cells in the core. However, throughout this workup, motor, frontal, and limbic brain functions will be described as if they lived in separate zones. And then, even though there are dozens of brain zones with very specialized tasks, I am going to reduce the whole grey mass into three major areas, with three basic functions, and propose the approximate refresh rate for each area.[2]

The rate of reset is pure hypothesis, based merely on the experiences of hundreds of patients and tens of thousands of hours that we logged, compared, and analyzed. But at least it's based on something. What we started with – the standard information at the time

---

[1] "The Chemistry of Mental Chaos," *Newsweek*, March 11, 2002, Newsweek, Inc., New York, p. 48.

[2] I will not describe all the in-between chemicals in each area that act as transporters or triggering molecules for dopamine, or the locations of dopamine storage bins and receptors, or those molecules that break up the very neurotransmitters that the brain has gone to great time and expense to put together. I am just going to talk about what dopamine does in each of the three areas, and the rate at which it comes and goes. We are going to **ignore** all those other details! Is this a great anatomy class or what?

Instead of these micro details, we will focus our attention on how dopamine processing in each area compares with dopamine processing in the other areas in general. And don't memorize too much – most of these details are just "facts." Like most scientific facts, they are likely to change again in the next few years. (See Appendix 5 and 6.)

– was based not on anything physical whatsoever, but on the idea that, since nerves were all made of nerve tissue, they should all behave in the same way. We like to think that our hypothesis, though young, at least corresponds to observations and is worthy of consideration.

Now let's pick up a scalpel and divide the oversimplified brain into three sections: the core, the front, and the sides.

The core

The central part of the brain is also called the limbic system, and is sometimes nicknamed the reptile brain, or lizard brain.[1] This is where the raw, non-thinking nerve[2] activities take place. The limbic system controls purely visceral responses, non-analyzed emotions such as rage, fear, and hunger. It controls processes like breathing and the movement of the digestive tract. It regulates critical, life-preserving processes that occur with no modifications from the logical, artistic, or will power parts of the brain. A command from this limbic center can override will power, reason, and habit. Think of raw animal passion, the shark that smells blood, or the mother protecting her child. "Act now, think later!" That's the motto of the limbic area.

The front

The frontal lobe, located in the forehead, governs mood, decision-making, and will power. This part of the brain also regulates ego and mental focus.[3] "I think, therefore I am," is the creed of the frontal lobe.

The sides/motor area

The sides of the brain have various functions: motor (movement), memory, speech, logic, vision, coordination[4] and more. In this discussion of dopamine, I will refer

---

[1] OK, if you are a brain specialist, you are possibly grabbing your pencil to start one of those scorchers beginning with, "Dear madam, are you aware that there are many parts to the limbic system, and the so-called reptile brain is actually only one part, etc." Right. I am writing this for people who do not need to fine tune. This is for readers who may well consider that the brain is an undifferentiated mass of grey matter, and that it functions as a telephone switchboard – the old 1950's understanding of the brain. Read on; you will find that the point I am making holds up, even though I don't break the limbic portion down into its dozens of parts.

[2] As noted earlier, nerves, neurons, axons, dendrites and the rest are going to be called nerves. Those of you who have learned that the brain has neurons and not nerves, pat yourself on the back.

[3] And a lot more. See the two preceding footnotes.

[4] Obviously, for you folks at the front of the classroom who have some background in brain study, I am including in the "sides" some functions that occur in the cerebellum, the site of learned coordinations. For you non-biology readers who are curious as to what I mean, here are examples of a learned, coordinated cerebellar activity: playing a memorized piece on the piano or driving a car. These activities require a steady motor flow with no noticeable thinking. In Parkinson's disease, this type of fluid coordination is lost. Dopamine is needed in this back part of the brain for these learned/coordinated activities.

In this chapter, since some cerebellar functions can be considered extensions of motor function and not a completely separate function, I am including this back-brain activity in with the general idea of

to this section as the motor area, and will ignore most of the other functions of the side brain. Motor function is the process most visibly affected in Parkinson's disease.

Actually, motor function has elements in the center and in the front of the brain, as well as on the sides. For example, the thought process involved in initiating reasoned movement might come from the front of the brain. Mood (also coming from the frontal lobe) affects the manner in which we move. For that matter, the substantia nigra's dopamine, which transforms movement thoughts into movement function, is located deep in the core. But for our purposes, namely, understanding how dopamine behaves differently in different brain areas, I will say that the motor area is located in the sides. You cagey readers will recognize that some non-motor problems of unmedicated PD, such as difficulty in recalling nouns when speaking and difficulty in daydreaming, items not strictly considered motor functions, are also governed by the sides of the brain, the part that we are calling the motor area.[1]

Most PDers who use DEDs (dopamine-enhancing drugs) are only concerned about the short-term assist to the motor area. They like to think that this is the main area targeted by these drugs. They are wrong. The motor area works at its best when the slowly changing limbic area is saturated. Furthermore, PDers in our program who decreased their DEDs too quickly experienced the exact symptoms that will be described in the scenario below when the limbic system, *not* the motor system, becomes stripped of dopamine.

---

sides-of-the-brain motor area. The principles that we're working towards with regard to dopamine still apply.

Now, for those who are truly up on their brain chemistry, I will admit that the oversimplification ignores the relationship of the midbrain and the motor area. I just lump everything together into one of three areas. I am not writing this as a technical book on brain function. I am describing a bare minimum of brain generalities so as to get across the idea that dopamine hooks up and releases at various rates in the brain sections that do various jobs. These variances are the key to the seemingly chaotic side effects of dopamine drugs. So please forgive the broad brush with which I paint the brain parts. Many in my audience have no background in medicine, even in science. My goal is to make a book that will enable people to negotiate the difficult path of drug understanding. Everything else, including nomenclature of obscure brain chemistries and anatomies, is secondary.

[1]While this oversimplification may be disdained by modern students of neurology, there is increasing evidence that a macro overview of the brain may actually be more accurate than the currently popular micro view. Although scientists in the early 20th century assumed that isolating individual molecules and labeling various brain components would lead to a complete understanding of the grey matter, it appears more and more as if the entire brain must be regarded as a unified construct in which molecules change shape and function and various brain areas interrelate and even change function, as needed. The role of mood in regulating the immune system, and the role of left-right hemisphere integration in mastering tasks as diverse as spelling, wrestling, and singing are starting to diminish our idea that we can compartmentalize and categorize the brain. It may someday be discovered that the brain is a microcosm of the universe, a mass of swirling changes that can only be understood and predicted by knowing its point of origin. This theory, that predictability is only possible if the origin is known, is gaining acceptance in physics, but it is not yet employed by biologists.

If history is an indication, however, sometime in the future we will be arrogantly mocking the biologists who are still trying to figure out the chemicals of the brain as if they were cogs in a clock. Of course, human nature being what it is, those mockers will, in turn, be mocked somewhere further down the road.

Dopamine roles in the three brain parts: a suppressor, precursor, and stimulant

How does dopamine relate to these three brain areas according to the current scientific thinking? According to the latest scientific model, in the limbic area, dopamine is a *suppressor* molecule. Dopamine stops signals from getting through. In the frontal lobe, dopamine is a *precursor* molecule for norepinephrine, a NT that does most of the frontal lobe work. Dopamine doesn't do much actual work in the frontal lobe, it is presumed, but is transformed into the mood/thinking NT norepinephrine, as needed. In the motor area, dopamine is a *stimulant*. Dopamine is thought to be a trigger in this zone, initiating actions in motor nerves.

Quite a change from dopamine's historical role as a mere muscle relaxant, the opposite of acetylcholine! No wonder dopamine-elevating drugs can cause such a wide range of responses, from euphoria to spasming. The specific brain response to any given dopamine-enhancing drug depends on exactly which brain sub zones are targeted by that drug. It also depends on the brain half-life of the dopamine-enhancing drugs (DEDs) and their dosage level. There are many sub zones, and, even within some of the small brain areas, there are varying dopamine responses.

For example, in the motor area, one group of dopamine receptors (the D2's) will accept only the dopamine agonists Permax and bromochryptine. In the very same brain area, there are other dopamine receptors (D3's) that can hook up only with Mirapex and Requip. This difference in dopamine receptors, even within the narrow confines of a specific region, such as the motor area, contributes to the different actions of DEDs; even within specific areas, each of the dopamine-enhancing drugs will behave slightly differently. And yet, in the big brain picture, all the DEDs have much in common.

DOPAMINE IN THE THREE BRAIN PARTS

Limbic Land – dopamine as suppressor

In the limbic area, dopamine acts as a suppressor molecule, or Off switch. Hundreds of nerve impulses constantly swarm towards the limbic area of the brain from the skin, muscles, gut, and sensory organs. (Let's ignore the nerves flowing out of this area.) The incoming nerve signals want to tell your brain what is going on in the world and report on the happenings inside your own body. The flood of incoming nerve signals is selectively screened: most nerve signals do not get past the gate; they never make it into the limbic area.

Dopamine serves as the screen that protects limbic land. Molecules of dopamine attach to the nerves of this area. The presence of dopamine suppresses and sedates the nerves. When there is adequate dopamine bathing the nerve surface, the nerve is content. The saturated nerve will not respond to incoming messages. When an incoming nerve signal is stymied by a dopamine-drenched nerve, it is as if the incoming nerve signal never happened. The incoming signal is said to be "inhibited," or suppressed.

When, because of some trauma, the dopamine in this area is dislodged, the exposed nerves spring to life, and nerve signals can zip through the holes where dopamine was displaced, flooding the limbic zone with nerve telegrams. In times of ease, most of the incoming nerve signals are stopped by the dopamine doorman. The presence of dopamine shushes the midbrain nerves and lulls them into complacency. Only if the

incoming nerve impulse is unfamiliar, unusually large, or coming from a particularly crucial body part, will it be able to get past the dopamine defense and into the limbic area. Once the incoming message arrives in limbic land, the impulse may be responded to, as needed.

Most responses in the limbic system are automatic. You do not have a choice about how you will respond. This is why it is crucial that most nerve impulses never even penetrate into this area. The limbic area is far too primitive for decision-making. Most responses to a nerve impulse getting into the limbic area are either Go or Not Go. Of course, if the decision is "Go," then the limbic area may or may not react on an animal (non-thinking) level.

The reaction of the limbic area depends on the quantity of signals coming through, as well as the source of the signals. If the electrical signal is small, with only a few nerve impulses slipping past the dopamine screen, the signal may be relayed to the frontal lobe for analysis. If the signal is alarming, the adrenaline switch may be flipped on and steps taken accordingly. If the signal is dire (as indicated by a large quantity of signals coming through), there may be an utterly unreasoned response: pure rage, or pure terror.

The higher the level of dopamine in the limbic area, the greater the likelihood that incoming responses will be ignored. And oppositely, when dopamine levels are low, incoming responses can pass into the limbic area more easily, implying great danger.

Anything and everything that stimulates any nerve of the body sends a neural impulse to the limbic area. If it weren't for the dopamine deadening of most of the incoming impulses, the brain would be constantly flooded, receiving more information than it could possibly handle. For example, the optic stimulation from a dewdrop and the feeling of your flannel shirt on your arm both send a nerve message to the limbic address. Without the dopamine barrier in place, both of those signals might constitute an emergency:

"Help!" screams the visual impulse in response to the dewdrop, "I'm blinded by the dewdrop!" And the skin sensor, detecting a flannel shirt warns, "Red alert! A shirt is hurting my arm!" Thanks to the screening of all but the largest, the unfamiliar, and the most critical impulses, these neural messages rarely get through to the brain. If you are healthy and have sufficient dopamine, your brain won't receive these mundane, non-critical nerve reports. But if dopamine levels have been stripped, as occurs during drug withdrawal, this hypersensitivity can result.

The dopamine in the limbic area suppresses most of the incoming impulses, nearly all of which are alarms, very few of which are pleasant. Most pleasant nerve impulses are actually only pleasant because we have time to think about them and make good associations. Aside from the sensation of a full stomach, there are very few impulses that are pleasant in and of themselves. In general, all incoming information is a red alert, a danger signal. Without dopamine in this area, you would be subject to too much stimulation, swamped with false alarms.

Therefore, we say that dopamine acts as an Off switch in this "reptilian" part of the brain.

Insufficient dopamine in Limbic land

If there is not enough dopamine in the limbic area, one is jumpy, edgy, raging, overwhelmed with sensory signals, and, most likely of all, fearful. *The extent to which*

you do or do not feel fear every moment corresponds to the amount of dopamine you have in your limbic system.

More dopamine means *less* fear, less awareness of aches, pains, and bothersome distractions. With more dopamine in the limbic area, there is less hunger, less panic. When the limbic area is filled with dopamine at just the right level, one can focus on higher thinking, pondering the meaning of life, or enjoying the company of friends.

The opposite happens when there is a decrease in dopamine: *less* dopamine means *less* ability to maintain equipoise. Less dopamine means less self-control. If there is not enough dopamine in this area, one might scream with pain, lash out in fear, fly into a rage at the drop of a hat, raven with hunger, or quiver with nausea and fear of eating.

If I'm being painfully redundant, here's why: most PDers imagine that their dopamine-enhancing drugs are simply movement enhancers. They have no idea of the real power of dopamine. They do not suspect that while their dopamine drugs appear to be stimulating the motor area, they are doing so in a large part by manipulating the limbic area, the fear center. The antiparkinson's drugs work primarily in the crucial Live or Die center of the brain. And unless a person absolutely understands the multiple faces of dopamine, he will not be able to make heads or tails out of the bizarre responses that eventually develop from the antiparkinson's medications. So, remember: dopamine is a suppressor molecule in the primitive brain. It suppresses information about the outside world and your relationship to it. More dopamine leads to calmness. Less dopamine creates dread and unreasoned response.

Slow motion dopamine changes

The healthy limbic system is partially saturated with dopamine at all times, assuring that most incoming signals will not get through.

We hypothesize that changes in dopamine levels occur very slowly in the limbic system. It may take up to ten weeks for a change in overall brain dopamine levels to come to equilibrium in the limbic area. It can take days for even a slight modification of dopamine increase or decrease to come to equilibrium in the limbic zone.

This hypothesis is not based merely on what happened with our patients. We've all seen examples of this slow-motion change in response. For example, when we get a new pair of shoes that fits differently from our old shoes, the foot sends a "new sensation!" warning to the brain. The dopamine-saturated nerve for foot pain lifts an eyebrow, but doesn't get overly excited. Possibly a small amount of dopamine is displaced by the new sensation. The brain notices this tiny change in the incoming nerve impulse. After several days, during which the incoming signals do not increase, the brain decides to move this signal into the "familiar, not a problem" category. Dopamine is allowed to settle back in around the foot pain nerve, and we never notice the sensation of the shoe anymore. This dopamine change was subtle and slow. It can take several days, maybe even a week or two, to become familiar with something new. It is good that this panic center of the brain responds slowly to change.

In an emergency, however, dopamine is stripped away more quickly. For example – as I write this from California, "earthquake country" – imagine the sight and sound of an earthquake toppling your house, shattering your windows and opening a rift in your front lawn as you sit, stunned, in the living room. These large, unfamiliar sensations flood your limbic brain with unusually powerful impulses. These signals, breaking past the

dopamine barrier, will cause lots of chemical changes in your brain. Adrenaline, the emergency neurotransmitter, also known as the Fight or Flight chemical, will start roaring through your body and brain. It seems that this combination of adrenaline and the sheer quantity of the incoming impulses will dislodge the dopamine that had been shepherding the drowsy limbic area. The dopamine in your limbic zone might scatter like chaff in a hurricane. The adrenaline will allow you to make lightening fast actions and turn off all pain-awareness nerves. The stripped limbic zone will allow you heightened awareness of the sights and sounds around you. The sheer quantity of nerve signals coming in will make you feel that you are experiencing more life-per-minute than usual, giving the illusion that time is slowing down. Your perception of time will alter so that seconds will seem like minutes, and you will have the power and impulse to act now and ask questions later. The emergency actions that you make will be powered by adrenaline. The limbic area, suddenly stripped of its dopamine, provides the fantastically heightened awareness.

After a few days, when the adrenaline level has climbed down and you've caught your breath, the wholesale displacement of the dopamine from the limbic area will start to be noticeable. For weeks afterward, you will be shaky and agitated, starting at small noises and lashing out at your mother-in-law while the limbic system remains out of sorts, until the dopamine that was scattered all through your brain during the emergency settles back down into its usual amount and location. It can take ten days before the dopamine in the limbic area even *begins* to resettle itself. It takes approximately ten weeks (and in some cases even longer, such as in cases of lasting emotional trauma) before the dopamine in the limbic area is restored to equilibrium after a shocking event.

We propose that there are short-term (a few hours) and long-term (ten weeks) reset buttons on the dopamine receptors in the limbic area. These delays, we suspect, are the reason why the limbic area cannot be restored quickly to its former state, even if there is plenty of dopamine present.

This time-lag delay in dopamine reattachment and dispersal in the limbic area provides a major clue in unraveling the mysteries of the DEDs. This concept is probably the single most important thing to remember for any person who is trying to make sense of drug increase or drug reduction symptoms.

Following are some examples of ways in which the pharmaceutical industry, though offering no neural mechanism, acknowledges this limbic delay. Directions for Prozac (an antidepressant) say, "Full antidepressant effect may be delayed until 4 weeks of treatment or longer."[1]

Instructions for Xanax (an antianxiety drug) note, "Wean patient with high doses gradually…to prevent withdrawal symptoms. A 2- to 3-month withdrawal may be necessary."[2]

L-dopa manufacturers explain in the drug insert, "Maximum effectiveness of medication may not occur for several weeks or months after therapy begins." They also state, on the same page under "information for the patient," "Therapeutic response may not occur for up to 6 months," and also, "Because of risk of precipitating a neuroleptic malignant syndrome, observe patient closely if levodopa is reduced abruptly or stopped."[3]

---

[1] *Physician's Drug Handbook,* 9th edition, Springhouse Corporation, Pennsylvania, 2001. p. 449.
[2] Ibid. p. 23.
[3] Ibid. p. 595.

The full benefits of Mirapex, an antiparkinson's dopamine agonist, may not appear for up to six months, and the manufacturer's insert on the drug warns, "Neuroleptic malignant syndrome (elevated temperature, muscular rigidity, altered consciousness, and autonomic instability) without obvious cause has occurred with rapid dose reduction or withdrawal."[1]

The extended period (up to ten weeks) that is needed to adjust up or down in response to limbic dopamine change and the trauma to this brain area that might occur if dopamine levels are abruptly altered make "approximately ten weeks" the single most important time frame to keep in mind when taking, increasing, or reducing any Dopamine-Enhancing Drug (DED).

The PD pioneers found this ten-week pattern coming up repeatedly in their charts. When we combed the drug company warnings and pharmacology books about drug chemistry, we discovered that this ten-week pattern for dopamine readjustment is known to those researchers and doctors who specialize in drug addiction. Regrettably, and in spite of the drug manufacturers' warnings and suggestions, not one of the pioneers' neurologists had any idea whatsoever about the ten week requirement for dopamine accommodation; all of them were hostile to the very idea of anything longer than a three-day adjustment period.

Variations on "slow" limbic change

The ten-week period is not the only time frame that operates in the limbic area. There is a range of time frames, as seen with this cigarette example: there is a short-term (twelve hour) dopamine receptor reset switch that causes the first puff of the day (or the first puff in twelve hours) to be more pleasurable than subsequent smokes, a ten week reset switch that determines the time boundaries of the worst symptoms of nicotine withdrawal, and then there are permanent brain changes and brain associations that are formed, so that decades after quitting smoking, a certain memory or trauma can trigger desire for a cigarette.

A small dopamine displacement, such as the one triggered by a new pair of shoes or a broken bone, can be accommodated fairly quickly, in a matter of days or weeks. After a large dopamine shift, such as occurs during an earthquake, physical or emotional trauma, or a drug regimen change, it might take at least ten weeks before the dust settles. Despite the wide variations, the rate of dopamine change is slow in the limbic area when compared to the fleeting neurotransmitter attachments and detachments found in most other brain domains.

This slowness of dopamine attachment and detachment from the dopamine receptors in the limbic area is a good thing – it prevents us from bounding back and forth between rage, fear, and bliss. Except for horrific emergencies (during which time adrenaline steps in and keeps us on an even emotional keel), the dopamine levels in the limbic area are adjusted gradually, over weeks and months. A healthy brain is usually able to process physical, emotional, and psychological inputs logically and according to habit without ever disturbing the barely-fluctuating dopamine levels of the crude, reactionary limbic system.

---

[1] *Physician's Drug Handbook,* 9th edition, Springhouse Corporation, Pennsylvania, 2001.p. 850.

This plodding rate of change is the reason that it takes several days of vacation before the stress of work begins to recede, and why a drug addict can often stop taking his drugs for several days before withdrawal symptoms begin to appear. In the former case, a vacationer, in the absence of stress, will slowly accumulate more dopamine in the limbic area and gradually blossom with an inner peace. In the latter case, during drug withdrawal, dopamine levels slowly drop and the limbic area gradually becomes over-stimulated, exposing the drug addict to free-floating fear and pain.

Even in the case of abrupt drug withdrawal, it may take two to ten days (depending on the drug and dosage level) before the dopamine free-fall even *begins* to be obvious. After dopamine levels descend down to the basement, it may be ten weeks before the limbic brain starts restoring dopamine (with native dopamine) to high enough levels to move the system back into something approaching a comfort zone. During the ten weeks while the dopamine levels are too low, the brain may be assaulted by sound, light, and pain. A person in this dopamine deficient condition may have shaking heebie-jeebies, paranoias, and no way to stem the flood of nerve input to this reptilian brain that acts before it thinks. But, although tortuous, this agony of withdrawal is not the most life-threatening situation that limbic area dopamine can produce…

The gravest danger: dopamine excess

The most dangerous neurotransmitter situation for a human is this: dopamine excess in the limbic area. An excess of limbic dopamine creates bliss at the most primal level. In this unreasoned state, there is no capacity for fear, pain, rage, or hunger. A person whose limbic area is over-flooded with dopamine may be utterly without capacity or desire for self-protection. While 21st century addiction researchers refer to dopamine as the neurotransmitter that regulates pleasure, a more accurate label would be "the NT that removes fear."

Walking off a cliff, self-immolation, or striding through a glass window can all appear as charming, even amusing, experiences, if there is no capacity for fear.[1] Death and mortality are not a concern if the limbic area is oversaturated with dopamine. For this reason, excess dopamine in the limbic system can be instantly deadly. Excess dopamine in the brain is the most dangerous neurotransmitter situation possible; it can kill faster than any other neurotransmitter imbalance.

Therefore, the body has built-in safeguards to ensure that there are *never* excessive levels of dopamine.[2] **If dopamine levels are ever, however briefly, elevated beyond**

---

[1] These are all activities that people have cheerfully performed under the influence of powerful DEDs such as speed (methamphetamine), an illegal street drug related to Eldepryl, an antiparkinson's drug.

[2] In Traditional Chinese Medicine, dopamine excess is the dreaded Kidney Yang Excess. Some schools of thought hold that there is no such thing as Kidney Yang Excess – there cannot be too much of such a good thing. However, that is pure semantics. What these people mean is that, by design, the body can never *naturally* exceed the Safety Limit. They also are making the point that a goal of life is to maximize Kidney Yang (the joy of pure Life Force).

When Kidney Yang is increased through meditation and focused prayer, there is no danger, and the sky is proverbially the limit. But when short-term increases in Kidney Yang are imposed on the body by chemicals such as heroin, cocaine, or other dopamine-enhancing drugs, this most precious aspect of mind-body is perverted into a dangerous brief caricature of wisdom and joy: the dreaded and "impossible" Kidney Yang Excess.

what the body deems a safe point, the body has stern and unforgiving ways of assuring that dopamine levels will never, ever, be too high again.

These ways are referred to as addiction if the source of the dopamine is illegal. These ways are referred to as "accommodation" or "tolerance" if the drugs are produced by the pharmaceutical industry. By any name, the processes are the same. These processes involve short-term and long-term changes in brain chemistry. Because of the grave dangers associated with excessive levels of dopamine, a brain that has experienced excess dopamine, even for a brief time, will chemically alter itself. The genetic expression of the brain cells is altered, thus altering the performance of those brain cells for a long, long time – maybe forever. These alterations are designed to lower the baseline dopamine levels so that an excess level of dopamine will never, ever, happen again.

The supreme danger inherent in dopamine excess is the reason why the brain has so many mechanisms for reducing dopamine levels. Killing the cells that produce dopamine, increasing the enzymes that break up or detach dopamine, and shutting down the nerves that receive dopamine are just a few of the measures the brain can take if the dopamine level is, even for a moment, excessive. The brain can also immediately reduce dangerous dopamine levels for the short term by setting in motion uncontrolled, even frantic, muscle activity or mental activity (including hallucinations and delusions), thus burning up some of the excess dopamine. By having the arms, legs, face, diaphragm, and heart muscles spasm and thrash about, dopamine may get used up more quickly and subside all the sooner to safer levels.

Dopamine allocation

Prioritizing

How is dopamine doled out amongst the various brain parts? It used to be assumed that any nerve that needed dopamine had a nearby private supply (vesicle), which was dedicated to a single nerve. When the storage bin got the "Open sesame" signal, dopamine could flow from the vesicle onto the nerve, do its job, and then immediately be carted back to the nerve's private reservoir. However, this model is of no help in figuring out how brain nerves access the floods of dopamine that surge indiscriminately into the brain in the wake of dopamine-enhancing drugs. This model may not even be correct.

The issue of dopamine distribution is a crucial one for the PDer. What happens in the brain when dopamine floats through the brain at random, as it does when a person takes certain dopamine-enhancing drugs? How does the brain decide which brain lobe gets to use the goodies? What is the mechanism for dopamine distribution, some method for deciding which brain area gets how much and at what rate?

Actually, the distribution of dopamine is not just an issue for drug-using PDers. Even in healthy people who supposedly have plenty of dopamine, it appears that when mood and external events alter dopamine levels in one part of the brain, it affects the entire brain. This is seen, for example, in the case when overall brain dopamine increases from elevated mood or even good weather. Good weather can decrease the amount of dopamine that is needed for temperature regulation. The subsequent increase in available dopamine can be employed in other areas of the brain. Movement, mood, and pleasure perception may all elevate slightly when the weather changes for the good after a long

period of poor weather. On the other hand, when the weather is cold, and more dopamine is needed in temperature regulation, we move more slowly, mood drops and the immune system is slightly suppressed. (The immune system is closely tied to norepinephrine (NE) levels – and dopamine is an NE precursor.)

Feeling sick or under the weather can affect our rate of movement initiation, and DED (dopamine-enhancing drug) withdrawal effects mood. All dopamine functions appear to be intertwined. A drain on dopamine in one part of the brain will affect other parts of the brain. There is a limited amount to go around, and the various brain parts share from a common pot. Dopamine distribution is in constant flux, and *all* brain parts benefit or suffer from healthy or incorrect dopamine levels, respectively.

In that case, how long does it take for dopamine to sort itself into various brain zones and, if there is a dopamine deficiency, which brain area gets the short end of the stick in the long run?

In our model, each of the various brain areas responds to overall dopamine change based on its rate of dopamine uptake and its refresh rate. For example, the limbic area can only change slowly to reflect an excess or shortfall in overall dopamine, and therefore it cannot take advantage of excess supplies as rapidly as the motor area, which uses dopamine quickly. This might lead us to think that the motor area, which responds in a flash to free-floating dopamine, would accumulate dopamine faster than the limbic area. And yet, this accumulation does not seem to occur.

Despite the faster rate at which dopamine can be processed in the motor area, over the long term it is the limbic area, even though slower to grab the dopamine, that seems to get more of the dopamine stashed away. Whether this is actually due to a prioritizing system or due to the limbic area's superior capacity for dopamine storage or both, we can only guess. But the net result is that dopamine can get stockpiled in the limbic area over time, whereas the motor area does not seem able to build up a supply.

Based on our patients' responses to their DEDs, we hypothesize the following: when there is excess dopamine floating around in the brain, the limbic area gets first whack at it. However, because the dopamine attachments form slowly in this area, the limbic area doesn't necessarily get to take full advantage of being number one on the priority list. The motor area, quick to respond, *appears* more affected by DEDs in the short term.

On the other hand, if there is a general shortage of dopamine, the brain will redistribute the supply so that, eventually, over weeks, the needs of the limbic area are satisfied. The motor area has to take a back seat; motor area dopamine needs may even be denied.

An example of this would be the sequence of the appearance of those Parkinson's symptoms that are related to dopamine deficiency. In PD, the entire system is low on dopamine, and yet the limbic area is able to function somewhat normally for years after the motor area begins to fade: it may be hard to get up from the sofa, but the lungs and heart keep working just fine. Susceptibility to anxiety may not occur until years after PD has been diagnosed. Temperature regulation, on the other hand, may be poor even before the motor problems become apparent. In other words, the overall dopamine levels are dropping, but the various systems that use dopamine do not decline uniformly – there is some system at work that prioritizes which area gets first grab at the dwindling

dopamine. We hypothesize that the limbic area is higher ranking than the motor area and gets first pick.

Limbic Review

1) Dopamine is a suppressor in the limbic area.

2) Dopamine attaches and detaches to limbic area receptors very slowly, except in cases of emergency, when it is displaced by adrenaline, or in cases of DED (dopamine-enhancing drug) decrease, when the accustomed supply simply disappears.

3) When dopamine in the limbic area is drastically decreased, it may cause fear, rage, shaking, tremor, nausea, oversensitivity to sound, light, touch, smell, and taste, tightening of the muscles (as if hypothermic [overly cold]), slowness of movement, insomnia, paranoia, and inability to think clearly, to name a few responses.

4) When dopamine in the limbic area is excessive, the opposite may occur: absence of fear, joyful illogic, insensitivity to pain, heat, or cold, and, very often, excessive motor or mental function.

THE FRONTAL LOBE – DOPAMINE AS PRECURSOR

In the front of the brain, dopamine plays an undercover part in the regulation of mood and will. The neurotransmitters that control most frontal lobe activities are (are?…were? Brain theory changes so fast these days…) thought to be serotonin and norepinephrine. So where does the dopamine come in? Dopamine is the precursor molecule for norepinephrine (NE). Dopamine levels help determine how much NE will be manufactured. For some time, researchers have known that most mood-altering drugs, such as the antidepressants and antianxiety drugs, which are designed to specifically increase serotonin or norepinephrine, have addictive properties that correspond to dopamine excess. Only recently have researchers realized that serotonin-boosting (SSRIs) and tricyclic mood drugs do cause a concomitant change in dopamine levels.[1]

Why should a serotonin-increasing drug cause an increase or decrease in dopamine? It may be that when serotonin levels are artificially boosted, as they are in the case of serotonin reuptake inhibitor drugs, dopamine that was allocated to turning into a frontal lobe neurotransmitter becomes surplus. This dopamine is freed up from its normal assignment and it becomes part of the dopamine reserves again, whence it causes excess levels of dopamine.[2]

---

[1] J. Glenmullen, MD, *Prozac Backlash: Overcoming the Dangers of Prozac, Zoloft, Paxil, and Other Antidepressants with Safe, Effective Alternatives*, Simon & Schuster, NY.

[2] The delayed response in the serotonin and norepinephrine enhancers may actually be due to the fact that their effectiveness is not due to serotonin or norepinephrine – it may be due to the way that these two NTs alter dopamine levels. Certainly, the authorities in the field admit that they don't really know how these drugs are working. The current model cannot explain the way these drugs work. Our hypothesis, on the other hand, not only describes what we've seen over the very long term with DEDs, it makes sense of some of these other drug mysteries as well. In the "definitive guide" to psychoactive drugs, Dr. Julien writes, "Although reasonable correlations have been found between drug-induced increases in the levels of norepinephrine and serotonin and positive, mood-elevating effects in people who are depressed, several limitations and inconsistencies in this pattern have also been seen.

"One major difficulty is that the time course of action is vastly different for the biochemical effect and the clinical response. Although neurotransmission of norepinephrine and serotonin is augmented soon

Where does extra brain dopamine go, in most cases? It is prioritized into the limbic system. Thus, in the presence of drugs that amplify frontal lobe function, drugs that ostensibly increase only serotonin (abbreviated 5-HT) and norepinephrine (NE), the levels of free-floating dopamine are increased. Subsequently, fear, rage, and hunger, the domain of the limbic area, are also decreased. One might even propose, despite the appearance that frontal lobe enhancing drugs are boosting 5-HT and NE, that it is the simultaneous boost to limbic dopamine that actually causes the improvement in mood.

Picking up the pace

The frontal lobe's chemistry changes faster than the limbic system's. Once the dopamine levels are at equilibrium, mood and focus, both governed by the frontal lobe, can last for a few days or a few minutes. This is the healthy pace with which logical minds respond to the rapidly changing, complex vagaries of life.

How long does it take for mood to change? Your mood can change quickly when, for example, you receive a thoughtless remark such as, "What happened to your hair?" or, "You look just awful!" As soon as you hear a negative statement, you can feel your mood start to change.

Going the opposite way, you can be sulking along, nursing a grudge, when suddenly an old song on the radio reminds you of happier times. By the time you start singing along, your mood has lightened.

It is possible to have a sustained focus in this area of the brain lasting for days, but for the most part, this is a product of training and habit. The actual chemistry of the area can change every few minutes.

Self-conscious awareness, another feature of this brain lobe, comes and goes quickly, with sleep and waking. The change from sleep to awareness usually takes just a few seconds, or at most, a minute or two; we propose that dopamine processes in the frontal lobe must be fairly quick.

Dopamine prioritizing in the frontal lobe

When there is extra dopamine floating around in the attic, it appears that the frontal lobe gets second whack at it, after the limbic area. If the frontal lobe had been feeling a bit low, and it gets a chance to grab some dopamine and convert it into NE, it can do so quickly. Eventually, of course, as the brain drifts towards equilibrium, the limbic area might take precedence, but because the limbic hook-ups change slowly, the frontal lobe might be able to use the extra dopamine in the meantime. This might explain the fairly rapid mood changes that can occur in response to strong DEDs. The short-term mood enhancement brought about by DEDs usually only lasts as long as the improved motor function. In fact, very often, a drug-using PDer can receive warning that his motor function is about to wear off – he senses the change in his mood that can precede the end of a dose.

---

after the drug is taken, the clinical antidepressant effect may not appear for three to six weeks." This quote is from the 2001 edition of *A Primer of Drug Action*, R. M. Julien, MD, PhD,, W.H. Freeman and Co., New York, NY, 1999, p. 126.

If it is in fact the DA that uplifts the mood, then the slowness of dopamine accumulation could account for the delayed effect of these drugs – drugs that supposedly act only on one NT. Realistically however, in a system as interrelated as brain NTs, there is no such thing as a drug that acts only on one NT.

It may actually turn out that DEDs improve movement primarily through the alterations that they make in the frontal lobe, not the motor area. This might explain, in part, why recovering PDers have such different motor responses to their first glimmers of native dopamine than they do to their drugs. This subject is discussed later in chapter 21.

Our hypothesis also states that if there is insufficient dopamine, the limbic area will eventually take what little there is, and the frontal lobe will be deficient. For example, when a body is shaking with fear or pain (limbic dopamine deficiency) and reacting out of pure primal instinct, it does not have the opportunity to indulge in moods, nor does it perform logically. This may be because the frontal lobe can't have a full complement of chemistry until the dragon in the limbic area is contented.

Of course, one can picture an exception to this rule: a person in great pain may be able to be temporarily lulled into a fleeting good mood by a pleasing distraction, even though underlying pain and limbic deficiency are present. When the distraction is gone, however, the pain will step forward again. This short-term quick fix, the redirecting of dopamine for frontal lobe usage, allows a shut-in, for example, to temporarily override pain, when a good friend comes to visit.[1]

Frontal Lobe Review

1) Dopamine in the frontal lobe is a precursor NT; it is converted, as needed, into norepinephrine. Based on the changes to DA levels after the use of serotonin-enhancing drugs, we propose that serotonin is also related to DA levels.[2]
2) Dopamine attaches and releases fairly quickly in the frontal lobe. We propose that the time frame may range from seconds to minutes.
3) Deficient dopamine can cause deficiency in the neurotransmitters of the frontal lobe (NE and 5-HT), leading to depression, moodiness, inability to think clearly, and lack of focus and will.

---

[1] In such an instance, when a person can temporarily override the limbic area with a quick snatch of dopamine to the frontal lobe, the person is often accused of having pretended to be in pain, simply looking for sympathy. This is not true – that moment of good mood may come at real cost. Subsequent to the burst of good mood, this person may have depleted some of the dopamine stock so that, soon enough, the limbic area will be suffering even more. On the other hand, if the spirits are lifted significantly, such as occurs in response to either a spiritual uplift or a placebo effect, the input can actually generate more dopamine, truly improving the situation.

[2] The relationship between serotonin and dopamine is not at all understood. It has been theorized that they are "opposites" because a drug-induced increase in serotonin will lead to a reduction in native dopamine levels. However, knowing as we do that dopamine is highly self-regulatory, and a reduction in native dopamine is the common side effect of any increase in pharmaceutical dopamine, it would almost seem as if serotonin is a dopamine-like compound, as far as the brain is concerned. That would explain why an increase in serotonin from pills leads to a paired increase in dopamine, but over the long-term, a decrease in dopamine.

Certainly, the side effects of serotonin-enhancing drugs, especially when taken at high levels, have many points in common with the side effects of dopamine-enhancing drugs, including ataxia (inability to coordinate muscle movements), tremor, myoclonus (twitching or spasms), confusion, agitation, diarrhea, cardiac irregularities and tremor. I suspect that these two compounds have a relationship that is much more complex than simply being "opposite:" most likely, they are two sides of a very similar coin. It may be that serotonin performs many dopamine-like roles. Also, one has to wonder wryly– does the idea that serotonin is the "opposite" of dopamine refer to dopamine's historical role as a muscle relaxant or to dopamine's modern role in providing pleasure? Certainly, serotonin, the major antidepression NT, is more like a cohort, and not an opponent of dopamine, recently renamed the Pleasure NT.

4) Excessive dopamine can cause excess neurotransmitters in the frontal lobe, thus leading to dangerously elevated mood and motor function (mania), intensity of thought and focus, delusions of physical prowess, and possibly even feelings of omniscience and psychic power.

THE MOTOR AREA - DOPAMINE AS STIMULANT

Lastly, we come to the motor area of the brain. It is now recognized by dopamine researchers that, in the motor area, dopamine is a stimulator. Isn't that fantastic? Dopamine is a suppressor in the core/limbic zone, a precursor in the front, and a stimulant in the motor area.[1] Dopamine is an NT multitasker!

Dopamine stimulates the nerves of the motor area. Its presence on a nerve receptor can initiate movement, stimulate speech, memory and laughter. It stimulates coordinated activity and integrates the logical and artistic sides of the brain.

Presto!

Dopamine molecules have a very brief active life in this area. Upon its release from the vesicle, dopamine is rapidly hooked up to its position on a nerve. Moments after the dopamine is hooked up, as soon as the nerve has had its chance at firing off, another chemical (called a reuptake enzyme) grabs onto the dopamine and detaches it from its nerve, scuttling it back to the storage sac. Depending on whether the nerve is for a slow moving or a fast moving muscle (a calf muscle compared to an eye-blinking muscle), the nerve will reset itself quickly or very quickly and be ready to fire off again as soon as the muscle is back into position. How fast can dopamine function in this area? It can function as fast as the blink of an eye. Literally. The mind decides it wants to blink. Dopamine is the NT that triggers the nerve that makes the thought, "blink!" Dopamine is the NT that converts the thought into action. As soon as this blink thought has finished, the dopamine molecules are ripped off the nerve cells where they set the blink in motion. They are carted back to their storage sac, where they are stored until another motor, memory, or musing thought is triggered.[2]

Dopamine works quickly in this motor area. How fast? It can work as fast as a twitch, as quickly as a thought. When we talk, dopamine initiates the speech centers, both the ability to speak and the choice of words. Those processes occur nearly as quickly as the thought behind the words – sometimes faster. When we fall asleep, dopamine levels in this part of the brain drop off sharply and quickly. When we wake up, it takes a moment for the full dopamine load to get moving again, but once it does, it is up on its toes, ready to go.

---

[1] To all you neurosurgeons out there who are steaming at this level of oversimplification, please, get your frontal lobe under control. This is a book for the general public. And to the general public, who may suspect that there is more to it than this, you are correct. There are dozens of special areas in the brain, not three. Still, for our purposes, you will find that these basic ideas about dopamine are sound – as sound as any science can be in this changing world.

[2] Acetylcholine is a part of this chain of events, it being the NT that actually trips the switch on the muscle activating nerve, but without the dopamine, the process won't even initiate.

A shortage of dopamine in the motor areas causes slowness of movement, slowness of the reflexes, slowness of speech, and slowness in processing sounds and ideas.

When there is an excess of dopamine in the motor area, there can be excessive movement and hyper-fast movement. An example of excessive movement is the twitching and grimacing that occurs in DED overdose. Another example is echolalia, when a person hears a word and then repeats that word dozens, if not hundreds of times, unable to stop. Excess dopamine in this area can create the fantastically quick movements that are recorded in the movies of the L-dopa experiments, in which the subjects pump their limbs literally faster than the eye can see. Excess dopamine in the motor area allows the extremely powerful and physically impossible movements, such as picking up a truck, that methamphetamine users have been known to perform while under the influence. An excess of dopamine in this area can cause muscular and mental activities that are literally impossible for a normal human to perform.[1]

Prioritizing

The motor area is the last area to get dopamine out of the native supply reserves. Until the limbic area and the frontal areas have enough dopamine, the motor area vesicles will have to struggle along with what little they can scrounge. However, if a sudden surge of dopamine washes over the brain (such as occurs with pharmaceutical or illegal dopamine-enhancers), the fast-acting motor area might be the first area to take advantage of it, snagging some dopamine for itself in the short term.

Motor area review

1) Dopamine in the motor area is a stimulant; it triggers nerve responses.
2) Dopamine can attach and disengage very quickly in the motor area.
3) A shortage of dopamine in this area prevents or slows movement, reflexes, and integrated thinking.
4) An excess of dopamine in the motor area can cause excessive, overly fast and/or random movement and speech.

[1] These activities, in which human bodies and brains perform in ways for which they were never designed, can cause irreparable damage to the body or brain. For example, one danger of the street drug Ecstasy (a dopamine enhancer) is fatal dehydration, which can occur after a full day and night of tireless dancing with no awareness of fatigue or thirst. But at the time of the "impossible" activity, the doer feels no pain and has no sense that anything fantastic is occurring. Also, the accompanying excess stimulation of the thinking areas can create hallucinations and delusions of heightened power. Ecstasy also creates permanent lesions in the brain. Not surprisingly, some PDers have discovered that this DED temporarily alleviates PD symptoms. In England, some PDers petitioned (in 2002) to have this highly dangerous drug made legal for PDers because it can "treat" Parkinson's.

COMBINING INFORMATION ABOUT ALL THREE BRAIN AREAS

The motor area only works if the frontal lobe is already up and running. The frontal lobe only works well if the limbic area is content. Here's a quiz: which brain area slows down first when we go to sleep, the mind or the movement zone? Answer: the moves slow down first. As we fall asleep, first our physical body grows calm (motor area), and then our self-awareness ebbs (frontal lobe). After dopamine is withdrawn from these two areas, and they are both operating at very low levels, sleep occurs. The body is maintained in sleep by the limbic area, which keeps the heart beating and the lungs pumping. The limbic area is also able to order up subconscious movement, such as the slow, mindless turning that we do in our sleep. When it's time to wake up, the frontal lobe wakens first, and we feel a stirring of consciousness. We remember who we are, where we are, and what lies ahead on the day's agenda. After that, the motor area kicks in; we start to yawn and stretch, and then open our eyes.

It would be terrible if this logical sequence were not followed: if the motor area stayed alert after the frontal area fell asleep, we might dance and sing while snoozing. If the frontal lobe was able to process thoughts while the limbic area was in a state of emergency, we could be trapped in useless indecision while the emergency roared around us. If the motor area could function at highest capacity while the limbic area was under stress, we might easily kill someone because we were feeling edgy.

Now, all of the above events do occur: people walk in their sleep, people become caught up in indecision while watching a child burn to death, and sometimes people act out their whims in a passionate manner with seemingly no override from the frontal lobe. However, these are all pathologies, dysfunctions. The basic program, the standard for brain function, is that the limbic area is the first one that must be pacified, then the frontal lobe can activate, and finally, the motor area can start to rock and roll.

Because the motor area can respond quickly to a surge of dopamine flooding through the brain, it may appear as if the motor area receives first priority – shuddering into action as the first flush of dopamine hits the brain – but over the long run the brain will insist that dopamine be accumulated first into the limbic area and then into the frontal lobe, while the motor area is not allowed to accumulate much at all.

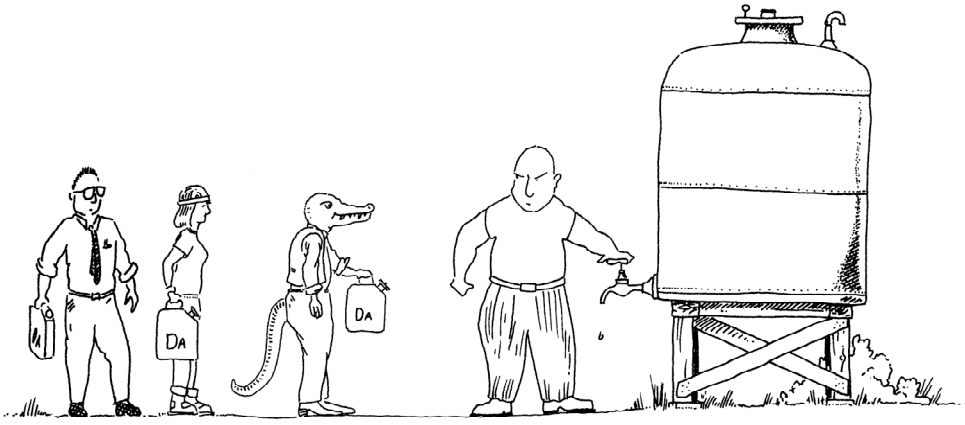


fig. 7.1
This drawing whimsically suggests that, in the presence of dopamine (DA)-enhancing drugs, the motor area – a lowest priority area – makes a swift move towards the front of the line, cutting in front of the frontal lobe and possibly getting ready to shove aside the limbic zone, thus disrupting the normal order of dopamine prioritization.

DOPAMINE MECHANISMS

This short section will discuss some of the more technical aspects of dopamine processes: the dopamine thresholds and windows. These must be somewhat understood in order to make sense of the graphs and charts that will follow in later chapters. Following that, there will be a brief proposal of how brain protection processes may cause PD drugs to appear ineffective, or alter the normal prioritizing of dopamine.

Thresholds

Dopamine, like many other neurotransmitters, works on the threshold basis. This means that there must be a certain minimum, a threshold level, of dopamine present on a nerve before any dopamine-related activity can result. Most people assume that a little dopamine gives a small result, a little bit more gives a little bigger result, and so on. This is wrong; until the level of dopamine rises up to the threshold level, no dopamine

business can result. As soon as dopamine supply goes over the threshold level, full functionality appears.[1]

Here is an example using pretend numbers: let's say that a single nerve has 200 dopamine receptor sites and a threshold of 100. If 99 of the receptor sites are filled, the nerve will not activate. If 100 receptors are filled, the nerve will fire off exactly once. If 150 receptors are filled up, the nerve will still fire off exactly once. That is how a threshold works – it gives an all or nothing response.

Dopamine evidently has some excess thresholds as well. For example, using the numbers in the above example, the nerve will fire off once if there are between 100 and 150 dopamines attached to the nerve. But, since excess dopamine is addictive, we can guess that there is a danger signal if and when too many of the receptors are filled. In this case, let's suppose that the danger signal occurs when 151 receptors are filled. In the presence of 151 dopamines, the nerve will go off exactly once, just as it would have if there had been 100 or 150 dopamines attached, *and* the nerve will send a signal to Safety Central to start reducing dopamine production. If there is far, far too much dopamine, the body may promptly institute visible methods for getting rid of some of the dopamine, methods such as dyskinesia. It may also activate invisible methods such as disabling the dopamine receptors or transporters.

Windows

The distance between the threshold and the danger signal, or Safety Limit, is called, in PD drug parlance, the "window." The effectiveness window is a dopamine level that is high enough to get a response but low enough so that side effects are not triggered. Keeping dopamine levels within the window is the goal of good medication management. The problem with this goal is that it is based on visible trauma and ignores the invisible brain damage.

[1] You PDers in the audience may say that this is not the case, that sometimes you can move slowly, and other times you move normally, thus disproving the threshold theory. In fact, you are using your waning adrenaline when your dopamine drops below the threshold at those times when you are moving more slowly. In the decades while your PD was developing invisibly, you were losing many motor functions and dopamine levels were dropping. You took up the slack with adrenaline. It is when you are finally too tired to care, or so shocked or frightened or sick that the body's adrenaline levels drop very low, that your Parkinson's disease becomes apparent and your dopamine deficiency becomes exposed. This usage of two neurotransmitters to do the job of one helps explain the partial movement that PDers can have.

During recovery from Parkinson's, the adrenaline levels drop to zero, and an unmedicated ex-PDer then finds that he or she does alternate between absolutely normal movement and no movement at all. The profound relaxation that occurs during recovery allows the adrenal glands to finally get some much-needed rest. The resultant dopamine flow is therefore very easy to observe; it is either above the threshold or below, with almost nothing in between. The abrupt cessation and resumption of motor function can resemble the On-Off of drug function, except that it only takes a few restorative minutes, or sometimes a nap, for abrupt resumption of full motor function. Another significant difference is that these no-dopamine periods decrease over many months.

LESS-KNOWN DOPAMINE ROLES TWEAK DRUG RELIABILITY

Other roles for dopamine

Dopamine plays other parts in the body drama. Dopamine is not just a suppressor, precursor, and stimulant. It also regulates body temperature, helps to ameliorate social stresses, and is depleted by infectious disease. People with Parkinson's are familiar with some of these situations; they notice that they cannot move as well in extremes of temperature, when under social stress, or if sick. What they may not realize is that dopamine plays a key role in regulating these body stressors.

It is important to bear in mind all of the influences that can be affecting dopamine levels if one is trying to maintain the best possible body function with the lowest possible level of drugs. For example, very often a PDer will think that he needs to increase his medication because he perceives that he feels stiffer or his drugs are not working as quickly as they were. He may assume the drug dosages are no longer adequate. However, it may be that the real problem, the reason he is moving more stiffly, is because autumn has arrived, bringing with it colder weather. The PDer may unwittingly be using more dopamine in temperature regulation, and so his drugs may not seem as effective. He may only need to turn up the heater or wear an extra layer of clothing rather than increase his medications. He may have been unable to detect that the temperature was growing colder – his DED medications were increasing his limbic dopamine so that he did not feel chilled. But the cold weather was forcing his body to use extra dopamine to keep the body temperature stabilized. Extreme heat also upsets the dopamine balance and can lead to worsened motor function.

It is worth noting that people are most often diagnosed with PD in the fall and winter, or after a surgery or illness.[1] Very often, they note that their symptoms ebb during spring and summer, or during vacations. This is due to the many roles played by dopamine. Simply observing one's physical movement is not an accurate indicator of the progression of Parkinson's disease, nor is it a way to determine appropriate drug levels. Many other factors must be considered as well in determining the cause of seemingly low or high dopamine levels on any given day.

To add to the complexity, consider this: dopamine is a precursor for norepinephrine, and norepinephrine, in turn, stimulates adrenaline receptors, including extra cerebral adrenaline receptors. It is conceivable, following this chain reaction, that L-dopa pills might create not just a dopamine response, but also a norepinephrine and adrenaline response. The fantastic array of side effects that can be set in motion by L-dopa or DEDs may be related to this branching, far-reaching set of dopamine derivatives.[2]

---

[1] Although most PDers are cold all the time, about ten percent of our patients went the other way, and were always uncomfortably hot. Curiously, several members of the "hot group" were first diagnosed with PD in the late summer, during the hottest days of the year.

[2] It is possible that within another year or two all of this information will be either found out to be wrong or found to be merely the tip of the iceberg. Candace Pert, in her *Molecules of Emotion*, presents a brilliant, well-supported hypothesis that extra-cerebral and cerebral neurotransmitters communicate across the entire body map. Not only that, but also the shape and function of these compounds, once thought to be fixed and static, may be tremendously flexible, so that various chemicals can do different jobs at different times. Stay tuned!

In conclusion, to wisely determine dosage levels, a person using DEDs must be aware of all of the influences that can deplete dopamine, the ways in which the brain prioritizes available dopamine, and how various brain parts manifest excess and insufficient dopamine.

Due to the mind-clouding traits of the DEDs, a person taking DEDs might be able to best gauge his medication needs by having a close friend or spouse carefully observing his external circumstances and physiological, emotional, mental, and physical settings. Otherwise, he may increase his drugs when putting on a sweater or eating a hot lunch might have met the case. Also, one must be aware that the medication simply cannot hope to mimic "normal" brain function.

Dopamine enhancing drugs alter the natural order

What happens when DEDs are introduced into the brain? The normal order of brain area dominance/prioritizing can be altered. It is these alterations in normal brain regulation that create some of the disturbing, baffling side effects of antiparkinson's medications. When DEDs come surging into the body, they do not follow the normal release pattern of prioritizing dopamine towards the limbic area first, the frontal lobe next, and the motor area last of all. Instead, the brain may act in unpredictable ways.

For example, the quick-acting motor area, which can take advantage of dopamine quickly if the upstairs is flooded with DEDs, may grab the dopamine before the limbic area does. In a case like this, a person who takes DEDs may thrash violently in his sleep or even sleepwalk. This response is not unusual with people taking too-high levels of DEDs, or in those who take their anti-PD drugs at night.

In another example, such a person might also have writhing, spasming dyskinesia from too much dopamine in the motor area, but not be able to tell that he is moving in an unusual way because his frontal lobe, also flooded, can't accurately assess the erratic movements and interprets them wrongly as signs of brilliance. This inability to recognize inappropriate physical, social, and even sexual behavior is often exhibited in PDers who are overmedicated.[1]

[1] Many of my medicated PD patients have acted out in socially unacceptable manners, including inappropriate sexual advances to strangers and friends, while under the influence of their PD drugs. While the spouses are usually furious or deeply grieved by these behaviors, they should bear in mind that anyone taking more than minimal – or possibly any amount – of PD drugs is not thinking clearly, and may even be stoned out of his mind. While he may appear to be superficially "normal," he may have no access to the parts of his brain where social inhibitions are stored. He may also not remember having done anything untoward ten minutes after the perpetration, or if he does, may try to justify it as being "divinely guided" or his "real self" expressing itself. Such behaviors can be a clue that the dopamine levels are grossly excessive.

Summary

This has been a long chapter, with lots of physiology and details about dopamine. But if you can keep these principles in mind, you or your loved ones may be able to judge more clearly whether or not you are overmedicated, undermedicated, or just right. Your MD may not be able to help you because he will see you once or twice a year for fifteen minutes and he can't possibly know how you are behaving most of the day. Furthermore, he may incorrectly think that hallucinations and spasming are normal symptoms of Parkinson's disease. Try to keep this in mind: in a healthy person's brain, in order to maintain the correct sequence of brain area stimulation and to ensure that dopamine is in the correct levels at all times in each of the correct brain areas, the tiny cells of the brain work with exquisite calibrations.

The allocation, engagement, and reuptake of dopamine are parts of an exact, precise mechanism. There is no room for error. Error in dopamine release causes pathological behaviors, everything from hallucinations to heart-stopping arrhythmias (heart spasms and irregularities).

Errors in the brain's dopamine regulation cause many movement, mental, and emotional disorders, not just Parkinson's. Schizophrenia, depression, and many other psychopathologies are caused by dopamine imbalance.

In distinct contrast to the elegant precision of healthy dopamine regulation, the dopamine-enhancing drugs deliver a dump truck of dopamine over the entire brain, bathing it in a random wash of dopamine. Although some of the DEDs direct their actions towards one brain area more than another, they can never hope to attain the level of discrete cellular fine-tuning that is required for healthy brain function. Instead, the massive surges of dopamine that are created with dopamine agonists, MAO inhibitors, alcohol, cocaine, opiates, methamphetamine, L-dopa, nicotine, Ecstasy and dozens of other legal and illegal chemical compounds inundate the brain with a flash flood of dopamine that cannot possibly be distributed correctly. The overloaded brain, flush with that most dangerous of neurotransmitters, **dopamine**, can act out in pathological ways.

Confronted with dopamine excess or deficiency, each brain area has its own ways of acting out. And you must remember an important linguistic distinction: in the case of people who are behaving strangely due to their PD drugs, these wrong behaviors arising from dopamine excess or deficiency are casually referred to as "side effects of antiparkinson's medications." In people who are taking illegal drugs, these same ways of acting out are condemned as "dangerous pathologies."

"There are some remedies worse than the disease."

Publius Syrus' Maxim 301

8. BECKY AND XANAX

NEW DRUGS, NEW DANGERS, AND A NEW APPROACH

When we last visited Becky, back in Chapter 6, she was taking Sinemet, Xanax, and Benedryl. It was June of 2000. We were just beginning to understand why people were having a hard time getting off their drugs. We were just beginning to formulate time frames for becoming addicted or for the onset of withdrawal symptoms following a drug decrease. Our hypotheses about these drugs, outlined in chapter three, were only partly formed, and tested on too few subjects. The three additional hypotheses from the preceding chapter were still percolating in our minds.

However, we were utterly certain by this time, based on our own experiences and bolstered by our researches into drug addiction and the role of dopamine in addiction, that Sinemet *was* highly addictive, despite the denials of Dr. Leslie and the dozens of other neurologists that we encountered. Becky's resumption of Sinemet was therefore probably causing semipermanent changes. If you will recall, at the end of Becky's journal in chapter six, she had just learned that Xanax also was addictive and could cause permanent brain changes.[1]

---

[1] Advantages of Xanax include "rapid onset…and good patient acceptance. Disadvantages noted include impaired psychomotor performance and alertness and the potential for dependence [addiction] and abuse. Treatment efficacy is therefore controversial" (*A Primer of Drug Action*, 2001 edition, p. 106). Becky's doctor had given a presumed PD patient, one who should have some level of psychomotor inhibition, a drug that *impairs* motor performance, in addition to its being addictive. Xanax is widely advertised as being non addictive. All the scientific writing that we found on the subject says that it *is* addictive.

Because this drug is so widely advertised to the public as being non addictive, I am including in this footnote a longish excerpt from *A Primer of Drug Action*, p.107. This extremely well supported book is a definitive guide for professionals working with psychoactive drugs. This excerpt, which describes the actions of all benzodiazepines, including Xanax, may help you to understand what Becky was dealing with:

"Sleep patterns can be altered markedly. When short-acting agents are taken at bedtime, both early-morning wakening and rebound insomnia for the next night are common. When long-acting agents are taken at bedtime, daytime sedation can be a problem. Impairment of motor abilities is common. This impairment is compounded by the drug-induced suppression of one's ability to assess his or her own level of physical and mental impairment.

"The cognitive deficits associated with benzodiazepine use are significant. In both children and adults, benzodiazepines can significantly interfere with learning behaviors, academic performance, and psychomotor functioning. Cognitive and generalized intellectual impairments can persist even long after the benzodiazepine is discontinued…

"Although benzodiazepines have a reputation for causing only a low incidence of abuse and dependence, the possibility of this adverse complication of chronic use must not be overlooked. When benzodiazepines are taken for prolonged periods of time, a pattern of dependence can develop, even following only therapeutic dosages. Early withdrawal signs include a return (and possible intensification) of the anxiety state for which the drug was originally given. Rebound increases in insomnia, restlessness, agitation, irritability, and unpleasant dreams gradually appear. In rare instances, hallucinations, psychoses, and seizures have been reported."

Bear in mind that this chapter, and even this entire book, is not written in a chronological straight line. We did not figure out what was going on with Becky as it was happening. Throughout this adventure with the medications we kept learning in retrospect what it was we had been seeing. In this chapter, Becky's journal notes will be interspersed with my reflections, many made in hindsight.

Here is Becky's next journal entry, one week after starting Xanax and restarting Sinemet:

> *June 27, 2000*
>
> A week's hiatus during MM's visit. Just read my previous entry and things have changed drastically. I feel much better now. My Sinemet dose has been cut to 2 or 3/day.[1] Felt rather better during tourist activities and dining out. But today I had a terrible phone fight with my son. When I was very sick I had nearly agreed to live with him, but put that on hold now that I feel better. Now he plans to come here and "see what is going on" as though he believes I am not responsible for myself. We love each other and have nobody else as family, and I don't know where it will all end. I am very upset. What can anyone do? If my son were in charge, he would count my pills and force me to take what the instructions say which is far too much. He claims that I will fall and no one will be with me to help (which is true to an extent). This is the end. I must choose between my son and my life, with no certainty either way.

Please note that she makes no comments about twitching or any other symptoms. During her first few weeks resuming the L-dopa she feels just fine. Due to her concerns about falling down with no one around, she signed up for a monitoring service that calls every morning to make sure she is OK.

> *June 29, 2000*
>
> Yesterday I went to the PD clinic. A visiting Japanese practitioner worked on me. I notice I type better today. The mother/son problem will be dormant for a while. I have worked my Sinemet down to 2 with no serious consequences. I take them at 8:30 and at mid afternoon. I take 1/2 a Xanax at night. Seems to work. One day at a time! This week has been "bliss and bane," in that I enjoyed having a guest but too many things happened at once. I intend to write a statement of my position for my son.

> *July 1, 2000*
>
> Slept well last night with the help of a calcium supplement and a Xanax. Seem to be maintaining OK on 2 Sinemet a day. Am sleeping, to the delight of the cat. Am eating at every opportunity but no weight gain

[1] Dr. Leslie had told her to start at two pills a day and increase her Sinemet daily by one pill a day until she felt better. By the third day, when she was taking four pills a day, she felt horribly overmedicated. She was confused and twitching violently. Her face was grimacing and her limbs were thrashing. She immediately cut back to 2 pills a day and felt saner.

so far, continue to look "scrawny," a word I hate.[1] Did various small projects like organizing my rolodex phone number file, which I could not have attempted a few weeks ago. Am working up to the idea of washing out my kitchen cabinets (but I avoided this even when I was normal!)

July 6, 2000 (As reported to me.)

"I take the Sinemet at 8:30. Within half an hour all my symptoms are much worse. Then, half an hour after that, everything smoothes out. When the pill wears off, in the early afternoon, I can fix it by eating. I get a big turkey sandwich from the deli and a cold beer. I take the Xanax to get to sleep. It works instantly."

"Within half an hour symptoms are worse." This drug initiation and wear-off phase, a half hour or so of discomfort while the medication is starting to take effect or ceasing in effectiveness, will be described more fully in chapter 21. At this time we had noticed the switching on and off phenomenon in other patients, but had not yet named it. The unpleasant Switching occurs at the beginning or end of a dose when the body switches from native dopamine condition to the drugged condition, or vice versa. Becky had been on Sinemet 15 days when the switching started to appear. Based on the appearance of Switching, she was evidently not able to abide even this small amount of L-dopa, but we did not understand that significance of the Switching at this point.

July 8, 200

For the past 2 days, I have not felt as well as I should. Does this mean that I should bump up the Sinemet dose to 3 a day rather than 2? Didn't sleep so well last night either. Felt the need for a second half of Xanax but didn't take it for fear of addiction. Only taking half a Xanax up until now. Got a haircut and lunch at the local deli. No appetite. Walking tired me out. I am force-feeding myself. I am so sick of the usual and don't feel like doing one of those Julia Child productions.[2] At 4 p.m. I watched "Oprah." Go on, laugh, but she supports positive things. The subject was people who stand up for what they believe in the face of powerful opposition. I don't have those kind (that kind…?) of guts! I can't even stay away from harmful medications! I want my comfort. Am going to try to get into a Pilates session again but have not made the move. What does this tell me?

July 11, 2000

Had a call from XX (her son), he plans to visit for two days at the end of this month. "We must have serious talks," (Whatever that means!). Any attempt to force me to do something will be a violation of my civil rights. What is the legal and medical record on forcing seniors to do what their children find reasonable but the seniors do not? We shall see. Felt

[1] She had lost nearly 15 pounds during her withdrawal from Sinemet. This is due to the nausea, the pacing throughout the night, and the constant shaking. She was 5'4" and down to 105 pounds.

[2] Julia Child is a famous TV chef.

pretty good today, but the book *Best pills Worst Pills* warns against seniors using Xanax. I only use 1/2 pill at a time and no more than 1 at most. But I already have damaged my brain with that damned Sinemet which I must now use it forever because I was casual in my use of it. I no longer have any respect for the medical profession because they clearly have no respect for seniors and just prescribe anything to get us out of the way. Yes, I am angry. Note about weight: Am keeping steady with nutrition bars and beer between meals. But what happens when I get sick of the taste (which is already happening)? A lot of weakness and not feeling well is the result of unrecognized dehydration and hunger.

In less than a month she has begun to frequently double her Xanax, from half a pill to a whole pill. However, even though she told me and put in her notes that she now takes a whole pill at times, she considers her usage rate to be half a pill.

July 12, 2000

Feel OK, but not as well as yesterday. I never feel quite RIGHT! I suppose I should count myself lucky – and I do – but it gets damned annoying. And it's all so unpredictable. The Breath Monster has been dormant for a while but who knows when it will re-appear.

July 13, 2000

Had off and on breath attacks yesterday, but they passed. Somewhat shaky this a.m. Had a late dinner yesterday which may have accounted for the breath attacks.

July 17, 2000

Slept erratically last night in spite of calcium and Xanax. Took a Sinemet an hour ago. Hasn't done much yet. News! I have gained 4 pounds! The beer and the nutrition bars are doing it!

July 20, 2000

Slept well with half a Xanax. Things are going pretty well. I take 2 Sinemet a day but I am not religious about it. Yesterday I was grocery shopping and wondered why my twitch was so active. I had forgotten about my afternoon pill and was 3 hours overdue. So I took it, late. I look back at the bad times and realize that they were really bad and that I feel so much better now. I consider the twitch minor. I feel pretty optimistic.

Her twitch had been gone, since getting off the Sinemet earlier, and a mere tremor had taken its place. She has been taking the Sinemet nearly a month now, and this is her first warning that the ticcing is resuming. Please note: the ticcing is happening because she is late with her pill, and yet, it only occurs because she has started taking the pills again. This means that the pill is causing the ticcing, even though the ticcing is most apparent (in this early stage) if she forgets to take the pill. Do you see how easy it would be for someone to assume that, since the ticcing ebbs

somewhat after taking the pill, the ticcing is proof that a person needs more pills, not less? But what is really happening is that pill-induced addiction has set in; the ticcing is now her brain's preferred way of complaining either that it wants more drug *or* that is has too much.

July 26, 2000

Read a play from the library, Marsha Norman's "'night, Mother," a play I do not recommend for anyone feeling depressed with a chronic disease, altho' it won the Pulitzer Prize in 1983. Massage this afternoon, and then on to the clinic.

July 28, 2000

Twitch is very active this a.m. Took the usual Sinemet at 8:30 but it did not improve things. Maybe the 2nd one at 2 will at least give the illusion of feeling better.

(No entries during the two days of her son's visit)

August 1, 2000

He left at 7:00 a.m. with cryptic words about "venting the past 30 years." I was not and am not the ideal mom. Enough already! Read an article this morning about Kava which is supposed to do the same thing as Xanax except it is a natural substance and non-addicting. However, you can't take both at once. Question: Is this true? If so, when can you stop taking one and start taking the other? Simultaneously? Taper off? Overlap? Feeling OK.

Over the next three weeks the entries describe the twitch as increasingly active.

August 21, 2000

I always feel as tho' I am working off surplus energy. Went on a Senior Saunter to Lake Lomond today. Fairly strenuous. Must have been 3 or 4 miles altogether up and down. Since the weekend, my twitch has been quite brisk. From time to time it stops briefly, prompting one of my fellow hikers to ask, "You don't twitch any more, do you?" I can get a measure of control over it by an iron will! But it doesn't last long. It makes me feel sort of wobbly-kneed. Is this a new phase? Walking it off helps. Strangely, in the evening, relaxing after a day of exercise, it all goes away.

August 23, 2000

The twitch continues to be increasingly active.

August 24, 2000

The twitch was very strong in my legs and hands. Breath was labored.

The twitching had now moved into her legs, a new development. Over the next few weeks she becomes concerned about the increasing breathing problems – they were the reason she had quit her Sinemet once before.

> *August 26, 2000*
>
> Am tempted yet fearful of eliminating one dose of Sinemet of the two I take. It seems to add to the twitch and it is certainly reawakening the Breath Monster, yet I have had such horrible experiences eliminating and/or increasing it that I hesitate to experiment. Those nights of fear and pacing, sleepless, were too ghastly. Did not sleep soundly last night. Twitch was too strong. Took half a Xanax but it did not work this time. Tossed and turned. Took some calcium. Tossed some more. 4 a.m. got up and made hot milk. It finally worked around 5 a.m. and I woke up at 6:45. All during this episode the twitch was annoyingly subliminal, rippling just under the outer skin.

This feeling of something crawling under the skin is caused by many of the antiparkinson's drugs. This movement is another way in which the brain can try to blow off excess dopamine. It is also possible that it is blood dopamine, and not brain dopamine, that causes the foul feeling of hostile electrical discharge in the skin of medicated patients.

It's now been just over a month since she started back on the Sinemet and began Xanax. She is having good days and bad days. The drugs are not having nearly as much of the desired effect as they had at first. The side effects, however, the switching symptoms, twitching, and breathing disorder, are all back.

> *Sept 1, 2000*
>
> I have stopped taking the Sinemet and started taking NADH, a health food store nicotine based dopamine supplement. Twitch is intensified, but no depression.

> *Sept 3, 2000*
>
> I just took a Sinemet because the twitch is exhausting. I began the day with NADH but it did not do anything.

> *Sept 4, 2000*
>
> NADH seems to stir up the twitch which makes it difficult to write this. Took a Sinemet yesterday hoping it would at least make me feel better (it did not). Took another one this morning but no results. What else can I do? J. said I would be uncomfortable if I tried to stop all at once. That is a mild description! This twitch is getting stronger and more constant. At night I take calcium, melatonin, and Xanax.

> *Sept 5, 2000*
>
> Yesterday it felt like the bad old days of twitching and discomfort so I had wine and cheese and then rice and chili and salad for dinner, ice

cream for dessert. Following all this the twitch and other presumable pill reactions lifted off and I felt nearly normal. Am confused. I started off the week taking 2 NADH and no Sinemet. I twitched a lot and felt symptoms like the Bad Old Days.[1] So I cut back on the NADH and added a Sinemet back in. Had "discomfort." Is taking NADH and Sinemet on the same day a no-no? Should you substitute NADH for Sinemet? Taper off the Sinemet onto the NADH? I am puzzled by the fact that around 4 or 5 p.m. I feel better and more relaxed. Is this an unconscious relaxation or what? I think the NADH causes twitch. Or is it the length of time between Sinemets?

Feel better after a brief walk, but the twitch is doing something new – it becomes a major spasm. Whenever I woke in the night it kicked in and was very unpleasant. It seems generally worse. The twitch no longer stops.

When I asked her, she was emphatic that stopping the Sinemet did not affect her speed of movement, her voice, or result in rigidity – the classic symptoms of Parkinson's. Also, she was certain that when she did not take the Sinemet, she had no breath problems and no facial grimacing.

During the few days when she had no Sinemet, she had no foot spasm either. The foot spasm was a brand new symptom that had started about two weeks after resuming Sinemet. Her energy was good, and only one problem was worsened because of having decreased the Sinemet a few times during the week – the Twitch.

In other words, without the Sinemet, she did not have her usual repertoire of excess dopamine symptoms. Her only problem at this time was the twitch, which, due to resumed exposure to Sinemet, now occurred whether her dopamine levels were either too low or two high. The twitch might possibly be a semipermanent condition brought on by the drugs. It also might be her addict brain's way of calling out for more drug. We had no way of knowing.

We have seen this in other people reducing their drugs as well: sometimes, the symptoms that the body uses to protest against too much dopamine will also be employed during drug withdrawal to protest about too little dopamine.

However, as far as symptoms of Parkinson's, which would have been characterized by poverty of movement, rigidity, or poor balance, Becky was feeling none. In fact, the NADH, a dopamine-enhancing drug, seemed to make her feel even more skittery than normal. She was walking quite a bit to use up the extra energy. She made a change in her medication: 1 NADH and half a Sinemet in the morning, and a whole Sinemet in the evening. She usually needed a full Xanax now to feel any effect. When she started the Xanax at the end of June, half a pill had worked like a miracle.

Sept 26, 2000

I notice that even the half a Sinemet seems to set off the twitch. The twitch is violent in the mornings. None of the meds help much. Sinemet tames the twitch, but it seems to cause the twitch. This week's ear acupuncture at the clinic was unusually refreshing. [She had received a

[1] The Bad Old Days is her name for drug withdrawal.

treatment for drug withdrawal symptoms.] The twitch was temporarily subdued. But last night and today the violent right hand/left foot twitch (I will not call it a tremor) is back and very annoying. Just took the morning Sinemet but don't expect much. This has not been a wonderful week. Re-reading Brideshead Revisited helped. Evelyn Waugh is a great writer. Wish I could get rid of this new tendency to favor my abdominal muscles for some unknown reason. It affects my posture and walking.

At 2:00 a.m. last night the twitch was horrible, took the usual half Xanax and calcium at 12:30, but they didn't work so at 2:00 took another half.

In hindsight, the Sinemet was probably causing the abdominal spasming.

October 7, 2000

No sleep. Nothing worked, not Xanax, calcium, nor aspirin. Intense twitching, by night and by day.

On Oct 11, in clinic, she told us that since reducing her Sinemet to 1/2 a pill in the morning and a whole pill in the evening, she could stand up straight again, had no Breath Monster, no facial grimacing, and no foot spasm, but she was very grouchy. The acupuncturists at the clinic noticed that there was much less violence to her foot movements this week.

She was having fewer symptoms of overmedication with the seemingly small reduction of half a pill in the mornings. What we learned later was that this reduction of a "mere" half pill, because it was 25% of her total L-dopa amount, was probably more than her brain could handle, thus the extreme twitching and grouchiness. She was five weeks into this reduction – she was at the nadir.

On Oct 12, 2000, Becky read *Prozac Backlash* after a librarian recommended it. This book confirmed her suspicions that Xanax was an addictive drug. She resolved not to take it any more.

The following week at the clinic she reported that she felt more agitated, "yucky, with scattered thinking," and complained more about the twitch. However, our observations and hers agreed that the twitch was actually smaller in power and amplitude. However, she said that after quitting the Xanax, "every little thing is driving me nuts. The twitch is smaller, but it's bugging me more."

This was our first hint that the Xanax may have been contributing to the tremor/ticcing. At this point, I looked up Xanax in my *Physician's Drug Handbook* and found that tremor is listed as an adverse effect of Xanax.

Oct 25, 2000

I need Xanax now and then. If I don't take it every day, it works. Xanax is good for overcoming the twitching to get to sleep and stay asleep but its claims as an antidepressant are not true. I may be damaging my brain further by taking it, but I am 72 years old so who cares? Same with Sinemet; the past two days 1 pill in the afternoon perked me up. So I am a junkie, so is everybody else. We are a nation of junkies. No wonder the

Taliban declared a holy war on us! Wish I could invest big bucks in the pharmaceutical companies so I could at least profit from my own destruction. Well, enuf of this! Somebody cheer me up! I was shaking so bad and in the midst of a panic attack, I took a whole Sinemet instead of the half pills I have been taking.

Oct 26, 2000

No sleep in spite of Xanax, hot milk, and Calm R Rest (a herbal mix). It is now 5:45 a.m. and the twitch is violent. My abdominal muscles are absolutely rigid.

Oct 28, 2000

No Xanax for two days, and no morning Sinemet. Twitch has mainly disappeared but I have a spacey feeling. I am having very different can't-quite-describe-it feelings with no pills.

Oct 29, 2000

Took half a Sinemet in the morning and half in the afternoon. Twitch was fatiguing and violent all day. I've been edgy and impatient all week: apprehension, short attention span. I have to keep pacing or moving unless engaged in something like watching a movie.

At this point she was experimenting with decreasing her Sinemet to try and get rid of the twitch. She tried taking only half a pill in the mornings and a whole pill in the afternoon. She tried the reverse. She tried taking half a pill both morning and evening. Looking back over her notes, she was averaging 1.25 pills of Sinemet per day for the last two weeks.

Some days she felt horrible, hunched over from abdominal spasm and nonstop twitching, and other days she felt long periods of calm. However, the dosage didn't appear to have anything to do with that day's symptoms. Sometimes she was able to sleep, sometimes she wasn't. The Xanax was unreliable as a sleep aid. She felt certain that it amplified the twitch, because in the short term, when she stopped the Xanax, the twitch decreased. However, a few days after stopping the Xanax, the twitch was worse than ever.

The problem was, we had no idea about the delayed responses of the limbic area from Xanax. Xanax is considered to "block limbic arousal." Evidently, with the Xanax gone, her limbic zone was slowly becoming "aroused." The sleeping limbic dragon was waking. We were at that time familiar with the ten week cycle of Sinemet, but didn't realize that with Xanax as well, to best predict what sort of day she was going to have, she needed to be looking at her medication over the past 24 hours, past several days, past ten days, and past 70 days. Also, we hadn't yet figured out that the people who were decreasing without going through hell were limiting their decreases to 10% at a time. We weren't sure if Xanax, like Sinemet, might have a delayed period before the full extent of the decrease become apparent. We just didn't know.

Nov 4, 2000

Taking only 1 Sinemet a day for one week now. I now have a clear pattern of how I feel at this level. I take NADH first thing and then all the nutritional supplements and breakfast. I am uncomfortable and I twitch all morning, just not feeling well. I do not want to take on any new projects. At noon I eat lunch and take 1 Sinemet. This allows me to feel better and the twitch to diminish. It lasts until around 6:00 pm. Dinner with wine extends the comfortable period until around 8 or 9. Mild symptoms until I go to bed at 10 and go to sleep until around 1 or 2 when the night twitch pattern sets in.

Because she had reduced her Sinemet for a whole week, she is now beginning to feel the creeping dopamine deficiency. While this makes her feel lousy, it also allows the afternoon Sinemet to impart a good feeling rather than one of excess. Prior to this, the pills were increasing her twitches. Now that she is in withdrawal, the pills decrease the twitching.

Another way to look at it is this: at the previous, higher dosage level (two pills per day), the morning Sinemet was pushing her brain levels of DA up so high that her afternoon dose was in the excess zone. But this last week, because she was now re-addicted, her baseline level was low; without the drugs, she felt lousy. So now, when she took only an afternoon pill, it merely pushed her up into the effective zone, not the excess zone. Meanwhile, her overall symptoms of overmedication were decreased.

Nov 7, 2000

Got so mad at my clumsiness that I threw the roll of paper towels across the room. Good thing that nothing more breakable was handy! Since our favorite narcotic, Sinemet, gives a few hours of relief, would 2/day be a setback? Is this the junkie talking? I am sick of waking in the night. I took a Xanax, first one in weeks. I slept through until 7:00! I am feeling very down and cynical. Went to the deli for a sandwich and beer. Feel slightly better but not a lot.

Nov 27, 2000

Took an extra half a Sinemet today. Felt slightly better. Keeping to one pill a day is hard. Just reviewed entries since 11-22-00 and realized I have been taking an extra half a pill all week!

Dec 4, 2000

Took Xanax but was plagued by four trips to the toilet in the night. I can't seem to make active decisions. Feel somewhat better but kind of drained of energy. Did not take the half Sinemet in the morning though I feel less than wonderful. I don't feel like cooking.

Sinemet can cause urinary frequency. Xanax had listed adverse effects of slowed reflexes, depression, confusion, unsteady gait and impaired coordination.

Dec 12, 2000

Taking Xanax every night again for over a week, and back up to one and a half pills of Sinemet. I woke up at 1:30 and the twitch was so bad even after taking Xanax that I tossed about for another hour. Then I thought of taking more calcium tablets because I was afraid to take anything else. It worked! That, plus cuddling up with the cat. But I am very twitchy this morning. Violent twitch in the right hand and left foot all morning. I am so sick of this. Days are long.

Dec 13, 2000

I am tired of feeling like hell when I get up after another miserable night of twitching and urination (5 times!) things are not good. When I walk it is as though I am limping and my biceps spasm. I am uncomfortable sitting, standing, or lying. I try to keep active with long walks but it is a drag. I cannot reduce my medication.

Theoretically and ideally, according to Dr. Leslie, I would take 1 whole Sinemet in the a.m., another at 2 p.m. and another at 7 p.m, but past experience with larger doses were not pleasant. But maybe I have changed? Everything seems to aggravate the twitch: too much medicine or too little, too much food or too little, anticipation of any sort, any deviation in routine, etc, etc. Painter came to give me an estimate and my twitch went crazy! Am in denial about the meds and have to face the fact. I am addicted. Now what? Mornings are a dead loss because of the foggy-headedness [a side effect of Xanax]. THIS ISN'T FUNNY ANYMORE! On the other hand, it does have the advantage of making most of life unimportant. An attitude of "who cares?" is developing. This is not necessarily positive!

Went to the store to avoid going stir crazy. When I walk my upper arm muscles cramp up and I have to fight walking stooped (or at least it feels that way.) I took my Sinemet this afternoon and it makes me briefly less twitchy but more spacey. It keeps changing its effect over time. Now it takes 2 and a half hours for the Sinemet to kick in. It used to work in half an hour.

This delay in effectiveness is due to the combination of a dropping baseline and a rising threshold, two addiction-based brain changes. The threshold is rising and falling over twenty-four hours but also creeping up semipermanently over many weeks. The increasing night time urination can, of course, be written off to the normal weakened bladder that can come with aging, but urinary frequency is a listed adverse effect of Sinemet, and historically, bladder stimulation was one of the first known effects of blood-borne dopamine.

Jan 6, 2001

This morning I have the usual slowness, lethargy. One can argue that it is the Xanax but it is the easiest of the sleep aids. I am sick of

martyrdom. Xanax makes me feel bummed out. If I am addicted to drugs why can't they be the kind that give a HIGH! Rats! I feel spacey and wasted and lunch was of no avail.

Xanax has listed adverse effects of drowsiness, light-headedness, insomnia, and nervousness.[1]

Jan 12, 2001

You can take too much melatonin! I took it several times last night, instead of taking Xanax, and I woke up slow, shaky, and slightly nauseous, and visually bothered. Pill-wise, it looks like Xanax is my only option. My pulse feels like a newly landed fish. That can't be good. I hate this life focused on my physical self! If all this is teaching me some spiritual lesson, the point escapes me. Am going to the hypnotherapist this afternoon. Will be a totally new experience. Have no expectations.

Feb 2, 2001

No more Xanax. I am learning more about it – it is a dead end. The hypnotherapy tape helps somehow. I listen to it at three in the morning; laying on the living room floor the twitching can stop for up to thirty seconds.

Feb 12, 2001

Feeling rotten all week. Got up today feeling lousy. This continued through the day. Felt like my stomach was in a vacuum cleaner. Vision was like I was drunk. Twitch was violent and relentless. Felt as tho I had gotten much worse. The Monster is lurking. I seem to be retreating to the Bad Old Days. The idea of eating gags me. General sick feeling. Mental malaise. All this in spite of taking Sinemet. Sinemet doesn't seem to work lately. Usually it kicks in an hour or two after taking it, but today was just solid discomfort. What's next?

Normally very quick witted, she is not capable at this time of understanding that she might be having drug withdrawal symptoms from the Xanax. I pointed this out to her, and she just repeated to me that she couldn't understand why the Sinemet wasn't working.

Xanax, a serotonin-enhancer, eventually causes a backlash decrease in dopamine levels. The use of Xanax had evidently lowered her baseline enough so that her Sinemet was not able to push her up above the threshold; her drugs appeared to be doing nothing.

In the short term Xanax may boost dopamine by blocking limbic arousal (preventing dopamine from being dislodged from the limbic area, so that the dopamine can accumulate there). However, the brain responds quickly to any sort of excessive dopamine accumulation by instituting dopamine reduction. Hence, the longer-term effect,

[1] *Physician's Drug Handbook*, p. 23.

and the one seen in rats after Xanax experiments, is an overall decrease in brain dopamine levels.[1]

> *Feb 20, 2001*
>
> Isolated hours of sleep, but mostly jerking around with no relief. Nothing helps, food nor melatonin nor herb tea. Friends urge increasing meds no matter how addictive. Strongly tempted. I am too old for it to matter. Exhausted. Everything is getting worse no matter what I do. Having trouble breathing.

> *Feb 25, 2001*
>
> I am not twitching. I feel dull and wasted. Some choice! I feel so up and down about this whole thing. It is torture. Two nights of nearly normal sleep.

This was followed by a worsening of the insomnia – the night terrors appeared. The Bad Old Days were in full swing. They lasted for nearly three months. She was maintaining her Sinemet. The terrors this time were the result of quitting Xanax. She had been taking the smallest dose available of Xanax, .25 mg, and she sometimes only took a half of that pill – a mere .125 mg. However, three weeks after stopping Xanax cold turkey, the full battalion of withdrawal monsters attacked.

> *March 3, 2001*
>
> Arm is jerking violently right now. Nothing worked for helping sleep and if I dozed, when I woke up the shaking was as tho professional torturers were at work! Bad night ahead. I am scared to go to bed. It was demonic the way I was jerked into violent shakes as soon as I dozed off last night. This is definitely a repeat of the Bad Days in 1999. I took my Sinemet at 3:p.m. and it only lasted until 7:45 then things went from bad to worse. Listened to hypnotape but was jerked out of calm half way through tape by vicious shaking. The Sinemet is my only comfort. I feel very virtuous not taking more than prescribed. What would happen if I took Sinemet at bedtime? Would this screw up my daytime schedule? At least I might get some sleep.

> *March 5, 2001*
>
> I decided to take something – anything – to let me sleep. I took two Walgreen's sleeping pills[2] and actually slept. I am still afraid to go to bed. I tried sleeping on the floor like two years ago. I was in de-tox torture but it did not work. The Sinemet is no longer reliable. Muscle problems and breath problems. Breakfast gagged me.

---

[1] J. Glenmullen, MD, *Prozac Backlash: Overcoming the Dangers of Prozac, Zoloft, Paxil, and Other Antidepressants with Safe, Effective Alternatives*, Simon & Schuster, NY.

[2] Diphenyl hydramine (Benedryl).

March 8, 2001

Torture! Constant twitching. No sleep at all in spite of melatonin, aspirin, herb tea, rice cake, and finally, half a Xanax (sorry)! How long can this go on? To death? I am desperate. Without sleep, time sort of blends into one stream. Very confusing. I feel awful.

March 12, 2001

As the evening Sinemet wears off I have snakes crawling under my skin. I can't eat, sleep, or read. Lost 2 pounds. I am sick and miserable all day. The ear acupuncture is confusing; it calms my twitch but then I feel drained of any energy. I dread bedtime. The bedroom has become a torture chamber. Sleepless horror. My muscles are spasming.

On March 12 I shared our team's observation that patients seemed to do the best who never decreased a drug by more than 10% at a time, and that it appeared that there might be a ten day period before one could assess whether or not a drug change was going to bring about an effect. Her previous rates of decrease, 100% or nothing, had not been successful with any of the other pioneers. She decided that she would try a slower method of reducing Xanax. She decided to take half a Xanax, twice a week, and see if the drug withdrawal decreased in intensity. The first pill helped her sleep for a few hours.

March 16, 2001

I took a whole Xanax and got a good night's sleep. But the shaking goes on violently when I am awake and I continue to feel awful. My legs and arms are so tired. I think we are on to something with this half a pill twice a week.

March 19, 2001

Took Walgreen's[1] and got a decent sleep. It is stronger than the twitch. Felt draggy but better than with Xanax. Definite link between amounts of food and fatigue level.

March 22, 2001

Felt as near to normal as I have felt in a long time.

Violent twitch in right hand all day but I'm eating again – Gained 2 pounds! Still feel pretty good after a long day. Walked as much as possible all day. Things began to smooth towards evenings.

March 25, 2001

Two Walgreen's made me sleep "normally" but was twitchy and still am. Restless twitch during hypnotize so it was useless. I HATE MY LIFE AT THIS POINT! Unclear thinking, unclear vision, uncontrollable twitch…What is next? All I can do is primitive housework. It is God's punishment (Ha!) for being a slob all my life because I sneered at

[1] Diphenyl hydramine (Benedryl).

housework. Terrible headlines in the paper: people are damaging their livers with Tylenol! What will they discover about Sinemet?

April 2, 2001

Slept comparatively well with 2 Walgreen's and a cup of hot milk. Felt shaky in the morning but it passed. After Pilates I felt fairly decent. (Tho I still shake.) I don't have that faint nausea.

Two weeks later she had a panic attack, not unusual during drug regime changes, and two days after that she slept through the night with no sleep aids whatsoever. After that point she started noticing a slight improvement in the ratio of good nights to bad nights. She was taking Xanax twice a week.

Although the drug literature states confidently that benzodiazepines neutralize the effect of Sinemet, based on the old (and unproven) "serotonin is the opposite of dopamine" theory, Becky found that now, after a night with Xanax, her Sinemet worked better.

After three days without a Xanax, the effect of the Sinemet was lessened. When, once a week, she went four days without a Xanax, the Sinemet did not work at all: she would be in slow motion and exhausted all day. Despite the current drug theory, Xanax seemed to augment, not neutralize, Sinemet.

She was having vision problems during this time: objects would transform into fantasy objects now and then. On April 23 and 24 she slept well, two nights in a row! She had no more hellish nights until April 30. On April 30 she had all the old symptoms of Sinemet excess: breath monster, snakes under the skin.

She stopped the Xanax completely at this point since it seemed that the Sinemet was now excessive again. The Sinemet *excess* problems had been barely noticeable while she was going through withdrawal from Xanax. Now that she was sleeping better and the Xanax withdrawal was behind her, the Sinemet was once again working very consistently; in fact, she was starting to have signs of excess Sinemet. She went from .125 Xanax twice a week to none, and with the aid of various other sleeping aids, she was able to stay off the Xanax completely.

This transition from overmedicated to undermedicated during a withdrawal, and then a return to overmedication at the new, lowered drug rate, will be discussed fully in chapter 14, Cycles of Change. After reading that chapter, you may wish to reread this chapter on Becky and Xanax – it will make much more sense in retrospect.

Summary

Becky's drug experiences:

Spring, 1998: She started our program and immediately began making very small decreases in her Sinemet. She had mild symptoms of lethargy and fatigue that passed within a few weeks of each decrease.

First of October 1998: She was symptom free and taking no medication.

Late October 1998: She took Sinemet for one weekend; her son was visiting and she wanted optimal energy to flaunt her newly-returned health.

Late October 1998: She found she could not stop taking Sinemet.

Mid-November, 1998: She developed a ticcing pattern that is still present. She was unable to stop or decrease the Sinemet.

September 1999: She developed breathing distress. Her ticcing had become quite violent over the last year. She had tried many times to reduce her medication and failed. She had even caught herself sleepwalking to take Sinemet at night.

October 8, 1999: She abruptly stopped taking Sinemet.

October 16 to February 2000: She suffered severe withdrawal symptoms, including hallucinations, extreme insomnia, and violent ticcing.

February 2000: The ticcing made sleep difficult; she started over-the-counter[1] sleeping pills.

April 2000: She realized the sleeping pills were causing her foggy headedness and confusion. Her pharmacist confirmed that these pills did have those side effects, and that they were addictive.

May 2000: She abruptly stopped the sleeping pills. This aggravated her ticcing and the extreme insomnia returned.

Late June 2000: Her son was coming to visit. She was afraid for him to see her in her exhausted condition. Her twitching had calmed down and was once again a mild tic, larger than a tremor, but not violent. Her neurologist prescribed Xanax and Sinemet. For two weeks she slept well and she did not mind, or even notice, that her ticcing was increasing in power.

July 2000: The Xanax was no longer as reliable for inducing sleep. The ticcing was worsening. The breathing distress began to return.

September 2000: Ticcing worsened within half an hour of taking Sinemet. Severe insomnia had returned. She felt sluggish and mentally slow (side effects of Xanax). She tried to reduce Xanax intake and failed.

November 2000: She attempted to reduce Xanax and/or Sinemet. All her methods were too abrupt, and she could not sustain the reductions. She seesawed back and forth, sometimes changing her drug regimen on a daily basis in an attempt to find stability with the drugs.

March 2001: She began a slow method of reducing Xanax and started taking over-the counter sleeping pills. While reducing Xanax, Sinemet benefits diminished.

April 28, 2001: She felt that the worst of the Xanax withdrawal (dopamine deficiency) symptoms were behind her. She had two days of feeling good.

April 30, 2001: Symptoms of overmedication from the Sinemet began: dyskinesia, cramping, and breathing distress. She had not had these particular symptoms of overmedication since she began trying to reduce her medication in November, 2000.

Becky had spent nine months attempting various methods for reducing Xanax and had been in misery during most of that time. She had experienced classic symptoms of addiction with the Xanax: 1) she had needed to increase her dose to maintain effectiveness, and 2) she experienced physical, mental and emotional symptoms of drug withdrawal when she stopped taking it. (Dr. Leslie had been enraged when Becky had asked if Xanax was addictive.)

[1] Over-the-counter means non-prescription. These sleeping aids were supposedly mild.

Basically, in May 2000, to help her with her drug withdrawal from Benadryl, a mildly addictive, over-the-counter pill, her doctor had given her a highly addictive, serotonin-enhancing, dopamine-altering drug, in conjunction with Sinemet, a DED. This led to symptoms of overmedication within a few short weeks.

Following her decrease in Xanax, during the depths of the drug withdrawal (when her baseline was still very low), her Sinemet did not appear to be helping. As her baseline slowly crept back up after ten weeks of withdrawal symptoms, her Sinemet was able to now breach the effectiveness threshold, and she perceived benefits from the pills. As the baseline continued to rise, she began noticing symptoms of overmedication again, as her daily doses pushed her up into the excess zone.

Becky is a humble, wise soul. Her self-deprecating sense of humor and her determination to help others by objectively chronicling her experience carried her through. Becky was the only person in our program, at this point, who was able to survive more than two years of drug reduction trauma, and she did it while living alone. Some of our other patients who had recovered from Parkinson's and started showing severe symptoms of drug excess had been at this point in and out of care facilities or were dead.

By the beginning of May of 2001, Becky and all of the pioneers had learned a lot about the medication. She understood now that her Sinemet was causing her ticcing (twitching) and the snakes crawling under her skin, and that it was contributing to her insomnia. She understood that she would probably always have some form of ticcing – the semipermanent result of having ever, however briefly, taken a dopamine-enhancing drug when she no longer needed it. She started a slow, 10% program of reducing her Sinemet and the rest of the Xanax. After every 10% reduction, she felt on the verge of slipping into the hell of withdrawal, but instead only hovered on the edge. She fought various degrees of depression, sluggishness, confusion, physical discomfort, and insomnia, depending on where she was in the reduction cycle, plus the twitching and ticcing that may well follow her to her grave, medicated or not, but she never descended into the full demonic agonies again. Every time she began to feel the slightest bit normal, she made another 10% reduction in her drugs.

By February 2002, Becky had not taken any Xanax for over six months and had been taking only 50 mg of Sinemet *one day* a week – practically nothing – for several months. She was no longer having drug withdrawal symptoms. For nine long months in 2001, from April until December, while carefully but steadily reducing her medication no more than 10% at a time, she had been hovering near the edge of drug withdrawal.

In February she was haggard, but her voice was strong and her stride was long when she made the effort; she had been plagued by "baby steps" since the Xanax reduction. While baby steps still occurred in times of anxiety, she was usually able to prevent them by paying attention to her posture. She had no slowness of movement and no balance problems. However, she was hunched forward somewhat, and sometimes tilted to one side. She now used a walker "just in case."[1] She was gaunt and had no stamina. Some days she walked several blocks to the library, the deli, or the drugstore and back, but other days she napped and puttered in her apartment. El Twitcho still appeared for five to thirty minutes when she first woke from sleep, and shook her "like a

[11] A "walker" is a four-legged walking aid that is pushed in front of one to provide stability.

rat" at times of great anxiety, but most of the time her shaking was once again a mere tremor.

She had aged. A person meeting her for the first time would have admired her hearty laugh and zestful appetite for life, theater, good friends and good times, but would have guessed, not incorrectly, that she had suffered much. Her eyesight had deteriorated during the preceding two years; she had been diagnosed with partial retinal detachment. Though her eyes were still bright and laughing, a gaze deep into her eyes revealed the inner weariness borne of too much pain and fear. She was easily exhausted and badly emaciated. On days of extreme weakness, it was an effort just to make herself a meal. Sometimes it took her two or three tries to hoick herself up out of a chair. But she was drug free and feeling more in control of her life than she had been in three years.

Here are her chart notes from that time:

> Sleep is better, though still up every hour to pee after 2:30 in the morning. No more pacing the floor, though there is some twitching, tossing and turning in bed. There is no more of the violent shaking. It's gone. I only have the small tremor, and even that is much smaller than it was. It's the worst at meals – my food falls off my fork. My appetite is good but I can't gain weight.

She felt promising surges of strength. Her eye doctor suggested that her retinal problems might respond to something as simple as in increase in leafy green vegetables. In early February 2002, she was more optimistic than she had been in years. And then, in late February 2002, her stalwart troop of friends, concerned about her frailty, weight loss, and fatigue, issued an ultimatum.

Becky's story will continue in chapter 24.

"Sweets grown common lose their dear delight."

Shakespeare's Sonnet 102

9. ADDICTION

THE WHY, AND HOW, AND WHEN OF DRUG-WROUGHT CHANGE

Addiction is a many-faceted field of study, with aspects ranging all the way from social acceptability to faith and physiology. Many of these facets are significant in a study of Parkinson's disease and Parkinson's medications. This is going to be a long chapter, a sort of mini-book. Grab yourself a sandwich and a glass of juice and settle in. After we're done with this chapter things will move along much faster.

Let me tell you where we are going in this chapter: We'll look at the mechanics of brain change – why, where, and how the addicted brain performs in the presence of too much or too little dopamine. Then come more details on drug withdrawal symptoms. After a brief look at the long-held (thirty years) suspicion among most researchers that the drugs used to treat Parkinson's somehow accelerate the illness, we'll consider why so many PDers falsely consider themselves immune to addiction. Observations from doctors and nurses regarding dopamine-enhancing drugs, with examples from case studies, will be followed by suggestions on how to track down the new research on dopamine.

This may seem like a lot of bother just to make the point that dopamine-enhancing drugs are addictive, but the details hashed out here are the ones that come up the most often when I lecture and the ones that meet with the most emotional resistance. Also, much of this material is crucial to understanding the subsequent chapters on what may be for some people safe methods of drug reduction. A person who is not taking antiparkinson's drugs may be able to grasp this material quickly and will resent the redundancy. However, many of my overmedicated patients have heard the same material every week for months before they began to get a grip on what I was saying. On their behalf, please forgive my heavy-handedness.

Drug reduction vs. drug withdrawal

I must distinguish between the phrase "drug reduction" and "drug withdrawal." Drug reduction symptoms are the changes that might occur even if drugs are reduced slowly and safely. These symptoms may be painful and difficult, but they are not life threatening if the drug is reduced correctly. Drug "withdrawal" refers to the terrifying and life threatening symptoms that can occur when an addict stops or decreases drugs overly fast after the brain has become dependent. As you can imagine, the line between drug reduction and drug withdrawal is very fine, grey rather than black and white.

In our program we had some patients experiencing drug reductions and others with drug withdrawal. One criteria we used for determining which condition a patient might be in was whether or not a patient was able to maintain mental awareness of what was happening and why. Another was whether or not the symptoms were life threatening.

Those who were, at worst, stiff or immobile, depressed, restless, and manifesting severe, exaggerated Parkinson's disease were considered to be experiencing symptoms of drug reduction. Patients were considered to be undergoing drug withdrawal if they were hallucinating, manifesting extremely erratic mental behaviors, suffering from severe sensitivities to sound, light, taste or smell, or experiencing diaphragmatic spasms or such violent shakes that their ability to breathe was affected.

Why does the brain set into motion addiction?

As mentioned in chapter seven, any excess of dopamine in the limbic area might turn deadly. The brain has a set of safety mechanisms that are triggered when limbic area dopamine ever exceeds the safe amount. I call this safe amount the Safety Limit. Even if the Safety Limit is breached only briefly, both short- and long-term dopamine-reducing alterations (addiction changes) may be set in motion.

Duration of brain change

Some brain defenses against dopamine are short term: a few hours or a few days. The majority of brain defenses against excess dopamine last for approximately ten weeks. Sadly, it appears that some brain responses to excess dopamine are semipermanent.[1]

Short-term brain changes

For a quick example of short-term brain change causing addiction, let's remember the humble cigarette once again. The twelve-hour change due to stifling of dopamine receptors via nicotine was discussed in chapter seven. Nicotine, you recall, is a dopamine agonist (a molecule that mimics dopamine). But, although nicotine from cigs resides only briefly in the brain, we all know how long-term addictive these little gaspers can be.

Long-term effects

In addition to the short-term (twelve hour) reduction in nicotine effectiveness following each cigarette, other, more lasting changes are also triggered by the dopamine-enhancing effect of the nicotine. These changes decrease native dopamine function and make pleasant memory associations. These changes cause the long lasting, "semipermanent" condition known as addiction. In some people, only a few cigarettes are needed to set in motion these long-term brain changes.[2]

This short-term damage also occurs with many antiparkinson's drugs. The pill's lifespan in the brain is brief, but the damage builds invisibly. Once the "honeymoon

---

[1] Modern researchers use the word "semipermanent" because there is always the possibility, however distant, that a damaged brain cell might someday heal itself. This usage reflects our radical departure from 20th century thinking in which the brain and all nerve cells were static, incapable of change or healing. This 20th century "fact" was based on pure conjecture. Now we have strong evidence that nerve cells and brain neurons can heal and change. Modern science vocabulary prefers terms such as "semipermanent" to old terms like "permanent." This new, more vague terminology is also less susceptible to being disproven.

[2] *Tobacco Control*, British Medical Association journal, as written up in *Santa Cruz Sentinel*, Sept 12, 2000, p. A1. This article noted that some people manifest symptoms of addiction after just two or three cigarettes, but other people require more smoking before the addiction sets in. The information about short-term DA receptor inhibition in dopamine agonists was from an article on the website of the National Institute on Drug Abuse, search words: dopamine/nicotine.

period" (two to five years) of good drug effectiveness is over, and the dose increases have begun, many patients find that the pill effects are less reliable. One pill may work and another pill the same day may not work. Although people often want to ascribe obvious events such as meals, naps, and exercise to these periods of "effective pill" or "ineffective pill," possibly the single biggest factor in determining pill effectiveness is the reaction of the brain against pills taken earlier in the day. These short-term changes in pill effectiveness may result from short-term brain shut offs, similar to those seen with nicotine. Even ignoring those changes that occur to the dopamine baseline over time, these short-term shut offs may be associated with longer-term brain changes such as emotional and mental conditioning, as is the case with nicotine. More research is needed to discover the way in which short-term dopamine excess makes long-term/permanent changes.

The long-term changes that occur from antiparkinson's drugs are both the ten-week variety and the semipermanent. These changes can lead to decreased effectiveness of medication. These decreases in effectiveness are typically answered with an increase in medication.

Mechanics of brain change

When the brain detects the slightest excess of dopamine, meaning any dopamine that goes over the Safety Limit, it can set in motion a cascade of dopamine-decreasing procedures. It might cut and slash dopamine production and speed up dopamine breakdown. Dopamine-producing cells may be killed or altered to no longer produce dopamine. Dopamine receptors may be dismantled, with instructions never to rebuild. Dopamine transport molecules may be broken down. Dopamine storage sacs may be shuttered. Enzymes that break up dopamine or detach them from their receptor moorings may increase. Whole nerves may be deadened. Any of the elements involved in the dopamine-promotion chain might be shut down or decreased. Any of the processes that the brain uses to decrease dopamine might be increased.[1] A few of these changes will be short-term, but most will be semipermanent.[2]

---

[1] A new item regarding dopamine receptor shut down is just in from the drug company that makes a glial cell neurotrophic factor (GDNF). This new product, being promoted as an antiparkinson's drug, forces dopamine receptors to accept dopamine even when they've been ordered to shut down. The GDNF overrides the brain's defenses. "Despite the fact that there were brain cells in the putamen [a brain zone in the midbrain] that [should] react to dopamine, they [PDer brain cells] were not using the chemical. The new drug stimulated the cells to react to the dopamine present," according to the manufacturer.

This research confirms our suspicion that not all PD-like symptoms are due to mere dopamine insufficiency. The refusal of PDers' dopamine receptors to do their job even in the presence of dopamine suggests that the overly simplistic "dopamine deficiency" theory of PD is wrong.

There are other forces at work in Parkinson's disease, including the PD sympathetic nervous response and addiction, both of which can cause some degree of dopamine receptor shutdown as well as decreases in other parts of the dopamine story. These decreases in tangential aspects of the dopamine system play a large part in keeping dopamine from being used by PDers even when dopamine is present.

[2] Cocaine and Ecstasy, two dopamine-enhancing drugs, have been shown to provoke genetic mutations, causing permanent damage to DNA, the cellular instruction kit that tells cells how to behave. The longer the time frame of drug use, the greater the damage. This research was announced by Giorgio Bronzetti, head of the Italian National Center for Research's (CNR) biotechnology department, and reported on December 5, 2003, Reuters news service.

Why does the brain have so many mechanisms to decrease dopamine levels? As noted in a previous chapter, excess dopamine in the limbic area has the potential to lead quickly to death. Dopamine excess is the most dangerous NT condition that can exist in the brain. The brain takes any breach of the Safety Limit very seriously. From whatever source, too much dopamine is too much dopamine, and the brain will "take steps."

Following any of the various types of brain changes described above, which were set in motion due to a perceived excess of dopamine (usually due to a dopamine-enhancing drug), the brain's net effective dopamine levels will be significantly reduced. When the drug that caused the excess is finally flushed out of the brain, these anti-dopamine measures will still be in place. These dopamine-prevention measures will have decreased dopamine activity below the level needed for normal brain function.

Let me say this again: brain cell alterations, in response to dopamine exceeding the Safety Limit, however briefly, will cause less dopamine to be subsequently available. The *necessary* amounts of dopamine required for healthy brain function will *not* be available. The brain will now have a semipermanent dopamine shortage. This drastic, semi-permanent change can occur in response to as few as two or three cigarettes, one dose of cocaine, one pill of levodopa, or one pill of Mirapex.

Which drugs cause these changes

Many psychoactive drugs, including cocaine, opiates, nicotine, methamphetamines, alcohol, antidepressants and anti-anxiety drugs such as tricyclic antidepressants and SSRI's (selective serotonin reuptake inhibitors), and **all of the antiparkinson's disease drugs** except amantadine and the anticholinergics, can cause fleeting and/or chronically excessive levels of dopamine in the brain. Therefore, they can all cause addiction.

How

Each of the various drugs, legal or illegal, that elevate dopamine levels, has its specific mechanism, or pathway. This means that each drug increases dopamine in its own way. The mechanisms described below are also used by the antiparkinson's drugs; which ones use which is described in the next chapter.

Methamphetamine works by stimulating the dopamine storage sacs, opening them up and dumping their dopamine into the system. This increased level of dopamine activity surpasses the Safety Limit.

Cocaine works by inhibiting the reuptake enzyme (the molecule that detaches an active dopamine from its receptor and escorts the dopamine back to its storage sac for reuse). The net result of cocaine's reuptake inhibition is that dopamine gets to linger longer on its nerve receptor, other dopamine molecules are freed up to stimulate other nerves, and the net level of dopamine-triggered activity goes over the Safety Limit.

By the time the cocaine is cleared away by the brain's garbage trucks, the addiction process has already begun; after even one use of cocaine has worn off, a user may feel less perky than before he ever took the coke, and his brain may have made a positive memory, linking cocaine use with enhanced happiness.

Nicotine works by mimicking dopamine, wedging itself into dopamine receptor slots so that the real dopamine can hook up with yet more receptors. This means that the

net level of dopamine receptors being used goes up. The brain perceives too much dopamine activity, pushing the brain over the Safety Limit.

Where

The active sites of the assorted DEDs (dopamine-enhancing drugs) vary from drug to drug: for example, alcohol goes to areas that regulate thought (cortex), movement coordination (cerebellum) and emotion (amygdala), and ventral tegmentum. (Don't be worried about the fancy terminology; I am including some of the more technical terms in case you want to look these brain areas up in a physiology book, and to make the point that dopamine is used throughout the brain.) Cocaine and amphetamines go to the ventral tegmental area, as well as to the "pleasure circuit" (the sublenticular extended amygdala and nucleus accumbens). The opiates go to the same areas as cocaine, as well as the regions where the brain's natural opiates, like beta-endorphin, are found. Crack cocaine goes to still other areas: the anterior cingulate and the prefrontal cortex.[1]

Each of the anti-parkinson dopamine agonist drugs goes to a different combination of dopamine receptors (known as D1, D2, D3, etc. – each of the various types of dopamine receptors gets its own number), which is why each of the agonists has slightly different effects and side effects (described in the appendices). The anti-dopamine response that the brain will use to defend against dopamine excess depends on the brain area and nerve type that is targeted by the specific drugs.

Some dopamine-altering drugs do not directly connect to dopamine-making areas. For example, the norepinephrine- and serotonin-enhancing drugs, though sometimes used with a hope of increasing dopamine in the frontal lobe, appear to ultimately decrease dopamine as an unwanted side effect.[2]

When it comes to location, L-dopa is the least discriminating of all the DEDs: it bathes the entire brain in a wash of dopamine.

Most other DEDs affect certain parts of the brain more than others over the short term. But regardless of where the dopamine starts its excesses, if it exceeds the Safety Limit, addiction will begin.

How much

In some people, a few cigs can start addiction, but other people are somewhat resistant. Different people have different degrees of addictability.[3]

How much DED can a PDer take before addiction begins? A question raised by many PDers is, "If PD offers me some degree of immunity to addiction, how much of the antiparkinson's drugs can I take before I start risking addiction? I understand that I have no immunity after the moment that my brain turns off its "emergency" standing, but if I still have Parkinson's, how much drug can I take without triggering addiction?"

---

[1] S. Begley, "How It All Starts Inside Your Brain," *Newsweek*, Newsweek Inc, NY, Feb 12, 2001, p. 40.

[2] J. Glenmullen, MD, *Prozac Backlash: Overcoming the Dangers of Prozac, Zoloft, Paxil, and Other Antidepressants with Safe, Effective Alternatives*, Simon & Schuster, NY.

[3] Why some people are more addictable than others may actually have to do with another factor: the relative strength of their sympathetic nervous system influences compared to their parasympathetic. This idea will be more fully developed in chapter 24.

Based on the withdrawal patterns that we saw in medicated patients who were at various stages of recovery from Parkinson's, it seems that even if the brain's native dopamine tank is set on "empty," even if the Parkinson's is severely advanced, the maximum amount of dopamine that a person ever needs is about 400 mg/day of buffered levodopa. A person with early stage Parkinson's who is willing to wait three months for the drugs to take effect may get a good result with much less; one patient woman found that after three months she was getting an excellent response with 150 mg/day of levodopa – one fourth of the starting amount recommended by her doctor.[1]

If someone needs more than 400 mg/day, accumulated over several months, to obtain a result, the amount above 400 mg is probably needed due to addiction changes and not to advancing Parkinson's.

As you will read in chapter 17, in *all* patients, whether their PD symptoms were severe or not, and regardless of their dosage levels at the time they began to reduce, there were distinct differences in the drug withdrawal symptoms when any reductions, however small, were made from levels *above* 400 mg/day than when reductions were made from levels of 400 mg/day or *less*. It does appear that possibly, inadvertently, we may have discovered the Safety Limit.

People with advanced PD whose symptoms are not fully alleviated by this level (400 mg/day) of dopamine must bear in mind that many symptoms of Parkinson's, ranging from constipation to sebhhorhea, are *not* dopamine related. Increased amounts of L-dopa will *not* assist with those symptoms that are not dopamine-dependent. Also, levodopa does not actually address the condition that activates the tremor. In order for levodopa to suppress the conscious self-awareness of vulnerability that turns the tremor on, the drugs must necessarily be taken at a level that alters the mind. This level of medication is inherently over the Safety Limit.

For those patients who protest that their levodopa was not effective until they took more than 400 mg/day, remember: it can take several months for these drugs to become fully effective. The limbic system is slow to accumulate and dopamine is a threshold neurotransmitter. Until the amount breaches the threshold, no results will appear. The slightest titrations with dopamine-enhancing drugs must be evaluated over ten weeks, at a bare minimum. This book's subtitle, "Patient experiences…" is in testimony to the years, not weeks, during which PDers patiently observed their snail's-pace drug changes.

Of course, once addictions have occurred and the baseline has dropped accordingly, a PDer may need to use more than 400 mg/day simply to feed the addiction. The PDer should not imagine that it is advancing Parkinson's that requires a drug increase; when L-dopa was first discovered to help PDers, people with advanced Parkinson's as well as those recently diagnosed nearly all received benefits from the drug at doses comparable to 300 mg/day of carbidopa/levodopa. Today, some doctors begin dosing at 800 mg/day even for the mildest cases.

[1] Actually, she appeared to have symptoms of very slight overmedication after four months – spastic toe curling and overly bright eyes. However, she was adamant that these symptoms were just signs of how "happy" she was.

After addiction has begun: The grim cycle

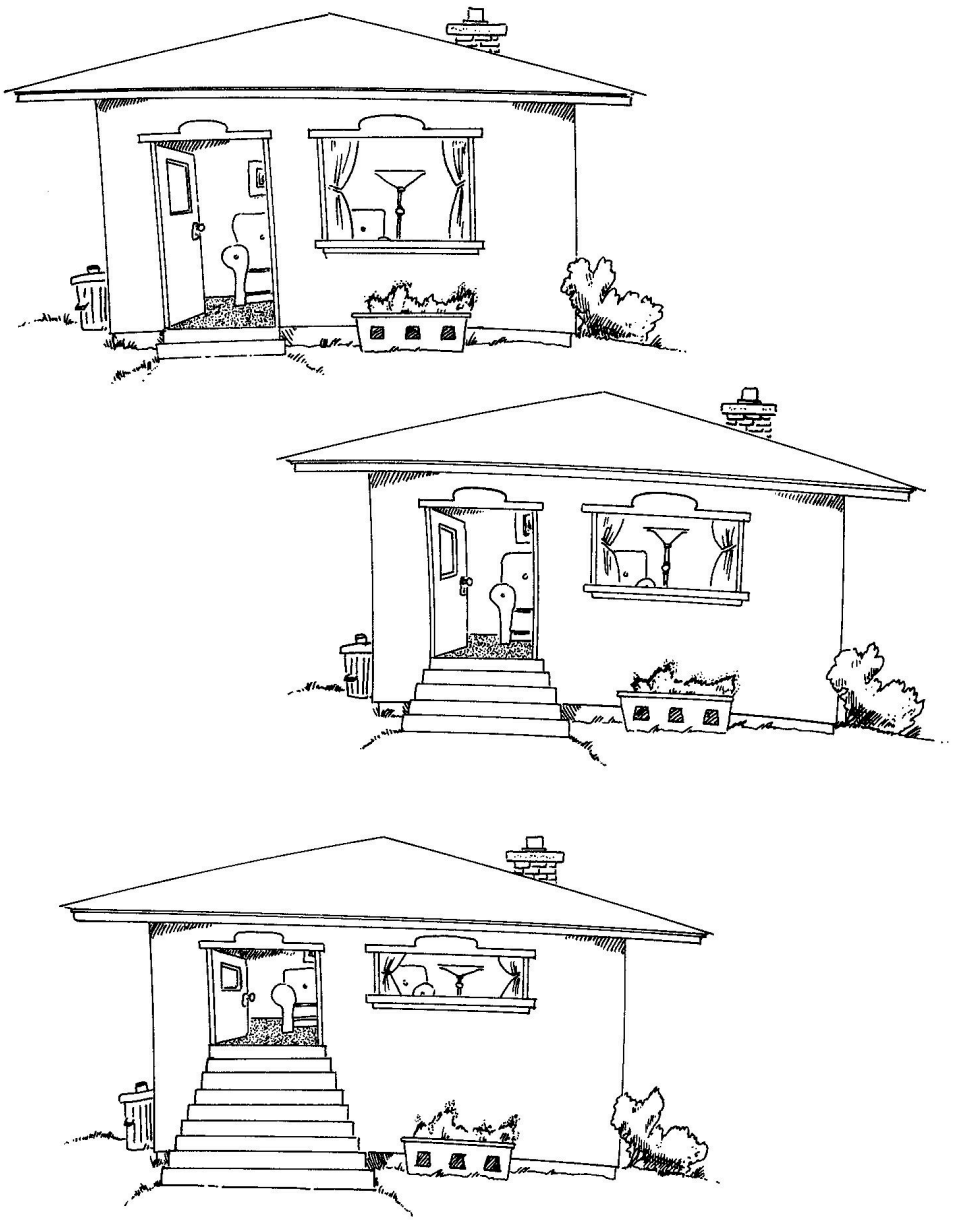
Because of brain alterations from external (drug) sources of dopamine, the brain becomes dependent on outside sources of dopamine. Because the outside sources cannot possibly provide the *exact* amount of dopamine that is actually needed, there is invariably a slight excess of dopamine when an outside source, such as opium, nicotine, or the antiparkinson's drugs, is applied. The brain responds to the excess by reducing some part of the dopamine system.

During a subsequent dose the brain then notices that, once again, despite its best efforts, there has been another incident of excess dopamine. So it spits on its hands, hauls up its slacks, and sets in motion more alterations to further decrease dopamine activity in the brain. When the drug wears off, the lowered dopamine activity feels distinctly unpleasant, or worse. So, of course, the brain needs even *more* outside sources of dopamine. In response to inevitable excesses caused by ever-increasing quantities of dopamine, the brain alters still further. The brain builds barriers against future dopamine excess. It constructs figurative moats and tall walls against the onslaught of future dopamine. Internal sources of dopamine are slashed. This means that the brain is even more reliant on external sources.

External sources of DEDs must be supplied in ever-higher quantities to jump the moats and breach the barrier walls. These higher quantities cannot hope to supply the exact, precise amounts of dopamine that will allow mere functionality – they unintentionally exceed the Safety Limit again. The defensive walls go higher still. And so on.

No drug is subtle enough to provide exactly enough dopamine. Because dopamine levels must cross over a threshold to be effective, too little dopamine does no good at all. The threshold must be attained or no business results. In an attempt to attain the threshold, drugs are invariably taken in a manner that exceeds the Safety Limit. With every application of external dopamine enhancers, the addiction process increases.

The addiction process makes it more difficult to attain the threshold. As the addiction increases, the baseline drops. More external dopamine than before is needed to lunge up above the threshold. And yet, with all these changes, the upper limit for safe dopamine activity, the Safety Limit, remains the same: a hair's breadth above the original baseline.

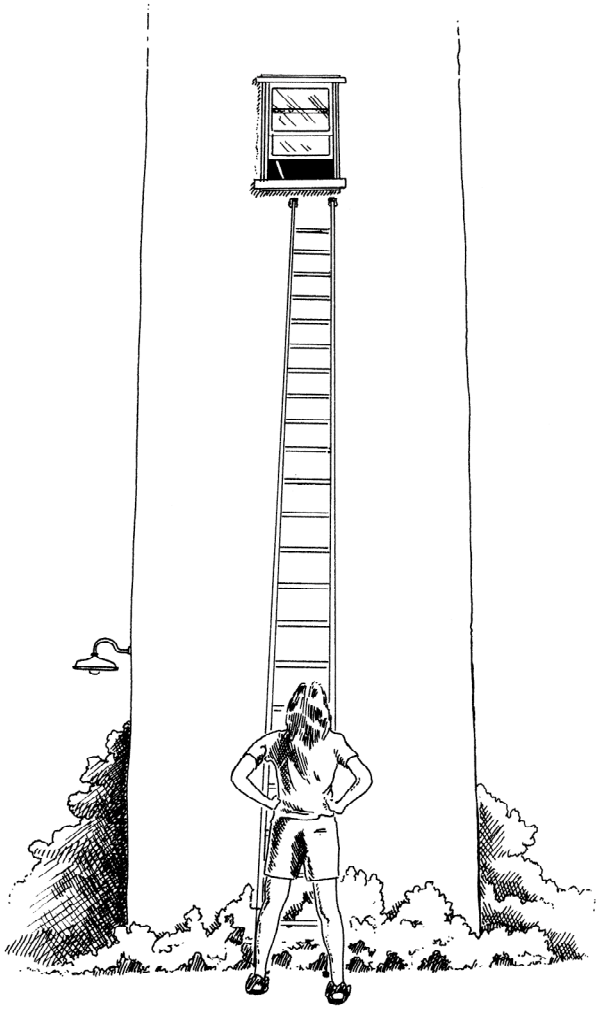


As the threshold rises relative to the baseline, it takes more steps (units of dopamine) to gain access to the door. In the pictures above, the lawn is the baseline and the top of the trashcan is the Safety Limit. As the threshold rises, all entries are at a level higher than the limit.

Ever-smaller windows

In much Parkinson's literature, the dosage amount that falls between the threshold and the onset of undesired side effects is often called a "window."[1]

As the addiction level goes up, the threshold rises. It requires increasing amounts of external dopamine to get a result. However, the level at which side effects commence remains the same. With the threshold rising and the ceiling (Safety Limit) staying in the same place, the "window" in which drugs are effective without being dangerous or having side effects grows ever smaller.



An ever-smaller window

[1] Please note, the distinction between threshold (short term change) and baseline (long term change) is ignored in this usage. In this case, "threshold" simply means "enough to get a response."

It would have been nice if the PD literature had used a new term such as "doorway" instead of "window." The "threshold" terminology is already common in biology, and means "amount needed to get a response." However, in biology, the term "window" typically refers to a time frame, as opposed to the meaning in Parkinson's literature, in which it signifies an amount. However, to honor this difference in linguistics, I have included artwork showing the situation both as a doorway/threshold (previous page) and again on this page as a window.

DRUG WITHDRAWAL

Symptom Review

1. When dopamine in the limbic area is drastically decreased, it may cause fear, rage, shaking, tremor, nausea; oversensitivity to temperature, sound, light, touch, smell, and taste; tightening of the muscles (as if hypothermic [overly cold]), and slowness of movement; insomnia, paranoia, and inability to think reflectively.

2. Deficient dopamine in the frontal lobe can cause deficiency in the neurotransmitters of the frontal lobe (NE and 5-HT), leading to depression, moodiness, inability to think clearly, and lack of focus and will.

3. A shortage of dopamine in the motor area prevents or slows movement, reflexes, balance function, and integrated thinking.

Confusing symptoms: drug reduction can look like PD

A common question that our patients have is, "Am I going through drug withdrawal, or is this the true face of my Parkinson's?"

Tremoring, slowness of movement or speech, anxiety or depression are all symptoms that might occur in PD due to a deficiency of dopamine. They also are symptoms that might occur in response to abuse of and withdrawal from certain drugs.[1] Does this mean that some of the dopamine-related symptoms of Parkinson's disease are similar to the symptoms of drug-withdrawal from illegal drugs?[2] Yes. When an addict's brain is in a sub-dopamine condition, an addict's body can develop symptoms that mimic parts of Parkinson's disease.

Since antiparkinson's disease medications can cross too far above the dopamine Safety Line, thus initiating addiction, the drugs that your doctor gives you can produce addiction symptoms and, when decreased, withdrawal symptoms. The best way to know if a non life-threatening symptom is from drug reduction, withdrawal, or PD might be to wait three months before making any further changes in drug regimen. Drug reduction and withdrawal symptoms will begin to ease up after three months or so; Parkinson's symptoms will not.

Limbic stress can lead to adrenaline

When an addicted person decreases or stops his drugs, he may have symptoms of limbic dopamine deficiency. Even if many brain areas are deficient in dopamine, the leader of the pack is always the limbic. In fact, limbic insufficiency can lead to some release of adrenaline. This may lead to motor area function via adrenaline even though overall dopamine levels are low. Therefore, symptoms of limbic insufficiency may be apparent even though motor function appears only somewhat impaired.

---

[1] Only the more powerful DEDs and serotonin enhancers can create symptoms of parkinsonism. The milder drugs of abuse, such as nicotine and alcohol, are not strong enough to trigger outright parkinsonism, although they can do significant damage to the dopamine system.

[2] Don't forget, there are many symptoms of PD that are independent of dopamine levels. But there are also dopamine-related symptoms as well. These symptoms are the ones that compare to drug addiction. The other, non-dopamine-related symptoms, such as the elevated adrenaline and muscle degeneration, are what make PD look characteristically different from drug addiction.

ANTIPARKINSON'S DRUGS CAUSE PARKINSONISM

Now we come to one of the most controversial points of this chapter. Although most of the older clinical neurologists cannot fathom this thought, and most patients are outraged when they find out, the fact remains: the drugs used in the treatment of Parkinson's disease can cause the incurable condition of drug-induced parkinsonism.[1] This fact is supported by research by the National Institute on Drug Abuse that explains the role of dopamine in addiction, and we have seen it in our research in patients who seem to have recovered from Parkinson's but have developed drug dependence. Again, because antiparkinson's drugs elevate dopamine, they have the potential to be addictive, to cause semipermanent brain damage, and to set in motion an illness that is currently incurable: drug-induced parkinsonism.

The mysterious increase in parkinsonism

An MD told me that twenty years ago, when visiting the VA (Veteran's Administration) hospitals, there would be usually about two or three men in a hundred who were tremoring or rigid with Parkinson's disease. Now when he goes to visit, nearly a third of the patients appear to have finger tapping and other symptoms that resemble Parkinson's disease and are being treated with antiparkinson's medications. Upon speaking with these patients, he found most have used antianxiety or antidepressant medications prior to their PD diagnosis. He feels strongly that these men are exhibiting signs of drug-induced parkinsonism rather than idiopathic PD.

Drug holidays

In the 1970's, it was recognized that the antiparkinson's drugs were addictive; when PDers no longer responded to their drugs at *starter dose*, or if their increased doses produced symptoms of overmedication, they were admitted to hospital and abruptly taken off their drugs.

After a painful period of drug-withdrawal, during which they were carefully monitored, they were once again able to respond well to their drugs at their starter doses. Unfortunately, as MDs became more cavalier in their prescribing and accustomed to the bizarre symptoms of dyskinesia, they allowed the drug levels to get much higher before suggesting a drug holiday. Then, abruptly stopping the drugs from these new, higher dosage levels often proved fatal.

Not understanding that it was their own prescribing patterns that had changed the relatively safe "drug holiday" into a lethal dopamine free-fall, some doctors in the early

---

[1] "Another unanswered question with levodopa therapy is whether or not this drug adversely accelerates the course of parkinsonism…There is continuing concern that ameliorating symptoms may be aggravating the disease. Thus, initiation of levodopa therapy is often delayed until the symptoms…actually cause an unacceptable degree of functional impairment. It leaves the clinician in a quandary about how to treat early stages of Parkinson's." *Primer of Drug Action*, Dr. R. Julien, p. 362.

Frankly, dear reader, I have not met many clinicians who are in a quandary about how to treat early Parkinson's. Most of the ones in my experience have blithely prescribed levodopa or agonists with a breezy nonchalance at the first hint of Parkinson's disease.

1980's announced that it was evidently unsafe to *ever* decrease any antiparkinson's drug by any amount.

Now, in the twenty-first century, even though the role of dopamine is better understood, and the value of a drug holiday, as soon as the lower doses become ineffective, is once again being proposed by bold researchers, insurance programs will not support this expensive hospitalized "drug holiday." Instead, despite the fact that drug holidays proved that the need for ever-increasing dopamine was due to addiction, most young MDs assume that decreased drug effectiveness and even symptoms of overmedication are simply signs of advancing Parkinson's disease.

Careless prescribing of addictive drugs

What is the consequence of the drugs in a person who has drug-induced parkinsonism? In a PDer, as we have seen, the drugs may be only mildly addictive. In a case of drug-induced parkinsonism, if there is no underlying resistance to addiction, the drugs can rapidly accelerate the progression of parkinsonism.

Although good solid research has pointed to a relationship between antianxiety and antidepressant medications and the subsequent onset of the tardive dyskinesias and bradykinesias that resemble Parkinson's disease, most doctors give out these drugs as quickly as their patients ask for them.[1]

In the year 2000, a Harvard MD wrote an excellent book, lavishly supported with evidence, about the dangers of permanent brain alteration from use of antianxiety and antidepressant drugs.[2] A year later, following the bombing of the World Trade Center in New York City, pharmacists reported that sales of Xanax, a popular and highly addictive[3] antianxiety drug, were skyrocketing.

In light of the dangers of mind-altering drugs, maybe doctors should be willing to prescribe a glass of wine at dinner or taking a day off of work when people report in with high stress levels. But the national mood seems to be "Treat my anxiety now, I'll worry about the parkinsonism later!" Living for the moment is the popular way, and the doctors and drug companies merely provide that which the public demands.

The Mandate of Heaven

There is an old Confucian maxim named The Mandate of Heaven that describes this law of supply and demand: "All institutions of man exist by the will of the people." In the context of doctors providing drugs rather than addressing root causes, this mandate suggests that the doctors are merely following the desires of the people. If people didn't demand instant relief from their tensions and worries, the doctors would not be prescribing fast-working (though addictive) medications, and the pharmaceutical companies would not have a market. So let us not blame the doctors or the inventors of

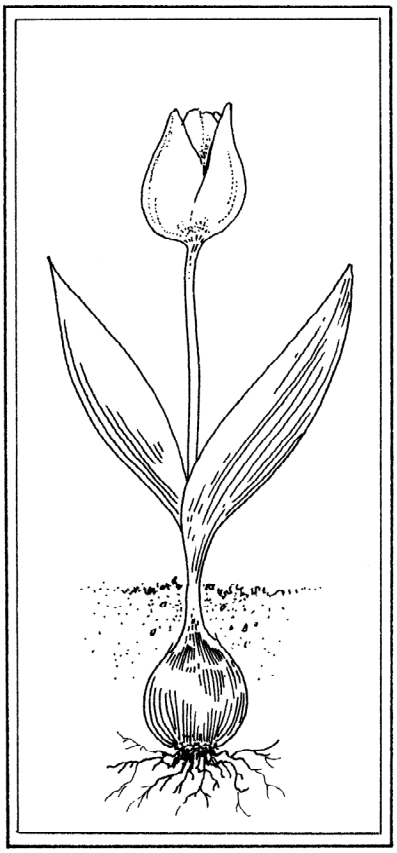
---

[1] In the late 1980's, tardive dyskinesia was deemed a response of hypersensitive dopamine receptors. This idea was in reaction to these tremors appearing in non-Parkinson's subjects subsequent to L-dopa use. This was prior to the discovery that dopamine was associated with cessation of fear and the brain changes of addiction. I am not aware what current idea is being promoted to explain tardive dyskinesia. I suspect it varies from one researcher to another and from one research sponsor to another.

[2] Dr. Glenmullen, *Prozac Backlash*.

[3] As noted in the previous chapter, although Xanax is promoted as being non addictive, I have worked with patients who are going through horrible months of agonies of withdrawal from this drug.

these drugs. Let's look at the root cause: if these dangerous drugs are being overused, it is because people are demanding quick benefits and not probing deeply into the true, fleeting nature of any "quick fix." Maybe some people in our drug-popping culture do not want to look too deeply into the roots of their problems.



CHANGES IN ADDICTABILITY

It is hard for the novice to brain science to understand how a person can be impervious to addiction, or how addictability in a given person is variable. A study on primates[1] (as noted in chapter one) proved that susceptibility to addiction was altered if social status was altered. It also showed that the chemical basis for the change in susceptibility in this case might have been due to change in dopamine receptor activity. (More about this in Appendix 7.)

Based on this study, and certainly based on everything we have seen with our recovering PD patients, it does seem that susceptibility to addiction varies from person to person. It even appears that PDers are, strangely enough, fairly impervious to addiction. *But*, the moment the brain begins to make dopamine, that immunity is lost. *Some patients who have remained at their accustomed levels of medication for more than 72 hours after those changes begin which signal brain recovery have suffered terribly from their medication*. But even stopping drugs completely prior to starting a recovery program cannot undo drug-induced brain damage; even if one got off medication promptly before starting he will probably never be completely healthy. He may always have symptoms of drug-induced dopamine deficiency. If one begins to reduce medication promptly after signs of recovery from PD have begun, *he will probably never again be completely normal; there may always be lingering signs of addiction, including life-long tremor*.

Patients who were already addicted to their medication even before starting the recovery program have *not* become completely normal after recovering from Parkinson's. They maintain a perpetual weariness, tendency to the hangdog absence of joy, complete with sadness deep in the eyes, similar to that seen in some recovered addicts of other drugs. Some have perpetual tremor, even though the other PD symptoms are gone. Many have found that, although they are no longer rigid, they are extremely weak to the point where they cannot use their limbs: arms that were once bent rigid at the elbow may dangle uselessly at one's side.

Some of these patients have found that they may always need just a hint of anti-PD medication to stay comfortable. A few people who have recovered who are in this group find that they can maintain healthy levels of mood and activity with doses as low as 25 mg/day of L-dopa or .25 mg of Mirapex. Compare these low numbers to the minimum effective dose of 300 mg/day of L-dopa or the 3 to 4.5 mg of Mirapex that is written up by the drug manufacturers.

All MDs in our experience insist that at levels as low as 25 mg/day of L-dopa, there is no effect whatsoever. However, as you will read in chapters ahead, a person who has been brain damaged (addicted) from L-dopa may never be able to come off the medication completely, always needing a few grains a day. These few milligrams are not to combat the Parkinson's – they are to counter the permanent decrease in dopamine caused by their inadvertent addiction.

[1] *Nature Neuroscience* 5(2): 169-174, 2002.

Am I addicted to my anti-PD meds?

Dyskinesias, facial grimacing and On/Off behaviors are all extreme symptoms of the brain acting out due to overmedication. These are visible signs of addiction. The invisible brain changes occur long before the overt ones show up. Any form of dyskinesia indicates that the brain is far over the Safety Limit. If the brain has been over the Safety Limit, it is safe to assume that changes will have been made in the brain and that dopamine levels may never again be normal, or even adequate.

This brain condition of semipermanent dopamine reduction (addiction), co-existing in the brains of PDers along with their PD condition of dopamine dormancy, means that PDers with drug addiction are actually combating two different illnesses at once – and they are both dopamine-related illnesses. The Parkinson's disease is, apparently, curable. The addiction is semipermanent: it may improve, but then again, it may not.[1]

Some patients in our program who were not yet having problems with their medication assumed that they were not addicted. They also assumed, incorrectly, that they could begin to recover from Parkinson's and then reduce their medication appropriately, by reducing a bit at a time, in response to any symptoms of overmedication that might arise. These people invariably became addicted. Once addicted, any further attempts at reduction set in motion those symptoms described in chapter seven for dopamine deficiency in the limbic part of the brain (drug withdrawal).

From the moment the brain switches over from the PD electrical pattern to the healthy pattern, even if their arms and legs are still ravaged by the decades of PD, even if the body has yet to relearn how to move using dopamine instead of adrenaline, even if dopamine levels are still substandard, it does appear that the moment that the brain resumes *capability* of making dopamine, the danger of addiction begins.[2]

People with Parkinson's aren't easily addicted

After all these pages of negativity, here's the good news: people with Parkinson's are not as easily addicted to dopamine-enhancing drugs as long as they still have Parkinson's disease.

A history of insufficient dopamine

Many PDers have noticed that, throughout their life, they have been able to stop using addictive substances without suffering from standard drug-withdrawal symptoms. These people usually attribute their lack of withdrawal agony to their superior will power. They are incorrect. The reason that this group of people can abruptly and even painlessly stop taking addictive drugs is – *they are not addicted*. Their dopamine system, even before the obvious symptoms of PD showed up, has for years been set at "Low." Their use of cigarettes, alcohol, or other drugs may have never even pushed their inadequate dopamine levels above the Safety Line. Even if they did go over the Safety Line, the

---

[1] Who knows, maybe someday cell death will be curable. At this point in time, it is not. But, "never say never," as the saying goes. Let us hope that even drug-induced cell death may some day be treatable.

[2] The physiological differences between patients who became rapidly (within 72 hours) addicted to their PD meds and the ones that seemed to have more time will be discussed in chapter 29.

addiction process, a subset of the dopamine system, was always functioning in a diminished capacity.

A history of reliance on the sympathetic nervous system

Another reason that they may not be addicted is that the entire addiction process may be a part of the parasympathetic nervous system. PDers don't use this dopamine-based system very much. They tend, since childhood, to have used the adrenaline-based, sympathetic system instead. This hypothesis will be further developed in chapter 24.

PDers aren't addicted to wimpy drugs

There will be case studies later in the book to support this statement, but for now, take it as read that, while PDers can become *habituated* to using a drug, they might not become technically addicted – at least not to the wimpier drugs such as cocaine, heroin, alcohol and cigarettes. The anti-PD drugs, however, all much stronger than these puny drugs, can force even a PDer's brain over the top.

PDers not being readily addictable is so important that I am going to repeat it: in the decades before their PD became apparent, their decreasing dopamine levels were supplemented by adrenaline, so that they could keep moving and performing even without a full dopamine compliment. Their declining dopamine levels were masked by the compensations of adrenaline.[1]

Decades of low dopamine

Their decades of lowered dopamine levels and their use of adrenaline instead of dopamine may be what prevent PDers from becoming easily addicted to cigarettes, alcohol, cocaine, or low levels of their antiparkinson's drugs. Even in the presence of these powerful dopamine enhancers, their DA levels rarely cross over the Safety Limit of their slumbering dopamine system.

---

[1] To be technically correct, it works the other way: in the presence of adrenaline, their dopamine levels were suppressed. After long-term suppression, dopamine-producing structures are dormant or dismantled. It is when the adrenaline system eventually is somewhat depleted (worn down from a lifetime of overuse) that the dopamine suppression and dismantling becomes apparent. But for the purposes of this discussion, because most PDers are fixated on the dopamine as being causative, we can speak as though the dopamine is the driving force. Actually, the difference is significant, but this book would be too long if all the technicalities were included. But this thought will be developed more, later in the book: it is not only the lowered dopamine levels with regard to the Safety Limit that keep the PDer immune to addiction, it is the entire package of dopamine suppression and the anti-addiction effects of the aberrant electrical system and long-standing injury. Similar anti-addiction properties are found in people who are in extreme pain – it has long been noted that such people can take opiates at levels that would knock out a moose without ever becoming addicted. There will be more about this in chapter 24.

"Just a question of mind over matter!"[1]

PDers regularly brag to me that they have stronger will power than normal people and give as an example that they stopped smoking cigarettes or using some addictive drug without so much as a sigh of regret. They kindly explain it for me by saying, "Just a question of mind over matter!" They are wrong. They could quit easily because they were never addicted. They were never addicted because their dopamine levels were never pushed above the Safety Limit. Very likely, if our hypotheses about parasympathetic/sympathetic responses to addiction are correct, their addiction system, in addition to their dopamine production, wasn't ever working at normal strength!

[1] Despite accusations from my cousin Michael that this drawing is of him, it is in fact a self-portrait of John Bateson, the illustrator, who sports a red mustache in the winter.

Recovery from PD = Goodbye, will power

When a person recovers from PD, he will no longer have his strong, adrenaline-based will power. When the adrenaline is turned off, the PDer is no longer driven with the stoic will power and intensity of purpose that may have characterized his life during his PD-developing years.

This drop-off in adrenaline often allows the recovering PDer to feel more deeply relaxed than he has since childhood. He may once again enjoy leisurely brain integration processes, rather than forcefully blasting his thoughts through to their quickest possible conclusion. He may, for the first time in his life, become able to ponder and muse. He may also, in this new condition, find that he has very little will power, even less than the average person.

Frontal lobe adrenaline

Many PDers assume that their lifelong ability to focus intently proves that they use their frontal lobe more than their limbic centers. They often point with pride to their vaunted ability to override their emotions via their will power. They are forgetting a crucial factor: they were using adrenaline in the frontal lobe, and not dopamine. Their brain may have very few connections for dopamine in the frontal lobe. If their reliance on adrenaline was fear based – which it usually is – they may not have been using the dopamine connections in their frontal lobe at all.

A PDer's minimal dopamine connections may only be those of the limbic area, maintaining basic systems of life support. When a person with PD recovers, he is often stunned by how his thought processes change. He may feel like a different person. His stoicism and focus may disappear.

It also appears as if the dopamine increase that occurs during recovery may go toward the limbic area rather than the frontal lobe. Dopamine in the frontal lobe is more related to love and harmony than it is to fear, ego, and survival. A recovering PDer may approve these qualities, but might not have developed a physiological basis for them – his frontal lobe activity may change from excessive (with adrenaline) to deficient. Because addiction is related not only to dopamine levels, but also to the *location* of dopamine levels, a person who is recovering from Parkinson's may be at higher risk than the general public for addiction.

Some recovering PDers, previously strong and self-confident of their ability to "take the drugs or leave them alone," have become some of the most tragically, heartbreakingly addicted people I have ever worked with.

It appears that their terrific addiction occurs if they fail to reduce their Parkinson's disease drugs in a timely fashion upon recovering from PD. "Timely," as you will learn, may be a matter of days for limbic system excess – and yet the reviving body may take months or years to restore itself to motor function. In the absence of motor function, most people are reluctant to reduce their PD medication, especially since it causes the unpleasant effects of drug reduction. Instead, they err on the side of taking their drugs for too long, with sometimes irreversible results. You can begin to see why we do not suggest a recovery program for anyone who is taking antiparkinson's medications.

I'm different

Foolhardy PDers often insist that, despite the experiences of other PDers, they are different. This supposed difference is often associated with their conviction that they are morally or spiritually superior.[1] They feel that, unlike others, they will still be able to use their will power and logic in the face of the terrors of addiction and drug withdrawal. What they are forgetting is that frontal lobe functions, for example, will power, play second fiddle to the limbic system. If the brain goes into a limbic-area crisis such as the crisis of drug withdrawal, a person who previously had terrific will power will not have physiological access to either his logic or his focus. When it comes to regulating behaviors, the limbic area always beats out the frontal lobe. In a limbic level crisis, the raw emotions always take precedence over reason.

Drug-induced lack of logic: poor self-assessment

A person under the influence of dopamine-enhancing drugs, even if he needs these drugs to help with his Parkinson's disease, is somewhat "stoned," or mind-altered. He is not capable of making good decisions or assessing his own condition. How many times have I been told by a person who is twitching, grimacing, and jerking, "I'm not overmedicated. I am not moving strangely. I'm sure I would be able to tell if I was…"

A PDer who wants honest, clear-sighted guidance in assessing his drug needs must be willing and able to work closely (daily) with a person who understands the dynamics of the drugs. Accurate analysis of medication levels requires daily, sometimes hourly, observation. Your MD cannot and should not be expected to provide this.

Squandering dopamine resistance for an On!

PDers who use their drugs to create a feeling of euphoria or even a condition of radiant health (On! time) are probably doing themselves a mischief. Though they are somewhat resistant to the addiction process by virtue of having very low dopamine levels and being in sympathetic nervous system mode, even PDers can abuse this gift of non-addictability and give themselves drug-induced parkinsonism by ever having their dopamine levels too high. Being On! is a mental form of dyskinesia and is therefore a sign of overmedication and addiction.

Help during drug withdrawal: higher powers

When the limbic area is deficient in dopamine and begins to exhibit its symptoms, the brain reverts to its most primitive behaviors. Sophisticated reasoning and will power are not available when the limbic region starts throwing its weight about. Therefore, a person going through dopamine withdrawal will not have access to his reason or his will. He may, however, still have access to his soul.

It is well known in addiction-treatment programs that those people best able to overcome their addictions are those who do not rely on their own reason or will power

---

[1] The tremendous ability to focus and process thoughts and actions that can be learned during a lifetime of adrenaline is often praised by society in general. Many PDers mistakenly attribute these "superior" qualities to spiritual advancement. In fact, the signs of spiritual depth – humility, acceptance of others, inner stillness, and unconditional love – are qualities that are very often lacking in PDers. As for the quickness of thought that PDers often mistake for wisdom, Mother Teresa once said, "No one ever got into heaven by being clever."

but instead put their life under the guidance and protection of a higher power, typically, a spiritual guide or religion. There is good sense to this – not only are will power and logic not accessible to a person whose limbic system is acting out, but research has shown that meditation and focused prayer cause the two brain sides to work more closely in synch, producing a calming effect and increasing dopamine production.[1]

As has already been noted, it appears to be *limbic* dopamine excess that is particularly dangerous. Focused meditation and directed prayer employ the frontal lobe. Meditation and/or perfect love balances the two brain sides and thereby stimulates the midbrain, increasing dopamine. When increased dopamine is internally produced and sent to the frontal lobe, as occurs during disciplined devotional exercises, a level of maximum beneficial dopamine is created – a level that cannot exceed the Safety Limit.

Whether or not dopamine is the neurotransmitter that is the actual, physical conversion point where the vibrations of life force or the bliss of Spirit are converted into a physical form is beyond the scope of this book, but it is possible that Parkinson's disease and drug addiction may someday yield clues about this hypothesis.

Considering this debate very briefly, unmedicated PDers, or those PDers whose drugs are not working well, often say that they feel unnaturally heavy inside, as if they are sinking into the earth. Their inability to move feels at times as if their body has grown dense, as if it cannot resist the gravity of the earth. Some recreational drug users who experiment with L-dopa have described the drugged sensation as being lighter than air, as if they are flying. Some patients even describe their dyskinesia as "floating limbs," though it looks for all the world like writhing.

As for this lightness of being, great saints through the ages have often floated during their moments of ecstatic union with the Divine. Athletes, when in "the zone" of supreme attunement with their sports goal, are able inexplicably to stay in the air longer than they should be able to when jumping or while taking running strides.

Is it possible that dopamine, through its electromagnetic influence and its increase during meditation, deep prayer, and joyful mental focus, is the neurotransmitter that causes this physiological lightness of being? And is it dopamine deficiency that creates heaviness? A corpse, even before the rigidity of death sets in, has a strangely heavy feeling. It is as if the density of the body increases when the life force departs.

Whether or not this density change is related to dopamine, or whether dopamine is directly related to spiritual awareness, is, as noted before, beyond the scope of this book. But prayer and meditation can increase dopamine levels, and this may be related to the fact that reliance on a power outside oneself can be helpful in overcoming addiction.

Ego- and fear-related qualities of adrenaline logic and will power may go into overdrive during times of limbic deficiency crisis as adrenaline guns the motor area, but their role is primarily motor function; the adrenaline surge may be inadequate to suppress the emotionally traumatic stress of drug withdrawal. The PDer who assumes he will be able to conquer his limbic area with sheer determination simply does not understand the forces at play in addiction.

---

[1] Brain scans of meditating nuns show increased activity in regions associated with focus (frontal lobe) and decreased activity in brain areas associated with sense of self. *Newsweek*, "Faith and Healing," Nov. 10, 2003, p. 47. There is much published research supporting this subject. I'm including this lone footnote simply because the above article happened to cross my desk while I was proofreading this section.

Placebo effect

A long-held notion of medicine that is being challenged lately is the placebo theory. A placebo is a pretend treatment, such as a sugar pill or dummy pill, which is given to an unsuspecting patient who believes that he is receiving the real thing. This is supposed to show whether the patient's response is due to the treatment or to the expectation of result. Some recent studies prove that placebos do not work. Other recent studies prove that placebos do work. So much for proofs. In the science world, "proofs" are about as unreliable as "facts."

Still more recent research indicates that whether or not placebos work depends on the *nature of the illness*. Also, the placebo effect has been shown to be related to dopamine!

For example, placebos do not work well on some virulent bacterial infections. They work very well on illnesses such as insomnia, depression, asthma, anxiety-phobic disorders, and Parkinson's disease, all of which have a dopamine component. In such cases, the placebos appear to work because *placebos cause a dopamine increase in the brain.*

The expectation of reward appears to manifest as a dopamine increase in all effective placebo studies. The dopamine increase then leads to the improvement in all of the dopamine-related conditions.

In one recent study, subjects who thought they were receiving an injection of dopamine but who were actually receiving a sugar injection showed a significant and immediate brain dopamine increase, as proved by PET scans. The subjects in this study were all people with Parkinson's disease![1]

What you do with this placebo information is up to you, but if you have been diagnosed with a dopamine deficiency disorder, you may want to keep in mind these two "expectation" principles:

1) If you meditate on having a pain somewhere in your body, you can create that very pain.

2) The fastest way to increase brain dopamine is to expect that you are going to feel better.

Placebo opposite: hopelessness

Where this comes into play in drug withdrawal is this same principle applied in the opposite direction: the negative symptoms set in motion during drug withdrawal make it increasingly difficult to access that part of the brain that is normally used to activate hope. Therefore, a person in drug withdrawal may have the opposite of a placebo effect: he may instead feel certain that he is going to get worse (paranoia). This may lead to a further dopamine decrease. This morbid direction of thinking makes the withdrawal even

---

[1]Fuente-Fernandez, R. et al. Expectation and dopamine release: mechanism of the placebo effect in Parkinson's disease. *Science*, 293, 1164 - 1166, (2001).

Hrobjartsson, A. & Gotzsche, P. C. Is the placebo powerless? An analysis of clinical trials comparing placebo with no treatment. *The New England Journal of Medicine*, 344, 1594 - 1602, (2001).

Fact or Artifact? The Placebo Effect May Be a Little of Both, S*cientific American,* W. Wyait Gibbs www.sciam.com/2001/1001issue/1001scicit2.html

worse, and the worsening makes it even more unlikely that reason, faith, and hope can be accessed. The downward spiral set in motion can lead even to physiological fatality or suicide.

TRACKING DOWN THE NEW RESEARCH ABOUT DOPAMINE

By now, you, the reader, have learned that the current understanding of dopamine researchers is that dopamine plays a role in addiction. What you also may have learned by unfortunate experience is that your MD is very likely unaware of the role of dopamine in addiction. I didn't know it either before I embarked on all this research. Your friends and family probably do not know that dopamine plays a key role in addiction. Most likely, the only one in your immediate circle who knows this is your fourteen-year old niece, who possibly learned it in health class.

Therefore, I am going to take a break from the terminology and visions of ever-increasing brain damage, and give you some references that you can use on the folks back home. These references were collected from various journals and papers, and they range from highly prestigious research journals to the lowly Sunday Newspaper Supplement. You can use these references to help bring your MD into the twenty-first century. Enjoy.

> "In the last decade, we've revolutionized our fundamental understanding of the nature of addiction. 'Contrary to popular belief, addiction is not just a lot of drug use,' says Alan Leshner, director of the National Institute on Drug Abuse. 'It's literally a disease of the brain…(because) they've found that all drugs have common effects on dopamine, a neurotransmitter involved in the experience of pleasure…Over time…you're taking drugs not because you like them, but because you must…Addiction is not like strep throat. It doesn't go away. Drugs change brain cells in profound, long-lasting ways.'"
>
> "Addiction: A Brain Disease," *Parade Magazine* (a Sunday Supplement to hundreds of US newspapers), Nov. 21, 1999, p. 11.

> "Opioid painkillers, which include morphine and heroin as well as prescription products like Percocet, Percodan and Vicodin, are so dangerous because they are so seductive. They work by throwing up roadblocks all along the pain pathway from the nerve endings in the skin to the spinal cord to the brain. In the brain these drugs open the floodgates for the chemical dopamine, which triggers sensations of well-being. Dopamine rewires the brain to become accustomed to the benign feelings. When an addicted person stops taking the drug, the body craves the dopamine again."
>
> R. Corliss, "Who's Feeling No Pain", *Time*, March 19, 2001, p. 69.

> "Early studies showed that cocaine blocks the transporters for three different neurotransmitters: dopamine, serotonin, and norepinephrine. Later, one vein of research suggested that cocaine's blockade of the dopamine transporter was most important for producing the drug's euphoric effects. By blocking the dopamine transporter, some scientists theorized, cocaine might raise the level of extra cellular dopamine in brain regions involved in the feeling of pleasure. This excess dopamine could continue to effect neurons in these regions, giving rise to euphoria."
>
> S. Stocker, "Cocaine Pleasurable Effects May Involve Multiple Chemical Sites," NIDA Notes, Vol. 14, Number 2, August, 1999, derived from research by Rocha, B.A., et al. (NIDA stands for National Institute on Drug Abuse.)

Here's an article that turned up from a quick search on "levodopa dependence":
Article Abstract for...Levodopa Dependence and Abuse in Parkinson's Disease
Olav Spigset, M.D., and Christian von Schéele, M.D.

Two patients with Parkinson's disease repeatedly increased their levodopa dosage on their own to 1500-2000 mg/day to reach and sustain a state of euphoria, regardless of the fact that dosages of 400-800 mg/day were sufficient to suppress their parkinsonian symptoms. Both were markedly unwilling to consent to recommendations of dosage reductions, and they readily accepted adverse effects such as hyperkinesias, anorexia, and hallucinations to achieve the positive mental effects. Thus, both patients fulfilled the diagnostic criteria for substance dependence. (Pharmacotherapy 1997; 17(5): 1027-1030)
http://accp.com/pharmacotherapy/Abs17_5/1027.htm addiction and abuse

Using the Internet

These days, general neuroscience journals, and not just drug abuse journals, teem with articles that explore the relationship between addiction and dopamine. Because of powerful resistance in the clinical (drug prescribing) neurology set to the idea that dopamine has anything to do with addiction, I decided, on a lark, to see how hard it would be to find research articles that had titles with the word "dopamine" plus any other word relating to addiction or addictive drugs. It took nearly half a minute for the computer to log on. After that, it was quick. Within seconds after typing in the two words "dopamine" and "cocaine," my computer search engine had pages of articles to choose from. The first article I found after typing in these two search words was

"Cocaine self-administration in dopamine-transporter knockout mice," *Nature Neuroscience* 1(2): 132-137, 1998.

The point of the article is not particularly applicable – it is an article on what happens to mice that have been genetically altered so that they don't have dopamine transport molecules. But my point is, the relationship between addictive drugs and dopamine already is well established in research labs in the late 1990s.

And for an example of what the federal government research is finding, the following is the first selection that appeared on the website of the National Institute on Drug Abuse when I did a search in The NIDA Notes using the word "dopamine." This website, http://www.drugabuse.gov, is a wonderful source for the hot breaking news about the role of dopamine in addiction.

Sora, I., et al; "Cocaine reward models: Conditioned place preference can be established in dopamine- and in serotonin-transporter knockout mice." *Proceedings of the National Academy of Sciences USA,* 95 (13): 7699-7704, 1998. (from the National Institute On Drug Abuse's website, http://www.drugabuse.gov)

Again this article is about what happens with mice with altered dopamine transporters, but the larger point here is that dopamine is associated with cocaine, and that point has been made.

But you needn't seek out the very latest news – even the old, cooled-off news of the last five years hammers home the point that dopamine is at the root of every major form of drug addiction. Dopamine has come a long way from being a sweet, unassuming

neurotransmitter that was considered back in the 1960's merely an "opposite" for acetylcholine (see Appendix 5).

Keeping up to date on your drug products

If you search for the brand name of your drug, you will likely find sites from the drug companies or patients who are telling the world what drugs they are taking. If you use the chemical name for your drug, you will more likely find research (often subsidized in full or part by the parent drug company).

For example, if you do a search for Sinemet, a brand name for carbidopa/levodopa, you might pull up the page produced by the company that makes the drug. These sites can be very valuable. I visited the Sinemet site three years after the new "controlled release" (slow dispersing, longer lasting) pills were put on the market. I was interested to learn there that even the manufacturer was warning that many people were having worse side effects from the slow release pills than they were from the basic pills. The web site was very specific about the types of problems that people were having. The manufacturer's recommendation was that, in such a case, people should revert back to the basic pill.

Not *one* neurologist in my community was aware of this problem. My patients who were having these very problems with their drugs were being assured by their MDs that the slow release pills had to be an improvement – therefore, if a person was having a problem with the new pills, they would be having even worse problems had they stayed on the old pills.

Do you trust the Internet?

How reliable is Internet information? That depends. You can find anything you want on the Internet. Some of it is pure flapdoodle. Read as much as you can, and read between the lines. Figure out if the writer is trying to make money from his web site. Is he selling something? If you are reading the results of research, be sure to notice who underwrote (sponsored) the research. Did the interested drug company do the study that led to FDA approval? Was someone trying to substantiate his own previous research by doing the study? Be wary, find reliable sources, and dare to put two and two together to get four. (Most national PD organizations are not reliable sources. Many of them rely on drug companies or drug company-sponsored laboratories for their articles or information, making them an unlikely source of unprejudiced information.)

Be informed

The problems that patients have had with their doctors have been, in most cases, more harrowing than the problems of drug withdrawal and addiction. I cannot make this point too strongly, that you and you alone must be responsible for staying informed in the ever-changing world of pharmaceutical drugs.

There is much emotionalism and superstition regarding addiction. Hopefully, the latest findings about addiction will help throw some light on a subject that has intrigued and tormented man since the beginning of civilization.

DOES YOUR DOCTOR KNOW ABOUT ADDICTION? WILL S/HE TELL YOU?

I am trying to take the high road here, and not point an angry finger of blame. But, it may be important for you to know that, sadly, a few MDs do know that dopamine plays a role in addiction, and some of these MDs choose to lie to their patients about the addictive nature of the medications.[1] This is not a slam at doctors. The discussion on "how much a patient should be told/how much information should be withheld for the good of the patient" is still very much an open debate in this country.

In some countries it is even illegal to give worrisome information to a patient. In China, for example, it was (and maybe still is) forbidden for a doctor to tell a patient that he has cancer. In the USA it is up to the doctor to decide how much to tell the patient; there is no AMA consensus.

This is not to say that MDs enjoy lying to their patients. But they are often taught in medical school to be misleading to their patients and not tell their patients everything "for the patient's highest good."

A corollary to this "don't give bad news" policy, to which many MDs subscribe, is that patients should not be told the known side effects of their medication, for fear that they will psychologically adopt the side effects. This is not a new attitude. In 1969, when the *Journal of the American Medical Association* first published that dangerous side effects were inherent with L-dopa usage and that the drug was unsafe at any level for any person, doctors responded with a storm of letters insisting that "such matters should be kept quiet, lest it disturb 'the atmosphere of therapeutic optimism needed for the maximal efficiency of L-dopa.'"[2]

Silent Nurses

This brings to mind a quick anecdote that I got from a British Parkinson's patient via email. She was diagnosed less than a year ago, and her only symptom had been tremor. Her doctor had placed her on 600 mg/day of levodopa. Because of ensuing dyskinesias, her doctor – not understanding how the drugs work – *increased* her drug dosages. By the time she contacted me, she was having violent symptoms of overmedication. Her dosages, one year after diagnosis with minimal symptoms, were 50% higher than the maximum recommended by the manufacturer!

I sent her the publicly available information regarding her drugs, their proper doses, and their known adverse effects – most of which she was having. She wrote back to me this story.

I paraphrase: "When I was diagnosed, I asked the PD doctor if there were any problems from the medications. I had read about them and I'd been to the support group, and it seemed that many people have horrible experiences with their medications. The doctor assured me that he had never seen any people have problems with their medications, and that the drugs he prescribed me were the perfect treatment for Parkinson's disease. The nurses in the room just stood there and never said a word. When

---

[1] On April 2, 2003, on Canadian radio (CBC), I tuned in to a discussion between three professors with PhDs in philosophy, representing Harvard, Toronto and some other university. The subject was "Lying." One of the issues was "When, if ever, is it OK for a doctor to lie to a patient?" There was no conformity of opinion on this issue.

[2] Oliver Sacks, *Awakenings*. Harper Perennial, New York, foreword to the 1990 edition, p. xxxii

the doctor left the room, the nurses both addressed me quickly, in whispers. They said that they spend most of their time helping people with side effects of the medication. Nearly all of the patient visits to the clinic were for problems that were due to the meds, not the PD. *The doctor had instructed the nurses never to mention this to the patients.*"

There are some defensive MDs. I was sent a copy of a letter written by an MD to his patient in response to a query on the relative safety of L-dopa compared to some of the agonist drugs. In the response, the MD stated that he had been using L-dopa for his patients for thirty years and it was perfectly safe. He assured his correspondent that it was the manufacturers of the new agonist drugs that were trying to make L-dopa look bad by claiming that it had side effects, but that, get this – *he would not have been using it for thirty years if there had been problems*. The problems that most people thought were coming from the L-dopa, he continued, were simply symptoms of worsening Parkinson's, and there was nothing to be done about it.

Another doctor, writing as the medication support doctor for a national Parkinson's organization (not in the United States – I do not wish to refer to this doctor by name, for obvious reasons), wrote in response to an inquiry about medication that he had done a brief search for articles about addiction to L-dopa on the Internet, and only four articles came up. Based on this, he concluded in print that L-dopa was not addictive, and the four cases on the Internet must have been exceptions. The illogic here is stunning. Because in his quick search on the Internet he did not find many examples of L-dopa addiction, he concluded that this condition did not exist!

In response to this, I offer that I have observed cases of violent addiction to L-dopa, where patients rapidly increased their doses despite doctors' orders to reduce and were subsequently unable to reduce their medication. Here are some quick examples:

Lila

Lila was in the hospital three times in two months. Her hospital visits started after she realized she was out of control with levodopa. When I first met her, she was 61 years old, had been diagnosed with PD less than two years earlier, and she had dyskinetic twitching in her shoulders and face. At that time she was taking a full hand of antiparkinson's drugs: 350 mg levodopa/day (three and a half 25/100 Sinemet) together with Permax (.5 mg three times a day) and Mirapex (.5 mg three times a day), Eldepryl (5 mg twice a day), and Robaxin (150 mg/day), which is used for acute, painful musculoskeletal spasms. She took this last drug to keep her dyskinesia at a lower level. In addition, she was taking Zantac (for intermittent heartburn, 300 mg three times a day, although the maximum dose for self-medication for relief of occasional heartburn is only 150 mg in 24 hours), Trazadone (100 mg at bedtime) to help her get to sleep, Minipress for high blood pressure, Adalat for her concerns about her heart, a drug for her high blood sugar and two types of asthma medication. She was also nearly 150 pounds overweight. She had lost nearly 100 pounds through eating smaller portions when she was first diagnosed with diabetes several years earlier. When she learned that diabetes could be controlled through pills, she embraced the pills and went back to her old eating habits.

Lila had never had times when her medication didn't work and certainly no On- and Offs yet her doctor, a general practitioner, had steadily increased her medications. The doc had added an additional antiparkinson's medication at every six-month visit

because he assumed that, with a degenerative disorder, this was appropriate. He also considered that Lila's lassitude was due exclusively to Parkinson's. Lila's history of obesity and diabetes was never considered significant after Lila was diagnosed with Parkinson's. After the PD diagnosis, any fatigue on Lila's part was obviously due to the PD and needed to be answered with increased doses of PD drugs. The fact that Lila had been diagnosed less than two years earlier and still had extremely mild symptoms did not seem to matter to the young MD when it came to Lila's drugs. Even Lila's dyskinesia was being treated with more drugs rather than with a decrease in antiparkinson's medications.

I actually did call this doctor about our common patient. I gave no advice but told the doctor that L-dopa and Eldepryl should not be used together, according to the literature. I stated that dyskinesia was a side effect of the medications and might indicate overmedication, according to the drug inserts. I also mentioned that most neurologists did not use two agonists together but usually prescribed either Permax or Mirapex, but not both. The doctor thanked me graciously, and added, "It's such a surprise to talk to an acupuncturist who actually knows something!" I am sure he felt he was paying me a compliment.

But back to the point of addiction: Following several months of treatment in our program, Lila began having symptoms indicative of recovery. Her dyskinesia became much worse; she was unable to stop twitching her shoulders and face, even at bedtime. She started taking another medication to help her sleep. I met with Lila's daughter and explained that the antiparkinson's drugs could cause insomnia. Lila told us that she would reduce her medication. For the next two years Lila tried to reduce her medication and honestly believed that she had done so. Her doctor told her to decrease her medications and planned an aggressive schedule of drug reduction that Lila was unable to maintain – the rapid reduction rate caused her to start having Offs for the first time. Without consulting the doctor, she increased her medication beyond her previous level. She went up to 700 mg levodopa/day, more than twice what she was taking before. She honestly believes that she has been consistently reducing her medication. Her dyskinesia is so violent that she falls down frequently; she broke her hip during one of these falls.

At the hospital after the hip replacement she was told that she obviously had fallen because she needed more medication, and they added another 100 mg of levodopa/day to her dose, which she gratefully accepted. That was over a year ago, and she has continued to increase her drugs since then. She alternates between crying and laughing when she explains that she is trying every day to decrease the medication.

Euclid

Euclid was a delightful fellow, an electrical engineer and a poet, and fond of dancing. The first time I met him, at a class I was teaching, he showed me X-rays of his feet. They clearly showed that the intermediate cuneiform bone was posterior to its correct location.[1] He subsequently started working with one of the PD team members.

[1] There happened to be an osteopathic surgeon at the class that day, accompanying his mother who had Parkinson's. The doctor had been attracted to our program because of his mother's particularly deformed feet. Such a sweet man, he had even brought study guides – plastic foot bones – to the class for the students to work with. He thought it significant that we had found a connection between feet and

After a year and a half of treatment, he was nearly off his medication and his PD symptoms were definitely fading. He was getting a bit limp in the legs as his muscles shifted from rigid to mushy, and this worried him a bit, but he assured us he would not succumb to the lure of the medication, come what may.

Euclid was taking100 mg levodopa/day, down from his highest level of 300 mg/day. He had been reducing slowly over the last year and had not appeared to have any adverse effects from the medication. However, just as he was beginning to feel deeply relaxed inside, as if his old rigidity was melting away, he suddenly became dyskinetic even at the low dose of 100 mg/day; he said that he felt overmedicated and was planning to decrease his drugs again in a few weeks.

The next time he came in, one week later, he was taking 500 mg/day. His reasoning? "I wanted to go dancing!" He was absolutely loony. He was writhing. He told us that he had no dyskinesia. He said that the jerking movements he was doing were under his control, and he did them because they felt good. If anything, they might be tremor, which would mean he "could" take more medication.

Furthermore, he had decided to drop out of our program, quit his job where he had two more years to go before qualifying for early retirement, and instead, sell his home, move into a care facility, get disability pay and take as much L-dopa as he wanted for the rest of his life. He could do this, he pointed out, because it was his legal right as long as he had Parkinson's! Furthermore, he just loved dancing.

Euclid had not had Parkinson's very long; he had been diagnosed only three years earlier. He had never had On-Offs from his medication, and he had even been driving his car just a week earlier and doing well at work, where no one suspected he had Parkinson's disease. But he was still taking levodopa when his recovered brain had made the transition all the way back to normal, and abruptly, without much warning aside from the sudden display of dyskinesia a few days earlier, the drugs overpowered his mind.

He sends us poetry now and then, the gist of which is that Parkinson's is incurable, he tried everything, but the drugs were the only answer. I understand through the grapevine that he has increased his drugs again. At the high levels that he is now taking, he is certainly addicted, and would probably suffer the respiratory distress that causes death should he try abruptly to stop taking his medication.

Shortly after Euclid lost control, his health practitioner told me, "I used to think you were being melodramatic when you talked about these drugs. I've known people who abuse bad drugs. I thought I'd seen it all. But I've never seen anything like this stuff. You weren't exaggerating. Everything you said was absolutely true. I'm heartbroken over this. I keep wondering if there wasn't something I could have said or done. I will never again work with a medicated patient."

Based on what we saw with Zoe, who – having no more PD symptoms – was never able to tolerate even a 2.5% reduction[1] in her daily dose for more than three days without going into crisis, Euclid also may not be able to tolerate any decreases in the medication at this point.

Parkinson's. He was pleased to see the X-ray, and agreed that the cuneiform bones appeared to be smashed together and also posterior. The three middle metatarsals were also displaced.

[1] She could not tolerate a 50 mg decrease. Her daily amount was 2000 mg.

Angus

When we saw the rapid change in Euclid, we were reminded of Angus. Angus was in his late sixties and had been diagnosed six years earlier when he started with us. He was taking 600 mg/day levodopa (six Sinemet 25/100). He started recovering and decreased his medication accordingly. He had slowly decreased to 150 mg/ day over a year and a half. He had never experienced drug withdrawal trauma, although every reduction was marked by a period of slowness, increased rigidity and agitation. However, these reduction symptoms only lasted a month or two. When they ebbed he found his mobility resumed at his pre-decrease level. His neck pain – his neck was the site of his most painful (and evidently drug-induced) dystonia – disappeared, and he considered this a greater miracle than his return to flexibility.

Shortly after he decreased again, to an average of 125 mg/day (done by alternating 100 mg days with 150 mg days), his entrance to the clinic was highly dramatic. He navigated through the street door into the waiting area and shouted to the embarrassed assembly, "I've got my sex energy back! I've got my sex energy back! It's great! I'm cured!"

We hustled him into a more private setting and asked how he was doing. His eyes had that telltale glow and he could hardly sit still. He told us all about it; his sex energy was great, he could dance all night, he had been to a party next door and had played cards faster and louder than anyone. He had more stamina than anyone in the neighborhood. Would we like to see him shuffle cards? His hands were working again, faster than ever.

We suggested that he might be concerned about his medication levels, but he dismissed us, saying, "The doctor says that the amount I'm taking now doesn't even do anything. Why should I reduce?" Then he glowered at us suspiciously. "Are you suggesting that the drugs are the reason I feel so good? Because you are wrong! This is the real me! This is the way I always used to be. I just changed my attitude, that's all. I am not going to decrease my drugs. They aren't doing anything, anyway. I may even increase them, just to show you!"

We called his wife and arranged a conference. We had never met his wife. Angus had joined the program before we made the requirement that any medicated patient must be accompanied at every session by a friend, spouse, or caregiver who could help track the patient's medication situation. Angus's wife came to the meeting, bitter that we were taking up her precious evening time. We suggested that Angus's new behavior was a bit over the top, and we suspected that the drugs were addictive, and what did she think? I am quoting her unexpected response as closely as possible:

"I don't want him to change anything. Every time he decreased his medication he didn't feel good, and I had to do his chores. I am sick of it. He uses those drugs and this program to lay around the house feeling lousy. We are going on a cruise in four weeks. I have been looking forward to this my whole life. I've been working full time my whole life even after he retired. I am sick and tired of taking care of him. What about me? I've never had any fun! I've never gotten to do what *I* want! It's my time to have a good time for once in my life! If he decreases his medication he will be a #*! drain on me during the cruise; I'll have to take care of everything. I won't have any fun. What am I supposed to do, take care of him the rest of my life? He's got this #*! Parkinson's, so he has an excuse to do whatever he wants and I have to work all my life!" At this point she expanded on her thesis with colorful language which I have forgotten.

I am embarrassed to admit that during her exposition my mind strayed a bit. I recalled Angus's supreme joy in his rediscovered sex energy. I mused as to whether his partner might not have her own ideas on that subject as well.

Mrs. Angus summed up: "So as far as I'm concerned, he can just take his drugs if they make him happy. I don't care what he does as long as he doesn't screw up our cruise. I don't care what he does after that. I just want him to be able to take care of himself for the next four weeks." She stood up, looked around for her purse, cocked an eyebrow at Angus, and he scurried out the door after her. He turned in the doorway and beamed at us. "Thanks for the great meeting! See you next week!"

The next week Angus appeared, grinning from ear to ear.

"You were wrong about the drugs. They don't hurt me at all. I've started taking 300 mg/day, and I feel even better than I did last week!"

He had a hard time lying still on the treatment table.

The following week he started taking 500 mg/day. He was rowdy and belligerent when he showed up at the clinic. He was talking in a super fast voice and couldn't stop. He said that he'd been to a party the night before and they were all "impressed" with how fast he talks. "They've never heard anyone talk so fast!" He was agitated, loud, and his arms and legs were in constant movement. His head was jerking back and forth. He could not lie down on the treatment table and quickly lost interest in being treated or even talking to his practitioner. He wanted to cruise through the clinic and see how everyone else was doing. He announced that he was going to start taking 1000 mg/day because he just knew that it would be the right thing to do.

We asked him to leave. His practitioner, Kathryn, called him several times during the next two weeks. He was happy to talk to her, but she said he rambled and didn't really make sense. "I'm not sure he knows who I am," she said.

Kathryn was devastated. Ten years earlier she had left her first husband after an anguished three years of trying to help him with his cocaine addiction, and she found the similarities too chilling. She said she would never again work on a medicated patient. She happened to see Angus a few months later, half-walking and half-running down the sidewalk, headed for the grocery store. His arms were jerking wildly, and his head was jumping back and forth.

She told us that he waved and he jerked his arms in her direction, but, she sighed, "I'm sure he didn't know who I was."

Back to the gist…

As for the *highly* regarded doctor whose specious argument against addictiveness in anti-PD drugs was based on finding only four cases of Sinemet addiction on the internet, I would like to point out that I never published, until now, the above three instances of addiction. But based on what I have seen and heard, I imagine that nearly every PD doctor in the world must have a few cases like this. I suspect that it would be people with drug-induced parkinsonism who are more susceptible to these forms of wild, euphoric addiction than those with idiopathic Parkinson's. (The exception is people in a recovery program, of course, who develop drug-induced parkinsonism from their drugs as they recover.)

However, most of us clinical health practitioners don't have the time to write up all the strange things we see. Most clinical doctors do not publish in journals. Furthermore,

journals only publish articles that are moving research forward. It is already known in research circles that L-dopa is highly addictive. It is simply absurd to think that, because only a few articles can be quickly found on the Internet following a cursory search, that this is proof that it is not addictive.

If you are going to take Parkinson's medications or if you are planning to decrease your medications for any reason whatsoever, you must accept the possibility that you already know more about dopamine than your doctor. You know more about dopamine than your MD might be willing to tell you. Your doctor may be incapable of accepting the idea that he has been prescribing dangerous medications.

In appendices 5 and 6 there is a brief history of the ever-changing "scientific facts" and theories of dopamine, the assumptions and the mistakes from 1950 to the present. Medical facts are often just pure guesses that attain the status of facts just because there is not yet a conflicting idea or because the guesses seem to agree with previous ideas that have gained fact status. This is the way of all research.

You are on your own

This recurring theme is back: your MD will not be familiar with these principles and may not be willing to listen to you. A common question from patients is, "How can I get my doctor to learn about these drugs?" I have no answer for you.

On your own, without MD support

If you intend to decrease your medication, whether it is to minimize your risk of developing parkinsonism or because you feel overmedicated, you will probably find yourself going through this difficult process without the benefit of informed MD support. Your neurologist may still be studying the information about dopamine from the

perspective of dopamine being a muscle relaxant. He may still be thinking of PD as a syndrome of excess muscle tone. This is not because he is ignorant or mean. It is because the field of brain research is too vast, too rapidly changing for any one person to possibly keep up with much of it. So if you want to prevent doing yourself a disservice with your medications, what you need to do is read the research even if your MD does not.

Summary

A*ddictive drugs, including the drugs used in the treatment of Parkinson's disease, may set in motion, in a much more permanent way than in mere Parkinson's disease, all of those symptoms of Parkinson's disease that are dopamine-dependent*. Also, many perfectly legal drugs, especially those that are used in the treatment of anxiety, pain, or depression, may change dopamine levels and set in motion drug-induced parkinsonism.

The huge surge in diagnoses of Parkinson's disease in the last twenty years is thought by many alert doctors to be the result of the burgeoning use of antianxiety and antidepressant medications. When the people who use these drugs begin to develop parkinsonism, they are usually misdiagnosed as having Parkinson's disease. Then, they are prescribed antiparkinson's medications, which further advance the addiction and increase the parkinsonism.

Your doctor may not know about the recent findings regarding dopamine and addiction. Even if he does know, he may feel that you should be protected from this information.

Keep in mind that research on dopamine is still growing rapidly. You must take responsibility for staying current about dopamine and Parkinson's disease. You cannot rely on your doctor to be up to date.

Finally, the production of dopamine in the brain is expectation dependent. Despair, such as that experienced during drug withdrawal, can very possibly decrease dopamine even beyond the decrease caused by addiction. And conversely, and wishing to end this chapter on a positive note, remember that the expectation of a good day can elevate dopamine levels.

"Anything green that grew out of the mould
Was an excellent herb to our fathers of old..."

"Our Fathers of Old," Rudyard Kipling

10. THE ANTIPARKINSON'S DRUGS

THE WAYS IN WHICH THE DRUGS AND POTIONS WORK

Now it's time to jump from generalities about dopamine to some specifics about the pharmacological actions of the individual anti-PD drugs. It was often difficult for my patients to understand how a perfectly legal drug could be addictive, and so I devised the following instruction method: based on the particular mechanism of action of each of the anti-PD drugs, I sorted these drugs and correlated them with well known addictive drugs that used the same mechanism. All my patients knew that cocaine, nicotine, and methamphetamine were addictive. By showing the relationship between the mechanisms of these street drugs and their parkinson's drugs, my patients started to understand why they couldn't reduce their medication without going into an addiction-like tailspin.

Most of the antiparkinson's drugs have "cousins," drugs that are well known to the person on the street, that use similar, though usually milder, mechanisms to elevate dopamine. Others have different mechanisms from their related illegal (street) addictive drugs but similar overall effects on the brain during use and withdrawal.

And so, here is a brief look at some of the dopamine-enhancing street drugs, their mechanisms of dopamine enhancement, and a comparison with some of the anti-parkinson medications that have similar mechanisms. This is brief because each of these drugs will be discussed in greater detail in the appendices.

Dopamine Agonists – Nicotine

Dopamine agonists have been used in the treatment of PD since the 1980's. Some of the more popular agonist PD drugs are Mirapex, Permax (also known as Pergolide), bromocriptine (Parlodel), Requip, and Cabergolene. These drugs are cousins of nicotine. Nicotine is, of course, a much milder and much less addictive chemical than any of the antiparkinson's drugs. After all, nicotine is not strong enough to allow initiation of movement in a person who can no longer move. Nicotine, in the small amounts delivered via tobacco, merely lifts dopamine levels for a short while. The amounts are not great enough to counter symptoms of Parkinson's disease. However, the gentle nicotine molecule works in the exact same way as the anti-PD meds listed above – it can assume the role of a dopamine molecule on a brain's dopamine receptor, tricking the receptor nerve into firing off as if dopamine, and not nicotine, was in the receptor slot. When the dopamine agonist takes on the job of a dopamine molecule, the remaining dopamines are freed to do their work elsewhere, so the net dopamine activity in the brain occurs at higher than usual levels – whatever "usual" happens to be.

Are the dopamine agonists addictive? Yes, indisputably. Cigarettes are internationally recognized as addictive. Nearly everyone knows that it can be very difficult to quit smoking. Nicotine provides a brief, mild feeling of well-being, but very

quickly after taking up the smoking habit, a user finds that he is irritable and irascible if he doesn't get his regular smoke. Nicotine is addictive because it is a dopamine agonist.

Word play

People who forgot to learn Latin, this author included, often misunderstand the word agonist at first. We imagine that "agonist" means "works against." Well, that's wrong. An ***ant***agonist is something that works against something. Note the prefix "ant" (as in anti) in the word *ant*agonist. Agonist is a completely different word!

The word agonist comes from the Greek "agon," meaning contest. Agonistikos[1] means "fit to compete." So "agonist" means, in a dopamine context, a chemical that competes with dopamine for the little dopamine receptor sites. A dopamine-receiving nerve doesn't care whether it is actually dopamine or some imposter molecule that is hooked up at the receptor site – once the receptor is filled with something, either a dopamine or a dopamine agonist, the nerve can work.

Actually, it would have been a boon if the person who named this class of drugs had used a more correct word instead of agonist. Agonist implies competition and suggests that if the agonist gets to the receptor, it is the winner, and dopamine is therefore, somehow, the loser. This is not the way it works. A dopamine agonist works like a look-alike drug. It is not exactly dopamine, but many receptors cannot tell it apart from a dopamine, and so it is allowed to stimulate a dopamine receptor, working hand in hand with dopamine to help push a nerve over the threshold. A more appropriate name for the agonists would have been "dopamine teammates," or "dopamine substitutes."

You may ask, since dopamine agonists work just the same as dopamine, why not use dopamine? Good question. Here's why: dopamine is a naturally occurring compound. The dopamine receptors in your brain are designed to attach quickly to dopamine, and the dopamine reuptake transporters are designed to remove the dopamine efficiently. But the dopamine agonist drugs, especially the ones that are completely synthetic, not derived from naturally occurring compounds, are complete foreigners to your brain. Your brain doesn't have a clue as to what to do with a synthetic dopamine agonist. These agonists are not a perfect fit into the dopamine slots on the receptor nerves, but eventually, enough of them can wiggle into the dopamine slots that your brain nerves go over the action threshold.

Then, after the agonists are in position, it is a tad difficult for your body to get rid of them: their free end doesn't match up with the dopamine reuptake transporters. Once the agonist has wedged itself into a slot intended for dopamine, it can stay there for a while.

Well, actually your brain has lots of defense mechanisms for getting rid of chemicals that it doesn't want, such as alcohol, cocaine, and dopamine agonists. But these mechanisms are much slower and clumsier than the elegant and lightening fast dopamine transporters. Consequently, the agonist drugs produce a much more gradual On, and a much more gradual off. The dopamine agonists are used instead of or in addition to levodopa because they can activate a dopamine receptor almost as well as dopamine, but your brain has a harder time getting rid of them.

---

[1] *Webster's New World Dictionary.*

NADH – straight nicotine

Some PDers in very early stage PD find that NADH, a pill form of nicotine available at health food stores, can provide enough dopamine so that they do not need the stronger drugs for a little while. Sometimes even humble nicotine, a drug much more mild than the mind-altering[1] antiparkinson's dopamine agonists, can provide a newly diagnosed PDer a small dopamine boost adequate to obtain the threshold for movement.

Cigarettes are hard to quit. The PD agonists are much, much harder to quit. This example of the relationship between nicotine and the antiparkinson's dopamine agonists may be helpful in understanding just how addictive the Parkinson's meds are. If you are taking Permax, Mirapex, Requip, or bromocriptine, remember: they work the same way as cigarettes, only much more so.

MAO inhibitors – Methamphetamine/amphetamine

Selegeline hydrochloride (referred to during its research days as both deprenyl and L-deprenyl hydrochloride) has many patent names: Eldepryl, Atapryl, Carbex, Selegeline, and Selpak. I will use the most common name, Eldepryl, when referring to this drug. Once in the body, this antiparkinson's drug rapidly breaks up into its active ingredients: amphetamine, methamphetamine, and other less understood compounds.

Eldepryl was promoted in its first antiparkinson's release in the mid nineteen nineties as "possibly preventing further degeneration of substantia nigra cells." This sort of hype is pure routine. Once the FDA approves a drug, there is no legal reason that an advertising campaign can't say something utterly hypothetical about it, such as "possibly prevents further degeneration." For example, it is perfectly legal to say that "possibly spinach prevents further Parkinson's degeneration," or even "possibly chocolate-dipped macaroons."

It is also legal to say that a drug possibly increases sex appeal. We see this perfectly legal but highly unlikely implication of sex appeal increase in ads for soft drinks and cars. This is all legal and above board. As long as the word "possible" is used, no illegality has been perpetrated.

Although it has been shown now through experience that this drug does absolutely nothing to slow the onslaught of accelerating PD, and in fact causes a special type of brain damage, the brief flash of advertising hype did its job – for several years people were asking for this hot new drug. Many doctors who got in the habit of prescribing this drug continue to do so.

Deprenyl is a slightly modified (liquid) version of Selegeline that is not legal in this country but which is available via mail order from overseas. The sales reps for Deprenyl insist that the special addition of a hydrogen atom here and there on the molecule make it safe and non-addictive. Considering it only works against Parkinson's because it increases dopamine levels, and it is the propensity to increase dopamine that

---

[1] The dopamine agonists used in treating Parkinson's are all strong enough to cause hallucinations and alter mental function. For example, the plant compound from which Permax was derived is historically famous for its mind-altering properties. During the Middle Ages, people who ingested this compound (a toxin given off by Ergot, a fungus that grows on rye) were considered to be divinely touched. St. Vitus's dance was the name given to the unrestrainable movement (dyskinesia) and appearance of ecstasy in those who inadvertently ate moldy rye. Cigarettes are not able to incite this level of bliss or movement.

determines whether or not a drug is addictive, this claim of "safe and non-addictive" is clearly misleading.

This drug works for PDers the same way it works for kids who take methamphetamine in order to party all night without stopping – it causes dopamine vesicles unrestrictedly to pour their dopamines out into the brain, where they slosh around stimulating everything in sight.[1]

Originally, the MAO inhibitors were thought to work because they slow down MAO. MAO is the enzyme that breaks up dopamine. This drug's apparent effect of allowing PDers to initiate movement was assumed to be occurring via this mechanism, in which MAO was tied up, and dopamine was allowed to last longer. However, with the finding that this product breaks up into amphetamine and methamphetamine, the guess as to this drug's action has changed. It is now suggested by the manufacturer that this drug's action is only in part affected by a decrease in dopamine breakdown. In the addictive drug research community, it is currently thought that the drug works by overstimulating the vesicles – a known function of methamphetamine.

Of course, when it comes to actually knowing what is causing what, all bets are off: western science is still, to a large extent, guesswork.

---

[1] The information presented by the drug company on this drug suggests that the drug works by slowing the reuptake of dopamine. This turns out to be only a guess, as newer research indicates that possibly this drug works by stimulating the vesicles, rather than by slowing reuptake. It doesn't really matter for the user or the doctor, but I thought you might enjoy knowing that a drug company does not need to know the mechanisms of the drugs they are selling. They only need to guess at what the pharmacodynamics might be. What they do need to prove, in the USA, is that the drugs are not fatal in the short term.

I find it appalling that most people assume that, if a drug is on the market, it has been proven to be "safe," without bothering to find out what is meant by "safe." The FDA seal of approval imparts a rosy aura in the minds of most American consumers, despite regular exposés and lawsuits that reveal that approved drugs are often, in fact, dangerous.

In *Prozac Backlash* you can learn that Prozac was found to be safe to use on mice or rats only if the animals were simultaneously given sedatives. Without the sedatives, the animals appeared to go berserk. However, because the Prozac did not harm the animals when accompanied by sedatives, the drug was approved. No mention is ever made in the prescribing literature available to doctors that explains that this drug may only be safe over the long run if given in combination with sedatives.

As for the dosing, Prozac was found to be safest when used at a very low level. Only in extreme cases should the drug be slowly boosted up to a higher level. In general (because they are often taught this in med school), many MDs feel that their patients are, for the most part, too stupid to take drugs that must be built up slowly over the long term. Neither are most MDs able or willing to work closely enough with a patient to determine whether or not a lower dose or a higher dose is appropriate. Therefore, when Prozac was first introduced with the sliding scale dosage instructions and pills of many finely graded dosages, it was not at all popular with prescribing physicians.

The manufacturers, throwing caution and moral guidance to the winds, rereleased the drug at only two dosage levels: high and highest. The safest level, the lowest dosage, is no longer even available. However, now that prescribing doctors do not have to worry about patient compliance, they happily prescribe this very dangerous, brain-altering drug to a population that wants happiness and wants it immediately.

This footnote has come a long way from pointing out that the original guess at what makes Eldepryl work has been changed. Please forgive me.

Methamphetamine

What is known about methamphetamine? This drug is not as well known as cigarettes to the man on the street, but it is well known in drug abuse circles, where it has various names including "speed" and "meth." It is related to Ecstasy. It is easy and cheap to make. Narcotics police are constantly busting "Meth" labs, but new labs spring up to take their place like mushrooms after the rain. Meth is a powerful, mind-altering drug. Methamphetamine, like its sister amphetamine, causes release of dopamine and norepinephrine from their storage sacs. Meth is a *very* powerful stimulant.[1] Meth makes a person feel unnaturally fast, capable, strong, and tireless. This drug is highly addictive and causes permanent brain lesions.

This group of drugs, the MAO inhibitors, is specifically prohibited from being used in conjunction with levodopa type drugs. From what I have seen, this prohibition is widely ignored. Drug reps (salesmen) seem to be completely unaware of this contraindication, and they promote the MAO inhibitors as a nice adjunct drug for L-dopa. Dr. Leslie (Rose and Becky's doctor) had many of his PD patients taking Sinemet together with Eldepryl.

Anticholinergics – Psychedelics

Next, let's consider the most commonly used anticholinergic drug, Artane, also known as Trihex. This class of drugs is falling out of favor with neurologists, possibly because they have been out for so long that the patents have expired and so the salesmen aren't pushing them, but also because they aren't as gratifying as the dopamine-enhancing drugs.

Anticholinergics work against the neurotransmitter acetylcholine (ACh). They work by blocking the receptor sites (the hookup sites) where acetylcholine is supposed to land on a muscle-stimulating nerve and trigger a "go" signal.

These drugs are mainly effective in reducing the restlessness and anxiety that is found in PD. Unless a patient has tremor or extreme anxiety, anticholinergics should not be used. When ACh is suppressed, the muscles can't move as fast, so motor function is slowed considerably. Also, the thinking processes are slowed as brain function, including anxiety, is reduced. Considering that most PDers already have a slowdown of motor function, it is counter-productive to use a drug that further slows the brain. This is why the anti-ACh's should probably only be used for patients whose tremor or anxiety is a greater problem than slowness and rigidity.

You might not be surprised to find out that this drug is often incorrectly prescribed to treat slowness and rigidity, even though this drug specifically and intentionally causes slowness. That is because in many doctor's books, this drug is listed as being used against Parkinson's disease without reference to *which* symptoms of Parkinson's disease it addresses.

Most neurologists – I am not making this up – have no idea that the various PD drugs have distinct functions and that certain drugs are best suited for specific symptoms. Most MDs will try any drug in the "antiparkinson's" category on their PD patients without regard to which PD symptoms are dominant. I have seen anticholinergic drugs,

[1] *A Primer of Drug Action*, p. 31.

which cause sleepiness and confusion and muscle weakness, being prescribed for people whose main complaints are fatigue, loss of balance and muscle weakness.

Belladonna

Scopolamine, the street drug most closely related to the anticholinergic anti-parkinson drugs, is becoming passé – just like the similar PD drug. The psychedelic drug scopolamine, an anticholinergic, is derived from the belladonna plant. Scopolamine is also found in the Datura plant, in jimsonweed (locoweed) and other plants. It has been used for centuries both as a poison and as a hallucinogen. Ever since the advent of LSD and the increased availability of psychedelic mushrooms in the 1960's, the scopolamines are no longer in fashion. Considering that these drugs were very often deadly, in addition to being psychedelic, it is just as well that they are falling out of favor.

The anticholinergics are not addictive in the same way as dopamine-enhancing drugs. However, the body does accommodate to them. Daily use of Artane, which suppresses acetylcholine, will cause the body to set in motion steps to *increase* the manufacture of ACh, in an attempt to override the effects of the drug. Then, if a person stops taking the drug, the excess acetylcholine causes insomnia, edginess, severe anxiety, and twitchiness for several weeks or more.

Cocaine – L-dopa

Cocaine's pharmacodynamics are different from those of L-dopa, but many of the effects on the brain during usage and withdrawal are quite similar. They are both fairly non-specific with regard to the activity region: L-dopa washes over the entire brain, and cocaine inhibits dopamine reuptake enzymes over a large area. The net result of both is similar: an increase in dopamine effectiveness spread over most of the brain. Did you know that L-dopa is beginning to be used on the street for its high? It is used both by everyday folks looking for a lift and by a few savvy doctors who enjoy the energy boost. Cocaine is often mixed with inert or toxic fillers, and pharmaceutical L-dopa or carbidopa/levodopa is fairly pure and the amounts are more reliable. (Presumably, the amounts are more reliable. However, some patients who have tried the brand name and the generic versions of Sinemet say that there is quite a difference between the various brands. Other patients say there is no difference at all.)

Cocaine

Cocaine works differently than the dopamine-releasing drugs (methamphetamines) or the dopamine helper drugs (agonists). Cocaine, as described earlier, works by blocking the reuptake of dopamine after it has finished its dopamine job at the nerve work site. The net result of cocaine use is that dopamine molecules that should ordinarily only stay hooked up for a few seconds get to stay in their position for a bit longer than they should. This frees up the other dopamines that are floating around, waiting their turn to be used.

Cocaine is a highly addictive, dopaminergic drug.[1] Both cocaine and L-dopa are mild anesthetics. People with cocaine addictions and those who are in cocaine withdrawal have characteristic, well-studied behaviors. These patterns of denial, paranoia, and deceit

[1] R. M. Julien, MD, PhD, *A Primer of Drug Action, A Guide to the Actions, Uses, and Side Effects of Psychoactive Drugs,* W.H. Freeman and Co., New York, NY, 1999, p. 126.

from cocaine abuse are so similar to the patterns that develop in abusers of L-dopa that I consider L-dopa to be more comparable to cocaine than any of the other drugs that elevate dopamine levels.

Street cousin summary

In summary, the most common anti-PD drugs, the agonists, MAO inhibitors, anticholinergics, and L-dopa have strong similarities to their street cousins: nicotine, methamphetamine, psychedelics and cocaine. Except for the cholinergics, these street drugs are highly addictive. They are all addictive in slightly different ways, and they all have different mechanisms for increasing dopamine, but every increase in dopamine will be met by the brain with addictive counter-forces that semipermanently reduce the ability of the brain to ever again go over the brain's internal plimsoll[1] line for dopamine. Even though the street drugs are illegal and the PD drugs are legal, your brain can't tell the difference. If you go over the brain's Safety Limit, you have gone over the line, and you will have to pay the price. Your brain may never be the same.

Medications not related to street drugs

There are also drugs that do not have a parallel on the street that are used in the treatment of PD. While they may not be technically addictive, most of them cause changes in the brain that lead to accommodations. This means that there will be a backlash if you stop taking them. A mere backlash might only last for a few months, compared to the true, dopamine-based addictions which can create changes that may well be lifelong.

The Antihistamines

Some people take mild antihistamines such as diphenhydramine (Benedryl, Tylenol PM, and others) to help with the insomnia of Parkinson's disease. These drugs are sold over-the-counter, but they have a strong effect on the central nervous system and should not be taken for more than two weeks, as it clearly says on the box. They are accommodative. They have a strong rebound effect when they are stopped. Rebound means that your previous symptoms may return with a vengeance when you stop taking the drug. For example, if you had insomnia before you started taking these pills and you took the pills for too long, then, when you stop taking the pills, your insomnia might be far worse than it was before you started taking the pills.

Mirtazapine

There is a powerful new "antihistamine," mirtazapine (Remeron), which has recently been approved for Parkinson's tremor. I have heard of this drug being described by neurologists as merely a strong type of antihistamine. This is a highly misleading

[1] Plimsoll was the British engineer who first started painting a line around the hull of a boat that indicated when the boat was safely loaded. If a boat was so heavily loaded that the Plimsoll line was under water, no longer visible, then the boat was too heavy to go safely to sea.

description. One of the actions of mirtazapine is to block histamine receptors, making it hard for histamines (the chemicals that cause sneezing and runny nose) to hook up. The major function of this drug, however, is that of SSRI/norepinephrine reuptake blocker. Most SSRI reuptake blockers can be addictive and they can cause tardive dyskinesia. Mirtazapine has been primarily used, not as an antihistamine, but as an antipsychotic, a sedating/heavily pacifying drug.

However, this drug caught the attention of the Parkinson's researchers due to its apparent ability to reduce tremor. The researchers figured that the tremor reduction was due to the histamine-blocking effect of this drug, which also causes extreme drowsiness. *The drowsiness induced by this drug leaves a person suspended in that "just falling asleep" state*. Because tremor ceases when sleep begins, this drug is able to decrease tremor. By keeping a person right at the edge of falling asleep, the tremor is subdued.

This drug was approved for tremor in late 1999, and it is not safe for anyone who must drive a car or perform any activity requiring alertness. The tremor rebounds with vigor as soon as the drug wears off.

The fact that it is also an antipsychotic was not known to any of the people I have met who were taking this drug. Also, because of its serotonin/norepinephrine enhancing properties, it is also, inadvertently, a dopamine enhancer and therefore addictive.

I have not known anyone who was able to continue taking this drug for very long. After repeatedly falling asleep while driving, my patients decided that the drug was not worth the benefit. After discontinuing (stopping) even a short course of the drug, there was a rebound effect: tremor was much worse for a month or more.

Amantadine

Amantadine, also known as Symmetrel, was first used as an anti-viral drug and appears to work by boosting adrenaline. The mechanism of this drug is unknown, and so it is impossible to make a comparison with street drugs. In Appendix 2, you will read some case studies that demonstrate how quickly the brain accommodates to this drug even though its pattern for withdrawal follows a slightly different timeframe than the dopaminergic drugs. This drug is considered to be very mild by some doctors. Why that is, I can't imagine, after watching so many people trying to stop this drug and failing. Typically, all benefit from this drug has ceased (due to accommodation) within three months, and after that it has powerful rebound side effects if discontinued.

Digestion Inhibitors

Comptan and Tasmar do not elevate dopamine directly. They work by shutting down the body's digestive process so that more L-dopa can make its way unmolested up to the brain. These drugs do not in and of themselves cause addiction. They simply gum up the digestion, often causing liver and/or kidney damage. These drugs are usually not used until a person is having On-Offs. The thinking behind these drugs is that advancing Parkinson's is the reason for the levodopa "failures." By eliminating digestive enzymes, thus insuring that the L-dopa gets delivered to the brain in extra-large quantities, they can increase the payload (and the damaging effects) of a given dose of L-dopa.

Curiously, these drugs are given by doctors as a "safer" alternative to increasing levodopa dose. However, this completely uninformed approach fails to recognize that these drugs only work by increasing the effective payload of levodopa – the very thing the doctor is trying to avoid. If a doctor has determined that what the patient needs is more dopamine in the brain, it makes more sense to take more levodopa than to take a toxic drug that shuts down gastrointestinal function.

True, with these digestion inhibitors, a person can continue to take the same number of levodopa pills that he was taking before and get more levodopa into the brain, but at what cost? So many people died in the first three months that Tasmar was on the market that they changed the prescribing recommendation: in the USA a person should have biweekly liver scans if he wants to use this drug. Canada has banned the drug. And at the brain end, the dopamine effect from taking the Tasmar was exactly the same as if the patient took a slightly higher dose of levodopa. Then again, Tasmar is recently patented and the patent for Sinemet has recently expired, but I do not like to think that this is significant.

NEW PRODUCTS

I get several emails a month asking me about exciting new "non-addictive" products or "harmless supplements" that help Parkinson's disease. I also hear regularly from patients telling me either that these exciting new (and usually expensive) products don't work in the long term, or else that they have turned out to be addictive after all.

I will list just a few of the currently popular ones.

Glutathione

This one has gotten lots of publicity from MDs who offer expensive shots of this drug. Patients who have reported to me after taking it say that they sometimes feel powered up by it but no more than if they ate several candy bars at one sitting. As soon as they stop taking the shots, they go into a slump for several weeks to several months, and then they find themselves right where they were before or a little worse.

NADH / nicotine patches / cigarettes

NADH is nicotine in a pill form. Regardless of delivery system (pills or patches, or the old fashioned chaw and cigarettes), nicotine is a powerful acetylcholine agonist, as well as a dopamine agonist. Nicotine, being an agonist for both of these movement-related neurotransmitters, can assist in imparting movement. It does nothing to deter the progress of Parkinson's disease, but it can help mask the symptoms, just as all dopamine-enhancing drugs do.

Do you think nicotine is addictive? Ha ha ha. Of course it is. So why is nicotine, in any form, legal? Nicotine is a naturally occurring compound. It requires an act of congress to make a natural substance illegal. There are powerful political reasons that nicotine has not been made illegal, plus our country's disastrous experience with alcohol prohibition.

NADH is sold as a supplement at your local health food store. NADH does not need FDA approval to be legal; since nicotine is a naturally occurring substance, it does not need to be tested and proven safe, nor can it be patented. You will find companies

trying to get around this limitation on patents by adding a hydrogen atom onto the molecule, thereby creating a patentable product (the hydrogen breaks off as soon as it hits the stomach and you have plain old nicotine again). This "enhanced" product can then be hyped and sold for much more money than plain old nicotine plant extract, which would be dirt-cheap. The patented forms are much pricier and are usually the only forms carried by health or vitamin stores.

There are side effects of nicotine that can make this form of PD treatment unpleasant: because nicotine enhances acetylcholine as well as dopamine, some people feel more edgy and restless when using nicotine. Others find it mildly beneficial.

OLD PRODUCTS

Macuna, fava beans

Macuna is in the same legal category as nicotine: found in nature, therefore unpatentable and legal until decided otherwise. It has a high L-dopa level. This herb occurs naturally, and therefore it is perfectly legal to sell it over the counter. It can be found in Ayurvedic herb stores. In India it is used as a brain and sex stimulant – similar to cocaine, but milder. Taking this herb can elevate brain dopamine levels quite high, thus its use in Parkinson's disease is explained. It is highly addictive under certain conditions. Despite the fact that it is natural and an "herbal" product, I repeat: it can be addictive.

I have seen several patients using this herb. Two were never able to reduce their daily amounts despite being clearly stoned even to the point of glazed eyes and illogical thought. One Macuna user visited the clinic just once. His wife told us that she pleaded with him constantly to stop taking so much Macuna because he was so strange all the time. He just laughed at her and told her she didn't understand. When we told him that Macuna was addictive, he laughed again and said that we didn't understand and that he could take as much as he wanted because he had Parkinson's disease.

Lance

Lance was a real fighter and survivor. He had survived polio as a child and was determined to survive PD as well. He was able to get off this herb. Lance took Macuna three times a day. His blood dopamine levels were measured in a lab test and his blood numbers were 10 times (!) higher than normal.

When I met Lance he seemed overmedicated. His eyes were unnaturally bright and his twitching seemed more like ticcing than like true tremor. Also, he seemed over-quick in initiating movements, and he bragged that, despite his Parkinson's, he worked out at the gym and ran miles every day, manifesting no symptoms of Parkinson's. I asked him what drugs he was taking and he said none. The next time I saw him he seemed even more overmedicated. He assured me again that he didn't like drugs and wouldn't take them. The third time I saw him I asked him again. Exasperated, he explained that he wouldn't take drugs because he didn't trust them and he didn't need them. He never took anything but herbal supplements. I asked about his supplements. He was taking Macuna and Deprenyl together: levodopa and an MAO inhibitor, together. Together, these two drugs are much more dangerous than either one on its own, and fatal interactions have

occurred in some patients. The makers of L-dopa state that it should not be used with MAO inhibitors.

I told him that he was, in fact, taking powerful drugs, whether or not they came in a pill form or as ground up leaves. He refused to believe me. I asked him if he could stop taking either one of his "supplements." He assured me that stopping would not be a problem, that they were sort of like vitamins: helpful, but not crucial for activities of daily living.

It took Lance eight months to get off the Macuna, during which time he felt all the usual symptoms of drug withdrawal. As he always put it, *"I'm still reducing the Macuna, but it's killing me, it's just killing me."*

He is now off the Macuna and still working to slowly get off of Deprenyl. For more than a year he has been going through agonies. "*It's killing me, just killing me*."

Another Macuna case study

A patient who visited our clinic had taken a Macuna extract that contained10% levodopa. She took 1000 mg/day for two weeks. (This would be equivalent to 25 mg/day of carbidopa-levodopa. Without a buffer such as carbidopa, levodopa is only about 25% as effective: one must take four times as much levodopa as they would carbidopa-levodopa, to get the same effect as Sinemet.) When she tried to double the dose, as instructed, she felt more rigid, and resumed it at the lower dose. She took the Macuna at this level for nearly four months before she stopped abruptly. Three months after stopping she had not noticed any traumatic reduction or withdrawal side effects. Her Parkinson's symptoms worsened; for example, she became more rigid. However, she did not experience the reduction symptoms of nausea, insomnia, paranoia, or longing for Macuna. She had not yet recovered from Parkinson's, and she was taking less than the "small amount" that is listed for levodopa in chapter 17.

Vicia fava, Fava beans

Fava beans (*Vicia fava*) also contain L-dopa. However, a very large amount of daily fava beans is required to give the same effect as the buffered carbidopa/levodopa. A few people with mild Parkinson's find that they benefit from this food. You can find articles about it on the Internet. Most people do not get enough benefit from this food to overcome their Parkinson's symptoms. If they do, they run the same risks as a person who is taking L-dopa. The brain does not differentiate between L-dopa from a veggie and L-dopa from a vial.

On the other hand, dopamine levels are subject to the placebo effect. If by taking fava beans you can feel better about yourself than you would if you were taking a drug, then by all means, do so. Possibly the positive attitude will help you increase dopamine levels naturally. Attitudinally-produced dopamine never goes over the Safety Limit.

The safe new discoveries

As you have learned, short of the placebo response, there is almost no way to pharmaceutically or herbally increase dopamine without quickly setting in motion an addiction response. Whether the dopamine level is raised through the mechanism of 1) taking the place of dopamine on the dopamine receptor sites (agonists), 2) increasing

dopamine release from vesicles (methamphetamines), 3) increasing dopamine derivatives (such as serotonin and norepinephrine) so that the dopamine supply backs up, 4) slowing the reuptake of dopamine (cocaine), or simply 5) inserting dopamine precursor (levodopa) into the brain, the final result will be that dopamine levels will temporarily go up. This rise in dopamine will trigger an addiction process. After that process has been initiated, the native dopamine levels will be permanently or semipermanently lowered.

Because addiction-lowered dopamine levels can cause slow movement, rigidity, poor balance, and tremor – in other words, parkinsonism – it is clear that any drug for treating Parkinson's disease that works by enhancing dopamine may eventually create parkinsonism.

As I was writing this chapter, I got an email from someone asking about a hot new form of a Deprenyl-like supplement for Parkinson's disease that is sold over the counter and which has "all the addictive parts removed, and only the effective, dopamine-stimulating parts remaining." He wanted to know what I thought.

Here's what I think: if a drug elevates dopamine, then it is addictive. How dumb do they think we are? Pretty dumb, evidently.

"To live by medicine is to live horribly."
from the introduction to Diaeta Naturalis, Carl Linnaeus (1707-1778)

11. THE BIG D: DYSKINESIA IN DEPTH

This chapter will touch on the significance of dyskinesia, the inability of a patient to perceive his own dyskinesia, and what dyskinesia looks like. I will also attempt to pound into a paste the fact that violent, super-human tremoring is drug-induced and not a symptom of Parkinson's. The redundancies in this chapter are intentional and are due to the second item on the list above – a person with dyskinesia cannot easily perceive that he is overmedicated. This redundancy is a last-ditch attempt to get through to a drug-addled patient. For the rest of you, a quick skim over this material should suffice, and then on to the next, more succinct chapter. For the deeply drug-lost, no amount of information will get through; the hammering and redundancies may simply be a waste of time. However, if even one person is able to see himself more clearly due to the textual repetitions, it will have been worth it.

Dyskinesia: serious brain damage made visible

Most of the changes that take place during addiction are invisible. However, when the brain is so severely overloaded that even addiction changes are too slow and inadequate to protect the brain, dyskinesia begins. Dyskinesia is a clear sign that brain damage is ongoing.

Due to the invisibility of most changes related to drug overdose, dyskinesia is the best objective tool that most of us have. A truly observant person may notice the other, more subtle signs of overmedication: an uncharacteristic lightening of mood, a change in sense of humor, untoward social behaviors and disinhibition. However, a drugged person, like a staggering alcoholic, may insist that these personality changes are by choice. By the time dyskinesia appears it is already too late to prevent ongoing brain damage, but the ease of eyeballing dyskinesia makes it possibly the single most valuable tool in determining whether or not a patient is overmedicated.

Manufacturers' warnings about dyskinesia

The drug books suggest that dyskinesia is an "adverse effect." Adverse effect does not mean "curious side effect." "Adverse" means "harmful." As an example of just how harmful it is, the warning insert for Carbidopa/levodopa states, "Muscle twitching or twitching of eyelids may be signs of overdose. Treatment of overdose includes gastric lavage [stomach pumping] and antiarrhythmics, if needed." The manufacturers of Permax say, "Toxicity may cause involuntary movements." Mirapex makers advise to "adjust dose gradually to achieve maximum therapeutic effect balanced against the main adverse effects of dyskinesia (and) hallucinations…" (Hallucinations can be signs of brain damage.) This last does not mean "strike a happy medium between movement and dyskinesia." These warnings mean "get as good a result as you can while staying far clear of danger signs: dyskinesia and hallucination." Eldepryl manufacturers warn, "In patients

who experience an increase of adverse reactions (including dyskinesias), reduction…is necessary."

To put this into a frightening perspective, most people with dyskinesias – and their doctors – accept monstrous levels of spasm and writhing as "normal side effects" of their drugs. And yet the manufacturers are alarmed at the merest twitching of eyelids. The researchers who should know best about these drugs suggest that at the first sign of any twitching, the drugged one is so dangerously top heavy that he needs gastric lavage, drugs to prevent heart attack, and, of course, a reduction in dose.

The wide difference between the alarm with which the manufacturers regard mere twitching and the insouciance with which doctors and patients alike dismiss violent dyskinesia should tell you that something is rotten in the way these drugs are being administered.

So, once again, risking redundancy, here it is – any sign of dyskinesia is a sign that brain damage is ongoing. There is NO acceptable level of dyskinesia. Consider every twitch to be another handful of brain cells being dispatched to the graveyard.

Drugged patients are not objective observers

The next point is this – an overdrugged patient is usually incapable of discerning whether or not he is having subtle dyskinesia.

I will say it again. If you have dyskinesia, you are overdrugged, and probably incapable of objectively noting the extent of your problem.

Therefore, if you are taking antiparkinson's medications, you are worthless as a judge of your own condition if you are overmedicated and having even the slightest twitching of the corner of the mouth or the tiniest curling of the toes. Even if you think you can detect these movements, your evaluation of them is suspect, and your decision-making abilities are clouded.

Now for a qualifier. A person with Parkinson's disease may be slightly more able to discern the dyskinesia than a person who is recovering from Parkinson's. Therefore, even though the rare person can tell when he is twitching prior to recovery, this does not mean that he will be able to so discern if he begins to recover.

Examples of poor judgment regarding dyskinesia

Now, in anticipation of patient protest, I will make some comments from case studies.

One woman who was deeply conscious of her facial appearance was keenly aware and deeply mortified that she had uncontrollable pouts and twitches in her cheek. She was not aware, however, that she was constantly rotating her shoulders.

Another person who knew that his arm was slamming uncontrollably into his backside was unaware that his head twitched from side to side. Nor had it occurred to him that his intestinal spasms, for which he was taking anticonvulsant drugs, were also forms of dyskinesia.

A drugged person may be aware of some aspect of spasming that is occurring. However, in most cases, he is not aware of the full extent of the twitching, and he is almost never aware of how hideous it appears to the outside observer.

Patients rarely mind dyskinesia, despite warnings

I have heard from many doctors that dyskinesia worries them, but it doesn't appear to bother the patient. Dr. Rafferty said, "Every one of my patients prefers dyskinesia to being undermedicated." In the most recent edition of *Parkinson's Disease – Questions and Answers*, it states: "Both off time and dyskinesia can be disabling. Most patients prefer dyskinesia to off time and most family members and physicians prefer for the patient to be off than have dyskinesia."[1]

As an example of the "family preference," I recall a 51-year-old patient who, pre-drugs, had been of a quiet and reflective nature, deeply devout: a kind doctor (MD) and a loving father to his four children. After ten years on Sinemet, he had dramatic Ons and Offs. During his Ons he was jovial to the point of buffoonery. He could have challenged any court jester and won Harlequin Honors. He happily played his violin for me and bounded around the room when On. When Off, he sat rigid in a chair, drooling, his voice a faint whisper. His devoted daughter begged me to help him reduce his drugs. Sad rage came into her voice as she sighed, "I hate it when he is drugged. He is a stranger. He is a fool. I want my father back. That man, that crazy man, is not my father. Even when he is just sitting there drooling he is more like my real father than when he has that creepy smile." As she said this, her father, just starting to come back On, beamed in amicable pride at her loving concern. But he did not share her feelings about the medication.

I have seen loved ones observing with horror the marionette movements of dyskinesia, while the twitching one laughed aside the concerns.

Therefore, I will say it again: a drugged person who is having dyskinesia is not a competent judge of his own condition.[2]

Next, as an adjunct to this mantra, any person who wants an objective evaluation of his condition with an eye to detecting overmedication must have a friend, caregiver, spouse or loved one who is able to observe him throughout the day and who knows what overmedication looks like. Because appropriate drug reduction, as described in chapters ahead, requires daily evaluation of symptoms and immediate response to the slightest indication of change in drug response, an objective observer is a key part in the drug reduction process.

What dyskinesia looks like

Dyskinesia has already been described briefly in chapter five. It would be impossible to list all the permutations of dyskinesia, but hopefully, the expanded list of symptoms below will be a nice starting point. I am only mentioning a few forms just to give you the idea. I have never seen two patients with identical dyskinesias.

---

[1] R. Hauswer (Associate professor of neurology, South Florida College of Medicine, Director, Parkinson's Disease and Movement Disorders Center), *Parkinson's Disease: Questions and Answers* (Third edition), Merit Publishing International, 2000, p. 94.

[2] Recently I received this email from a former patient: "I was invited to the local PD support group to speak about my own experiences during recovery. They all seemed so normal at first, but then it dawned on me that *nobody* smiles that consistently, and sure enough the stories of problems with the meds began to surface." Those strange, blissful smiles were caused by the listeners' medications.

Starting at the bottom and moving up, dyskinesia can include the following: uncontrolled toe spasming, curling, tapping, ticcing, or dystonias; foot spasms, cramps, random movements (violent, smooth, or writhing), ticcing, or dystonias; ankle rotations or wiggles; leg spasms, cramps, or jerking, bouncing strides, kicking, rising up on the toes when standing still or walking, overly fast walking, uneven or loping gait with or without one leg moving more exaggeratedly than the other; rocking from one buttock to another when sitting; hip pain, spasms, or cramps; buttock twitching; intestinal spasms (may feel like gas pains); spastic bladder, rectum, abdominal or stomach muscles; back cramps or spasms, torso wiggling, spasms or rotations; diaphragm spasms, uneven breathing, heart arrhythmias (irregular heart beats); shoulder twitching, shrugging, or rotating; random arm movements, overly fast arm or finger movements, arm flapping, spasms, ticcing; hand clenching, flapping, finger ticcing; neck twisting or cramping, choking, head bobbing, repeated chin thrusting; tongue thrusting; pouting, grimacing, lip spasms, grinning, teeth clenching, cheek spasms or cramps; eyelid twitching or ticcing; forehead lifting; eyeball rolling or spasming; upper body writhing, jerking or twisting; lower body writhing, jerking or twisting; and whole body fits and starts.

Also, overly rapid or over loud speech, helplessly repeating a word or phrase, or strangely quick or darting movements that were previously (pre-drugs) not a part of one's repertoire may indicate gross overmedication. The unmedicated speech pattern of PD is a slow, faint plod – words may take several moments before they drop off the lips. Impossible to understand rapid speech or stammering is a form of dyskinesia, not PD.

Dyskinesia can take the form of psychomotor excess. In this case, the patient may wish to show off how well he can run, jump, or perform feats of strength and manual dexterity when medicated, or even compete with others. There is typically high dudgeon on the part of the patient if the suggestion is tendered that it is the drugs, not the patient's fabulous, can-do attitude, that are creating the supreme motor skills. Dyskinesia can

To capture the feeling of the writhing type of dyskinesia, the artist drew this series. These stills were derived from a fluid demonstration. The model was demonstrating a sampling of the arm and head movements plus the facial expressions made by Rose, a patient described in chapter one.

In the artist's words, "Maybe the reader can put the pictures together into a flip book to get a sense of the rapid transitions and flailing movement.

occur in one muscle or in groups of muscles. It can be intermittent or repeat hundreds of times at impossibly fast speed.

Uncontrolled movements: twitches and tics

Twitching is uncontrolled muscle movement.

Ticcing is *repeated* uncontrolled muscle movements. A tic is "any involuntary, regularly repeated, spasmodic contraction of a muscle, generally of neurotic origin."[1] Another definition states that a tic is a "spasmodic muscular contraction (where)…the movement appears purposeful, is often repeated, is involuntary, and can be inhibited for a short time only to burst forth with increased severity…It can be tonic (from constant muscle tensing) or clonic (from alternating tensing and relaxing)."[2] When Oliver Sacks tested L-dopa on patients who had been previously rigid (paralyzed by sequelae of a viral sleeping sickness), many of them developed powerful, rapid spasming that resembled a grotesque parody of the quavering, delicate Parkinson's disease-type tremor. He properly referred to this as ticcing.

Ticcing can be an adverse effect of nearly all Parkinson's disease medications.

Conscious control

One may be able to consciously restrain dyskinetic movements. Many people who were rigid prior to taking medication protest that the rhythmic movements induced by the drugs are not dyskinesia because they are "by choice," and "feel good." They often point to the fact that they can stop making the moves with a conscious effort. However, any pill-induced, involuntary *urge* for movement is dyskinesia, even if it can be consciously restrained.

I hope I have conveyed something of the unawareness of the person in the throes of overmedication. The dreamy expression seems to exemplify the desperation of the situation; the outside observer can see this person is moving irrationally, but often the subject is not aware of it."

Even if the subject is aware that he is making excessive movements, he is rarely aware of how dreadful it appears to the observer; he imagines that it is subtle, or graceful.

[1]*Webster's New World Dictionary*, World Publishing Co, NY, 1970.

[2]*Taber's Cyclopedic Medical Dictionary*, F.A. Davis Co, 1989.

The physiology of dyskinesia

Dyskinesia occurs when the body uses up excessive dopamine by burning it off in muscle movements. Dyskinesia occurs in those muscles that are the least able to resist. Usually, the muscles least able to resist are those already weakened or damaged by illness or prior accidents. Limbs that have been weakened by injury or previous illness, including polio, frequent sprains, bone breaks, poor posture, Parkinson's disease or other health conditions, may be the brain's most likely choice for working off dopamine excess with dyskinetic bursts. People with a history of heart disease or angina might have heart arrhythmias. People with a history of asthma might have throat, chest, or diaphragm spasms. In general, dyskinesias seem to begin in those parts of the body that are weakest in that particular individual.[1]

Similarities with tremor: dyskinesia near the tremor zones

Because the body uses the weakest muscles to work off its brain excess, tissues in the vicinity of the tremor lines are often called on to serve dyskinesia duty. If there is still some residual amount of connection between the tremor areas and the brain, these areas alongside the tremor tissues may also be used. The rhythm of tremor may even be used as a starting point for rhythmic dyskinesia (ticcing); the brain may employ a pre-existing tremor – a site of weakness – and the theta wave rhythm to get relief from excess dopamine.

Drug-related twitching, as with many drug adverse effects, may have two causes; both are opposite sides of the same coin. It can begin in response to excess drugs and may return in an amplified form if the body is deprived of the drugs.

Tremor can begin as a manifestation of the brain's theta wave in atrophied tissues that have only a small, tenuous, residual connection, if any, with the command centers of the brain. The tissues immediately adjacent to the tremoring ones, though not quite disconnected from the command center, are yet weak, and thus easily employed by the brain's drug relief center, should excess dopamine ever be detected. In the case of such excess, the powerful dopamine-discharge mechanism can create a large, ticcing parody of the tremor using tissues adjacent to the tremor area and moving in a pattern similar to the established tremor. Therefore, a dyskinetic, amplified version of the tremor may occur in

---

[1] Curiously, once this habit of crazy, excessive muscle movement begins, the brain will continue to perform it, as if the brain has learned to associate this movement with correct or even pleasurable behavior. Although one's conscious mind may find the dyskinesias appalling, the altered limbic area has determined that jerking this muscle around is a good thing that should be performed as often as possible, and especially when under stress.

Driving home the point made above, many people who develop spasming or ticcing in response to elevated dopamine levels will continue to perform these motions for the rest of their life, even if the drug is discontinued. In fact, these patterns, developed as a way to combat traumatically excessive dopamine levels, will be employed if the limbic area is traumatized by a *decrease* in dopamine. In other words, the same movement patterns, once they have been learned, will be employed by the brain in times of stress, whether the stress is coming from excess dopamine or deficient dopamine. Unlike the relatively short-term (ten week) brain changes of addiction, the limbic brain's movement patterns are very hard to change. They may last for a lifetime.

the same areas as the pale, resting tremor of Parkinson's. It is nearly impossible for a drugged PDer (or an unobservant MD) to distinguish between them.

Once this ticcing behavior has been established, the body may also use this new dyskinetic ticcing anytime that it wants to express displeasure, such as when the drugs are excessive, when they wear off, or during times of drug reduction or withdrawal.

To complicate matters, any person who has ever taken antianxiety or antidepressant drugs for more than a few weeks may, at some much later date when the pill use is long forgotten, manifest a ticcing form of tardive dyskinesia that also resembles tremor. Tardive dyskinesia is discussed in further detail later in this chapter.

Violent ticcing, often called (erroneously) violent tremor

A particularly violent form of tremor-like movement can be triggered by the medications. It is actually a form of dyskinesia. Most people call it violent tremor. However, it really should be called violent ticcing, a form of dyskinesia.

Violent ticcing is much more powerful than the tremors of unmedicated Parkinson's disease. I have patients with these hammering, drug-induced exaggerations of ticcings. When they are shown videos of unmedicated patients with their classic PD tremor, they are just astonished at the feeble, quavering, pathetic tremor. "Is that what a tremor is like?" they ask. "That's nothing like what I have! My shaking shakes my whole body; it shakes me like a rat! That video *can't* be showing tremor. What I have, now *that's* tremor!"

Drugs ease the violent ticcing and also cause it

In any given drug dose, the medication can both cause ticcing and temporarily ease it as well.

While a dose of drug is starting to be absorbed and the drug levels in the brain are in the ideal zone, just above the threshold line and below the excess line, the dose of the drug may stop the tremoring. However, as the dose continues to be pulled through the blood-brain barrier, raising dopamine levels above the excess line, the violent ticcing may begin.

An illusion of pills curing ticcing

Because the addicted brain uses its limited repertoire of tricks to complain when dopamine falls below the threshold level, it may well be that the body will use violent ticcing to protest when the dose wears off. A pattern of violent ticcing both prior to a dose and during the high point of a dose may occur. These periods correspond to when the dopamine levels are below the threshold or in the excess zone, respectively.

A dose that begins below the threshold and eventually rises into the excess zone may create this illusion: ticcing due to deficiency (induced by addiction) will be followed by a brief period of good feeling and no ticcing while the brain level is in the narrow window of On time. This period of good feeling in the first flush of dopamine (anywhere from ten to ninety minutes after taking a pill) convinces many people that the ticcing is indeed a part of Parkinson's, and the pill is the remedy. The subsequent increase in ticcing, anywhere from twenty minutes to two hours later (while the drug is at its *highest* levels in the brain), is then incorrectly perceived as insufficiency of medication. The usual (wrong) assumption is that the effects of the dose have worn off prematurely.

Following along the timeline of the drugs in the brain, the excessive levels of drugs will eventually be answered with a crash in dopamine levels (the hangover phase) that is worse than the original condition. In such a case, when the drugs wear off completely, stressing the brain, a body that ticced violently to protest an excessive drug may tic violently again to protest the sinking of dopamine below the threshold. A person trying to make sense of this – "ticcing before a pill, ticcing during a pill, and ticcing after a pill" – will usually conclude, wrongly, that his medication is simply too weak, and may increase his pills further.

Repeat

The muscular, powerful dyskinetic variation on tremor is not tremor. Most people who develop it call it tremor. It is actually a form of ticcing. Ticcing is sometimes mislabeled as tremor by undereducated neurologists.

Violent freezing: an extreme form of dyskinesia

Violent freezing is a form of dyskinesia in which the whole body is seized in a one-muscle or body-wide muscle cramp. It can be powerful, painful or not painful, brief or long lasting. It looks like supreme immobility.

This violent freezing is caused by extreme overmedication, as opposed to the freezing of PD which was described in chapter five. The word "freezing," unfortunately, has been adopted to describe both this drug-induced pattern and the sudden lack of movement initiation that is inherent in unmedicated Parkinson's disease.

One type of violent freezing is actually a form of excess muscle tension rather than lifeless rigidity. It occurs when a person's brain dopamine levels rise above the excess zone, into the dyskinesia and freezing zone. Though these periods of intense immobility are called Offs, they are actually a form of dyskinesia, or wrong movement, and are triggered by drug excess.

An example of violent, drug-induced freezing occurred in Rufino back in chapter five, when, an hour after taking his evening drugs, most of his body became "like stone" and his arm was jerking like a piston. Laurel, about whom you will read later, had violent freezing dystonias in her hips when her medications were at their highest. In this type of dyskinesia, a body part may twist into a horrible spasm and stay there for minutes or hours. Due to the immobility created by these spasms, many people assume that violent freezing is a sign that their drugs are not working and respond by increasing their medications.

Shut Down: a type of violent freezing

Another form of violent freezing is the Shut Down. In this type of Off, there is no obvious cramping but the body becomes extremely rigid, as if the motor function of the brain has completely shut down. This is probably a protective mechanism, as it seems to occur most often when the PD medications are at their highest brain levels, a few hours after taking a pill. This Shut Down may be preceded or followed by frantic dyskinesia or it may not.

Just so you don't try to pigeon hole this symptom in an attempt to prove that this immobility during peak drug hours is a form of pill failure, I will add that the Shut Down may be preceded or followed by normal movement, apparent wearing off of medication,

violent freezing, weakness, or fatigue. Everyone is different. The best indication that immobility is coming from drug excess and not Parkinson's is the timing: if it occurs more than half an hour after taking the drug, and eases up within six hours after taking the drug, it is a symptom of overmedication – an extreme form of dyskinesia, and one that represents gross overmedication.

A Shut Down may occur because the brain is temporarily stopping all motor function rather than experience a level of motor function so frantic that harm might occur. It may feel just like unmedicated PD, or even worse.

Sometimes during a Shut Down a person can shuffle and drool, and look just like severe Parkinson's disease. There may or may not be an excessive gleam in the eye or lack of coherent thought to prove the point that this Shut Down is from drugs, and not PD. Still, if you need proof that the Shut Down is due to drugs rather than PD, take note: if a person can move better in the morning *prior to his first dose* than he can during six hours following any other given dose, it may be because the drug is so excessive that the body is going into severe shock. We consider this Parkinson-like complete immobility from drug excess to be a form of dyskinesia – literally, "wrong movement."

Clarity of vocabulary

If you hope to make any sense of your drug responses or chart your way through drug reduction, you will need to determine whether your behaviors are coming from too much dopamine or not enough. In the case of violent freezing and Shut Downs, too much dopamine and not enough dopamine can look very similar. Your MD, during your brief visit twice a year, will probably not be able to distinguish between the two based on your description, thanks to our poverty of vocabulary.[1]

Drug-induced addiction causing parkinsonism → increased freezing

Via the addiction processes, drug excess can cause parkinsonism. A symptom of parkinsonism is freezing. In other words, the medication can make two kinds of freezing: drug-induced freezing corresponding to a specific dose, and an increase in the ordinary tendency towards freezing that accompanies parkinsonism. This double potential to cause freezing of either kind explains why all of the antiparkinson's drugs list "freezing" as one of the adverse effects of the medication.

Tardive dyskinesia

Nearly all of the dopamine-, norepinephrine -, and serotonin-enhancing drugs can cause tardive dyskinesia. Tardive means delayed, or late, as in the word "tardy," and refers to the late or delayed onset of the tapping, tremor-like movement; this symptom may not even appear until years *after the medications have been stopped*. It is a long-term problem: it is not dose related. It is semipermanent.

Tardive dyskinesia most often takes the form of finger tapping motions that can superficially resemble the tremor of Parkinson's disease. However, it does not necessarily occur along lines of PD atrophy. Also, unlike a resting tremor, tardive dyskinesia can

---

[1] I request that one of you eager young neurologists, anxious to get your PhD in research, should invent some new words so that drug excess immobilities have different names from non-drugged immobilities. You will get your degree and the rest of us will be able to communicate.

appear more like a nervous tic. Any PDer who was ever overmedicated at any time, however briefly, may have – soon or someday – lifelong tardive dyskinesia that may or may not resemble his old tremor even if he recovers from Parkinson's disease or stops taking his drugs in a timely fashion.

What does tardive dyskinesia look like?

Tardive dyskinesia is the restless, constant tapping motion of the hands, feet, arms, legs, or neck that is seen in patients who have taken antipsychotic, antidepressant, antianxiety, or dopamine-enhancing medications at some time in the past. This drug-induced tapping is so common now that, when we think of people in mental institutions, we picture them in our mind's eye as having a glazed or vacant look and restless patterns of finger tapping and other repetitive movements.

These repetitive movements are not historically a necessary adjunct to depression, anxiety, or other mental disorders, nor are they necessarily a part of many psychiatric disturbances. These modern nervous tics are often caused by the medications that are used for these disorders. If you have taken antiparkinson's medications and eventually recover from Parkinson's disease, you may find that you will have similar tapping, ticcing movements for the rest of your life. These permanent afflictions are caused by damage that dopamine-altering drugs do to the brain.

While PD appears to be curable, the drug-induced parkinsonism movements such as tardive dyskinesia are not – at least not yet. Bear this in mind when you imagine that you must increase your medications to help with your tremoring; you may already have permanent ticcing due to your medication.

When do dyskinesias begin

Dyskinesias do not always occur in the first few years of drug therapy – though they can. Eventually, however, if drug use is excessive, they will probably arise.[1] They develop from long-term use of the drugs and also from short-term excessive levels of the drug. The data on dyskinesia is still changing.

From the 1970's to the 1990's, it was thought that most PDers could have up to five years before they developed dyskinesias (known as the five year honeymoon). Now, in the first decade of the twenty-first century, the general consensus in the literature is that half of the people taking L-dopa develop dyskinesias within two to five years. This may be due to higher prescribed starting doses; a prescribing theory that I have heard with increasing frequency is that newly diagnosed patients should start with three pills per day and take an additional pill every day until they start to feel good. Considering that these drugs can take several months before the full effectiveness appears, it is no wonder that some patients, after following this regrettable suggestion, have starting doses of 3000 mg/day levodopa even though the manufacturers suggest as little as 300 mg/day as a possible starting dose.

---

[1] The warning insert for Carbidopa/levodopa states, "Muscle twitching or twitching of eyelids may be signs of overdose. Treatment of overdose includes gastric lavage and antiarrhythmics, if needed."

Can you imagine? The manufacturers suggest that even simple muscle twitching of an eyelid should be met with stomach pumping and heavy sedatives. How do most PDers then defend their decision to take more medication to treat their grimacing and violent ticcing? I suspect that some of this defensiveness relates to the unsuspected emotional attachment that many PDers have to their medications.

Furthermore, some doctors do not understand that when they prescribe two antiparkinson's drugs concurrently, they must greatly reduce the doses of one or the other or both. It is no surprise then that we are seeing demographics of earlier onset for dyskinesias and other drug side effects.

I have even had patients who were put on the highest levels of anti-PD medication after being misdiagnosed with PD by a general practitioner or a surgeon in the emergency room. Thirty percent of the patients who come to me for help with their Parkinson's do not even have Parkinson's disease.[1] If their diagnosis is doubtful to me, I send them out for a second opinion and insist that they see a neurologist. Usually, by the time they learn that they do not have Parkinson's, they are addicted to the drugs and have beginning signs of parkinsonism, in addition to whatever their actual, misdiagnosed problem happened to be.

These should-be-criminal wrongs might be avoided if all doctors took the time to read the drug information, and if they would stick to their specialty instead of confidently misdiagnosing. Please excuse my warmth, but I have seen patients in irremediable agonies and I have had patients die, simply because their doctors could not be bothered to learn to diagnose correctly or to read the drug directions provided by drug manufacturers.

The onset of dyskinesia varies from patient to patient, from drug to drug. Dyskinesia or other side effects may begin when the drugs grossly exceed the Safety Limit. Any sign of dyskinesia is a sign of gross overmedication and addiction. The dyskinesia can be subtle at first, the slightest twitching of the toes or uncontrolled lifting of an eyebrow, but even such slight movements are proof that brain damage is occurring.

Modern Parkinson's

Because there is so much confusion about the dyskinesias of Parkinson's disease, and because even many doctors now associate excess movement with Parkinson's, I will risk redundancy to mention again that good old-fashioned Parkinson's disease, the historical, *pre-medication,* "poverty of movement" version of Parkinson's disease, is very different from the active, twitching, spasming, fast-talking Parkinson's that you see today.

Repetition

Many people consider the dyskinesia or painful violent freezings to be the hardest part of their Parkinson's to bear. But in fact, these are not a part of their Parkinson's. The wild, disordered movement, often considered even by modern doctors to be the worst part of Parkinson's disease, is not a part of Parkinson's disease, but is dyskinesia *caused by the medications for Parkinson's disease*.[2] Sorry to go on and on about this, but this issue comes up again and again and again.

---

[1] This number matches recent brain autopsy studies. Almost a third of supposed PDers are revealed – during autopsy – to have no symptoms of PD-like changes in the midbrain's substantia nigra. In other words, they were misdiagnosed.

[2] Because so many patients and their doctors just assume I am wrong about the drugs causing the movement, I am pleased to use a reference by the doctor who is the National Medical Director of the National Parkinson's Foundation, Inc. This article is very easy to find, and the author's conservative position and authority are irreproachable. Many people assume that, since I am an acupuncturist, I am rabidly anti-drug, and that I am probably part of some cult that advocates "acupuncture instead of real

In my years of working with PD patients, dyskinesia is the concept that they have the most trouble with. This is a real problem because PDers suffering from dyskinesias are reluctant to reduce their medication. They often insist on increasing the medication to "treat" the dyskinesia. And in the short term, for maybe up to three months, an increase in medication may reduce the dyskinesia or make it seem less problematic. (See Superdosing.) However, within a few months, the problems are even more severe.

Parkinson's causes rigidity and poverty of movement. Parkinson's can cause tremor and an adrenaline (stress or anxiety)-related, fast, exhausting, frantic, but reinable exaggeration of the tremor. The medication causes dyskinesia and excess movements. The super-fast hyper-speed ticcing that is an inhuman, machine-like amplification of the tremor movement is a form of dyskinesia; it is not tremor.

SUMMARY: COMPARING TREMOR AND DYSKINESIA

When trying to distinguish between tremor and dyskinesia, keep in mind that tremor is like trembling. The tremor in the chin looks like a person who is trying not to cry. Chin tremor does not look like a person who is chewing on four-day old biscuits. Tremor happens in an animal that has been injured and is lying very quietly, maybe in shock. Tremor occurs when you are too cold or when you are frightened, and it is vibratory, not muscular. Tremor is helpless like a startled baby. Resting tremor ceases briefly when the tremoring limb is used in an activity. In times of adrenaline stress when action is inappropriate, the tremor can become amplified. These amped-up movements are along the exact same areas of the body as the resting tremor, but they are larger, more powerful, and do not respond to conscious control. They ebb when the situation changes, and they are not dose related.

The medication can cause excess movements in nearly any muscle of the body. They can be rhythmic and repetitive, in which case they are often confused with tremor, but they are more commanding than tremor, and unlike resting tremor, they may or may *not* cease when the shaking limb is called into action. Because the learned patterns of dyskinesia, especially ticcing, can become part of the behaviors your brain uses to indicate stress, these patterns may not necessarily be dose related – they may occur when your drugs wear off, as well as when the drugs are at excessive levels.

Questions and Answers

These are actual questions that I have received after presenting the above material on dyskinesia.

Q. I have a really bad tremor, but I know it is caused by the Parkinson's disease and not the medication, because after I take my L-dopa pills, it stops for almost two hours and then comes back worse than ever. I think I should increase my medication, but the last time I did that the tremors got worse. Did I not increase enough?

---

medicine." No, I'm not an anti-drug nut, and I study the same journals and authorities that your doctors do (or should), but with the difference that I am a researcher, so I have to read more articles more closely, and stay much more current. So, please read the article on Curing Parkinson's Disease in Our Lifetime, Part 3, by Lieberman, MD, National Medical Director, in the Fall 2000 issue of *Parkinson's Report*, the magazine of the National Parkinson's Foundation, page 10. The author talks about the "involuntary, writhing movements (dyskinesias) that result from prolonged and excessive levodopa use."

A. Remember, your pills are at their most effective about two hours after taking them. The shaking that starts two hours after taking your pill is being caused by the pills reaching maximum effectiveness. In other words, your shaking is not tremor, but is pill-induced dyskinesia. Note that your shaking got more powerful after your last drug increase.

Q. Why do you think that this worsening of my shaking isn't being caused by the worsening of the Parkinson's disease?

A. Parkinson's disease, in an unmedicated person, never attains that violent level of shaking that you are exhibiting. When I was talking about violent ticcing, the other members of the class were pointing at you. Also, I have seen people who argue just as you are doing, and who have been shocked and amazed when, by decreasing their medication enough, for a long enough period of time, their violent tremor abates and they once again have the weak, fluttery tremor that they used to have.

Q. How much drug reduction is necessary, for how long, to enable the shaking to decrease and be restored to the normal resting tremor? How long before it stops altogether if I recover?

A. It is impossible to answer that question. Everyone is different. Because you are dyskinetic right now, you have damaged your brain. You will probably have some level of tardive dyskinesia (finger tapping, twitching, ticcing, or tremoring) the rest of your life even if you were to stop taking your meds and recover from Parkinson's disease.

"Doctors prescribe medicine of which they know little, to cure diseases of which they know less, in human beings of which they know nothing."

Voltaire (1694-1778)

12. Leading Up to a Solution

Another Death; The National Report

What was happening in the meantime for the rest of the people in the project? By June of 2000 we'd had nearly fifty patients sign up. We'd had two patients who had been recently diagnosed with PD and who had been on very low levels of medication. These two had experienced what we were beginning to recognize were symptoms of recovery. They quickly had gotten off their medication and had some moderate, passing difficulties, just as Becky had had during her very first experience in getting off the drugs. They had never used the drugs again. A few others had gotten off their drugs more slowly and then followed a course similar to Becky's when she chose to give herself a "little boost:" increasing need for the drug and rapidly worsening adverse effects.

We had some patients taking high levels of medication who were still in the early stages of gradually reducing their medication but appeared to be making progress and avoiding Beckyesque pitfalls.

Those who had joined the program and were not taking any PD medication found that their recovery moved along pretty much as is described in the recovery handbook: recovery was painful, slow, and fatiguing but lacked the terrors and paranoias that some medicated patients were having.

The undrugged stories were somewhat predictable. Every medicated case was unique. Most were dreadful, but a few were astonishing; Buzz's story simply floored us, for example. He quit his high level of medication abruptly as soon as he detected recovery-like changes and, against all odds, survived the nightmare.[1] He subsequently made a straightforward and unusually fast recovery from Parkinson's almost as if he had never been drugged. The only indication that he had ever taken the drugs was the telltale (and probably permanent) tardive dyskinesia, the rapid hand shaking that appears to be the long-term Kilroy Was Here of all dopamine-enhancing drugs.

We were beginning to learn from working with the patient researchers that there were good ways to attempt med reduction as well as bad ways. This chapter chronicles this period and introduces some of the early findings as we stumbled on them in our regular visits with the pioneers. We hope the tragic loss of some of our pioneers, described here, will illustrate the dangers involved in using medications for Parkinson's disease once the PD has begun to recede via the recovery process.

[1] We do not advocate this method. Abrupt cessation of these medications can cause death. This person's case will be described later in great detail as an example of what *not* to do, and why.

Failure, for the most part

Most of the medicated patients were struggling, unable to tell if they were getting better or worse. Their attempts at reducing their drugs were futile; they could not tolerate the powerful symptoms of drug withdrawal. They kept waiting for a sign from their symptoms or leadership from their doctors, but they got neither, and their overmedication got worse and worse. A tragic few, like Zoe, were lost forever in a nightmare world of pills, confusion, and relentless, writhing torments. Two people, sweet Rose and one other, had died from the effects of overmedication.

There seemed at this time to be no way to guess who would be successful in reducing their meds and who wouldn't be. It appeared to be one big crapshoot, one with potentially lethal consequences. And if a few were successfully getting off their meds, the majority was not.

Drugs, not attitude, were the problem

Some patients entered into the project brimming with optimism. Others were shoved, sulking and skeptical, into our clinic by an adamant spouse. But curiously, attitude was *not* the strongest predictor of recovery; whether or not a person was using anti-PD drugs seemed to be the single most critical factor; their attitude was a distant second in importance. Most people simply couldn't reduce their drugs, not even by a slight reduction of one or two pills a day. (Back then, we didn't know what the word "slightly" meant.)

After two years, we began to recognize that the all-at-once and the pill-a-day decreases were formulas for failures. The 10%, two-to-ten weeks reduction program, hit upon by chance, seemed the most successful. The 10% rule that we came up with will be described later in this chapter.

There were exceptions to the 10% rule. If a person was already manifesting a significant number of recovery symptoms, they might already be too far addicted to make any reductions. Zoe, for example, could not tolerate a decrease as small as 2.5%. Also, if a person's symptoms of overmedication were too severe, the 10% rule might be helpful, but every single decrease in what might be a multi-year process could very likely set off a ten-week round of withdrawal agonies similar to Becky's.

Therefore, although we had found what appeared to be a safe way to reduce medications for some people, there were still many qualifiers. It was not a one-size-fits-all proposition.

The most curious item noted above was that those drugged people whose subtle electrical patterns had been restored to health, *whether or not their mobility had yet improved*, were the most at risk. If they were not taking steps to decrease their medication, they might end up deranged, dead, or wishing for death within a year or two. By this time we had seen several examples, in addition to Becky, of the change in addictability that occurred after a person began to recover.

Samantha

Samantha (Sammy to friends) had been recently diagnosed and had only taken Eldepryl, an MAO inhibitor, twice a day for a few months. She was 55 years old at the

time. After a month in our program, she started noticing changes in her feet and decided to cut back on her medication. After two months of steady, very small reductions in her dose, she only needed to take it every Monday morning, to help her face the workweek. None of the reductions had been excessively difficult, although she felt more tired than usual. After two months of taking it only on Mondays, she quit taking it altogether, or so she thought. During this time she started showing increased signs that she was recovering.

One weekend, leaving for a camping trip with friends, she decided to take Eldepryl on Saturday and Sunday to have more energy for hiking. She saw me the next week and said that by Monday she deeply regretted taking it. On Monday she had felt strongly hung-over, confused, and deeply longing for the drug in a way that she had never felt before.

She didn't take any more for two months, when she again took some on a weekend in which she had a difficult personal situation to deal with. After that weekend ended and she had even stronger yearnings for the drug and an even deeper feeling as if hung-over, complete with a reappearance of parkinson-like symptoms, she swore to never take the medication again.

Lingering memories of Eldepryl, or any DED

Sammy's drug was Eldepryl, not L-dopa, but we saw the same pattern with all the PD drugs: it was not too difficult to reduce the medications if a person still had Parkinson's *and* was not yet showing signs of dyskinesia (signifying severe brain damage from the drugs). *But* if the person appeared to be recovering from PD and still took the antiparkinson's medication, the medication behaved like a classic addiction temptress.

Without the reining influence of the Parkinson's (anti-addiction) brain pattern, the drugs imparted brief periods of strength, confidence, and even euphoria. And when the drug wore off, the person was plunged into a state of depression and extreme weakness, and experienced feelings of confusion, grogginess, incompetence, and even paranoia. In addition, using the DEDs (dopamine-enhancing drugs) after the PD was gone seemed to invite a quick return of parkinson-like symptoms: the dreaded drug-induced parkinsonism.

The drugs seemed to call to a person more strongly when the symptoms of Parkinson's disease were gone. For most people with Parkinson's disease, the pills have no particular allure; they are just a necessary part of the day.[1] But *if* a person who seemed to be recovering from Parkinson's disease in the slightest indulged in a little "harmless" pill taking on the weekend, the drugs called with increasing enchantment, even though the after-effects, when the drugs wore off, were more unpleasant each time.

Ongoing Parkinson's disease seemed to protect a person from rapid addiction. Recovery from PD, on the other hand, made a person susceptible to addiction and withdrawal symptoms.

Sadly, many of our ex-PDers seemed to have a lingering, subconscious memory of the joy of the medication. When times were bad, either from illness, grief, or cold weather, they yearned for their powerful meds, meds that were stronger than any street

[1] The exception is those people who take the drug to create an On! feeling, as noted in chapter three.

drug, and which they could get, legally, because they had had, at some point, a diagnosis of PD. I have gotten so many phone calls asking for "permission" to take the drugs.

"My father died last week, I'm a wreck and I need Sinemet" or "I had a bladder infection and now I think I have Parkinson's again, can I take the pills?" are examples of the way these phone calls usually begin. I have to remind the patient that I have nothing to do with their drugs, and I'm not their MD. But anyone who thinks he will never want drugs again after getting off should be aware of this long-lasting effect.

The National Institute on Drug Abuse

At about this same time, the National Institute of Drug Abuse had made its major breakthrough in the study of addiction: all addictive drugs increased dopamine; addiction was due to diminished dopamine levels that occurred as a rebound against the drugs. Their work meshed perfectly with what we were seeing.

What we had that they didn't have was a group a people who had originally needed their drugs due to an organic (occurring naturally, within the brain) lowered dopamine level – a group that also didn't get addicted. The patient base for the researchers on drug abuse was people whose dopamine levels had been lowered primarily in response to drugs or toxins. These people could not reduce their addictive drugs without suffering. Ours could – as long as they still had Parkinson's.

We were seeing something that no one had ever seen before – reduction of otherwise addictive medications in people who were not susceptible to addiction AND the rapid change in their addictability if they started using medication again after they recovered their normal dopamine system.

This was actually very significant. Many researchers on drug abuse were, and still are, looking for social, educational, and political reasons for drug abuse/addiction. What we were seeing was that people who were not addictable in one brain condition (Parkinson's disease) were highly addictable when that brain condition was removed. Their susceptibility to addiction had nothing to do with their social, educational, or political standing.

However, we were in no position to publish this information in drug abuse journals. Our premise was based on the curability of Parkinson's disease. That premise was still unacceptable: according to neurologists, if our patients got better, it was because they had been misdiagnosed. At the time, the SPECT[1] was just beginning to be used, and even PET scans were expensive and rare. Only one of our patients had ever had one. We applied for various grants so that we could pay for before-and-after scans of our new patients, but our grant applications were never even given consideration. Morale sank.

The 10% plan

We had been doing the math. Several patients who had reduced from 1000 mg a day down to 900 had seemed to have the easiest time with their reductions. Other patients who had started from lower levels and reduced daily intake by the same amount, 100 mg, were in agonies. We postulated that it wasn't the absolute amount of the decrease, but it was the percent change. We looked at other charts and found that this thinking held up;

[1] SPECT scans are an improvement over the old PET scans. Both are radiological scans that can show density of molecular activity.

patients who had been taking approximately 500 mg/day had handled a decrease of 50 mg/day pretty well; they had felt lousy, but never went into withdrawal hell. After two to ten weeks at the lower level, they were doing as well as they had been before at the higher level. We shared this idea with all the patients. Some of them tried it themselves, and others did not.

It soon developed that most patients who never reduced by more than 10% of their current dose, no matter what their current dosage level was, might have an easy or a miserable time during each drug decrease, but they were able to get through the next few months without entering into a ferocious world of mental chaos. They also felt good enough after each decrease to start another round of decrease.[1]

In contrast, most people who decreased by more than 10% had gone through hell and resumed drugs at the previous levels or higher. Sometimes even a decrease of 15% propelled one into the abyss. It had long been recognized that strong narcotic drugs were best reduced on a 10% plan. It now appeared that dopamine-enhancing drugs were also "10%" drugs! We finally had something that might really be helpful. Morale soared.

Birdie

Birdie was 65 years old. She had moderate PD, not advanced, not early. She was taking quite a bit of medication: 500 mg/day levodopa, 4.5 mg/day Mirapex (an agonist), several pain relief drugs, and several "good stiff drinks" every day. She lived alone, her children lived nearby. She was very active, always getting out and about, and considered herself to have a rich and busy life. She did not feel that her quality of life had been affected whatsoever by her Parkinson's; the drugs were working very well for her. She came to us because of her increasing lack of voice.

Beginning of recovery

She responded well to the treatments at our clinic. Sensation returned to her feet after decades of numbness and her movements became more fluid.

She was not at all interested in decreasing her medication over-quickly because she preferred to "live life to the fullest." She had no qualms or squeamishness about taking whatever drugs were recommended, as long as they would allow her to be independent.

Unfortunately, at the same time that she started having more feeling and motion in her feet, she started having dyskinesia for the first time. Violent cramps appeared in her feet and legs about an hour and a half after taking her pills. As the medicine reached its peak, she would be screaming with pain as her legs tied themselves into knots.

The young doc's diagnosis: "dyskinesia from overmedication"

We pointed out to her that the spasms corresponded to the effective time of the meds, and asked her to work with her MD. Young Doctor Williams, a neurology/movement disorder specialist fresh out of school, recognized that her symptoms were coming from her drugs. He told her that she was very overmedicated and she should decrease by one pill a week until she felt better.

---

[1] Full details on the ten percent program are in chapter 17.

At this time we had already seen people nearly die from decreasing at this rapid rate, and we told her so. She told her doctor, and he assured her that one pill a week was very, very slow. Compared to what his older colleagues were recommending, his prescribing was ludicrously overcautious, he felt.

Considering that he was probably thinking of a decrease in terms of 1 to 3 hour half-life, one pill a week was very slow indeed. If he had been thinking in terms of ten-week accumulations, he might have done the math differently.

At any rate, in the first week of her decreasing her meds, her response was not too severe, merely causing some periods of immobility. We warned her that the withdrawal, which resembles a sort of super-Parkinson's, plus having all the features of nausea, paranoia, pacing, violent versions of the side effects, and bouts of fear-stricken whole body rigidity, may not even *start* to appear until about seven to ten days after the meds have been reduced.

But Birdie's leg cramps seemed a little less intense after reducing her medication, and that's what she wanted, so she was determined to decrease by another pill (100 mg) the next week, to further decrease the spasms.

We warned her that, based on what had happened with other patients, the true benefit and possible hell of the drug reduction might not be apparent for possibly ten weeks, so there was no way of knowing if she was now at the right med levels after only one week. But she was still having some mild leg cramps during her On times, so at the end of the first week she reduced another pill. Halfway through the second week, she started to feel short of breath plus having classic signs of drug withdrawal, and her leg cramps were amplified by a new sort of terror – a fear that she was going to die from the leg cramps. Her thinking now was that this was further proof that she was still overmedicated. After all, her doctor said that her leg cramps were definitely caused by her medication. She reduced by yet another pill. This made a reduction of three pills – 300 mg, 60% of her original dosage – in a mere three weeks. She was sliding downhill fast now, and about to enter into actual drug withdrawal.

A 60% reduction

Birdie had decreased by 60% in three weeks. By the middle of the third week we figured that the first reduction of the three was really beginning to show its effect and the second reduction was just barely making itself felt. She hadn't even begun to notice yet the effects of the third reduction. But at this point she was having trouble moving, and she was screaming from the leg cramps. She was both undermedicated, as evidenced by the immobility, and doubled up from leg cramps, a symptom that the doctor said was due to excess medication. (We have not met a doctor yet who knows that during drug withdrawal, the body will mimic, from sheer terror, the same symptoms that it exhibited in the face of excess medication. Both responses are a panic response by the brain to a condition of extreme danger.)

Drug withdrawal

Birdie's children had her admitted to the hospital. By this time she was taking powerful antispasmodics for the leg cramps, cramps that were now so powerful that she thought her leg bones would break. She was imbibing several extra stiff drinks a day plus taking a new prescription, an antianxiety drug to help her with the horrors that were

overwhelming her. She was frozen stiff most of the day, gasping for air, and she had no speaking voice.

Young Dr. Williams reversed his position: based on her agonies, her original problem must have been undermedication rather than overmedication. He ordered her to take six pills a day since her starting dose had been five pills. For the next few days, as the incoming flood of L-dopa combined with the antianxiety pills that were probably just beginning to take hold, she started to calm down.

Gross overmedication

Within two weeks, her levels of dopamine began to rise back to her previous level – where she had been clearly overmedicated to begin with – and then beyond that level to an even higher level. Within three weeks she was having rapid-fire spasming in her fists and face, both common dyskinesias of L-dopa, and the powerful clenching of her feet was literally pulling them into balls. She could not stand and she was deeply confused.

She had several problems going on at once. She was continuing to recover from Parkinson's – once the recovery process begins it cannot be turned off, and it will continue even if you stop having treatments – and she was increasing her medication. Also, because fewer than ten weeks had passed, she was apparently having some remaining symptoms (terror and paranoia) of drug withdrawal when the short-term effect, the flood of each dose wore off. She increased her medication again because the fleeting high seemed to give her precious moments of oblivion during which she didn't notice the pain from the extraordinary foot and leg cramping.

Birdie's doctor ups the drugs

Birdie was entering into a dangerous land – the land of people who are taking L-dopa who do not have a Parkinson's disease electrical pattern in the brain. She was now addictable and had no defenses against L-dopa. She had rejoined the rest of the world, dopamine-wise, and was as susceptible as the people in the *Awakenings* study. Those monstrous situations that Oliver Sacks had described, which were met with jeers of derision from fellow MDs who decried his statements as impossible, started happening to Birdie.

We have seen over and over that if a person decreases dopamine levels for a few weeks and then dumps a high level of dopamine into the system, both withdrawal and overmedication will occur. The limbic area, which, remember, is very slow to change, will still be in a panic, sending out distress signals of terror, which can include shaking, nausea, paranoia, wracking pain and sensory overload. The body may, in these times of stress, also incorporate any patterns that have been learned by the brain to use in times of low-dopamine stress, such as tremoring, hunched posture, drooling, and all the usual symptoms of Parkinson's disease.

The high levels of dopamine flooding the brain will also be picked up in the motor area, which responds quickly to dopamine. The motor area doesn't care that the dopamine levels have been low for several weeks. In the presence of even briefly excess dopamine, the motor area will fire off with dyskinesias, freezings, and hallucinations – all the tricks that it has ever learned to use up the extra dopamine.

In this situation, when a person decreases for several weeks and then increases, the slow-changing limbic area is undermedicated and the motor area is overmedicated. So

this person may alternate between the worst symptoms of drug withdrawal and symptoms of overmedication. Birdie was having all of these problems. This explosion of symptoms is utterly baffling to anyone, including an MD, if he doesn't understand the underlying process.

Birdie's doctor, observing her, decided that, for some unknown reason, Birdie's Parkinson's disease was accelerating at a faster rate than most. Remember, her MD assumed that the half-life of these drugs was measured in hours. He assumed that whatever he was seeing was the result of that day's pills. He had no reason to suspect that she might be reacting one day to drugs that she had increased or decreased weeks ago. He decided she should increase her medication again. She increased again, from 600 mg/day levodopa to 700 mg/day.

After two more weeks, the withdrawal symptoms abated. She was no longer subject to paranoia, nausea, and terror. Within another week, the full effect of seven pills a day was starting to be seen. Suddenly her dyskinetic leg cramping, foot balling, hand clenching and arm spasming was literally mind crushing. She was shrieking in pain. Her body was contorted with spasms. She was screaming and sobbing.

We have seen that people with PD can tolerate a fair amount of overmedication. If the drugs are increased slowly enough, *and the person has PD*, the brain seems able to accommodate somewhat to the drugs. A dose of 700 mg/day does not usually trigger such violent symptoms, so quickly, in a PDer. In general, most PDers with advanced PD don't seem to suffer this level of side effects until they have been above the 800 mg/day of L-dopa level for a sustained period. (Please, do not use this 800 mg number as a guideline. Every person is different; every person has a different tolerance for the medication.)

The point I am trying to drive home as if your life depended on it is not the 800 mg/day number, but the fact that *if a person has recovered, and their electrical circuits are no longer running backwards (in PD fashion), the body has very little tolerance for drug overdose*.

Overdose was now happening with Birdie. Most PDers, if they increase by 200 mg of L-dopa, will not go into screaming torments. But we have seen that if a person has begun to recover, even a 50 mg increase, *even just staying steady or reducing too slowly,* might not be tolerated. Hideous side effects can occur no matter what the drug level, if a person is recovering.

We lose Birdie

At this point the MD decided that she needed drugs to stop the spasms. She started taking anti-epileptic drugs and powerful sedatives. She was taking antispasmodics and combining them with "good stiff drinks."

Her children were certain that our clinic, and not the good doctor, certainly not the FDA approved drugs, had caused her problems. She was sent home to live with her daughter. She wanted to come to the clinic, but her daughter, a registered nurse, would not consider it. Her acupuncturist was allowed to make house visits to give massage treatments on the steely knots in her arms and legs. The spasms and knots persisted despite the powerful anticonvulsants and anti-epileptic drugs. Birdie died just over a year later, in 1999, in a convalescent home.

Another death

Right around this time I heard from health practitioners in both Florida and southern California; they each had had a patient who had appeared to be recovering. Their patients had each been told by their MDs that they were, respectively, overmedicated and misdiagnosed. In both cases, their doctors told them (respectively) to reduce and stop taking the medications immediately. Both patients obeyed and subsequently went into shock.

Failure to stabilize

The Florida patient ended up in a hospital where the medicos ended up deciding that his new unpredictability of response to L-dopa (after 24 hours) proved he did not have Parkinson's. He was put on assorted drugs, one after the other, to determine if his problem was psychological or physiological. They changed his drugs daily. He got worse and worse. He nearly died in the hospital where they were trying to "stabilize" him. He spent his remaining days in a care facility, heavily drugged. He died within the year.

Failure to obey orders

The one from southern California was told to stop taking her medication, as she had evidently been misdiagnosed. The sudden stopping was highly traumatic. She went back on her medication, and her doctor pronounced her a "problem patient," a patient who wouldn't obey orders. Her doctor simply couldn't work with her under these conditions.

Failure to decrease the drugs

Meanwhile, at our own clinic, we had Zoe and one other patient who had been lost to the drugs – they didn't die, but they were illogical, writhing day and night, and in Zoe's case, suffering from a life-threatening respiratory condition that kept sending her back to the hospital.

Every time Zoe's MD or the hospital staff told her to reduce her medication, she increased it. Her doctor's suggestion – that she decrease by 30% – was ludicrous in light of what we knew, even at the time, but of course we could not say anything in opposition to her prescription. I did share information with Zoe and her husband, explaining that a 10% limit had seemed to work for some recovering patients, but her husband was understandably unwilling to risk even a 50 mg/day decrease (2.5%) because even this small decrease seemed to worsen the suffocation. Zoe, due to the life-threatening dyskinesia in her diaphragm, was really too far-gone for help.

Zoe's doctor

You may be thinking, correctly, that we had a moral obligation to contact Zoe's doctor. I did that. This particular doctor was so hostile to our project that he refused even the offer of a business card. He sneered at the business card, which had the word "acupuncture" on it. When I calmly informed him that it was extremely rude to refuse a proffered business card, he gingerly took hold of it, and snarled, "There's a Chinese man in my office; I'll give it to him." No, this doctor was not interested in hearing anything from us about drugs.

We couldn't find local doctors who were interested, and we couldn't give advice to our patients. We were limited to reading to our patients out of the drug textbooks or drug inserts, and sharing case studies with them without making conclusions. Legally, we were not in a position to offer any advice. We kept telling people to work with their MDs, because that is the law, but the MDs were clearly uninformed. I soon received more emails from around the English-speaking world with tragically similar stories. By the end of 1999 we realized that our suggestion to "work with your MD" was possibly a death sentence.

Broken hips

There were variations on the medication problems. Not all the problems were stemming directly from tardy reduction in medication but were coming from the physical weakness that can occur during recovery from Parkinson's. When this weakness appeared in a medicated patient and caused an interface with the traditional medical establishment, it was a formula for tragedy.

It was becoming a common email story: a PDer would have a change in symptoms that corresponded to recovering. Recovery symptoms include extreme weakness; this occurs when previously rigid muscles begin to "melt" into ineffective softness. These patients were unable to move due to their supreme weakness, whether or not they were medicated, unmedicated, or attempting medication reduction.

Due to temporary immobility from weakness, the spouse would frequently take it upon himself to carry the recoverer to the bathroom or bedroom. After the aging spouse accidentally dropped the recoverer, the recovering PDer would be admitted to hospital with a broken hip. In my own private practice, I had three patients with broken hips in two years. My emails indicated that this was happening around the world, in similar circumstances.

Trouble in the hospital

It was during the hospital visits that the real dangers began. Inevitably the MDs would note that the person with a broken hip was a PDer. Concluding that the cause of the fall had been undermedication, the MD would typically prescribe a whopping dose of antiparkinson's drugs. If the patient had previously been taking medication, the doctor would increase the drugs by 50 to 100% (!!!) of the highest level ever prescribed for that particular patient.

Not immediately, but within a few days to a few weeks, that patient would be hallucinating, thrashing about, or doubled over in spasms. This seeming intolerance for the medication would lead to experiments in which one medication would rapidly be exchanged for another. A person might be on three different drugs in one week, and all the while, due to the slow adjustment of the brain to the medications, the brain might still be reacting against something that had been ingested weeks ago. The most common response of the MDs was a desperate attempt to "stabilize" the patient. Again, the stabilization was presumed to be complete in three days.

Off to the nursing home

These disastrous attempts usually ended with the patient being placed in a care facility with mandatory prescriptions for high levels of anticonvulsant and antispasmodic drugs, together with the usual PD drugs.

Also, following the hip replacement surgery, the doctors typically wanted their patients walking as fast as possible. Following my patients' hip replacement surgeries, their surgeon – not their neurologist – demanded that they be given extra-high doses of levodopa. The idea was to get the patient walking again quickly. The doctors in every case prescribed an increase in antiparkinson's drugs – *regardless of whether or not the patient was manifesting any signs of dopamine insufficiency*.

If the word "Parkinson's" appeared anywhere on the patient's chart, the surgeons – a hip doctor usually knows next to nothing about these drugs, by the way – fixated on the diagnosis of Parkinson's and assigned galloping doses of L-dopa to their post-hip replacement patients.

When I went to visit my patients in the hospital, they would be gazing at me out of over-bright eyes, beaming away, arms fluttering, head rolling on its stalk, face jerking and grimacing like the worst excesses of St. Vitus's dance.[1] They were utterly unaware of pain, or the need to walk, or even to think. They were stoned, hopelessly stoned. Their doctors, noticing their disinclination to practice walking, and ignoring their grotesque side effects, usually increased their dosage further. No logic in the world could convince these doctors – and believe me, we tried – that the patient was showing signs of overmedication. Nope. If "Parkinson's disease" was on a patient's chart and that patient wasn't walking, the doctor had carte blanche to prescribe all the antiparkinson's drugs he liked, in an attempt to maximize movement.

In addition to this "the more the merrier" attitude towards the antiparkinson's drugs, all the doctors we have heard of from our Internet patients and practitioners evidently imagined that drug stabilization occurs in three days. Then, after a failed attempt to "stabilize" the patient, they conclude that the patient is either psychotic or misdiagnosed. Several patients were told that they obviously never had PD and were ordered to stop all the medications immediately. They went through the usual trauma and died.

From around the world, I was hearing horror stories from people who were recovering from PD. Their meds were suddenly far too strong, but if they tried to reduce them quickly they went into shock, and their MDs had absolutely no idea what to do.

Morale sank.

Personal responsibility

I paced the floors, I prayed, and I agonized. The nightmare experiences of my patients were ever-present in my thoughts. Rightly or wrongly, I felt a responsibility for every single one of those disasters.

---

[1] "St. Vitus's dance" (spasms and gyrations of presumed spiritual ecstasy) was the name given in the Middle Ages to the gleaming eyes, frozen smile, and uncontrolled twitching and flailing of limbs that occurred as a side effect of eating moldy rye. Ergot (a rye fungus) causes the excess movement and the delusion of spiritual brilliance. One of the first pharmaceutically produced dopamine agonists was derived from Ergot.

My dilemma

I could not give any advice about the meds. If I did, I would lose my license. If I didn't, patients might die. Even if I did break the law and tell the patients what they were doing wrong and what they should be doing right, 1) they might not be able to follow my suggestions, 2) if my advice conflicted with that of their doctors they would probably follow their doctor's advice, and in either case, 3) the patients would be just as overmedicated as before *and* I would lose the right to practice Asian medicine. I could be throwing away my right to practice medicine and most likely the patients would be making mistakes, even dying, just the same.

Family members

I knew from the attitudes that I had seen from patients' family members that patients' families and friends would override my observations anyway. Very often, the children or brothers and sisters of the PDer in question did not know that their loved one was involved in our program until they ended up in the hospital. When they learned of our program and contacted me for information about the patient, these loved ones were usually raging with frustrated helplessness. When I tried to share information about the known, written drug instructions, and pointed out that the doctor was not complying with the manufacturers' instructions, they were furious – at me.

Coach

Coach nearly died at this time. His PD symptoms had rapidly evaporated in response to treatment. He felt terrific until he suddenly went from being undermedicated to being overmedicated. In Coach's case, the dyskinesia happened to occur in his heart muscle. He started popping nitroglycerin for severe angina.

Coach was actually getting very good medical advice – his wife was a doctor; she was absorbing all of the information that we could give her. She was able to make adjustments to his medication in good time, and he had reduced his drugs, down to 200 mg/day of L-dopa from 400. Then, due to a bout of flu, he felt so punk that he increased his medication to 300 mg/day levodopa. As his flu symptoms waned, he suddenly went beyond a glimmer of native dopamine into a flood of dopamine for three solid days. He felt glorious! And the life-threatening heart arrhythmias began along with a desperate, agonizing fast reduction in order to stop the heart spasms.

There will be more about Coach in another chapter, but the point is, even patients who were trying to stay ahead of their dopamine levels by decreasing their drugs in anticipation of recovery were running into problems if they didn't move fast enough – and there was no way of knowing just how fast was fast enough.

Ups and downs

Ups

The primate research was a big morale booster for us. Even if the MDs were still insisting that our recovering patients had been misdiagnosed, research had shown that brains could change in addictability due to external changes. (Technically, for all you non-biologists, a foot injury is a form of external, or "environmental," injury.)

We could use our patients' change in addictability as possible proof that something inside their brains was changing, something to do with dopamine receptor activity.

This seemed like a reason to celebrate. Our medicated patients who became suddenly intolerant (addictable) to the medications were the best possible proof that our program was altering people's dopamine levels. We could be nearly certain that we were using an effective treatment to reverse Parkinson's disease. We had possibly found the cure for an incurable illness. Morale went up.

Downs

By June of 2000, the deaths of Rose and Birdie, the virtual loss of Zoe and others, and the reports of deaths coming in from the Internet weighed heavily. We became more somber in our approach to the medications. We no longer paid any mind whatsoever to the prescriptions of the scoffing doctors who had mocked our program and assured their patients, "Just keep taking your medications until you don't need them anymore. If you recover from Parkinson's, you can stop then."

Most pioneers determined to decide their drug dosings for themselves. Others, more cautious, made special appointments with their doctors to ask about safe methods of drug reduction. Some doctors sniffed at the entire idea and refused to give "permission."[1]

Well meaning and dangerous

Those well-meaning doctors who wanted to show support for alternative medicine were much more deadly; they invariably advised their patients to reduce their drugs in a way they considered to be slow and careful. Their ideas of slow and careful were usually far wide of the mark. By the time these unfortunate patients went into withdrawal shock, about ten days after beginning the reduction, they were down to no pills at all, and they had no way to get their brains quickly up to the levels needed to prevent the nausea, respiratory distress, heart spasms, violent shaking, and screaming tortures of hypersensitivity to light, sound, and touch.

Doctors, startled by these symptoms, which they considered, every single time, to be symptoms of abruptly worsening Parkinson's disease, invariably gave the patients even higher doses of their drugs than they had been taking before at the maximum. Within ten days at these higher levels, the patients would be having amplified patterns of those very symptoms of overmedication that had driven them, several weeks earlier, to want to decrease their medication.

Typically, at this point, as violent dyskinesia kicked in, the doctors would start pumping anti-epileptic drugs into the patients' bodies, or antispasmodics and muscle relaxants. Following this, as their patients' brains continued to act out against the now

---

[1] Although well-meaning doctors may tell you the contrary, an adult in the United States is not required by law to follow the suggestions or prescriptions of his doctor. As long as he is not a danger to himself or others, a person is allowed to refuse medical interference. He is not supposed to take prescription medications without a doctor's prescription. But he is legally able to refuse those prescriptions. If a person decides to stop using prescription drugs, that is his option. If, in order to stop safely, he must titrate down slowly and carefully, that is his legal right. But maybe the doctors who outright refused to work with their patients were the smart ones; their patients, in general, working on their own, did better than the patients who got emotional support and incorrect suggestions from their would-be helpful medicos.

extremely excessive DED levels, and their mental responses became fogged by the suppressants, they were hospitalized or put under convalescent care. From there on out, it was just a matter of time, usually less than a year, before the patient died.

Indecision time

We weren't sure how to proceed. We weren't sure if we should even continue the project. And yet, our instructions for the treatment of PD were on the Internet, being shared in chat groups, and articles had been published in several journals. People around the world were treating PD, but they didn't know about the horrible risks from the medications. We'd put information out into the public domain about how to recover from Parkinson's: we could not easily make this information disappear. At the same time, patients had no idea what to do about their medications, and the instructions from the doctors were usually deadly. What should we do about the medicated patients?

Warnings go out

We shared details of these lost pioneers with our surviving patients. We warned everyone about the difficulties, even tragedies, that might lie ahead with drug reduction. I was also regretfully obliged to warn them, based on what I had seen and heard, that their neurologist might be uninformed or misinformed about the medications. Even so, I could not advise in any way, shape, or form, other than sharing public information and past case studies. They were on their own.

No more medicated patients

We made the decision, which still stands, that we would no longer accept medicated patients into the program.

A solution emerges

We agreed to continue working with our current patients, whether medicated or not – they were getting better, and therefore they were most at risk, and we had a responsibility to them.[1] Our small army of grassroots researcher-patients, both in California and abroad, continued diligently recording their experiments in medication reduction, using charts and journals. We were able to fine tune the 10% finding. We discovered the cyclical pattern of overmedication, reduction, fleeting emergence of correct drug levels, and the return to overmedication. We formed hypotheses to make sense of these empirical findings.

The crucial patterns of "drug failure"

We discovered and named the patterns of overmedication that had previously been ascribed to "drug failure," but which, in light of our new hypotheses, were actually due to drug excess. These patterns were the key to determining any given patient's current location on the slowly moving cycles of medication accommodation. The next chapter will describe and name some of the patterns we saw.

---

[1] Patients who were overmedicated were met with each week, but they did not receive treatments.

Slow progress

Our project was still creeping forward. Though all of us on the team were acupuncturists, and all of us had neither the desire to be involved with these drugs nor the right to prescribe medication, we decided to keep working with the remaining drugged patients. Without our ever intending it, we were present as witnesses as our patients uncovered a slow, macrocosmic way of looking at dopamine.

Morale went up.

"Though this be madness, yet there is method in it."

Shakespeare's Hamlet

13. THE HIDDEN SIGNIFICANCE OF ON-OFF PATTERNS

INTERPRETING THE DAILY ONS AND OFFS

There were rhythms in the daily On-Offs patterns of people who were somewhere between moderately overmedicated and somewhat undermedicated. We named these patterns daily Build Up, daily Deficit, Roller Coaster, Turnaround, Crashing, and Sliding. These daily patterns helped prove our hypothesis that dopamine accumulates in the brain. They also provide signposts along the way of drug reduction.

Special meaning for Off

For purposes of interpreting drugs' adverse effects, the term "Off" in this chapter may refer to any time when adverse effects appear during what should be the ideal effective period from a dose. In other words, over the course of one dose's effective period, a person may go from On to not moving (the traditional meaning of Off) or he might go from On to a condition of dyskinesia. The dyskinesia, in this case, will be considered a form of Off. He may go from On to freezing, or from On to dystonias or a condition of intensified Parkinson's symptoms: all of those non-On conditions that occur during the dose's effective period are forms of Off. Any and all adverse effects, including wild exaggeration of tremor (ticcing), will be referred to as Offs for the purposes of this chapter.

DAILY PATTERNS

The patterns that repeat day after day are "daily" patterns. If, for example, on Monday, the first dose works fine but the second dose of the day doesn't work or works only briefly, and then again on Tuesday the morning dose works and the second dose doesn't, and Wednesday plays out predictably the same, this repeated pattern of working and not working is a "daily" pattern. A daily pattern repeats itself, more or less, over a span of twenty-four hours.

Subsets of daily patterns include the daily Build Up and the daily Deficit. In the daily Build Up, the doses become less effective/predictable over the course of the day; in the daily Deficit, doses increase in effectiveness/predictability over the day.

Patients whose charts were completely erratic, with no daily predictability, were apparently grossly overmedicated: if these patients reduced their medication, they would eventually start having "daily" or "consistent" dose-related patterns. [1]

[1] Patients who take drugs at night have almost no chance of figuring out what their body is doing with the drugs. There is never a time when the drug levels recede far enough to determine a baseline. In *Prozac Backlash*, Glenmullen points out that a study of cocaine-using rats proved the following: brain damage was less in those rats who had intervals during the day with no cocaine. All rats in this study each

The Daily Build Up

The most common daily pattern is a decrease of pill effectiveness over the course of a day. Most On/Off patients who have a daily pattern find that their first pill of the day is the most reliable. Over the course of the day, the pills become less effective. Often the afternoon pill or the last pill of the day works only for a few minutes, works like a roller coaster, doesn't work at all, or causes a violent Off.

Build Up

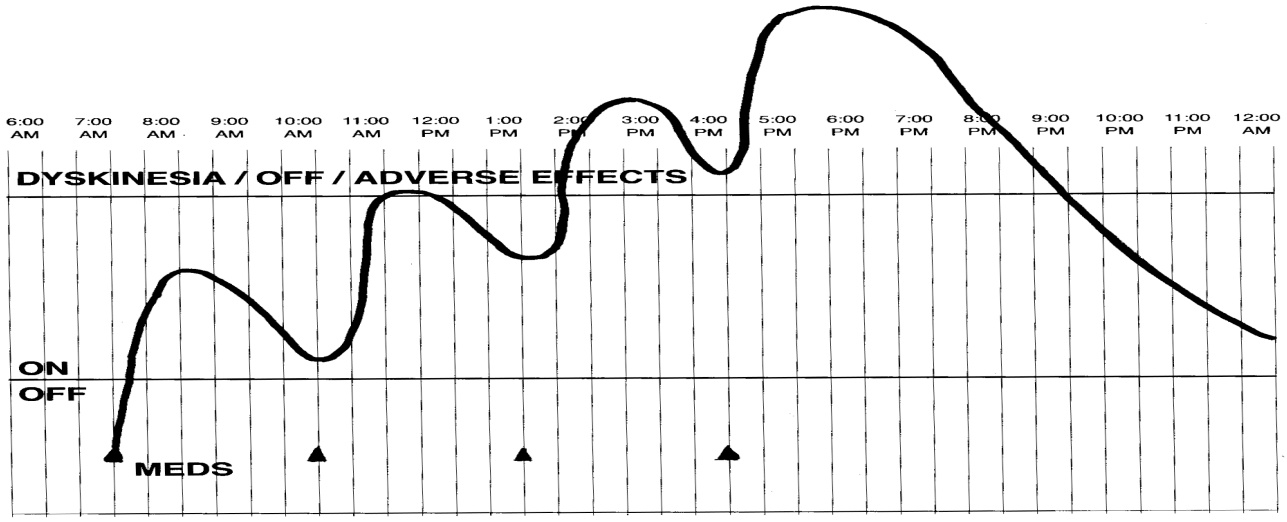


fig. 13.1[1]

In the above graph, the third pill of the day has problems – possibly dyskinesia, maybe some ticcing, or Off time. The fourth pill of the day will probably have some freezing or appear not to work at all. The Off from the fourth pill will be the result of excess medication, although it will look for all the world like pill "failure."

Common neurologist suggestions to treat late-in-the-day pill failure include: "Double your evening dose," "Take your doses closer together in the evening so that your body doesn't have a chance to come down," and the most dangerous, "When you go Off, supplement your evening dose by grinding up an extra pill and dissolving it in a little

---

received the *same* amount of cocaine spread over the course of the day. Some rats were given the drug in small amounts all day and all night, and the other group was given larger amounts, less frequently. The premise of the research was to show that smaller amounts, taken more frequently, would do less brain damage. This premise was intended to support the "gradual decrease" method of drug rehabilitation in drug addiction clinics. However, what they found was the opposite. Rats who took large amounts but who had many hours between doses fared much better in terms of avoiding long-term brain damage. The conclusion of the study proposed that possibly there is a brain-healing process that *can* occur, but which will *only* occur after the cocaine is effectively out of the brain. By providing small doses constantly throughout the day, the cocaine levels never got low enough to allow the brain to initiate healing.

This sort of thinking does have a parallel in antiparkinson's medications: those patients who have erratic, rather than daily (repeating) patterns, or even have hallucinations, are usually those who take the drugs both day and night or take them at excessive levels, never having a rest time when the brain can presumably rest or do repair work. An example of charts that are completely erratic due to overmedication can be found in chapter 18; Sonny, described in chapter 18, had erratic patterns until he decreased his medication. When his medications were reduced to a more appropriate level he began having daily Build Ups again.

[1] These charts assume a pill every three hours, half-life of four hours.

water. Drink up the dissolved pill to get an immediate, extreme On."[1] These suggestions usually work – for a few days or a few months (see: superdosing footnote, p. 243) – then the daily Build Up syndrome worsens even further.

It wasn't until patients began reducing their drugs and the opposite pattern, the daily Deficit, arose that we realized that an accumulation of medication was the reason for late-in-the-day pill failure.

As patients *reduced* their drugs, sometimes the first pill of the day didn't work and the last pill of the day worked better than it had in years. For those *reducing* their medication, the pills needed to accumulate over the course of the day in order to attain the effective threshold. Alarmingly, those patients *recovering* from PD who did not reduce their meds in a timely fashion often had drastically *worsening* of daily Build Ups.

The daily Deficit, a reversal of the build up

Those reducing their drugs often had a daily Deficit; the pills increased in effectiveness over the course of the day. Sometimes the first pill didn't work at all. Even if pills were effective later in the day, the following morning the first pill(s) might not work.

This pattern of deficit most often appeared after a long period of drug insufficiency (zero drug effectiveness) due to dosage decrease. After experiencing weeks or months of rigidity, slowness, or other signs of drug insufficiency, during which the drugs might appear not to be working at all, the drugs would begin to work again, even if only slightly – in a Deficit pattern.

This pattern, in which the pills taken *later* in the day were sometimes *more* effective than the first pill of the day, usually only lasted a few days. After the brain's accommodation to the lower dose was complete, all the pills might work, or the daily Build Up pattern might reappear.

Again, in a daily deficit the first pill of the day might work or might not, or it might work only briefly. The second pill might have a better result than the first. However, after one, two, or even three pills, the medication might be more effective, later in the day, than it had been in a long time. The afternoon pill or the evening pill might work wonderfully for a change.

[1] This grinding up of the pills is specifically warned against in the information supplied by the drug company.

Deficit

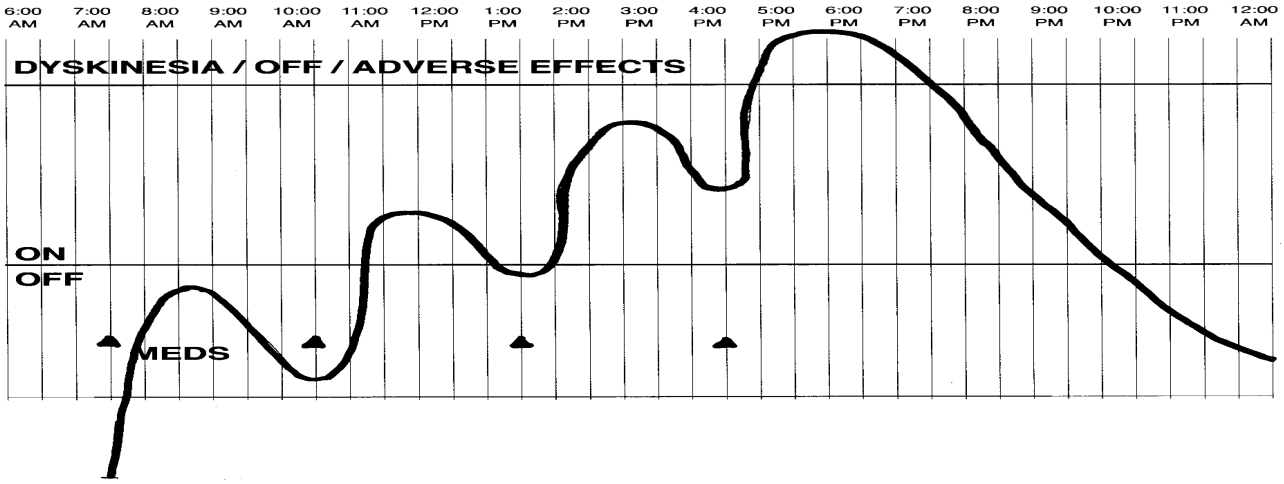


fig 13.2 In fig.13.2 the shape of the dopamine line of the Deficit is the same as the line shape of the Build Up; the only difference is the baseline, or starting level. During a Deficit, the baseline is so low that the first dose can't cross the On threshold.

Adverse effects even during a Deficit day

The daily Deficit occurs when the dopamine level in the brain is so low that one pill is not sufficient to surmount the threshold. The accumulated effect of two or more pills over the course of the day is enough to get over the threshold. When a daily Deficit pattern is occurring, Ons may still manifest adverse effects, but there may be fewer adverse effects than before.[1] If a person is recovering from PD faster than he is reducing his meds, he may have more adverse effects even when the brain is accommodating to the lower doses.

Even in a Deficit situation, with prompt decreases, the Ons may still be fraught with dyskinesias or other adverse effects. This occurs because of addiction damage: if a person has built up an addiction level so high that their movement threshold is almost the same as the Safety Limit, any movement might be accompanied by adverse effects.

But thankfully, even our people who at first had dyskinesias with their daily Deficit On times found that their addiction-elevated thresholds began to subside if they continued to make appropriate drug reductions over months and years. Eventually, even pioneers who had been subject to violent dyskinesias were able to have Ons without side effects if they steadily decreased their drug levels. It seems so obvious in hindsight; the Offs, freezings, and pill "failures" were due to excess dopamine accumulating in the brain.

[1] Sadly, since ticcing can rapidly become semipermanent, the supremely annoying but not life-threatening ticcing *may* become ever-present, despite the events of recovery, correct medication levels, or no drugs at all.

Build Ups and Deficits from sum total reduction, not specific dosing patterns

In the big picture, the reduction/withdrawal symptoms, Build Up, and the Deficit pattern weren't affected so much by which one of the daily pills had been reduced; it appeared that the total drug amount per day was significant, but not the time of day when the pills were (or were not) taken.[1] The choice of which pill to decrease or stop appeared insignificant in the long run.

There were exceptional days: sometimes, due to illness or unaccustomed stress, any drug user might have a day or two when none of the pills seem to work very well. This is not a daily Deficit; this is a lousy day.

Turnaround

The transition from daily Deficits to daily Build Ups can occur as quickly as overnight. Although the elegant patterns of Build Up and Deficit make it obvious that daily accumulation of the drug plays some role, other "random" factors such as illness, the full moon, or even a good dinner can moderate the effects of dopamine. Consequently, a person may lurch abruptly from a Deficit to a Build Up, or may wobble back and forth between these two conditions for several days, as factors other than pills play their parts.

It's often hard to tell where one is in terms of overmedication and undermedication. In the short term one might feel as if there is no pattern whatsoever to drug results. One might emerge from months of withdrawal and drug insufficiency into a perfectly normal day. The next day might also be all songs and roses, or one might descend briefly again into hell.

Sometimes, instead of a logical, anticipated daily improvement in the Deficit pattern, a person may be on a seesaw of symptoms for a day or two. A very common pattern is one in which a person in drug reduction feels bad one day, better the next, and worse again the next day. This ambiguous period in which a person isn't certain of whether he's getting better or worse is called a Turnaround.

Laurel

This is excerpted from an email that came (in mid-January) from Laurel, in England:

> "*I went through a bad patch at the beginning of December. I felt as if I hadn't got the strength or will to do anything. I was at crawling point a lot of the time. Then in the ninth week after my last reduction, I suddenly felt OK again. On the Tuesday I didn't switch Off* [from deficiency] *at all – quite amazing when things had been so difficult. Instead I was dyskinetic most of the time. I thought it was another turning point and was about to do my next reduction when I seemed*

[1] The resumption of native dopamine, most often noticed first thing in the morning, can react unpredictably with pharmaceutical dopamine. This native dopamine may also alter the effect of some morning pills.

As you will read later, the movement afforded by native dopamine is distinctly different from the movement that is induced by the drugs. For recovering patients, after their few minutes of healthy movement waned, even the first pill of the day might be unpredictable. Keep in mind that this is a book of generalities, and that each patient had a slightly different story to tell.

to hit rock bottom again. I was confused – should I reduce or not? I finally decided to go ahead anyway and stopped taking the 50 mg I was still taking overnight. I've started sleeping better and funnily enough my 3 o'clock dose in the afternoon is much more effective. This is the dose that has always given me the most problems. I'm now down to 600 mg from the 900 mg I was on when I saw you in June. Still a long way to go – I get very impatient at times."[1]

Laurel's case demonstrates the transition from drug reduction (crawling, or Off most of the time) to a mere deficit that could be overcome during the course of the day. Her afternoon dose worked for the first time in years. Also, the drug-induced insomnia that had plagued her for years, and which was exacerbated from drug reduction pain, was easing up.

Some history of her case will help. She had been severely overmedicated when she first contacted me, with powerful, painful dyskinesia and violent Offs alternating throughout the day. Ever since Tui Na treatments restored feeling in her feet, she had been slowly reducing her medication.

Every time she reduced her medication, she went through a phase where she could hardly move, then got movement back, and eventually had excruciating dyskinesias again. Most gratifying in the above email is that her late afternoon pill, which hadn't worked in a long time (producing instead a hideous dyskinetic spasming in her hip like "a drill digging all the way into the bones"), was once again effective. She was sleeping better, too, meaning that her end-of-the-day drug levels were much better.

Her afternoon pill hadn't been working even after several previous rounds of drug reduction. This suggested to us that, even after suffering through several withdrawals, her drug threshold level was still so high (due to addiction) that if her morning pills worked, they would put her over the Safety Limit by the afternoon. When, at lowered doses, her afternoon pills started to work correctly – instead of causing paroxysms of pain – we knew that her threshold was starting to drop down into a much safer range.

However, she still had a long way to go: we know now, in a person who has apparently recovered from PD, as little as 50 mg/day of levodopa can cause what appears to be permanent addiction. Laurel was still taking 600 mg/day, with every indication that she was recovering.

More about Turnarounds

Laurel's email described a Turnaround with seesaw days. Why did she suddenly have one wonderful day followed by a dreadful day?

She seems to have made the abrupt switch from undermedicated to OK to overmedicated in two days time. This speed is not uncommon; we've seen many two and three day Turnarounds. The Turnaround transition from undermedicated to overmedicated can be extremely fast once the brain level of dopamine crosses into the limbic appeasement zone and over the movement threshold. It may even be that feeling

[1] This was a brave decision, to look another round of withdrawal in the eye and go for it. By doing so, she probably saved herself much grief by deciding to go ahead, take the chance, and reduce again. This may sound melodramatic, all this hyperbole about the risks of a few days more or less on the drugs after they have become effective again, but by the time you've finished this book, you might understand how important "just a few days" can be in working with these medications.

good again and moving well after not feeling good for so long triggers a placebo response that elevates native dopamine, thus boosting the process along. And after that, overmedication can occur within hours, and if, God forbid, a person no longer has Parkinson's disease, ferocious addiction might set in within 72 hours.[1]

This return to addiction may include freezing, of course, and pill failure – so how is one to know if one has suddenly become overmedicated or if one is still suffering from dopamine insufficiency? They both look so similar! *The timing of the pill failure is the best way of knowing the cause of the Off. If the pill failure is in the first pills of the day, with better pill effectiveness in the afternoon, it is probably due to insufficiency. If the Offs or severe adverse effects begin later in the day, or one and a half to four and a half hours after taking a pill, they are probably due to overmedication.*

Laurel's decision

In Laurel's case, on Sunday she could move a little, on Monday she felt great and by Tuesday she had dyskinesia. By Wednesday, she felt horrible: she wasn't moving. The conventional wisdom would be, of course, that she wasn't moving because she was badly undermedicated. But what about the possibility that she was already horribly overmedicated again? On a hunch, based mainly on her Tuesday dyskinesia, Laurel made another decrease in her medication. And instead of dropping back into immobility from this small decrease, she noticed she was suddenly feeling better than she had in a long time.

Her hunch that she could tolerate, and even needed, another drug reduction so soon after resuming movement (pill effectiveness) paid off: due to her quick adjustment in medication, before she became addicted again, the next round of withdrawal was not as painful or long lasting as the previous one.

Addiction protection, again

Does it matter if a person who *still has* Parkinson's disease spends a few extra days at the overmedicated level? From what we've seen, yes, but the addiction response to this overmedication will be relatively slow.[2]

Does it matter if a person who is recovering from PD, whose brain is changing, *whether or not the changes are, as yet, visible,* spends a few days at the overmedicated level? Yes, just a few days can be dangerous and even deadly. This has been mentioned before, and it will be discussed again. I really want to hammer this point home.

---

[1] I do not mean, "God forbid a person recovers." The oath is in reference to the qualifier that such a person is still taking drugs. My editor has warned me that irony is dead, but I trust the reader to understand my meaning.

[2] This point is mentioned on behalf of any PDer who is not in a recovery program but who might be having drug-related problems and is reducing meds to get them back down to a healthy level.

QUICKIE PATTERNS

There were patterns that were neither the daily, 24-hour, repeating rhythms nor the ten-week grind. These were quick (taking minutes or a few hours) anomalies that seemed to be related to a single dose of the medication, regardless of what had happened the day before or the hour before. We named one of these the Roller Coaster and the other the Crash. These drug responses might occur in one dose or another, or all the doses. They were predictable results of severe overmedication. Their seeming unpredictability had more to do with vagaries of daily life, mood, and weather than with the exact timing of any given pill.

These symptoms, which, like daily Build Ups, help to prove that a person is gravely overmedicated, are helpful signposts in the drug reduction cycle. Of course, these patterns might worsen due to a Build Up and lessen during a Deficit, but in any case, these dose-related, quickie patterns are proof of drug excess.

Roller Coaster

Sometimes a person goes Off just when the drugs should be at the highest level in the brain. He takes a pill, goes On, and at some point in the next few minutes or hours, goes Off for a while. Then, *before taking another pill*, he may go back On. During what should be the highest point of the On, there can be an abrupt Off or freezing. Sometimes this Off can be extremely intense, featuring violent freezing, or it might feature all the usual symptoms of Parkinson's.

The curious bit is that *before* the next pill is taken the Off might end, or at least diminish, and there might be an On, a partial On, or at least a feeling of relaxation. Then, after this second On from one dose, there is a final Off. After that, no more Ons, or not many, will occur again until another dose is taken.

The mid-dose Off usually comes during what appears on the graph to be an ascent into the highest point of the drug concentration in the brain; for example, if the person usually got three hours of On time from a dose, the roller coaster Off might occur right around an hour or an hour and a half into the sequence. Sometimes these mid-cycle dips would be a few minutes. Sometimes they would be the majority of the pill-effect period. The overall illusion created was that a person went On, or started to go On, and then instead went quite Off for a short while or perhaps the duration of the hoped-for On time.

Patients describe this phenomenon: "I thought my evening pill was on the verge of working, I felt the usual change in my brain, but then it never did work. Instead, I felt worse than if I hadn't taken a pill." Or, "It sort of worked, but it wasn't reliable. Three times I thought the pill might really kick in, but then I'd go hard Off again, instead. I hate it when they don't work."

Pill failure is pill excess

In fact those pills weren't failing. They were working too well. The incoming dopamine was so great that the brain was hitting some sort of upper threshold above which no movement can occur – the Shut Off zone that we proposed earlier.[1]

The On at either the beginning or the end of the dose might be so brief that it seems that the pill isn't working. But if the severity of the Off increases during the time when the brain dopamine levels are highest (an hour or two, or sometime in the four hours after taking pill), one might logically assume that what happened was *not* a failure on the part of the pill to raise dopamine levels but an extreme Off from dopamine going too high.

Whether or not a person Roller Coasters or merely has severe dyskinesia and shaking or other adverse effects may vary from person to person, and from month to month as the brain tries out new methods to try and get rid of the excess drug. Typically, the longer a person has been using the drugs, the more likely it is that freezings will occur rather than the milder adverse effects such as dyskinesia and shaking. However, there are no hard and fast rules; every brain is unique.

A Two-Line chart

Charting two opposing lines: dopamine levels vs. On time

If the *dopamine* level is charted for the Roller-Coastering pill, the line soars up into the highest portion of the graph, above the upper threshold for movement. A graph of dopamine levels shows a steep climb, and then, as the brain institutes drastic measures of dopamine elimination, a steep decline down into the realm of mere excess, and then plummets down into the normal On zone or below.

The graph line for *movement* during this period would go in almost the opposite direction from the dopamine saturation line: when dopamine is the very highest, the movement is at the very lowest. Therefore, although the graph of dopamine saturation is a nice, predictable up swell followed by an eventual down slope, the graph of movement can look like a Roller Coaster.

---

[1] As an aside, some pill unpredictabilities might have been coming from their other drugs, as well. For example, some of my pioneers were taking anticonvulsant or anti-epileptic drugs to moderate the intensity of the cramps. The irony here, which I'm sure you are noticing, is that these people were taking pills to make them relax or become limp while they were simultaneously taking powerful stimulants that were making them cramp up. Some were also taking pain relievers to deaden the pain of the spasms. Dopamine may function as a mild anesthetic, so, in a sense, they were doubling on pain relief as well. Amazing.

Roller Coaster

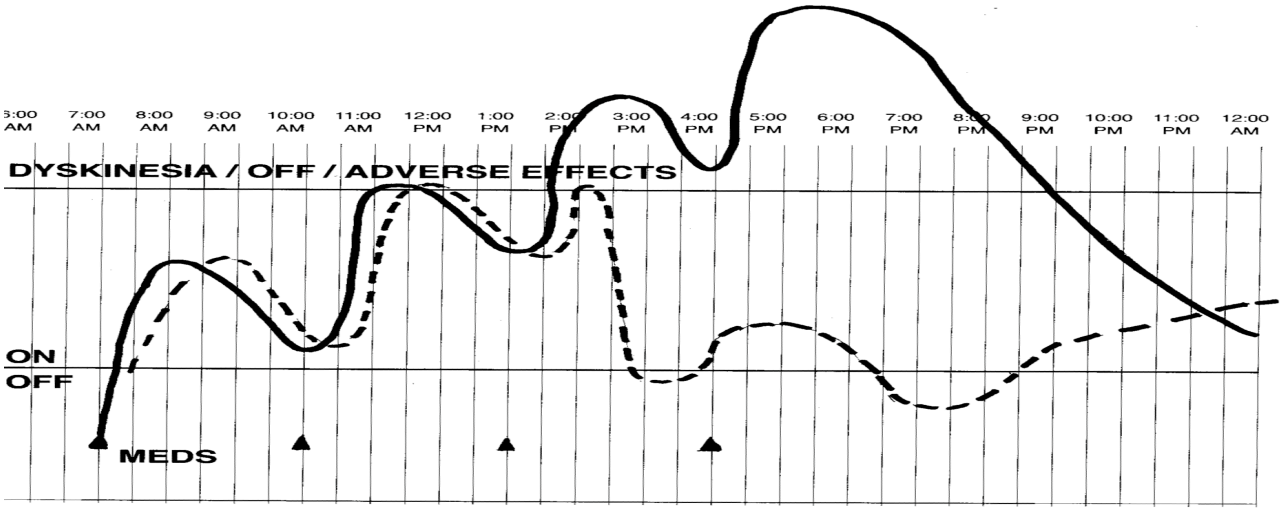


fig. 13.3

KEY
- - - - dotted line = movement
____ solid line = drug-induced dopamine levels

The solid line is the dopamine level; this line is the same as the one for the Build up and the Deficit. The dotted line shows the mobility; when the dopamine level rises higher than the hour numbers at the top of the graph, it is possible that a person will freeze, go Off, or manifest full-blown Parkinson's – in other words, it will appear as if the medication is not working. Not until the dopamine level drops down into the area below the hour numbers on the chart will the drug user be able to move again. Based on appearances, it seems as if the drugs didn't work – the person went Off instead of On.

Laurel again

Laurel often had Roller Coasters so abrupt that she didn't notice any On time at all other than a faint sensation that some change was occurring. Laurel often wrote to me about her afternoon Madopar (L-dopa plus a buffering agent, similar to Sinemet): "I thought this pill was going to work, I felt the change-up starting, but then the pill didn't work after all. In fact, the pain and dystonia got much more intense than usual. The pain was immobilizing, and only decreased three hours after I took the pill, after which I went back to just plain Off. *It's more painful to have a pill that almost works and then doesn't than to not have had any pill at all.*"

In Laurel's case, even if there was no obvious On time at the beginning or end of the dose, we still call her problem a Roller Coaster. That hint of pill action right when she hopes for an On, her sense that a "change-up" is happening, is indication that the levodopa has gotten into her system. The ebbing of the fierce dystonia and pain three long hours later just before she goes utterly Off is the decline of her dopamine levels back down below the upper threshold, into the realm where movement might be possible. This

phase is the second On at the end of a Roller Coaster, even though she got little real movement out of it.

Therapeutic benefits of brain Shut Down

The extreme Offs during Roller Coasters may be therapeutic in the short term: following a "failed dose," the next dose, or even the next several doses may work better than usual. It may be possible that the extreme dopamine-destroying measures instituted after the flip of a Shut-Off switch help to speed up the breakdown of dopamine, so that the day's accumulation can be reduced more quickly.[1]

It does appear that by having an Off period during a time when medication levels are clearly peaking, the brain gets a reprieve somehow: it often accepts the next dose of medication more serenely.

If one is having Roller-Coastering symptoms, he may be horribly overmedicated.[2]

Mid-chapter summary

The Build Up, Deficit, and Roller Coaster ideas were new interpretations of dopamine "failures." The first two demonstrate that dopamine levels at any given time of the day might be affected by dopamine that had been taken many hours earlier, and that motor function might be negatively affected by an excess accumulation of dopamine over the course of a day. The Daily Deficit, even more than the Daily Build Up, proves that dopamine action over the course of a day is cumulative.

The third, the Roller Coaster, proves that times of highest dopamine saturation can produce Offs: even after going Off mid-dose, a person might go back On again without taking another pill, showing that moderate dopamine levels may be more beneficial than extremely high levels.

---

[1]Some patients have expressed doubt that the brain actually has a Shut Off switch. They point to the specific example of Superdosing to prove that there is no such thing as a fixed danger level, above which a drug will fail to work.

Superdosing is the use of a very strong dose to get an On time when the usual dose fails to work. Some people who, due to Build Ups, get no On time from their evening dose are instructed by their physicians to either double their evening dose or crush a pill, dissolve it in water and drink it down, together with their usual dose. Though these techniques are specifically warned against in the drug literature, they both work in the short term.

The question arises, if there is a ceiling, or Shut Off switch in the brain, how is it possible that these Superdoses can exceed the Shut Off switch and create an On time?

It appears that even the absolute Shut Off is calibrated for a certain amount of dopamine excess. The Shut Off barriers constructed by the brain are effective when the anticipated amount of excess appears. However, the sudden appearance of a monstrous dose, unlike anything experienced before, can overwhelm the protective mechanism. While the brain is thus overwhelmed, the dopamine performs its normal job of appeasing the mind, sedating the tremor, and initiating movement.

Of course, even these Superdoses soon become ineffective. The brain simply constructs an even larger barrier, probably by killing off more dopamine receptors and dopamine-producing cells.

Typically, my patients who have used these Superdoses have found that they work for several weeks, or even a month or two, before they, too, become fairly ineffective.

[2] However, this is only a theory. I am not a prescribing physician, and I do not give advice regarding prescription medications. Furthermore, no one should adjust their drugs based on what they read in a book of unproven theory. If you suspect you are overmedicated, please work with your physician. Legally, only he can advise you.

Crash

A crash is a short-term, dose-related event. It is the easiest event for most patients to understand. Humans have been experiencing crashes after drug- or alcohol-induced highs for thousands of years. Sometimes referred to as "paying the price" for a few hours of fun, the crash is a venerated tradition. In the world of addiction, crashing has many names; the best known, perhaps, is "hangover." Crashes in people who still had Parkinson's were often less severe than in our patients who were recovering.

The crash is the miserable feeling that many patients have when a pill wears off. PD-drug users who have On-Offs often notice that after any given pill wears off, they feel much worse than they did before the pill ever started working. When the pill wears off, they do *not* go back to feeling how they felt before the pill kicked in. They feel worse, sometimes quite a bit worse. The crash of worsened mood and movement may last for a few minutes or several hours. It often causes a delay in onset of the next pill.

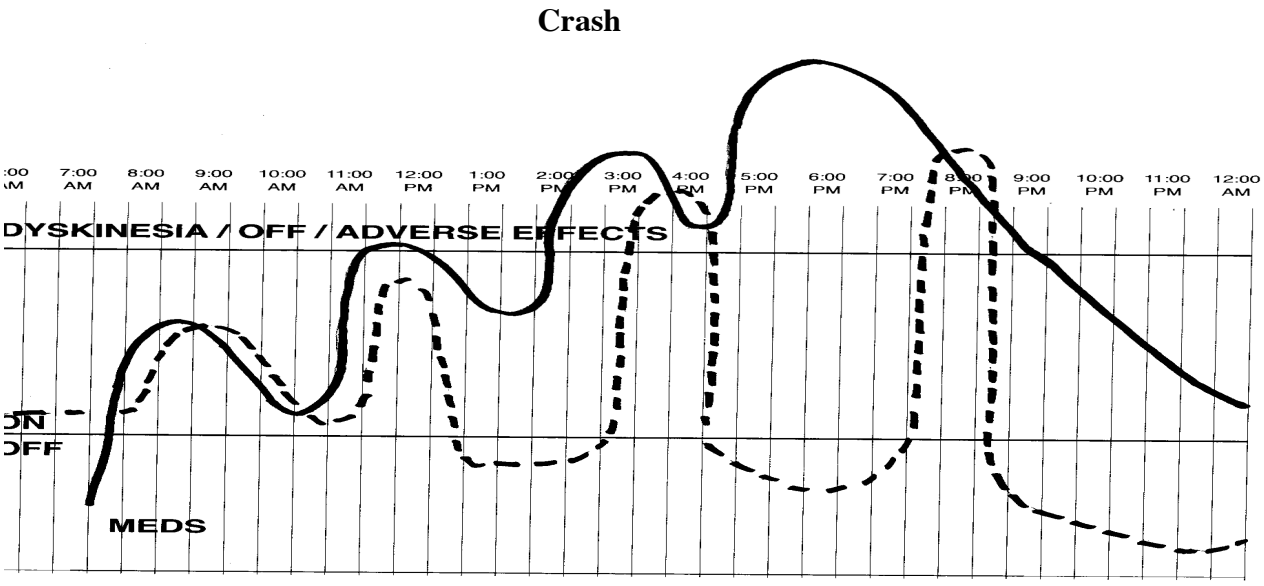


fig. 13.4

In fig. 13.4 the solid line indicates brain dopamine levels and the broken line is movement. Note that in this graph the On times (the broken line, when it goes up into the On zone) are followed by increasingly deep and longer lasting Offs, even though the accumulating dopamine in the brain (the solid line) is increasing. These worsening Offs are crashes, similar to hangovers, and are the result of brain processes that intentionally make decreases in the dopamine system as a way to counteract the excess dopamine from the drugs. Notice how, in this "Crash" graph, the Off that follows each dose is more severe than the last. Notice that the last dose of the day barely gets any visible response at all before the movement level crashes down into a profound Off.

The crash response can worsen throughout the day in response to the Build Up. For example, many people can move a little in the morning before their first pill. After

the first pill wears off, they feel worse, having no ability to move, or less than before taking the pill (a crash). After the second pill wears off they may feel even worse than after the first crash. Crashes may worsen in intensity throughout the day. Many patients find that their last crash of the day is the most severe; it can last for hours.

Worsening Crashes

If a person is in a med reduction phase and is barely having any effect from his medication, there may be no Crashes at all. However, even during drug withdrawal, there may be some faint suggestions of a Crash several hours after a dose. How is this possible? These Crashes may be caused by the fact that, even though a person is reducing and therefore having less medication than he is *accustomed* to have, and therefore experiencing drug withdrawal, his dopamine levels might still be, in fact, higher than the Safety Limit. His motor threshold may be so high that he cannot move without being considerably over the Safety Limit. When he reduces his drugs, he may be under the threshold and therefore not be able to move, but he is still over the Safety Limit and subject to crashing. This creates a condition with simultaneous symptoms of undermedication (immobility) and overmedication (crashing).

Hangover: familiar example of a crash

Alcohol is an effective, dopamine-enhancing, short-term treatment for depression in exactly the same way that dopamine-enhancing drugs are an effective, short-term treatment for Parkinson's disease. Over the longer term (many hours), alcohol and antiparkinson's medications are depressants, although in the short term (a few hours), they are both antidepressants and stimulants.

The crash that follows a too-high[1] dose of medication is exactly like a hangover. Any excess amount of DED, whether medical or recreational, can cause a crash. A perfect, correct amount of medication, one that does not trespass above the Safety Limit, will not cause a crash. Such moderation is scarcely possible in the exquisitely balanced brain, but PDers have an advantage: in PDers, the Safety Limit appears to be only half-awake. PDers can take quite a bit of medication before they start crashing. Therefore, given their relative immunity to crashing, if a PDer does have a crash following a dose of drugs, he is probably highly overmedicated.

Crashing is deceptive: it looks like undermedication

The crashing PDer may think he is undermedicated: his pills may not seem overly effective, and he may not be having much On time, but if a PDer is worse after a dose than he was before, we propose that he is over the Safety Limit and doing himself a disservice; he is overmedicated. Overmedication may cause brain damage and parkinsonism.

You will read more later about instances in which a person can appear to be both undermedicated and overmedicated at the same time. A person in the throes of drug

[1] "Too-high" means anything over the Safety Limit. The Safety Limit is a microscopic increase above the normal dopamine amount.

withdrawal who is still over the Safety Limit and having crashes after a dose wears off is in this doubly disheartening condition.

Withdrawal vs. a crash

A person who is experiencing mild drug reduction symptoms or even withdrawal may be able to derive some small benefit from his pills even though the relief may be brief. Such a one may imagine that he is crashing after his pill-induced benefits subside. This may not be the case. Going from lousy to OK and back to lousy again is not a crash and may not signify overmedication. A crash is the distinct worsening of symptoms following a dose compared to the symptoms *prior* to the dose. A return to drug withdrawal symptoms, back into hell, is not necessarily a crash – it might be merely a resumption of the day's status quo.

Twenty-four hour crash or Build Up?

Sometimes a patient will take an extra pill during the day. Very often, this is a well-meant attempt to be more sociable during a rare family get-together, or even an attempt to impress the doctor at the biannual check-up. In the next few days, some or even all of the doses may not seem to work as well.

Patients ask me, "Is this a crash or a Build Up?"

My answer is, "Why bother to give it a name?"

The whole thing is very moot. The main thing to recognize is that by increasing one's daily pill intake, one might have more Off time in the days that follow. It doesn't really matter whether this is from a severe crash, Build Up, or both, or even from the post-special-event letdown. In fact, as you will read later, these distinctions between various drug "failures" on any given day are all grey rather than black and white. Only when looking at the On/Offs over a period of many days do meaningful patterns occur.

Tracking changes over months, not days

Short-term changes in dosage and long-term changes in dosage all converge on any given day, and pills taken weeks earlier contribute to the overall brain response. It is almost pointless to worry over exactly which pill has caused what. This is why the daily patterns that opened this chapter were described as *repeating* patterns. Only when the drugs are taken at the same level for many weeks so that the limbic part of the brain's dopamine level is at equilibrium, and every day is similar, is there maximum value in labeling the daily patterns. To try and guess what is happening on any given day while ignoring the events of the preceding three months is a waste of time.

Switching

Switching is another dose-related symptom. Switching is the uncomfortable time at the beginning or end of a pill's effective period. It occurs as the body makes its transition from unmedicated to medicated, and then again when the pill is wearing off, during the descent from medicated to unmedicated. During the former, some minutes of extreme symptoms may occur just prior to the On. Later, during the latter, extreme symptoms appear again, just prior to the Off.

We will discuss what this looks and feels like, and what might be causing it, but not until two chapters down the road. We will need to go off on a tangent about how it

feels when the body starts producing dopamine again before we can do justice to Switching. We are only mentioning it here because it fits the dose-related patterns, and because we will refer to it in the next chapter, in our discussion of how these patterns can be used as signposts.

The Slide

The Slide is the ten-day period during which a drug change slowly starts to manifest. The beginning of the slide may be imperceptible. The end of the slide is the certain feeling that something is different. The number ten is only an average: the actual time ranges from two to twenty days. When I will refer to the "ten-day slide," keep in mind the larger range. The range can vary from one drug reduction/increase to another. Subsequent to the Slide is the actual *beginning* of the drug reduction, withdrawal or drug increase symptoms.

Many people have tried to reduce their meds rapidly, often based on an MD's well-meaning suggestion. What they don't realize is that, although the motor symptoms may change quickly over a few days or a week in response to this reduction, the limbic area, the "reptile brain," will have a delayed response to a sudden change in dopamine levels.[1] These quick-change patients may, of course, notice a decrease in movement a day or two after reducing the medication and assume that this is the extent of the change. But this quick change in superficial mobility, however painful, should not be confused with the long-term brain change required to bring the system to equilibrium with the lowered dose. The failure to account for the slide is the cause of most of the trauma and death associated with abrupt changes in dopamine levels.

The lizard stirs

It can take approximately ten days for the slumbering limbic lizard to even detect that there is serious mischief afoot. The beginnings of the drug reduction Slide may be imperceptible. The end of the slide is the suspicion that something deep and unsettling is churning in the brain.

It takes this long for the dopamine loss to reach a point where the limbic area *starts* to suspect an unaccustomed level of fear, pain or hypersensitivity. It can take ten days before this part of the brain even *begins* to have an inkling that there are significantly lowered levels of dopamine. After the ten-day slide into drug withdrawal begins, it may take ten weeks to feel the full effect, the full horror of the dopamine shortage. During this ten-day slide, while the brain is invisibly heading down the slippery slope into dopamine deficiency, the patient might think that he his doing just fine.

Some patients, experiencing a feeling of relief from the more violent symptoms of overmedication, may state that they feel better, not worse. We've named this brief period of feeling great immediately after making a drug reduction the Vacation. The Vacation often lures people into making another, premature drug decrease, with dangerous results.

Despite a few days of feeling better, despite a brief Vacation from the ups and downs and Ons and Offs, at the bottom of the slide the reckoning awaits: reduction begins in earnest.

---

[1] This is the case with L-dopa and also with the agonists and other drugs. Specific amounts for the time frames and doses of various drugs are in the Appendices.

Climbing up a slide

The opposite is also possible. A medication start up or increase may take at least ten days for the effects to *begin* to appear. It can take *months* for the full effect of a new level of drugs to be in place.

An Up Slide and a Down Slide

If a PDer realizes, two weeks into a reduction, that he has reduced drugs overzealously, he cannot quickly reverse the symptoms. Even if he changes his mind about reducing his drugs and resumes taking them at the pre-reduction level, it may take ten days (or from one to twenty days) for the limbic system to reverse itself and slowly climb back up the slide. The Slide masks the transition that may be occurring in the limbic area. This Slide function is actually a good one, in everyday life. The slide allows us to maintain limbic stability in the face of a traumatic event, so that we don't begin to act out until the emergency is over. Even better, a traumatic event may be soon tempered and the limbic depletion reversed before we even detect that the inner animal was ever threatened.

But in a case of drug reduction, there is to be no rescue from the abrupt change – from the intentional dopamine decrease. The Slide will occur slowly and unseen, until finally the full scale reduction or withdrawal erupts, and then there is no way to appease the monster without climbing slowly back up the steep Slide, suffering all the while.

Danger! Changing dosage too quickly

One cannot restore dopamine to the limbic area any more quickly than one can strip it away. In fact, the terrors of withdrawal can unleash the emergency neurotransmitter, adrenaline.

Adrenaline whips through the brain and dislodges dopamine from the limbic zone. This is how the limbic system is supposed to work in response to adrenaline. During an emergency, a dopamine-stripped limbic zone will allow for a heightened sense of sound, smell, touch, and sight. Even if the "emergency" that triggered the adrenaline is actually an internal crisis of dopamine deficiency, adrenaline still does what it does best: increases the heart rate, opens the lungs, and *strips dopamine away from the limbic area*. This additional depletion of dopamine via adrenaline, in response to the terrors of withdrawal, can make the upward slide, following a "correcting" drug increase, much more harrowing and slower than the downward slide.

When people reduce their drugs too quickly and slip into a dangerous, life-threatening version of drug withdrawal, mere administration of L-dopa may not help them; it may take too long for the incoming dopamine to assimilate in the limbic area.

For example, imagine you make a drug reduction and after three days of feeling good, you make another, larger decrease. Ten days later, your body starts acting out: your diaphragm goes into spasm and your heart rhythm is dangerously irregular. You panic and start tossing back handfuls of pills. Maybe you start taking four times as much medication as you were taking before. Will it help you? Probably not. If it takes ten days for increased dopamine to climb back up the slide, to slowly saturate the limbic area and turn off the withdrawal process, *but* you've had your fatal heart attack on Day Three of your panicked drug increase, all the incoming dopamine in the world isn't going to do you any good after Day Three: you're already dead.

Patients are poor judges of Slides

The Slide seemed to be the most difficult concept for patients to understand. Their spouses and caregivers can understand it, but drug addicts have tremendous difficulty thinking in the long term. They might intellectually accept that there could be silent changes occurring deep in their brain. But when it came time to decide how much medication to take on any given day, at any given moment, they rarely grasped any concept larger than the way they felt during their last dose. For the most part, they usually thought in terms of how they felt from hour to hour. A drug-addled mind cannot think in the long term.

Some patients think that the term "Slide" is too gentle. They think that "ticking time bomb" is a better description.

Slide review

When increasing dopamine dosages, it can take several days to several weeks for dopamine to even begin to build up in the limbic area. Therefore, an over-fast drug reduction ***cannot*** be remedied in a dose or two, or even a day or two. If you have reduced too quickly and then reinstate your old, higher dose, you must be prepared to spend up to three weeks in agony while your brain slowly, oh-so-slowly creeps back up towards the threshold. Not until you regain the threshold will the withdrawal symptoms decrease.

Actually, sometimes a person *can* rise back up out of a reduction in one or two doses. Other times it can take a long time to Slide back up. What determines the time frame? The determinant is probably how far a person has dropped below the threshold. Is there any way of knowing if you are a thousand grams or just a few milligrams short of feeling good? No. The limbic system, when disconnected to the reasoning centers, does not have a graduated response rate. The unmoderated limbic system is Yes or No.

Short of having disastrous, life-threatening symptoms, there is no real way of knowing if you are just below the surface or plumbing the depths.[1] The threshold makes it impossible to judge just where you are in drug withdrawal. If you are close to the threshold, a very small increase will bring you back out of the confusion. If you are deeply deficient, it may take weeks before an increase will reveal an effect.

An inexact science

Please, don't expect any of these patterns to follow hard and fast time frames. The main reason for warning you about the slide is that, although there may be some changes in *motor* function immediately after a dosage is altered, these changes may not signify anything to do with the *limbic* area.

Knowing that there is a slide function for the limbic area can help you self-assess where you are in terms of drug addiction. If, during discouraging drug withdrawal times, you find that your pills are even mildly helpful, this is an indication that you are actually not doing too poorly; you may be hovering just below the threshold. If, on the other hand, you decide to resume drugs at former amounts but it takes months before your symptoms go back to their prior levels, you may have been deep in a limbic hole. This slide has

[1] A patient in Sacks' experiment gave a nice description of this. She recounted that, prior to taking L-dopa, she was merely crawling on the surface of the earth. While using L-dopa, she was soaring thousands of feet in the air. After she was taken off L-dopa, due to life-threatening side effects, she was ten thousand feet under the ground – much worse than she'd been pre-dopa.

nothing to do with worsening or improving Parkinson's. It is a function of how the brain responds to excess or insufficient dopamine.

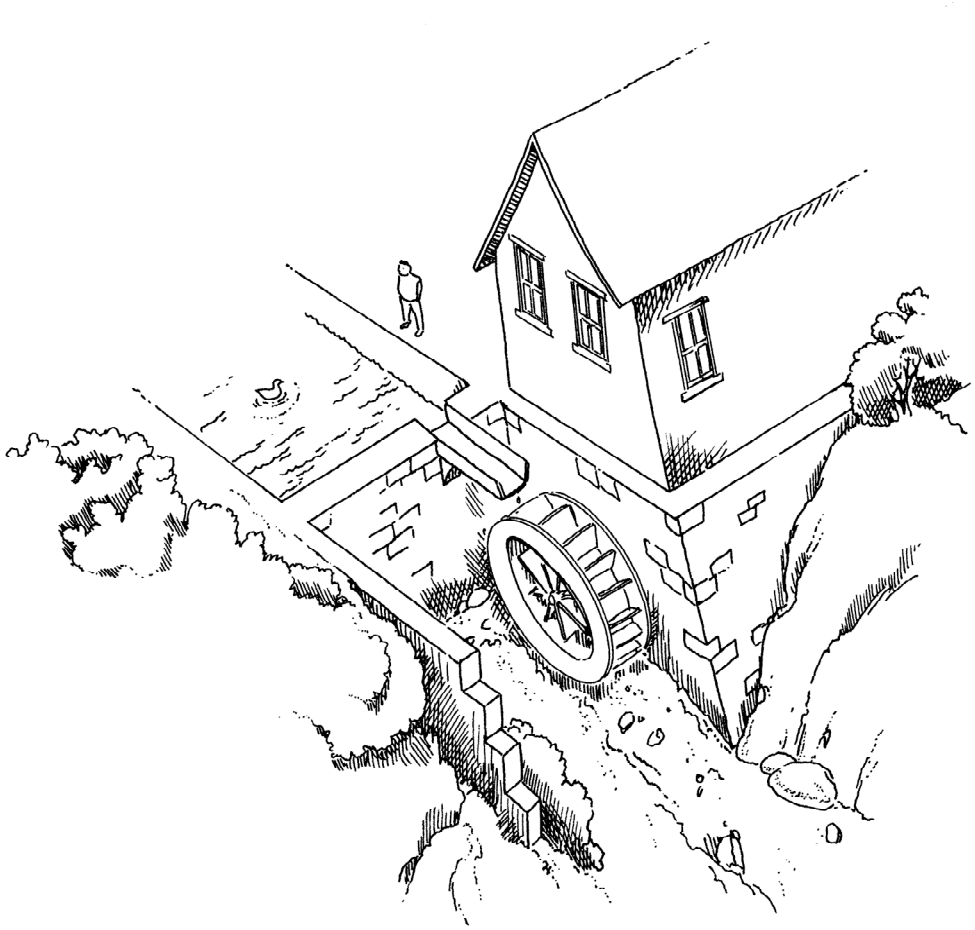
Peek-a-boo

During the Slide, a person may notice a faint change in mood or motor function that lasts a short time and then recedes. Many external events, and not just the daily dose, might be affecting the limbic area. Mood and motor function may teasingly come and go.

There may be a day that starts off poorly, boding ominously of drug withdrawal, and within a few hours, the barometer changes back to All Clear – or the reverse. There is a tendency for PDers to attribute every single alteration in mood or movement to their drugs. They forget: even people who do not have Parkinson's have ups and downs.

Responding to drugs despite withdrawal

Of course, don't forget that in response to a flood of dopamine the motor area may possibly snag a bit of dopamine even while your limbic area is staggering helplessly. Therefore, a fleeting, faint increase in motor function in response to a surge of drugs may not be a completely accurate indicator of limbic dopamine levels. If one's threshold for motor function is higher than the Safety Limit, the brief spasm of motor function in response to a massive dose of drugs during a drug withdrawal phase may even be a time of simultaneous overmedication with an underlayment of undermedication.



Thresholds: The water wheel example

Just looking at the water wheel won't tell you whether or not the reservoir is nearly full or nearly empty. The water wheel is your motor function and your mood. The reservoir is the limbic area. Unlike with the duck pond above, there is no window into the brain reservoir: it is shrouded in mystery. You only know that the brain reservoir is too low when your motor and mood wheels stop turning; you do not know by how much it is too low.

Safely undermedicated

Then again, even if you are hovering just below the threshold level, just below the effective dosage, in a relatively safe but uncomfortable (even painful) dopamine range, the effect of the daily dopamine drugs may not be visible. In Laurel's email, she wrote that she was at "crawling stage" for most of her withdrawal from 650 mg/day down to 600 mg/day. She had been taking medication throughout this time. It appeared, judging by motor function, as if she was not taking any medication at all, despite her 600 mg/day.

Variations: turnaround on the Slide

Sometimes the brain can respond to a drug decrease within a few days, increasing the baseline amount without ever going through any drug reduction symptoms whatsoever. If, after several days of drug reduction, just when a person is expecting to start feeling dismal, he has a return of facial grimacing or dyskinesia, he may need to act quickly and reduce his medication again, even before withdrawal symptoms appear. Many times we have seen people experience an unexpected improvement in their overall condition – even a return of dyskinesia, an improvement that apparently warranted another reduction – even before the first reduction's symptoms became manifest.

Impossibility of certainty

Do not be rushed into action by slight changes that may flicker on your mood screen. Experiencing one or two hints at impending drug withdrawal that unexpectedly clear up after a day or two does not necessarily mean that you have turned around and should reduce again. On the other hand, if dyskinesias or any other sign of overmedication hove into your radar screen after having been gone for a while, you may be overmedicated again. If so, you may wish to take timely action to prevent brain damage.

One cannot assume, on the basis of one bad day, that a withdrawal is still in full swing. On the other hand, if after ten weeks of misery the sun comes out, only to retreat again behind clouds of immobility, this darkness does not prove that one has re-descended into the pit. Maybe today was bad and tomorrow will be fine again. It may even be that one has swung from being under- to overmedicated!

It would be impossible to convey enough information in this short book to enable a person to predict the course of their drug reductions. The seemingly limitless range of possible responses to a drug reduction is based on hundreds of factors, some obvious, some subtle. For that matter, not all drug reductions will even set in motion a distinct decrease in pill effectiveness. One person may have several pain-free rounds of drug decrease preceded or followed by several difficult bouts with reduction. Because limbic levels are hidden behind a threshold and drug response varies per dose and per person, it is nearly impossible to guess what shape any given reduction might take.[1]

---

[1] Actually, one can learn to anticipate. Some of my patients' spouses became quite good at guessing just where their partner was in the drug reduction cycle. There are evidently hundreds of cues to work with. Tuned-in spouses were aware of everything that might be influencing their partner's drug responses: birthdays, holidays, weather, the strange 23 day cycles that seem to effect PDers, world events, spiritual malaise or hope – these all seemed to have a magnified effect on a teetering limbic system. Very often a spouse would pick up on a very slight, subtle alteration in behavior and adjust the dose with good success. You will read more about this in the next chapter.

However, if a person was able to anticipate recovery from PD based on changes in the circulatory system, sleep patterns, behavior, or movement, or could anticipate becoming overmedicated based on a change in the daily patterns, and responded to these anticipations by making a prompt, slight drug reduction in advance of addiction, their withdrawal struggle appeared to be be minimized.

On the other hand, if a person disdained to start a drug reduction at the first sign of overmedication or recovery, he was usually setting himself up for an agonizing, drawn-out withdrawal or the hell of increasing overmedication.

Summary

The patterns that were recognized and named by the pioneers gave reasonable proof that dopamine was indeed accumulating in the brain at a rate independent of blood half-life. They also suggest that mechanisms may exist by which the brain can temporarily shut down all movement or accelerate the rate of dopamine dismantling. These patterns would seem to prove that "failure" of a particular dose may be due to dopamine rising above a certain danger level in the brain and not due to the failure of the drug to make its way into the brain (the conventional assumption). These patterns also suggest that Ons, Offs, and ups and downs may be occurring in response to previous doses: doses taken hours, days, weeks and months earlier.

There is tremendous variability in the rates of change: these patterns have been assigned approximate time frames, but these time frames are not hard and fast; they are not carved in stone nor cast in iron.

There are daily patterns, single-dose dependent patterns, two-to-three day patterns (Turnaround) and ten-day patterns (slides), in addition to the ten-week limbic adjustment. These variously timed events occur simultaneously. Therefore, the effects of any given pill may reflect the doses taken ten weeks ago, ten days ago, two days ago, earlier this morning, and an hour ago.

These drugs are neither unpredictable nor unstable: they simply must be understood in the context of their accumulated history.

Looking Ahead

In the next chapter, the patterns discussed in this chapter will be used as signposts in the cycles of change that occur during slow, sensible drug reduction.

Chapter Review

Daily Build-up – a daily pattern of increasingly ineffective doses
Daily Deficit – a daily pattern of increasingly effective doses
Roller Coaster – Offs, tensions, or pill failures that occur during a time of highest drug saturation
Turnaround Days – a bad day/good day/bad day pattern that occurs while the brain toggles from undermedicated to good to overmedicated
Superdosing – extremely high doses that override the addiction (protection) barriers
Crash – feeling worse after a dose wears off than before the dose started
Slide – a slow, (two to twenty day, generalized as "ten day") period during which symptoms begin to change
Switching – the worsening of symptoms just as a dose is beginning or ending

Walking the fine line between overmedication and undermedication

"What goes up must come down."

Sir Isaac Newton

14. CYCLES OF CHANGE

AN OFT REPEATING LOOP OF UPS AND DOWNS

No dose stands alone

Drugs taken up to ten weeks prior influence the daily patterns, single dose patterns, and several-day patterns. Although patients were trying to make daily explanations for their Build Ups, Deficits, Roller Coasters, Crashes, and Slides, the ten-week perspective was always more significant.

Ten weeks, more or less

Ten weeks is approximate, of course. It may take six weeks or fifteen for the drugs to come to equilibrium. Severe adverse effects of the drugs might linger for half a year or be semipermanent.[1] However, the most common time frame for the worst of withdrawal or the maximum benefit from increase does seem to be very nearly ten weeks to the day, as if there is a timed mechanism, a reset button deep in the limbic zone that has ten weeks as its refresh rate. However, due to the vagaries of weather, other health issues, relationships, and expectations, there may be other time frames to work with as well. I may use the term "ten-week phase" in this book. You will understand that this is an estimate.

Many of the current manufacturers' instructions for dopamine-, serotonin-, or norepinephrine-enhancing drugs hint at a two to three month period before the drugs reach full effectiveness. Our patients who were reducing drugs saw a marked shift in reduction symptoms within a similar time frame. Whether or not the ten-week period has to do solely with accumulation or solely with reset buttons, is a combination of both, or is reliant on some process of which we have no idea whatsoever doesn't really matter for our purpose. What does matter is this: the pill you are putting in your mouth today may be working in conjunction with all the pills you have taken in the last ten weeks.

Knowing that every dose was being affected by doses up to ten weeks earlier made the whole process of drug reduction even more daunting. How could one possibly

---

[1] We had first read the "ten weeks" number in *A Primer of Drug Action, Non-Technical Guide to the Actions, Uses, and Side Effects of Psychoactive Drugs*. It was with regard to one phase of cocaine withdrawal. It stated, "The withdrawal phase, which lasts 1 to 10 weeks, is the period of maximal relapse potential and drug craving which…may be associated with reduced dopamine transporter levels and reduced frontal cortical activity" (p.126). The rest of the text made it clear that the drug craving might be semipermanent, and yet, it had been repeatedly observed that week one to week ten was the time frame when a person was most at risk of relapsing into drug use. My patients often want to use this ten week number as an exact, inviolable number, and are often discouraged when they continue to feel like the underside of a shoe for eleven weeks, or even fourteen. However, even patients who feel miserable beyond the ten weeks do notice that right around ten weeks they can tell that something, however subtle, is improving.

track all the cumulative effects during a drug decrease? Maybe we needed a system to keep track of all these factors. We found, instead, a cyclical pattern that gave us what we needed.

CYCLES OF DRUG REDUCTION SYMPTOMS

The overmedication phase (adverse effects, dyskinesias, build ups) that bestirs one to initiate a drug reduction in the first place may recur again, after a person has gone through the various phases of drug reduction. The cycle appears thus: Overmedicated; Drug reduction; Vacation/Slide; Withdrawal; Back and Forth and/or quick Turnaround; Feeling Good; and back to Overmedicated again. The timeframe for completion of this cycle was, very often, ten weeks. The patterns repeat themselves over and over, following nearly every drug reduction.

The patterns described in the previous chapter could be used as signposts in the confusing welter of drug reduction symptoms to show where a person was in the cycle. Although symptoms on any given day might not be significant, what were significant were the repeating daily patterns, or subtle *shifts* in the daily patterns or dose-related patterns. Amazingly, if a person could think about his medication in terms of months and not days, and understood the significance of the patterns, he might be able to adjust his drugs to appropriate, maximally beneficial/safe levels without undergoing ferocious drug withdrawal symptoms.

Details of the cycle

Here, complete with signposts and (estimated) time frames, is a more detailed look at the cycle of drug reduction that might occur following a moderate decrease.

1. Overmedicated – This phase is marked by adverse effects: dyskinesia, overly bright eyes, illogic, unwarranted exuberance, daily Build Ups, Roller Coasters, freezing, or painful dystonias whether On or Off (most pain-causing dystonias are side effects of the medication).[1]

Time frame – How long might this phase last? If no changes are made in medication levels, this phase will last indefinitely and will most likely continue to worsen. Even if the medication is reduced, these symptoms may still continue for a few days.

2. Vacation and Slide – The symptoms of overmedication may begin to retreat: a possible decrease in dyskinesia, tension, insomnia; fewer Build Ups, Roller Coasters, or drug failures.

If one begins to feel downright healthy and well balanced, this is referred to as a Vacation, or the calm before the storm. While these overt signs of improvement are occurring, the brain may be invisibly sliding down into a state of limbic insufficiency.

Time frame – This phase may last one day to twenty days, ten on average. Usually within ten days there are hints of drug reduction symptoms. If a drug reduction was inadequate, there may be a return to symptoms of overmedication within a day or two of

[1] Drug excess symptoms include *any* form of adverse effect, even if an MD says it's "normal."

the Vacation. Such an abrupt return to overmedication does sometimes occur. It may signify that another immediate drug reduction is in order.

3. Limbic Stress – This phase abuts the dreaded agonies of drug withdrawal. During this time drugs may appear to be ineffective, or if they work, they may provide uneven coverage. Possibly only a few of the daily doses will be effective. It is just as likely that none of the doses will work.

Symptoms include: nausea, free-floating anxiety, insomnia, immobility, shaking with terror, or frantic, restless pacing. Any learned symptom of dyskinesia may appear, including ticcing and spasming of all muscles, including lung and heart.[1]

Time frame – These symptoms usually worsen over a period of up to ten weeks. After ten weeks these symptoms may abruptly cease or they may begin to ease up ever so slightly. The change after ten weeks may be so subtle that there may be only a sense that a change is pending, even though the symptoms are still raging. Prior to this, there may have been a sense of impending doom and ever-worsening symptoms.

After ten weeks there may be moments during which a person begins to resume brain clarity, even remembering, at times, why he made a drug reduction in the first place. The time when a person is most likely to succumb to resuming the drugs at the previous, overmedicated level is in the span from ten days to ten weeks after decreasing the medication. After ten weeks have passed, a person might begin to see that, just possibly, his body is starting to readjust to the decreased amount of drugs.

4. Back and forth – This sometimes-tortuous phase can feature fewer drug withdrawal symptoms, or the drug withdrawal symptoms become erratic. Just when one starts to be certain that the withdrawal is over, there may be a day, two days, or alternating days of drug withdrawal, even while days of relative peace and normal movement are occurring.

Some doses of drugs might be effective or partially effective. It may be that the afternoon or evening doses are more predictable than they have been (daily Deficit). The effective doses may be less prone to crashing than they were before. The switching phase of the drugs may or may not be much worse, depending on whether or not a person is producing more native dopamine. This phase may precede or take the place of the Turnaround, which is the time of transition from insufficient drugs to excess.

Time frame – This phase may last anywhere from a day or two to several months. In general, a person who has never been addicted and who makes prompt reductions at the first flush of feeling good or at the first inkling of dyskinesia or grimacing will spend less time in this phase than a person who has become addicted. If no addiction has ever occurred, a person may be able to get through this phase in a matter of days or even hours. It can be quite abrupt.

[1] A complete list of drug reduction/withdrawal symptoms is in Appendix 3.

5. Feeling good – This phase might start small and build or burst forth overnight, fully blown. The medications may suddenly switch from unpredictable to utterly predictable. Symptoms are: "fantastic!" and "feeling human again!" This stage is particularly dangerous. After the long, slow slog through drug withdrawal, the temptation can be to linger as long as possible in this medication-induced glow.

It is far safer to be perpetually undermedicated than to ever, however briefly, be overmedicated. Therefore, if a person who previously had Parkinson's disease is suddenly, because of his drugs, feeling on top of the world, it should be clear to the meanest intelligence that something is wrong. Although the person may feel that he is finally balanced again, he is in fact on the verge of overt overmedication.

Time frame – It may be a matter of hours, or it may be a matter of up to three days, but inevitably, the good feelings of this period will make way for symptoms of overmedication. In fact, it is overmedication that is causing this glee. The turnaround, the abrupt transition from feeling lousy to feeling great, is usually an indicator that a person is overmedicated.

If a person *were* perfectly medicated, neither excessive nor deficient, he would not feel particularly good. He would feel a peaceful pause from the rigors of drug withdrawal but he would also recognize faint but unmistakable signs of those particular Parkinson's disease symptoms that are dopamine-related: primarily, slowness of movement, thought and speech, and depression.

If the person is also recovering from Parkinson's, he might be feeling extreme weakness and/or the exhausting but emotionally satisfying symptoms of Parkinson's recovery (pain in previously numb areas, lack of muscle tone in areas previously rigid, extreme fatigue, unexpected susceptibility to emotions, sadness, and tears, lassitude, etc.). In either case, he should not be "feelin' good!"

One should be either slightly affected by Parkinson's symptoms or even experiencing the rigors of recovery, neither of which feel particularly good; feeling good – in a medicated person with Parkinson's – is a subtle sign of drug excess, so the overt symptoms of overmedication are just lurking around the corner when one once again feels good following a spate of misery-inducing drug reduction symptoms.

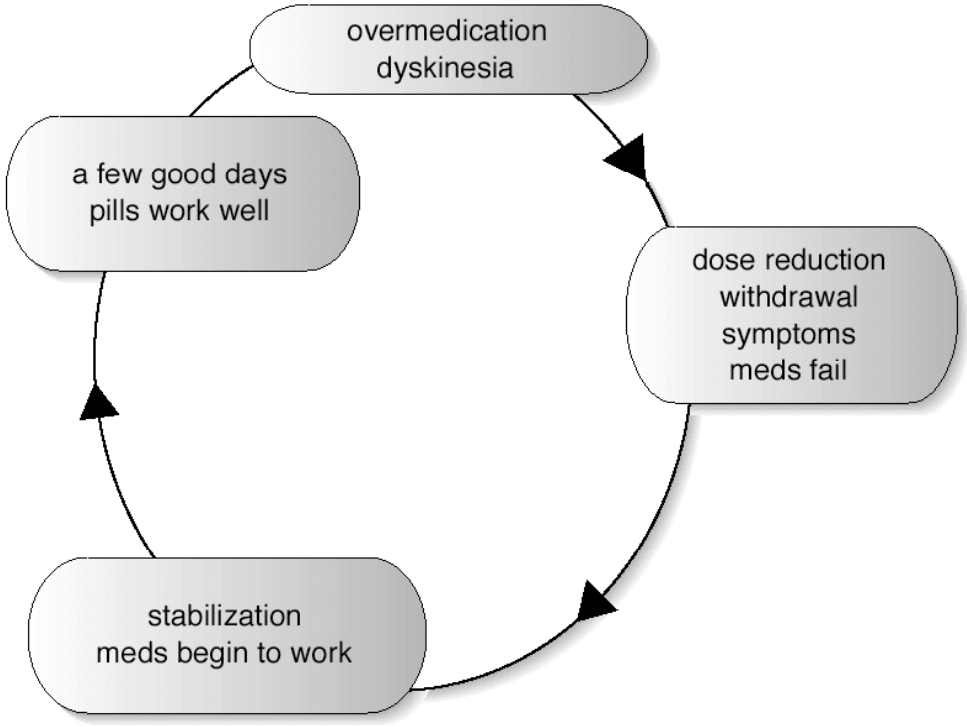
The rule of thumb is this: *if you have Parkinson's and are taking medications, if you feel good, you are probably overmedicated.*

Only when a person is completely off the drugs can he finally risk feeling truly joyous. Only then can he be certain that his joy is coming from his true nature and not from an overloaded limbic system.

Again, this phase usually lasts one to three days.

6. Overmedicated – This phase is marked by overt adverse effects: dyskinesia, overly bright eyes, illogic, unwarranted exuberance, daily Build Ups, Roller Coasters, freezing, or painful dystonias whether On or Off.

Time frame – How long might this phase last? If no changes are made in medication levels, this phase will last indefinitely, and will most likely continue to worsen. If the medication is reduced, these symptoms may continue for a few days.



The drug reduction cycle

This cycle might be repeated after every drug reduction. The most obvious way to know that a person has completed a cycle is the return of inner peace, or worse: symptoms of overmedication. As soon as a person feels good, he might need to make another drug reduction and begin the cycle anew. This disheartening realization – that there could be no pauses during this cruel cycle – was almost the hardest thing for recoverers to bear. The very hardest thing was the fact that, if a person had ever been overmedicated, however briefly, he might never recover completely from the drug-induced brain damage. Instead, after recovery from Parkinson's and getting completely off the drugs, he might linger forever in the Back and Forth stage of the cycle: on some days he may manifest no Parkinson's symptoms; on stressful days, PD may appear full-blown.

A lifetime of permanent back and forth

A person who has become addicted to the medication may always be in the back and forth stage after he gets down to a small amount (none-to- 150 mg levodopa/day).[1] If a person has ever spent too many hours on the drugs at excessive levels, he may, for the rest of his life, have days when he feels good alternating with days on which he feels miserable, even if he gets off the drugs completely and has no Parkinson's symptoms, per se.

Although he may eventually get off the medication and be able to live well on a maintenance dose of 25 mg levodopa/day or a minute dose of whichever DED he prefers, for the rest of his life he may always hover at the edge of dopamine sufficiency, on the lip

[1] For comparable amounts with other drugs, please see chapter 17.

of the threshold. Any higher dose will push him over the edge into adverse effects and ever-increasing addiction.

The very small dose of DED may be required to compensate for the addiction-induced brain damage. This person may never be truly at peace if he is not taking the drugs. Without the medication, a person who was addicted may find that he simply cannot bear the tardive dyskinesia, the painful, heightened sensitivity and depression that may ever be his lot, should he stop the medication completely. And yet, even at extremely low doses, the medication may cause dyskinesia, mental imbalance, and gradually worsening parkinsonism. This person may forever have symptoms of overmedication, even at microscopic doses, but without the medication, his discomfort may be too great to support a life worth living. A person in this difficult situation may find that the slightest change in the weather, the onset of flu, or the mood fluctuations of everyday life can send him into a tailspin that can last for days.

In our experience, anyone who takes dopamine-enhancing drugs for a period longer than three days after having manifested even the slightest signs of recovery from Parkinson's is at risk for a lifetime of back and forth even if he gets off the medication or is somewhat stable at an extremely low level such as 25 mg levodopa/day.

Perpetual ticcing or tremor

If a person's tremor was caused by or increased by medication, he may, for the rest of his life, manifest shaking at the slightest sign of stress, or he may develop constant tardive dyskinesias even if he recovers from PD – just like some people develop years later after having taking antianxiety drugs and antidepressants. A person who had tremor with his Parkinson's who *ever* took the antiparkinson's drugs will probably be especially susceptible to tardive dyskinesia even if he recovers from PD. As for the rest of his Parkinson's symptoms, they may continue – symptoms now of drug-induced parkinsonism.

Pleading for mercy

Strangely, or maybe not so strangely, patients who were starting to feel truly good again after months of withdrawal despair would beg me to "allow" them to postpone the next drug reduction. I always reminded them that I had nothing to do with their drugs. Each person had to make his own decisions about his medications. I was not able to say anything one way or the other, except to point out what people had done that appeared to be effective, and what people had done that appeared to be a formula for disaster. And still, after I explained all this, they would cry and ask if I couldn't make an exception in their case.

No matter how many cycles of drug reduction they had experienced, each cycle seemed new at the time. The limbic area does not seem to have much ability to reason and remember. It cannot remember that the bad days of drug withdrawal have been followed, every time, by an improvement in overall health and drug function. The limbic system lives one breath at a time. It only knows that the drugs made it happy, and that during reduction it is desperately unhappy.

Laurel, as described in the last chapter, made the brave decision to make another reduction straight away. She was atypical. Most patients working without a drug gatekeeper chose to postpone making subsequent drug reductions.

Feeling good was bad, feeling bad was good

Feeling good was a sign of overmedication? Offs were caused by too much medication? This was painfully counterintuitive.

Nearly all the doctors, except for those most recently out of school, would treat the Offs as if they were symptoms of insufficient medication. The crazy idea that having Offs, Build Ups, and increasing periods of immobility were signs of drug excess was the opposite of common sense. Nearly every patient struggled with the idea.

When patients were Off, they had only one thought: how to get On. When Off, no matter if the Off was due to a Build Up or was occurring in the middle of a Roller Coaster, More Drugs always looked like the answer.

The spouses could understand the premise. The people with the drug addiction really struggled with it. They might be able to understand it while they were in my office, but they could not understand it the moment that the Off began. Remember, anyone who is so far gone that On-Offs are occurring *is* addicted, and therefore somewhat addled.

Blurred signposts

I found one thing to be fascinating: patients were usually very honest when writing up their journals or filling in their graphs. However, in my office, when confronted with the evidence of their own charts or journals, they were, addict-like, often careless with the truth.

For example, Becky would look me in the eye and tell me that she had reduced her medication. I would point to my copy of her weekly journal and show her that she had increased. She would be baffled every time.

A few addicts were honest if they were Off. Most were not capable of objective, reflective self-analysis whether Off or On.

Hua To

Hua To Huang had been slowly reducing his medication for over a year. He had been hideously overmedicated when he started working with me. His dyskinetic contortions of face, neck, arms, legs, and torso were pitiful. He was only 40 years old. He had been diagnosed several years earlier. He appeared to be recovering: his facial expression returned, his sense of humor came back, and some of his movements appeared to be more fluid. He had never had tremor and had no signs of hand-tapping tardive dyskinesia. Of course, the drugs were masking much; we had no way of knowing what his underlying PD symptoms actually were.

He was deeply sincere and scrupulously honest in his self-evaluations when Off. At such times he could objectively report to me that he had been overmedicated during the week and tell me about his dyskinesia. However, when he was On, he was unable to tell me whether or not he had been overmedicated. Sometimes, even while his head jerked violently back and forth, he would tell me that he hadn't been overmedicated in the last week, and no longer had dyskinesia. If I pointed out that he was having it right before my eyes, he would try to explain to me, "Basically, what is happening is, I am moving right now but, basically, it is not a problem."

His compassionate wife might plead with him to tell the truth, but he had no idea what she meant. On the other hand, he was able to record faithfully in his charts that he had been over the top during his Ons.

Altered self-awareness

It almost seemed as if there was a split personality involved in some of the medicated patients. It was as if the medication allowed a person to feel confident in his confrontations with the world. A person under the influence was able to behave with confidence and joy, seemingly unaware of his drugged condition. And yet, it seemed as if there was always another person inside the cheerful (or tormented) shell that was able to recognize the drugged person as a mere puppet.

I suppose that this is not so strange at all. We all know of people who, when drunk, are supremely self-confident and a friend to all, can see no problems with their behavior and may not even consider themselves to be under the influence. And yet, many an imbiber, if you look him in the eye and ask with sincerity whether or not he is drunk, may answer happily in the affirmative. One part of him knows that he is drunk, and yet the part of him that is interacting with the world may feel supremely confident that it is his true, brilliant self that is manifesting.

For this reason, alcohol is considered (in drug study circles) to be a "disinhibitor" as well as a depressant: alcohol makes a person feel as if his inhibitions are gone, as if his drunken behavior is more honest, capable, loving, and intelligent than his everyday, inhibited behavior. Of course, as we all know, a drunk has less clarity of thought, is less capable of physical function, is more susceptible to mood swing, and is less able to assess his own performance than a sober man.

The drunken *illusion* is one of increased openness and affability. The *truth* is, a drunk is addled. And, most significantly, there is usually a second personality, a higher awareness, hidden behind the mask of drink. This inner person knows perfectly well that he is, in fact, a wise man cloaked in the temporary garb of a stupid drunkenness that thinks itself clever.

The inner wise man

In the case of PDers who were recording their daily symptoms, it appeared that it was usually the wise man, the objective man behind the drugged man, who kept the diary. It did seem that people taking antiparkinson's medications were able to objectively write down their symptoms of the day. And yet, during an interface with me or their spouse, they might not be able to assess their past week's symptoms with any degree of objectivity whatsoever. I would be reading their journal, noting that every dose of medication was causing dyskinesia, but when I turned to the patient to say, "Looks like a lot of dyskinesia this week," the jerking, grimacing patient might respond with, "There was no dyskinesia this week."

How then, if a person's objectivity is altered during drug use and during drug withdrawal, will he be able to make use of these signposts? Correctly interpreting these signposts might require a painful degree of honesty and symptom analysis. This brings us to the need for a helping hand.

NEEDING HELP

Nearly all the successful patients, the ones that stayed in the program and especially those who got off their medications, were the ones who came with a partner. The friends, caregivers, or spouses of the patients provided critical support. Very often, people going through medication adjustments would be confused, argumentative, hostile, and even violent when spouses would tell them that they were having dyskinesia or other symptoms of overmedication. The valiant spouses who kept on in the face of this seeming ingratitude and helped to analyze every hour of their PD partner's life for several years were equal heroes in the experience.

Of all the medicated patients that were trying to reduce their drugs, only two people who lived alone succeeded in getting off their medications.[1] All the others, those who lived alone and those whose spouse/partner felt that the PDer needed autonomy with the drugs, failed to successfully reduce their medication. Worse, those who were living alone or trying to go through drug withdrawal without the support of a friend or spouse were the ones who were most likely to go crazy and use the drugs to create euphoric On! episodes, ultimately ending up in care facilities.

Expecting the doctor to be the support person

Even the relatively objective spouses had trouble at times determining where a person might be on the cycle of drug decrease. So many questions arose: if a person was not showing signs of improvement nine and a half weeks into a drug reduction, did that mean that the reduction had been too severe? What if a person felt great after the evening pill but still had horrible Offs in the morning – was this person in a Deficit, a Turnaround or simply in a moderate stage of drug withdrawal? Their best hope at figuring it out was by having records of every behavioral change over the last ten weeks.

No doctor can be expected to follow the dozens of daily symptoms and guess whether or not a person is sliding up, sliding down, on the cusp of overmedication or nearly done with withdrawal. No one can guess whether or not a particularly difficult withdrawal phase is due to an over-ambitious reduction or the result of having lingered too long in Feeling Good in a previous reduction. There are so many variables! Unless he lives with you, it is not reasonable to expect your doctor to be your support person.

An example of medication gatekeeping

To illustrate how difficult it can be to decide whether a person is undermedicated, overmedicated, in withdrawal, or simply suffering from Parkinson's, and to drive home the idea that a drugged patient cannot go this alone, let me share from Sophia's purple journal.

[1] Taylor Paul and Buzz. I am referring in this statement to those people who had been on dopamine-enhancing drugs for more than a few months. A few people such as Elaine and Sammy, who had been recently diagnosed and were only taking very small, starter doses of medications at the time they joined our program, were able to get off the drugs quickly by themselves.

Hjalmar

Sophia was right beside her husband Hjalmar every step of the way. It was she who decided when and by how much he should reduce. She kept the little purple notebook of his doses and any symptoms that might be significant. Hjalmar had been diagnosed with Parkinson's disease eighteen years earlier, in 1980. His original symptoms were right hand tremor, no right arm swing, and utter lack of facial expression; he was only 47 years old at the time of diagnosis.[1] Dr. Rafferty had been his neurologist from the start, and there was warm, mutual respect between these two men who had been doctor/patient for nearly two decades.

At the time of this journal excerpt, Hjalmar had clearly started recovering from Parkinson's (Dr. R had remarked on his many striking improvements), and had been slowly reducing his medication for over three years. He was down to 300 mg levodopa/day (3 of the 25/100 Sinemet, regular) from a high in 1998 of 1100 mg levodopa/day (combined Sinemet regular and Sinemet CR), Artane (an anticholinergic drug, 5 mg three times a day), and Mirapex (a dopamine agonist, .5 mg three times a day).

After he had been at 300 mg levodopa/day for several weeks (after having decreased three weeks earlier from 350), Sophia noticed some strange new patterns emerging: drooling, shuffling, and inability to feed himself. These symptoms seemed to actually get worse two hours after each dose, but she felt that they were clearly signs of Parkinson's, not symptoms of overmedication. Hjalmar continued to worsen for another two weeks, and so she increased the drugs slightly to an average of 325 mg/day. He got much, much worse, so she decreased for two weeks to 275 mg/day. He got worse still. So she increased again to 300 mg/day. This made him frantic and yet he continued drooling and unable to move. So she decreased again. He got still worse. She went back and forth over several months. Here are some of her notes towards the end of this difficult time. She made notes every day and created a week summary from the daily notes.

As you read through this, try to determine for yourself whether Hjalmar is in drug withdrawal or overmedicated. Remember, adverse effects of Sinemet can include drooling, slowness of movement, incontinence and hallucinations. Symptoms of drug withdrawal can include drooling, slowness of movement, incontinence, and hallucinations. Parkinson's disease can have symptoms of drooling, slowness of movement, and incontinence. I have put the dosage changes in italics.

> 6-22-01: 300 mg/day all week. Very weak, drooling, leaning forward, peeing in the bed, can't get up from chairs, confused regarding urination in the daytime, peeing on the floor. Squirmy dyskinesia. *Reduced 3 p.m. pill in half due to dyskinesia yesterday*. Last night dry at bedtime.
>
> 6-29-01: Couldn't figure out how to put his clothes on. Stuck to the floor all the time and squirmy dyskinesia after just one pill in the morning.

[1] Photographs of Hjalmar's return of facial expression were included in the article: J. L. Walton-Hadlock, "The Use of Yin Tui Na and Stomach Channel Acupuncture Points in the Treatment of Facial Immobility in Parkinson's Disease," *The Journal of Chinese Medicine*, No. 69, June, 2002, p. 43-47.

7-6-01: Wakes up fine, but after one pill he is stuck to the floor, anxious, bent over. One day *he forgot his 3:00 p.m. pill* and he was less stuck in the evening.

He seems to be better with fewer pills, but his last reduction was so recent, and he is drooling and bent over so much of the day already, and last time when we reduced he could barely move. Reluctant to reduce again.

7-13-01: Walking slowly, different than the freezing, this is a completely new pattern of slowness. He must be overmedicated. One day *he took only 200 mg* and did so much better, and in the mornings, prior to taking his pill he does better than an hour after taking it. But he can no longer get in and out of bed by himself, he is profoundly weak, and slightly paranoid, I think. Last night he said, "I'm afraid I'll get pregnant." He's confused in general. *Reduced to 200 mg/day.*

7-19-01: Very weak. Needs help throughout the night, falling down even with his walker. Drooling, incontinent, cannot get into bed. He can't even wash his hands or face. He's getting crazier. He was fighting me when I tried to help him, gripping me too hard. He can't feed himself. He is much worse this week; evidently he did not need a reduction in medication after all, he must have needed an increase. *Back up to 300 mg/day.*

7-26-01: He's stuck, drooling, incontinent, singing, laughing, noisy all night. His right hand doesn't work at all. He simply can't use it. Up every hour to pee at night, smiling away. He fights with me when I try to help him to the toilet. May not know who I am. I think it was a mistake to increase the drugs. *I reduced him down to 200 mg/day* and the next night he slept better.

8-3-01: A rotten week! Had helpers over, and he was fighting with them. Found him in bed one morning with only one diaper on, don't know what he did with the other one or his pajamas. Needs help with everything. For the first time in his *life* he was too weak to take communion at church. He needs help with everything. It's as if he's given up trying. He's drooling and weak and incontinent.

8-10-01: He so much worse, *tried giving him Mirapex* – he hasn't taken that in a year, but he's so bad. After two days with Mirapex he was much worse, craziness, hallucinations, thought he had a pink bunny in his hand. Wanted me to get rid of the Coke in his hand. He wasn't holding anything. Thought he was up on the roof. He was sitting in his chair. More stuck. *Stopped the Mirapex.* I forgot his afternoon pill on Tuesday and he made more sense that evening, so ***Wednesday I quit giving him any pills.*** Simply don't know what to do anymore. He's crazy with fewer pills, he's crazier with them. I know this is too fast, it might be dangerous, I can't take it anymore. But he's been dry at night the last three nights. Still getting stuck and drooling.

Now, based on the neat and clean theory of easy to interpret cycles, just what was going on with Hjalmar? He had not had any obvious days of Feeling Good, and yet, based on the laughing and singing all night, together with the hallucinations, Sophia finally decided that the real problem was, in fact, overmedication. You need to bear in mind that Hjalmar had been one of my first patients, and every single dose reduction had been an unpredictable adventure. At the time that Sophia decided to stop the drugs cold,

we had only recently determined the 10% rule to prevent life-threatening withdrawal, and we had also seen a few instances where a person in recovery needed to make reductions faster than 10% in order to avoid hysterical mania. (In these latter cases, a person went from undermedicated to riotously overmedicated, seemingly overnight. In these cases, the problem was not life-threatening withdrawal, but mind-threatening overdose.)

Sophia was trying to guess, from the maze of symptoms, whether Hjalmar was at one extreme or the other, or whether he was simply manifesting the true face of his Parkinson's disease. The "squirmy dyskinesia" and hallucinations decided her: he was over the top.

> 8-17-01: One night he completely soaked the bed. Don't know how much fluids he had. Four nights later, slept through the night, and dry! Yes! Daughter Mary stayed with him for a couple of hours on Tuesday and says she will never do it again. She thinks she hurt her back trying to pull him up out of his chair. He went for a walk with Dwight this week, but still needing help with almost everything. DROOLING all the time.
>
> 8-24-01: Washed himself and got himself partly dressed. Eating better by self but still wants help with everything. Drooling still terrible. Got out of chair by himself but needed help the rest of the day. Did a good job at the NovaCare facility Weight Training for Seniors program, raised to level 4, but slept the rest of the day. On Tues the bed was dry and he walked three times around the (.33 mile) park loop with Dwight, but needed help with everything the rest of the day. Weds, nothing special, but he can't convince himself that he can do anything without being helped. Fri, at NovaCare increased two of his weights to level 5 – still can't do anything but drool at home.
>
> 8-31-01: Everything the same. Sure would be nice to see some improvement. Weds, got off toilet by himself. Sat. absolutely grinned at his new haircut. (At the time Hjalmar had started our program he'd had no facial expression for over twenty years even when medicated.) We had company for an hour or so and I only had to wipe his drool once! Rest of the day needed help with everything. Fixed his own lunch one day!
>
> 9-7-01: Drooling might be slightly less. 3 times around the park (no walker, as usual).
>
> 9-15-01: Went to Community Theater last night, didn't drool too much. Friday he took himself to the bathroom. Needs help with everything, still drooling. Last night couldn't even eat pizza, the easiest thing to eat. Wanted us to feed it to him. Arms increasingly rigid. Did I do wrong to stop the medication?
>
> 9-28-01: Less drooling the first half of the week. Walking a mile in the park three times a week. Helper comes in three times a week to give him a shower. He is having trouble feeding himself. *But two times this week I had a real conversation with him, the first real talks we've had in nearly ten years.*

This week, at his weekly acupuncture session, he expressed an opinion for the first time as to what he wanted help with: "Could you do acupuncture on my mouth or lips? Sophia has a hard time with the drooling." This thoughtful remark nearly reduced Sophia to tears. Until now she had never been able to tell if he even knew what she was

going through. The "two conversations" that they had had were about his concerns for her, wondering what would happen to either of them should the other one die first. He wanted to know if she had made any plans in that direction. After nearly twenty years of mind-altering drugs he was sincerely curious and concerned about the future and aware of the needs and feelings of people around him, for the first time in a decade. He was also self-conscious about his drooling for the first time.

> 10-4-01: Terrible drooling, needs help with everything, but sometimes could feed himself. Walked a mile in the park two times, did his weight program. His sleep is fantastic, and his voice is reasonable.

His voice had been inaudible prior to starting our program, and although his voice had returned, it had disappeared again around the time that the drooling, hunched posture, and unpredictability of the medications started, back at the beginning of summer.

> 10-18-01: A good week. He got dressed by himself two times this week, one time he had a great big booming voice. Better able to feed himself. Got up and down from chair by himself once this week. I think he can't see real well – he sits around with his eyes closed. Did fine at the six month MD visit, though he didn't swing his arms when he walked. Dr. Rafferty was not real curious as to why we'd quit the drugs, didn't ask us any questions.
>
> 10-26-01: <u>Ten weeks with no medication!</u> On Saturday morning Hjalmar noticed that he was cold, and then later that afternoon that he was hot. Even with his ice cold feet he has never noticed temperature in his whole life! Slight improvement in dressing and eating, balance is lousy but no falls, still difficulties with chairs, toilet, drooling, and initiating movement. Sleeping a lot. Overall, much more calm and manageable than those last few months on the drugs.
>
> 11-2-01: Same this week, lots of drooling, sleeping. Better bladder control. Lots of talking in his sleep. His mental clarity is fine. In Sunday school he made two contributions to the discussion, and they were good, cogent points. He hasn't contributed in years. People in church were commenting, "He's in there again." Or, "His mind is back!" He's using his hands better this week. He got his underwear and T-shirt on by himself. He's extremely weak. He has trouble initiating movement, although he walked 1.25 miles one day. He simply cannot get up from the toilet or chairs. Much of the time his voice is OK.

Hjalmar got his flu shot, a process that stimulates the immune system and which can induce mild symptoms of illness. Whether it was the flu shot, the onset of cold weather, or the Thanksgiving dinner, he had a wild week. This was also the fourteenth week after Hjalmar had stopped taking any drugs. Here is the report of the week's symptoms:

> 11-30-01: Yelling in the night that his shoes were on. Hallucinated holding a coffee cup in his hand, didn't know what to do with it. Didn't remember our 30-years neighbor, eating imaginary cookies, going through the motions. Sitting on the stairs, didn't know how to get down. Pooped on bathroom floor and didn't

know it. Says he doesn't know how to play the radio or get out of bed. No incontinence, though, and less drooling. Walked about a mile, three times. One crazy day!

12-7-01: Several times reached out and turned on lamp by himself. Dressed himself several times, got into chair by himself. The week is getting better. His chronic shoulder pain is less frequent. Urination good all week. Feeding himself is slightly improved. No hallucinations.

Hjalmar told me with wry pride at the weekly visit, "Today I made my own toast and poured my own juice!"

12-21-01: Getting himself dressed. Washes up self in the morning. Only needed help with food one time. Needs help to get out of the chair but can get off stool and the high bed by himself. Also, can get out of lift chair by himself. He gets stuck in doorways or in the bathroom, but just needs a shove to get going again. One hallucination: laying in bed, thought he was stuck in a vacuum cleaner. I think that he may have been only half awake, dreaming. Dreaming is allowed! Drooling bad some days, less on other days.

I included some bits from Sophia's journal that extended beyond the ten-week period because it demonstrates just how extremely slow recovery can be. Also, it makes the point that at ten weeks (Oct. 26) there was not an abrupt return to perfect health. However, at ten weeks there was the beginning of recovery from the drug withdrawal. The most significant change was that he started to make sense; he was capable of meaningful conversation and was concerned for the welfare of those around him.

In retrospect, he probably would have done better had he reduced his medications more quickly – after first noticing recovery symptoms – instead of taking three leisurely years. Sophia, nearly 70 years old, had been understandably reluctant to make reductions, despite his increasing dyskinesia, if Hjalmar was at any time slow or having difficulty moving. This meant that she only decreased the drugs when adverse effects of the drugs became harder to bear than the weakness and other recovery symptoms. By the time he took the last of his medications, he was clearly suffering from overmedication. This overmedication and prior decades of overmedication have doubtless contributed to a serious level of brain damage. Hjalmar may never recover the degree of movement that we are hoping for. However, he does continue to improve.[1]

---

[1] As for the rest of Sophia's journal, which records Hjalmar's very slow recovery of physical function, it is too long to include here. It must be read with the awareness that he had lived with Parkinson's for over twenty years by this point, and he had been taking mind-altering, powerful stimulants day in and day out for most of that time. It was to be a long, hard haul for Hjalmar before he could engage in much physical activity. However, he still feels strongly that Sophia had made the right choices for him, and that joining our project was a good decision. As Sophia regularly points out, "All the other people who were in the support group when we joined are long dead. We must have done something right."

When Hjalmar most recently saw Dr. Rafferty, he used his walker. Although he had used a walker for years, even before starting our program, he had made it a point of pride to never let Dr. Rafferty see him using it. Dr. Rafferty said that he was sorry to see Hjalmar reduced to using a walker, after he had done so well for so long. (Footnote continued on next page.)

I included the above notes from Hjalmar and Sophia's journal to show just how difficult it can be to know whether or not a person is struggling with withdrawal, overmedication, Parkinson's, or all three. Had Hjalmar been going through this alone, he could never have made the difficult decisions regarding drug reduction. In fact, many times during the preceding years, when Sophia had made a small reduction in his drugs, she would find Hjalmar in the kitchen between meals, looking through the drawers.

"What are you looking for?"

""I'm looking for my pills."

"You already had your morning pill. You'll get another one in two hours, at 11 o'clock."

"Oh. OK. I want another pill."

Half an hour later Sophia would hear footsteps in the kitchen. There would be a replay of the above dialogue. Half an hour later, they would perform the scene again. Sophia quickly learned that she had to hide the medication and never let Hjalmar see where she kept it. During many drug withdrawal phases, Hjalmar didn't even know that she had made a small reduction, sometimes just chipping away at a part of a pill. And yet, within a few days of starting a reduction he would start looking through the kitchen drawers, feeling as if he had forgotten to take his pills.

Hopefully, this vignette conveys the difficulty in evaluating drug symptoms that may be influenced by drugs that have been taken up to ten weeks earlier, combined with symptoms of Parkinson's disease and symptoms of recovery. If the above sketch helps even one person realize that drug reduction is challenging and should only be entered into with a true friend, partner, or spouse, then the preceding pages will have been well spent.[1]

Dr. Rafferty, an outspoken opponent of Asian medicine, had attributed Hjalmar's improvements over the last three years to Sophia. "You certainly do take good care of him," he said. "He's doing better every time I see him." So when Dr. Rafferty, a deeply caring, warm-hearted man, saw Hjalmar using a walker and learned that Hjalmar was still not taking his antiparkinson's medications, he was saddened.

He asked, "Are you happier, not taking the pills and having to use a walker?"

Hjalmar replied forcefully, "I have my mind back. That is worth more to me than appearing to move well. Besides, I've made a lot of progress. My first symptom was my loss of facial expression and my tremor. I don't shake anymore, and now I can smile again."

Dr Rafferty suggested, "No, you can't."

Hjalmar, who had long awaited this opening, said simply, "Oh, yes I can…" And flashed a radiant smile at Dr. Rafferty.

Dr. Rafferty stared. Hjalmar grinned bigger, and started to chuckle. Dr. Rafferty looked slowly back and forth from Sophia to Hjalmar. He concluded their biannual visit by saying, "I guess you know what's best, and I'll see you in six months."

[1] What is not mentioned in the journal notes about Hjalmar is this: like most people with Parkinson's he had been tremendously driven, with powerful intensity of purpose, boundless strength, and stamina. That was not the reason he did so well in our program. I suspect that what enabled Hjalmar to survive the drug reduction and the recovery from Parkinson's were qualities more rarely seen in PDers: humility and acceptance that a plan of great beauty and love lies hidden behind the unfathomable will of God. Throughout his years with me, he never veered from his conviction that God was with him, and that every lesson he learned from Parkinson's was a blessing. Though he strove for health and battled his Parkinson's, on a deeper level he accepted his illness and embraced his battle with the same humility with which he had accepted his great physical strength and power of his earlier, pre-Parkinson's days. Those qualities of strength and purpose, so often treasured by PDers, were deemed by Hjalmar to have been only temporal gifts from God, to be used in manifesting God's love. (Footnote continued on next page.)

Mark

Mark and Margaret are another example of how a partner can be of help during these difficult times. I will have more details on Mark's very impressive, slow and steady recovery, thanks to Margaret, in a later chapter. But for now, I will point out that one time, when Mark made a joke after weeks of staring at the walls, Margaret made an immediate, slight reduction in his drugs. Her suspicion that Mark was on an upslide was exactly right: four days later, he was having dyskinesia after his first dose of the morning. Another very small dose adjustment was all that was needed to plunge Mark back down almost to the edge of drug withdrawal, where he hovered for another ten weeks. Had Margaret not already initiated one reduction based on the simple observation that Mark had made a joke, Mark would probably have been subjected to a more violent up and down than he subsequently experienced.

Margaret was extremely tuned in to tiny alterations in Mark's behavior. Hjalmar's wife was also. Sonny's young, second wife was also very aware of any change, but her extreme resentment of the extra work required of her while Sonny was in withdrawal made her want to delay every drug reductions until a "more convenient time" or until he was in agonies of dystonia and dyskinesia. She frequently pointed out to me that she had not planned on spending retirement taking care of Sonny, and she was bitter that life was "passing her by." Sonny's reductions, when she got around to making them, were usually painful, long, drawn out agonies of withdrawal.

Hjalmar again

For another example of a spouse detecting a positive change amidst the darkened clouds and adjusting drugs accordingly, let me insert this quickie about Hjalmar that occurred when he first got down to 300 mg/day, just before he sank into the confusing welter of symptoms described earlier.

Sneering at sports

Sophia, like Margaret, was getting good at seeing patterns in Hjalmar's drug decrease cycle. She was especially excited when the following, new benefit/symptom of drug decrease occurred:

"Last week," recounted Sophia, "a friend got tickets to the Giants game up in San Francisco. Hjalmar didn't come. He's always thought spectator sports were a waste of time, but I am a complete baseball nut. So after the game – it was a great game – when I got home, Hjalmar was just sitting there in his chair, smiling, with this wry sort of smile on his face, like he used to get years and years ago before he ever had the Parkinson's. So I asked him what he was smiling about, and he said, 'I'm just picturing fifteen thousand adults sitting around getting excited watching a few grown-up men in funny clothes throw a ball back and forth.' That's my old Hjalmar! That's just how he used to talk twenty years ago! It's his real self! I don't care if he can't move very well, I've got my husband back!"

When God took away his strength, Hjalmar, like a shorn Sampson, grew closer, not further, from God. Despite Parkinson's disease, Hjalmar still loved Him.

By following these quick glimpses of her husband acting like his old self after decades of contented illogic, Sophia was able to summon up enough confidence that things were going the right way that she could override hers and Hjalmar's fear of further drug reductions. This little event was just one of the changes in Hjalmar that helped her make her subsequent bold decision to stop the drugs when they became so unpredictable at what Dr. Rafferty called "very low levels."

Summary

There is a pattern to drug withdrawal. The drugs are not the utterly unstable and unpredictable monsters that they seemed to be when they were first discovered. However, the pattern is an extended one, a several month cycle, not a several hour cycle.

After a drug reduction, while a person is going through this long, ten-week cycle, there are signposts – daily and hourly symptoms – that indicate just where a person might be in the cycle. These signposts may be obvious, but they are more likely to be subtle.

A person who is about to step onto the path of drug reduction should be willing to seek daily help and support from someone who is willing to travel alongside. If there is no such person available, one is probably better off never to attempt reducing the medication, as the subsequent ups and downs and backs and forths invariably lead to extreme excesses of the medication. And finally, while I am shooting off warnings, no medicated person should attempt to go through the recovery process: the potential risks of recovering while still medicated are worse than Parkinson's disease.

Statement of purpose: review

In case you are wondering why I am writing this book, considering that I am stating that no medicated person should enter into the recovery program in the first place, let me again state here that I never said that this book would provide a safe method of drug reduction for any and all people who want to recover from Parkinson's. This book is an adjunct to our strongly worded warnings that we will not accept medicated patients into our treatment program.

But hopefully, this book has become much more than that: the *preceding* chapters may serve as a warning for those patients, recently diagnosed, who are considering taking medication for their Parkinson's symptoms and whose doctors may be uninformed as to the nature of the drugs and the correct dosing. The details on drug reduction in the *following* chapters may be helpful for those who need to decrease their medication slowly and safely because they are suffering adverse effects from excessive levels of medication.

All of these chapters, fore and aft, are not intended as guides for people taking antiparkinson's medications who now want to stop taking medication and try to recover. For most of them, because they are already taking drugs, it may already be too late for them to stop the drugs and to try to recover.

However, for those people taking drugs, and for those whose drugs are becoming problematic, they or their doctors might wish to extrapolate from the experiences in this book to come up with drug programs that will help to best manage the drugs, minimizing adverse effects and optimizing/extending the years during which the medication might be helpful. Of course, even if the drugs are taken correctly, the symptoms of a person with Parkinson's disease will continue to worsen. But by using the drugs correctly one might avoid or postpone adding painful and unnecessary dyskinesias, dystonias, and parkinsonism to the steady decline of idiopathic Parkinson's disease.

I also hope, although I am not overly sanguine about this last, that this book may possibly open a door for MDs who are questioning the current thinking about Parkinson's disease medications. I am especially excited about the possibility that the final hypotheses in the last chapters of this book, in which I propose a relationship between the parasympathetic nervous system and addiction, might some day lead to a better understanding of addiction in general. If this study of Parkinson's medications can eventually help those who struggle with addiction of any kind, then the grassroots researchers and myself will have been rewarded beyond our highest expectations.

I repeat, if a person is taking medication, he should not enter into a Parkinson's recovery program.

"It is cocaine," he said, "a seven-percent solution. Would you care to try it?"

Sherlock Holmes speaking to Dr. Watson, in "The Sign of Four,"

Sir Arthur Conan Doyle

15. A TEN PERCENT SOLUTION

I have frequently alluded to "small" decreases in medication, and 10% decreases. This is the chapter in which I get specific about what, exactly, I mean by small, and explain the 10% rule. Before I tell you my ideas on "small" doses, let's see the specific recommendations the drug manufacturers have for drug reduction. Here they are, straight from the books:

Levodopa: "Because of risk of precipitating a symptom complex resembling neuroleptic malignant syndrome, observe patient closely if levodopa dosage is reduced abruptly or stopped."[1]

Sinemet (buffered levodopa): no suggestions.

Artane: no suggestions.

Eldepryl: no suggestions.

Amantadine: "If drug is being taken to treat parkinsonism, warn patient not to discontinue abruptly because that might precipitate a parkinsonian crisis."[2]

Permax: no suggestions.

Mirapex: "If drug needs to be discontinued, do so over a 1-week period.

"Neuroleptic malignant syndrome (elevated temperature, muscular rigidity, altered consciousness, and autonomic instability) without obvious cause has occurred with rapid dose reduction or withdrawal of or changes in antiparkinson therapy."[3]

---

[1] *Physician's Drug Handbook*, p. 595.

[2] Ibid, p. 30.

[3] Ibid, p. 850. I am especially gratified about this warning. The original (1997) insert information for this drug did not include this warning. I wrote to the manufacturers of Mirapex in late 1998 stating that three of my patients, after rapid decrease of Mirapex, had experienced symptoms of headache, heat and/or pressure in the head, followed by symptoms of slight stroke (short-term partial paralysis, short term loss of or difficulty with speech, short-term personality change, and other symptoms consistant with mild stroke). The Mirapex people did respond and wrote asking for further details. In the next edition (2000) of the *Physician's Drug Handbook*, I saw that the available information had been amended to include the above quote.

The reason I noticed this in Mirapex patients and not in Requip patients is solely geographic; my hometown is just south of UC San Francisco, a location where Mirapex had some final years of testing. In the first few years after Mirapex and Requip, two *highly* similar drugs, were released, it seemed as if Mirapex was most often the agonist of choice on the west coast and Requip on the east coast. Most of my Requip-using patients were east coasters, visiting me only briefly. Today (2003) the drugs seem to have become generally distributed.

Requip: “Although not reported with repinirole (Requip), a symptom complex resembling neuroleptic malignant syndrome (elevated temperature, muscular rigidity, altered consciousness, and autonomic instability) has been reported with rapid dose reduction or withdrawal of antiparkinsonians. If this complex occurs, stop drug gradually over days and reduce frequency of administration to twice daily for 4 days and then once daily over remaining 3 days.”

Do you notice anything curious about all of the above? With the exception of Mirapex and Requip, not one of the drug advisories gives specifics about how to reduce the medication. I wrote to two of the companies asking for specifics on rate of drug reduction. While not identifying myself as a doctor, I did write that I had patients who were taking these drugs. One company did not answer me at all, and the other sent me the usual insert information, together with the reassuring comment that “This drug has been approved by the FDA.” In other words, most of the companies that make these drugs either do not know or do not publish information about the specific rate at which one should reduce these medications.

Only two companies made suggestions; in both cases, their suggestion that these drugs be reduced over a one-week period is ludicrous. The one for Requip is ridiculous – they suggest that if a person has already had a meltdown from stopping the drugs too quickly, then he should, after having stopped, reduce slowly. What on earth is that supposed to mean? And maybe they did not read my letter carefully; I pointed out that the three patients who had experienced stroke-like symptoms had not had these symptoms until three weeks after their precipitous decrease. Given this, what would these agonist makers have you do after your stroke: start back up on the drugs again and come off them again over a week?

It only makes it worse when they admit that the drugs can precipitate a crisis in rapid reduction, and then imply that a leisurely, one-week reduction will prevent this sort of crisis. Here’s why: this one-week suggestion comes from makers of the two antiparkinson’s drugs which specifically need a gradual build up of eight to twelve weeks when starting to take the drug in order to prevent powerful adverse effects. In other words, they know perfectly well that a person must start these drugs at levels approaching one tenth of the effective dose and work up to the full dosage over months. And yet, they suggest that a person can safely reduce these drugs over the course of a week?!

I am certain that NO testing was done to prove these reduction numbers. Since they know that over-fast reduction is dangerous, one would think that they might at least have experimented with reduction rates before proposing that, while one must increase the drugs slowly over *months* to attain an effective dose, one can safely stop taking them in a week!

One of my patients was involved in the testing that was done on Mirapex. Not one mention was ever made to her or anyone that she knew in the test group regarding testing for drug reduction safety.

Therefore, as you can guess, our little band of pioneers had to figure out the hard way just what the word “slowly” means. As already noted in earlier chapters, we found that a drug reduction of 10% appeared to be safe. This chapter is going to give details on what we mean by 10%.

10% of the current dosage

Patients who did the best usually reduced their medication by approximately 10% of their *current* dosage.[1] This means 10% of the amount the person was taking immediately prior to each reduction. This does NOT mean 10% of the highest amount of drugs ever taken. As you have guessed, this means that each reduction was a little bit smaller than the one before. Sometimes, due to the size of the pills and difficulty in breaking the pills, it was necessary for pioneers to be approximate with their reductions.

The weekly amount is more significant than the daily amount

Also, as the reductions got smaller, sometimes it was just impossible to get close to the 10% number. That was when we discovered that it really doesn't matter if the daily amount was decreased by 10%. What seemed to matter was that the overall, *weekly* amount was reduced by 10%. Because a slide into drug withdrawal usually took ten days (which is more than a week), it appeared that the limbic monster could be appeased as long as the *weekly* total of drug reduction was no more than 10% of the previous weekly amount. This finding was a huge help; in the beginning of this research project, people were literally rubbing their pills down to size with an emery board. Once we figured out that the limbic brain didn't have a clue what was going on from day to day as long as it got its ten-day supply, the pioneers had much more flexibility in their dosings. For example, in the chronicle below, watch what happens when the doses get down below 575 mg/day.

Even if you know how to calculate 10%, please read through the entire scenario below. I have tried to include many of the various irregularities that occur while people are reducing their medication. Therefore, the example below is a combination of several patients' charts, and covers a wide range of circumstances. Please do not think that anything that involves humans and drugs can ever be so simple as to conform to an exact 10% plan.

Please be aware that the example below only represents the kinds of numbers used by people in our study who got off their medication with a minimum of drug withdrawal symptoms. These numbers are not recommendations. These numbers are not supported by any drug company. You must work with your doctor. These numbers are presented as research curiosities only. They are not suggestions. Thank you. If you don't understand the significance of this paragraph, please find someone who does: maybe your doctor would be a good place to start.

This example of Waldo, a fictitious patient, is actually a composite of the reduction sagas of three patients (Hjalmar, Sonny, and Mark[2]). For purposes of this

---

[1] Math question: How does one calculate 10%?

Math answer: To calculate what ten percent of a number is, multiply that number times one tenth (.1). Another name for ten percent is one-tenth.

[2] Sonny and Hjalmar were both taking slightly more than 1000 mg/day when I met them. Mark was taking 300 mg/day of levodopa and an anticholinergic.

fictitious example, we will say that Waldo was taking 1000 mg levodopa/day when he started reducing his medication:

First reduction 1000 mg/day ➔ 900 mg/day (a 10% reduction)

Explanation: To calculate 10% of 1000 mg/day, Waldo multiplied 1000 times .1 (one tenth). The resulting answer was 100. Therefore, he decreased his medication by 100 mg/day: he went from 1000 mg/day to 900 mg/day, a decrease of 10%.

Second reduction 900 mg/day ➔ 800 mg/day (11%)

Explanation: This time, Waldo multiplied 900 times .1 and got the answer 90. Another way to do this math is to remove the right-hand digit of the number. In this case, removing the zero at the right-hand edge of 900 yields the number 90. This means that this reduction needed to be a decrease of 90 mg/day. However, it is too difficult to cut a 100 mg pill down to a 90 mg pill with any sort of precision. Therefore, Waldo reduced by a whole pill (100 mg) rather than by a cut pill (90 mg). However, by checking the math, one can determine that 100 mg is not a significantly higher amount than 90 mg. In fact, a reduction of 100 mg/day, from the existing level of 900 mg/day, is only a reduction of 11%. Eleven is close enough to ten that Waldo made this reduction without going through any withdrawal problems. His decrease took him from 900 mg/day down to 800 mg/day, a decrease of 11%.

Keep in mind that 10% is an approximation. Eleven percent, or even fourteen percent, is close enough to ten percent that the limbic area probably won't notice it. On the other hand, a number like thirty percent, for example, is *not* close enough to ten percent to avoid withdrawal symptoms, in our experience.

Third reduction 800 mg/day ➔ 725 mg/day (9.3%)

Explanation: Three months later, Waldo was starting to feel good after eleven weeks of drug reduction misery. He now had two choices. He could either reduce by a whole pill (100 mg) or he could reduce by three fourths of a pill (75 mg). Most patients find that cutting a pill any finer than one fourth or three fourths is simply not accurate.

Here is the math for the two possible choices. If he reduced by a whole pill, it would be a reduction of 12.5%. Here is how he did the math. Using his calculator, he divided 100 (the amount of one pill) by 800 (his daily amount). The answer was .125. To form a percent from this decimal number, he must move the decimal place over to the right two times. Then, .125 becomes 12.5%.

His other choice was to reduce by 75 mg. He divided 75 by 800, and found that this resulted in a number .093, or 9.3%.

Wilma, his wife, decided that 9.3 was closer to 10 than 12.5, and so chose for Waldo to make the smaller reduction. From what we have seen, he could also have made the larger reduction of the two with no problem. He reduced by 75 mg/day, going from 800 mg/day down to 725 mg/day.

Fourth reduction 725 mg/day ➔ 650 mg/day (10.3%)

Explanation: This time, Wilma wondered what another reduction of 75 mg/day might be in terms of percent. When she divided 75 by 725 (his current daily amount), the answer she got was .103, or 10.3%. This was just right, they both agreed, and so on this reduction, he reduced by 75 mg/day, bringing him down from 725 mg/day to 650 mg/day.

Fifth reduction 650 mg/day ➔ 575 mg/day (11.5%)

Explanation: Waldo and Wilma were getting the hang of it now; Wilma divided 75 (for 75 mg of pill) by 650 (his daily dose) and got an answer of .115, or 11.5%. They felt that this was reasonable, as he had easily survived the preceding decreases, and so he decreased by 75 mg/day, bringing him down from 650 mg/day to 575 mg/day, a decrease of 11.5%.

Sixth reduction 575 mg/day (4025 mg/week) ➔ 525 mg five days of the week, and 500 mg two days of the week, for a total of 3625 mg/week (9.9%)

Now, for this reduction, Wilma had to take an entirely different approach. Waldo was taking 575 mg levodopa/day. If he reduced his pill by 75 mg/day, it would be a decrease of 13% (75 divided by 575 = 13%). She felt that 13% might be a bit too much.

However, if he only reduced by half a pill (50 mg), the reduction would only be 8.7 % (50 divided by 575 = 8.7%). She felt that 8.7% was too small a reduction, as she was eager to get him off the pills. She also felt that 13% might be too much. What should he do?

When she calculated how much pill he needed to reduce to make a 10% reduction, she found he would need to take 518 mg/day. Because the pills come in 100 mg units, it would be very tricky to break off 18 mg from a 100 mg pill.

Some days more, some days less

Happily, such exact breaking is not necessary. Because the limbic brain apparently only cares that it gets 90% of its dopamine pills *over the course of the week*, Waldo and Wilma only needed to figure out how to reduce 10% of his *weekly* amount, not his *daily* amount.

How much was he taking per week? In one week he was taking 575 mg (his daily amount) times 7 (seven days in a week).

575 mg x 7 = 4025 mg.

Rounding numbers

Instead of figuring 10% of 4025, which is rather an awkward number, Waldo and Wilma, who were good with math, just "rounded down" the 4025 to a nice, even 4000. It was easier to figure 10% of a nice, round number like 4000. In fact, it was extremely easy: ten percent of 4000 is 400. Now, how did Waldo reduce his medication by 400 mg during the course of the week?

Remember, because the pills come in 100 mg, the easiest way to reduce was in increments of 50 or 100 mg. The smallest possible accurate pill breakage was to 25 mg.

What Wilma wanted to do was, somehow, over the course of the week, give Waldo 400 mg less than he had the week before. They could have done this in many ways: He could have taken 100 mg less on four of the days, and left his dosage the same on the others. Since, in this particular case, he wanted each day's dose to be as similar as possible, he chose to take 50 mg/day less on five of the days (that's a total of 250 mg less over five days) *and* 75 mg less on two of the days in the week (that's 150 mg less for two days). Adding these two numbers together (250+150 = 400), he had a total of 400 mg less than he had taken the weeks before.

The weeks before he had taken 575 mg every day. This week, the week of the 400 mg/week reduction, he took these amounts: 525 mg Monday, Tuesday, Thursday, Friday, and Sunday (which was a 50 mg decrease on those days), and he took only 500 mg on Wednesday and Saturday (a 75 mg decrease). His total mgs for the week was 3625 – a decrease of 9.9%. This was almost exactly a ten percent decrease, and although his physical symptoms reflected the daily dose variations, his limbic system never even suspected that some days the dose was smaller than on other days.

Tricking the lizard

Please do not jump to the conclusion that it was always best for the daily doses to be as close in size as possible: there can be advantages in having a much smaller or larger amount on one or two days of the week and compensating by increasing or decreasing (respectively) the other doses of the week.

For example, sometimes a person realizes too late that he is in a Turnaround, on the verge of being overmedicated, and probably should have decreased sooner. Not wanting to be overmedicated, not even for a day, this person could take most of his next week's reduction immediately, over the next two days! This means that there would be two days with a greatly reduced medication level. This could prevent overmedication. Then, during the latter part of the week, in order to prevent sliding into a withdrawal, the person could take just enough medication so that the total reduction *for the week* was only 10%, even though the beginning of the week had almost a 50% reduction rate. Here is an example:

Seventh reduction 3625 mg/**week** ➔ 3275 mg/**week** (9.7%)

Return of dyskinesia!

Continuing along from the previous reduction, Waldo was taking 3625 mg/week. Five weeks after the last reduction, Wilma noticed on Sunday night that after his last dose of the evening he was twitching and grimacing. Waldo had already done the math; he anticipated that he should reduce his medication by 10%, which would be approximately 360 mg/*week* (not day, but week) for his next reduction.

Here was the plan: he was going to spread this reduction of 360 out over the entire week, and reduce by 50 mg each day: 50 mg times seven days in the week would equal 350 mg – pretty close to the target of 360 mg which would have been 10%.

He could do this 50 mg reduction easily; the pills would break into 50 mg units very nicely. This would mean that on the days when he previously took 525, he would only take 475. On the days when he took 500, he would reduce down to 450.

This would give him a reduction of 350 for the week, which was very close to his goal of 360. When he did the actual math, he found that this reduction of 350 mg/week was a reduction of 9.6%, which was close enough to 10% to go ahead. That was his original idea, before the dyskinesia appeared.

However, Wilma was deeply concerned about the sudden reappearance of dyskinesia. She vetoed the idea of a 50 mg/day decrease. He needed to take steps quickly to prevent having dyskinesia again the next day.

Times and amounts

Let's take a moment to explain the time and doses of his daily medication. His 475 mg/day would be taken in this way: 100 mg at 7:00 a.m., 100 mg at 10:00 a.m., 100 mg at 1:00 p.m., 100 mg at 4:00 p.m., and 75 mg at 7:00 p.m.

But because Wilma (but not Waldo) was concerned about the sudden appearance of dyskinesia, the next morning, Monday, he did not take his first pill of the day or his second pill. He only took his third pill of the day and his fifth pill of the day. That meant that he had reduced by 300 mg in one day! If this rate of reduction was continued through the week, he would have been reducing at a rate of nearly 60%! However, they had no intention of taking this rash and drastic step.

Because he had taken only 40% of his daily dose on Monday, he had no dyskinesia on Monday nor any on Tuesday morning after his first dose of the day. However, just to be on the safe side, and thinking that it is always better to be slightly undermedicated than to be slightly overmedicated, Wilma did not give him half of his second dose on Tuesday.

They had been planning to reduce by 350 mg over the course of the week. By reducing by 300 mg on Monday and 50 mg on Tuesday, he had finished his reduction of 350 mg by Tuesday, only the second day of the week! Over the rest of the days of the week he took the same dosages he had taken on those days a week earlier. As a result, his total reduction for the week was exactly what they had planned: a nice, safe reduction of 350 mg. But by jumping quickly into the reduction rather than spacing it out over the week, he never experienced any days with dyskinesia after making this reduction.

By Wednesday he was moving extremely slowly. On Thursday he was also moving slowly, if at all, but by Saturday he was moving so well that they realized he might need to make yet another reduction, sooner than expected.

They had been waiting to see how he felt by the end of the week; if he had been moving more and more slowly by Sunday, he was probably on a mild slide into another very mild reduction phase. If that was the case, he was right on schedule, and his 10% reduction would not be a difficult burden. If this had been the case, then over the course of the next week they could redistribute his reduction to make each day's dose similar to the other days, as they had previously planned.

If, on the other hand, he had been moving well by Saturday or Sunday, or even Friday, what might he do? If he had unexpectedly rebounded smartly from his reduction earlier in the week and was moving well by Sunday, he might need to make yet another

decrease in his drugs. This would indicate that he had gone through an entire reduction cycle in the course of less than a week!

This is often the way when dyskinesia suddenly appears at the end of a long drug withdrawal phase: there may need to be one, two, or even three rapid (less than ten weeks apart) reductions in a row to prevent overmedication. Had he been having dyskinesia by Thursday, after making those powerful reductions on Monday and Tuesday, Wilma might have suspected that he was not Sliding down, but was actually recovering dangerously quickly from his Parkinson's and his addiction; he might even need to have a day or two with no medication to see what was going to happen next. We will consider that contingency in depth later on, when we look at the case study of Viktor. For now, since he was moving somewhat slowly again on Sunday, he stayed at the level of his seventh reduction. He continued at that level for three weeks.

After that first week with all the reduction occurring on Monday and Tuesday, he switched over to a more consistent schedule: for the next two weeks he made the 50 mg/day reduction each day, as he had originally planned. In other words, since after the sixth reduction he was taking 525 mg on five days and 500 mg on the other two days of the week, this time, for this seventh reduction, he took 475 mg on five days of the week and 450 on the other two days. This kept him at 3275 mg/week, as planned.

The reduction was more painful than some of the others. For several days, Waldo was sobbing from pain and immobility. He called out for Wilma to help him all night long. Her journal shows that he called to her for help more than six times an hour throughout the night. He was sleeping in the lounger chair in the living room. He couldn't move by himself at night, and needed help to use the bathroom or to shift from his always painful (despite advil and bedtime brandy) position to another one, equally painful. This extreme level of disability and pain is not unusual with safe, slow drug reduction. He was abjectly miserable, but not psychotic nor suffering life-threatening symptoms.

Only at the end of three weeks at this reduced level of 3275 mg/week was he feeling *almost* good: he wasn't having dyskinesia, his appetite was good and he was finally sleeping again, getting up to five hours of sleep a night. He would have preferred to be getting more sleep, but he realized from talking with other people in the clinic that 5 hours is actually pretty generous during a drug reduction. Waldo did not feel ready to make another drug reduction. Wilma decided he should.

Eighth reduction: 3275mg/**week** ➔ 2925 mg/**week** (10.6%)

Explanation: This week Waldo again reduced by 350 mg/week. Wilma wondered what percent it might be this time if he decreased by half a pill per day again. When she did the math, she found that 50 mg/day was equal to 350 mg/week. When she divided 350 by 3275 the answer was 10.6%: just right! He had been taking 475 mg on five days and 450 on two days of the week. For his eighth reduction, he decreased by 50 mg/day, which put him at 425 mg/day five days of the week, and 400 mg/day on the other two days of the week.

Ninth reduction: 2925 mg/week ➔ 2800mg/week (4%)

Explanation: On this reduction, Waldo and Wilma both wanted to get rid of the annoying quarter pills. They wondered if there was some way he could take the desired amount and be using whole pills or half pills. They figured that, if on the five days when he was taking 425 mg/day, he went down to 400 mg/day, that would be a decrease of 25 mg times 5, or 125 mg. However, a reduction of 125 mg/week was only a 4% decrease. That would not be enough of a decrease to keep with their 10% plan.

However, they considered how he was doing in general; on average, he was making a reduction every two to four weeks, based on the fact that he felt somewhat OK by the end of two or four weeks, experiencing neither Build Ups nor severe withdrawal symptoms. They decided that, rather than wait a full four weeks to make this 4% reduction, one that would bring him down to 2800 mg/week (a neat 400 mg/day with no messy pill breaking), and he would make this small, 4% reduction just ten days after his last reduction.

The reduction went well. However, within a week after making this small, 4% reduction, he had a bit of twitching and dyskinesia on the Sunday evening. Wilma immediately set in motion his tenth reduction.

Tenth reduction 2800 mg/week ➔ 2500 mg/week (10.7%)

Explanation: Wilma enjoyed not having to break the pills into quarters, and so she wondered whether or not he could manage this reduction using only whole pills and half pills. She decided that by reducing by 300 mg this week, he could make this work. To reduce by 300 mg, he could make a decrease of 50 mg on six of the days of the week. That would make it easy: he had been taking 400 mg every day; now he would take 350 every day for six days, and on the seventh day he would take 400. He would be taking four doses a day on most days: three doses of 100 mg and one dose of 50 mg. He decided to take the smaller, 50 mg dose in the evening. This way, he would have more time to rest his brain during the night. Though the smaller dose at bedtime might mean difficulty falling asleep, they planned to have a dinner high in carbohydrates and possibly a cup of hot milk at bedtime. These tricks sometimes enabled him to get to sleep even when his brain was cranking and his body stiff from the lowered dose.

However, because the dyskinesia had reappeared, they had to abandon this nice, neat plan. Instead of spreading this decrease out over the week, he made most of the decrease over the first two days of the week. On Monday, though he did not much like the idea, he only took one dose of medication: his afternoon pill.

We need to pause in this chronicle and take a side trip into dosage timing and amount before we can continue Waldo and Wilma's story.

Times and amounts

When he had gotten down to 400 mg/day, during his ninth reduction, they had rearranged his dosages. Before this, he was taking pills five times a day, every three hours. At the ninth reduction, he started taking his pills four times a day, 100 mg at each dose. He spread the pills out so that he was taking them every three and a half or four hours instead of every three.

Shortly after this time he had started having the extreme fatigue in the mornings that is typical of recovery from Parkinson's. No matter what he did with his drugs, he

simply could not rouse himself at 7:00 a.m. He was terrified, but to Wilma it looked as if he might be in the Deep Sleep stage of recovery. This was exciting: if his body was doing such a lot of repair work, then this was further confirmation of recovery. This was also frightening because he was still taking a considerable amount of medication, and Wilma knew that he needed to be off his medication before his brain started making its own dopamine. He was still taking 400 mg/day, and it might take quite a while to comfortably reduce down to nothing.

Because he was not able to move in the mornings until he abruptly sprang to alertness at 9:30, she stopped giving him his first dose of the day at 7:00. She figured it was just a waste of a pill and a strain on his body to be ingesting stimulants when the body was clearly trying to rest. He started taking his first dose of the day at 9:30 or 10:00 and taking the rest of the day's pills three hours apart. He took a pill at 9:30 a.m., 1:00 p.m., 4:00 p.m., and 7:00 p.m. By having a big lunch at 2:00, a light dinner at 5:30 and a snack at bedtime, he was able to have plenty of protein at these meals without interfering with pill metabolism.

Now, back to their response to the dyskinesia.

Monday decrease (continued)

Because this tenth reduction coincided with the reappearance of dyskinesia, together with the onset of Deep Sleep in the mornings, Wilma felt he needed to try something drastic to bring his drug levels down quickly. The dyskinesia had appeared on Sunday; on Monday, instead of taking his first dose of the day at 9:30, he waited until mid afternoon to take his only dose of the day. In other words, to prevent overmedication, he only took 100 mg for all of Monday.

This certainly worked – he was barely able to move or speak on Monday or the next day. This was uncomfortable, even painful, and he wanted his drugs. Wilma kept reminding him that it could have been much worse; after all, moving and speaking are just functions of his brain's motor area. His limbic zone, the zone that could have sent him into terrors and hallucinations, simply snoozed through the whole thing. He was cranky and in pain because of his relative immobility. His extreme leg pain sometimes reduced him to tears. Even so, Wilma pointed out, he was not in the agonies of withdrawal.

They had planned to decrease by 300 mg this week. Had there been no appearance of dyskinesia, he would have taken 350 mg on Monday. Instead, because of the Sunday evening dyskinesia, he only took 100 mg: a reduction of 250 mg for the week, all taken in one day!

On Tuesday he cautiously took his pills as planned, at 9:30 a.m., 1:00 p.m., 4:00 p.m., and 7:00 p.m., reducing the evening dose, as planned. He had no dyskinesia, so on Wednesday, he started in again to take four doses. However, after his Wednesday 4:00 p.m. dose, he was dyskinetic. They had no idea what to do: if he decreased again, he might risk the withdrawal symptoms that he was trying so hard to avoid. If he failed to reduce and let himself be overmedicated, he would certainly have more problems down the road, as he became addicted.

Social crisis

To make matters worse, his 65th birthday party was planned for the following weekend. Everyone was coming to visit. If he decreased rapidly, he might be a drooling, immobile blob over the weekend. His wife wondered if he should take extra medication over the weekend to be at his "best" for the company, and to take some of the hospitality burden off of her. What to do?[1]

A major social event looming at the same time as dyskinesia appears is a fairly common occurrence. This may be in part because the anticipation of a social occasion can cause an increase in either adrenaline, if stressful, or dopamine production, if joyful. Given this natural increase in mobility during times of emergency and times of gaiety, several people have taken advantage of the upcoming social event to initiate an early drug increase, or even a slightly larger one than usual. I have many such instances to choose from in this composite example that I am writing up here, so I will use in this example the path followed by the person who had the fewest problems.

Brave decision

Wilma and Waldo decided to decrease his medication again. Evidently, even with the large decrease on Monday, he was still showing faint hints of dyskinesia by Wednesday. He was not sure how to proceed at this point, so he averaged the amount of medication he had taken in the last ten days, including his Wednesday medication that had started him twitching. He used the last ten days because that is the average length of time of a Slide, and he wanted to know whether or not he was sliding. Here's how he figured this:

Wednesday	300
Tuesday	350
Monday	100
Sunday	400
Saturday	400
Friday	400
Thursday	400
Wednesday	400
Tuesday	400
Monday	400

His total for these ten days was 3550. His daily average over these last days was over 350. Due to his extreme decrease on Monday, he was already, over the last ten days, averaging 350 mg/day, which had been his goal for this next reduction. Therefore, he might safely assume that he was done sliding, and was nearing equilibrium with the

[1]All three of the people on whom this composite example is based had memorable social crises during their drug reductions. Hjalmar had his 70th birthday party, attended by friends and family. He took an extra 50 mg of levodopa that day though he knew he would be dyskinetic the next day.

Mark had a daughter receiving her Master's degree in two weeks; his wife Margaret said, "I don't care if Mark has to go in a wheelchair, he needs to make another reduction and he will do it. But no matter what condition he is in, we are GOING to that graduation; we wouldn't miss it for the world.

When Sonny started having dyskinesia three weeks before Christmas, his wife Evelyn announced that they would make no decreases until after New Year's Day, because "the holidays are hard enough without having to deal with Sonny going Off all the time."

350/day amount. And yet, even with this new low average, he was having dyskinesia. Maybe they needed to rethink this reduction and do another one immediately. It does often happen that two or three quick, back-to-back reductions in a row might be needed. Maybe this was one of those times.

Why was Waldo so overmedicated, and so suddenly? It could be that the ninth reduction, the one before the last, of only 4% was not adequate. It could be that the upcoming birthday party, complete with grandkids and chocolate cake, was causing him to produce more dopamine than usual. Maybe, compared to his rate of drug decrease, he was recovering far too quickly.

Once the recovery is set in motion there is no way to slow it down. Ceasing treatment will not help. Once the body is given permission to fix the old injury, it charges ahead like a child running to the ice cream truck. Once a person starts to recover, he is going to recover. About the only thing a person can do to slow this inevitability down is take dopamine-enhancing drugs, thereby doing himself a mischief and causing permanent brain damage. In such a case as this, the patient will still recover from Parkinson's, but he will possibly continue having symptoms due to drug-induced parkinson*ism*. Waldo didn't care one way or the other, but Wilma wanted none of this parkinsonism stuff. He reluctantly agreed to make a big reduction in his medication.

Completely ignoring the math, Waldo did not take his morning dose for five days, and he did not take his afternoon dose every other day for these five days. This became his 11th reduction. After his 10th, brief reduction (which had lasted only three days before he was dyskinetic again), he had been taking pills at a rate that would have been 1750 mg/5 days. During these five days of rapid decrease, he only took 950 mg/5 days. This is a decrease of a whopping 46%!

Eleventh reduction 1750 mg/ **5 days** ➔ 950 mg/ **5 days** (46%)

Explanation: In order to be certain to avoid the dyskinesia, which had reared its ugly head within two days of a large decrease, Waldo took the drastic steps described above for five days. Over these five days with the greatly reduced doses, he was not moving well and he was in pain.

Part of the pain was the horrible throbbing he was starting to have in his legs as he regained sensitivity in his extremities.[1] This painful part of recovery seemed all the worse because his brain was accustomed to use levodopa to mask the incoming pain signals.

Levodopa is a mild anesthetic: in addition to weaning his motor area off the stimulation of the drugs, Waldo was also weaning himself off of the sedative effect of the medication. Therefore, in addition to undergoing bouts of immobility and tightening of his muscles (reduction symptoms), he was also experiencing the sharpness of hundreds of reawakening nerves, nerves previously pacified in part by the medication.

This heightened sensitivity, the exact opposite of Parkinson's, prevented him from sleeping. He was becoming exhausted, and Wilma was at the breaking point. Wilma decided to give Waldo a glass of brandy at bedtime.

[1] Restoration of blood vessels, proprioception, and temperature sensitivity in the extremities are some of the most painful aspects of recovery. Unmedicated patients may find these recovery symptoms very painful. A recovering person who is reducing medication may find them even more so.

Alcohol is a very mild dopamine enhancer, compared to levodopa. She was deeply concerned about his developing another addiction, and therefore worried about him drinking the brandy. But when she thought it through, she decided that alcohol, though famous for its addictiveness, would not cause parkinsonism and tardive dyskinesia – two forms of permanent brain damage. Alcohol might affect his liver, but so might levodopa affect his liver. He was up against what she saw as two evils, and alcohol was clearly the lesser of the two. They were both quite gratified to find that he was able to drop off to sleep more easily after a snifter at bedtime. He was able to get several hours of sleep before starting in on calling to Wilma for help "every #%*! ten minutes!"

The Alcohol question

Had Waldo and Wilma thought more deeply about the addictiveness of alcohol, they might have realized that, since he was in a condition of dopamine deficiency during his drug reductions, he was most likely not risking any addiction at all from the alcohol: addiction only occurs when one goes over the Safety Limit, and on these days of extreme drug decrease, with their immobility, pain, and insomnia, he was not anywhere near the Safety Limit. He was on safe territory, as far as addiction was concerned.

Back to Waldo.

Waldo, despite his very large medication decrease, did not fall into the dreaded abyss of drug withdrawal. Maybe he was being helped more than he realized by his slow but steady apparent recovery from Parkinson's. Instead of withdrawal, he merely went through the usual symptoms of extreme pain, feeling helpless and scared, and calling for his wife to help him shift positions throughout the night.

Waldo was in a lot of pain, especially in his legs and shoulders, but everyone else in the clinic who was at his stage in recovery, *whether medicated or unmedicated,* was also experiencing new pains very similar to Waldo's. Wilma decided that the achiness and stabbing sensations in Waldo's legs, his frequent urination, his inability to support his own weight (he was starting to become limp), and his emotional fragility were coming from the recovery and not merely from the drug reduction. He did not have nausea or hallucinations, nor was he shaking violently from terrors. Wilma decided that he was experiencing recovery, primarily, and secondarily experiencing drug reduction. She couldn't be sure, but after meeting with and hearing stories about patients going through addiction and actual drug withdrawal symptoms, she did not feel that Waldo was going through over-fast reduction or drug withdrawal.

Twelfth "reduction" 950 mg/ **5 days** (190 mg/day – his quick, emergency reduction) ➔ 250/**day** (which is equal to 1250/5 days) (This constituted an increase of 31% over the preceding five days, and a decrease of 29% compared to the five days before that.)

Explanation: This was an attempt to moderate between the radical reduction of the last five days. The twelfth "reduction" was actually a slight increase over the amount he'd been taking for the preceding five days. However, compared to the amount of medication he had been taking *ten* days earlier, it was still a substantial decrease. In fact, compared to the amount he was taking after his tenth reduction (2500 mg/week), his new drug level of 1750 mg per week (250 mg times seven) was still an overall decrease of

29% for the last two decreases. This was an average of 15% for each decrease: slightly on the high side, but not extremely so. And now he was down to 250 mg/day, after originally having been taking 1000 mg/day.

He felt he was almost done with the medication. Wilma decided to not give him his morning dose and reinstated his afternoon dose in its place. She used his unmedicated mornings to give them both a more accurate picture of what was really going on in his body than she could have seen had he taken drugs first thing in the morning. He was still sleeping a tremendous amount, and often slept past what would have been his first dose.

Of course, the hardest part was yet to come. We will leave Waldo at this point, because individual cases diverge very quickly from any generalization when the dose gets down to the last little bit. Lets give thanks to Sonny, Hjalmar, and Mark who merged into Waldo while demonstrating the 10% principle and the equally important principle that, despite good intentions and good solid theory, the safest drug reductions do not always occur in a neat straight line.

The last little bit

The reduction of medication from approximately 300 mg/day of levodopa down to zero is a special range. Some people try to go quickly through it. Some people struggle mightily, getting down to zero and then going back to 200 mg/day, and then back and forth between zero and 200 mg several times before they finally stop for good. Others get to 100 mg and go crazy.

In our experience, the most dangerous time is when a person is taking approximately 100 mg/day levodopa or similarly small doses of the other anti-PD drugs. This is when our patients were most likely to want to drop out of the project.

Curiously, this is also the time when spouses and friends would remark, "His brain is back!" It was as if the curtain across the mind that is created by the drugs begins to part, just a bit, when the medication is reduced down to about 100 mg/day of levodopa.[1]

At this terrible stage, a person retreats behind the familiar curtain of drug haze when the drugs kick in and is painfully jerked back to reality when the drugs wear off. This is a different reality than a person had during Offs and times of undermedication when taking higher over-all levels of the drugs.

At higher levels, even though the motor area staggered through Ons and Offs, the frontal lobe of the brain was never actually clear of drug-induced mists. However, when the daily average of medication is at 100 mg/day or less for more than a few weeks, the brain may be somewhat free of the drug now and then, and especially during Off times. These flashes of reality can be very painful for the patient, both emotionally and physically. On the other hand, friends and family are often thrilled or else uncertain how to react to this mixed blessing: the return of the consciousness, the return of "the man I fell in love with," combined with the obvious psychic, physical, and emotional pain being experienced by the loved one. So we will tackle the "last little bit" in a chapter all its own.

---

[1] For other medications, please see the individual drugs in the appendices and chapter 17, The Last Little Bit.

Conclusion

In the meantime, I hope that this composite journal chronicling the drug reduction adventure of our fictional Waldo has opened the eyes of anyone who said to himself "10%? OK, that will be simple enough." For anyone who imagined that he could simply do the math, multiply by .1, count fourteen days, do the math again, count to fourteen again, one two buckle my shoe, three four out the door, knit one purl two and be home without a hitch, I hope that this chapter has given you pause.

One test reader asked why I had written the above sentence in closing this chapter, as it made no sense to him. So let me put it another way: if you think that there is an easy, formulaic way to neatly and simply get off the medications, that it will have a neat rhythm to it, like a poem, or that it will be as easy as following a recipe or a knitting pattern, you are wrong.

Reducing dopamine-enhancing medication is very difficult. The brain does not respond the same way every time to the same process. The timing and symptoms of each reduction cycle can be different from the ones before.

Most people cannot do it alone.

Legally, only a doctor can help you and advise you in this extremely difficult process.

What your doctor recommends may be harmful.

If you are taking medication, you are not a good candidate for recovering from Parkinson's disease; you may be better off having Parkinson's disease than recovering from Parkinson's while using drugs and thereby becoming addicted.

"And oftentimes, to win us to our harm, the instruments of darkness tell us truths, win us with honest trifles, to betray's in deepest consequence."

Shakespeare's Macbeth

16. STRANGE BEHAVIORS

THE HITTIN' AND THE CHEATIN' AND THE LIES

In addition to the expected discomforts (an understatement) during their 10% drug reductions, we also saw uncharacteristic violence, defiance, and deceit creeping into our patients' behavioral repertoire during their drug decreases. These events were not necessarily bad – in fact, since they happened so frequently, we were eventually able to recognize them as additional signposts in the drug reduction cycle.

What's going on? PD or withdrawal?

These behavioral changes usually occurred after the ten-day downhill slide into drug decrease was finished and the reduction was in full swing. These changes helped serve as an alert that the patient was experiencing mind-altering drug reduction/withdrawal symptoms. By confirming that a person was in reduction/ withdrawal, these uncharacteristic behaviors showed that any parkinson-like symptoms might actually just be stemming from a brain reaction against drug reduction. Therefore, any seeming worsening of Parkinson's symptoms that occurred at the same time might be safely considered also to be a part of the drug decrease phase and *not* signs of advancing Parkinson's.

The main question for drug reducers is usually, "Is this symptom a sign of withdrawal or is it Parkinson's?" These extra clues, unpleasant though they are, can give comfort and assurance that any physical and mood problems are at least as much due to temporary drug reduction trauma as to anything else.

Although from an objective, analytical standpoint, these behavioral changes can be helpful, from the emotional perspective they can be devastating. Anyone planning to embark on this journey should be forewarned about these aberrations. This quick chapter will share a few of these unexpected potholes on the rocky road.

Physical violence

Sonny

Sonny was 61 years old when he joined our project in March 1999. He was diagnosed in fall of 1989. His symptoms at that time were left arm not swinging and loss of dexterity in typing. He developed symptoms on the right side after five years.

His symptoms (when Off) in March 1999 included rigidity in both arms and hands (worse in the left), a little rigidity in the left leg, and lack of arm swing. His feet scuffed the ground. He would trip and fall "maybe once a year." He had On-Off cycles four or five times a day. Sometimes the Offs were "minor, sometimes major." During a major Off his voice was faint and he drooled, which inflamed his young (second) wife. His handwriting, always small, had become micrographic.

His medications, when he started with us, included the following.

Daily Medications:

Sinemet: 1000 to 1200 mg levodopa/day (five to six 50/200 Controlled Release)

Sinemet: 50 to 200 mg levodopa/day (half to two of a 25/100 Sinemet)

Permax: 4.75 mg/day

Tasmar 300 mg/day

Klonopin (also called clonazepam and Rivotril. This anti-convulsant, used as an adjunct therapy for schizophrenia and mania, which Sonny described as a "pain killer," was taken twice a day to reduce his dyskinesias and intestinal dystonia.)

Naprolan (naproxen, an anti-inflammatory pain reliever)

Vioxx (rofecoxib, an anti-inflammatory pain reliever)

After working with me for several months and noticing improvement in his condition, Sonny had begun reducing his medications. After nearly a year of slow, steady reduction, Sonny came in to my office for his weekly visit with a hangdog look and swollen face – as if he had been crying. His wife was drawn up in her stiffest manner. I asked my usual first question, "How was your week?" and settled in for his report.

He quavered, "I need to be committed. I can't stay at home any more. I don't deserve to be home. My wife has been so good to me. She's been helping me move when I get stuck, she's helping me with everything, she's helping me with the medication, she's doing it all for my own good. I don't appreciate what she's doing for me, what she's going through. She deserves better than this. I think I need to stay in a care facility from now on. She shouldn't have to put up with me anymore."

There was a silence in the office. We all looked at each other. I sighed inwardly and mounted what I hoped was a professional expression on my face, trying to assemble a look both helpful and non-judgmental, and offered a statement of fact: "So, you hit your wife."

They shot each other a puzzled look. How had I known? And why wasn't I more shocked?

"Well, in fact, yes. I hit her…How did you know? I've never done it before. I've never done anything like it before. We've been married twelve years and we've never had this situation. I'm turning into a monster."

"Yes," I said, "and you're the second PDer this week who hit his wife for "withholding" drugs. When a couple walks in with puffy eyelids, the PDer is looking guilty, and the spouse is looking like a martyr, that's usually what's happened. Were you asking for extra pills and she wouldn't give you any?"

"Yes."

"Do you want to talk about it?"

"No."

The familiar story came out. Sonny had been doing very well, but just two weeks earlier he had reduced his pills yet again. It was his fifth reduction over a period of nearly a year. As the drug levels finished their slide and he began feeling the effects of the most recent reduction, he decided to increase his pills back up; he had felt that they were reducing too fast.

His wife was in charge of the pills, and she wanted him to hold his ground. After all, he was still able to move, his dyskinesia was greatly reduced, and his dystonias had stopped so completely that he no longer needed the anticonvulsants or the Naprolan. He was completely off the Tasmar, and he wasn't even in the weak stage yet where he couldn't get out of bed or a chair by himself. He was still having Ons and Offs throughout the day, as he had been even before they started treatment, but after this last reduction his brain started to crank at him; he wanted those pills. They had gotten into a screaming fight, and to make his point, he'd hit her. As soon as he did that, he stopped in his tracks and announced that he needed to be put away. From what I had been seeing in others at his stage of drug reduction, the violence was pure routine.

I assured them that this was perfectly normal behavior, that I had seen it before, and that it was very hard to reduce medication. They wondered if he would do it again as they continued the reductions. I said I didn't know. His wife offered that if he ever hit her again, she would paste him one. There was a silence, and then I asked the usual second question: "So, how was your sleep this week?" They never brought up the subject of hitting again.

Dishonesty

Shortly after Hjalmar first started reducing his medication, he shuffled into the office for his weekly visit with a benevolent smile and his eyes shining with drug-induced, transcendent love for all mankind. He always took his second pill at 9:00, so his weekly appointment at 11:00 ensured that I would always see him beaming away, like a levitating saint blessing the people. Sophia was snorting steam.

"I have just had it with him, I have just had it!"

Hjalmar continued radiating joy. It seemed that the words "Bless you, my child" were trembling on his lips. In a minute he would've started strewing candy and coins if he'd had any. Sophia kept up her tirade. "He is driving me crazy! What am I supposed to do? Watch him every minute?" Her voice started cracking.

It was the old, old story: the drug sneak. "On Monday I gave him his 9:00 pill, only it was a 150 mg instead of 200. Then he went in the living room to read the paper and I went in the back room. I heard him rummaging around in the kitchen, so I went to see what was up, and he was getting into the pill drawer. He said he needed to take his 9:00 pill, and I told him he'd already taken it. So he went in the living room, and I went in the back room, and ten minutes later I heard him sneak in the kitchen. There he was again, getting out the pills. I told him that I'd just told him he'd taken his pill and that it wasn't funny, and he just acted all innocent, like he didn't remember that he had his pill, and then he said he didn't remember that we just had the same conversion a few minutes ago! And then, after his 11:00 pill, he did the same thing! I've taken to hiding the pills. And now he's going through all the drawers in the house when he thinks I'm not looking. He says he hasn't had his pills yet when he knows perfectly well he's already had them. It's driving me bonkers! I'm ready to kill him."

I glanced at Hjalmar to see how he was taking this. If he had smiled any more broadly he would have fallen off his chair.

"Well, sounds like you are doing the right thing by hiding the pills. Is he having serious problems? Is he freezing up worse than usual? Why is he needing the pills?"

"He's doing just exactly the same. He's stuck half the time, but he's getting around. He brought in the firewood yesterday without using his walker, which he hadn't done in years. He's not thrashing around so much in his sleep. He's doing perfectly well, but he's lying to me about his pills. I used to think I could trust him! That's the hardest part; I can't trust him. (Tears started to roll.) I used to think I knew him so well. (Sobs.)"

"Well," I said, "he's stoned out of his mind, of course, and will be until he's able to safely get down off his meds a little more. He's medicated; he doesn't know what he's doing. He's not himself."

"He'd better not be! I'm gonna kill him. I can take anything but not lies!"

"You're doing a great job, Sophia," and turning to Hjalmar, I asked, "And how was your week? How are you sleeping?"

These events are so commonplace in the drug reduction stories that I can now tell them without getting shocked. But the spouses are invariably shocked and disheartened by the lying, hitting, and hostility that are part and parcel of drug reduction. I have asked my patients to get together to form support groups outside of their weekly meetings at the clinic, but only rarely have they done so. I suspect that these behaviors are so scandalous that they do not wish to discuss them with anyone.

Lying to the doctor

There is another form of dishonesty that often arises during drug decreases that is much more worrisome to me: lying to the doctor.

Hua To announced in a weekly visit, "I reduced my drugs again this week. Basically, I am feeling much better. I also saw Dr. Pender last week. I told him I increased my medication."

"Why on earth did you do that!" I gaped.

"Well, basically, he asked me why I was doing so much better, so I told him I had increased my Permax to 13 pills a day from 10. If I had told him that I had decreased, going from 10 pills down to 6, he might have gotten mad at me."

"Why on earth would he have gotten mad? He noticed that you were doing so well. Why did you lie to him? "

"Because I disobeyed him. He expected me to increase, so I told him I did."

This is such a common scenario that it worries me. As a primary care physician, though not an MD, I am concerned that there is such a high level of fear towards the medical profession that patients lie to their doctors. Also, these lies go onto patient's charts. If Hua To ended up in hospital for some unanticipated reason, the staff would understandably give him medications at whatever level was on the doctor's chart. This had happened in Rose's case, with a deadly result.

The fear that Hua To felt towards his MD is all too common. I do not know what to do about this problem. This deserves a book in its own right.

My patients at the clinic greatly enjoyed sharing with each other their animosity towards their doctors, especially the many doctors who responded happily to a patient's visible, apparent improvement but who became outraged when they learned that the improvement corresponded to patient-initiated decreases in their drug regime.

Lying about hallucinations: doctor-appeasing strategy

We did learn one helpful bit of information that, regrettably, belongs here in the section on dishonesty: the only condition under which 100% of the doctors approved of drug reduction was hallucinations. My patients all started taking careful note of the hallucinations or even inventing fictitious hallucination stories so that they could get past the Angry Doctor situation. Evidently, it worked like this:

"Harlan, you're looking great! (Or, Hunter, you look terrible!) What are you doing differently? "

"I've decreased my medication."

"Why did you do that!" the doctor would reply, with puzzled or angry overtones, on the verge of the Stern Father act.

"I was having hallucinations. Now they've stopped."

"Oh," the doctor would say, backing down, head nodding in sympathetic understanding. "Well then, OK, that's fine."

I do not recommend lying to your doctor. This whole aspect of patient/doctor relations fills me with sadness. However, you may need to be aware of the hallucination gambit.

Bravado

"I can quit anytime!" was Brad's battle cry after he had quit yet again for a quick three days to prove that his meds weren't addictive. I had seen this routine many times before in other patients. It usually occurred just after a patient ended the drug reduction cycle and started feeling good again. These boastful assurances were meant to justify an indefinite delay before the next drug decrease.

I can't tell you how many times a person has stopped taking their L-dopa for three days to show me that they can do it and to prove that everything I quote about L-dopa's addictiveness is pure nonsense. I see this all the time. Sometimes they even go a full ten days just to say that they didn't notice any sort of slide. And then they will start taking the pills again and realize that they are not feeling as well as they used to at their old dosage level (because they are slipping into a withdrawal phase, and it will take at least ten days before the pills begin to work as well as they did before). So at this point the person will double or triple their dose in order to get back to the good feeling that they remembered before they quit. When I ask why they are taking so much, the answer is invariably, "I already proved to you that I can quit anytime. I proved that I won't have any withdrawal symptoms when I quit, so it doesn't matter if I take the drugs or not."

To which anyone would logically counter, "Then why did you start taking them again?" (Or else a common variation, "And why did you double your daily dose?")

And the answer to this is always, "It makes me feel better, and it obviously has no long-term effect on me."

There is no one quite so predictable as a drug addict.

Irrational self-confidence

L-dopa imparts an unrealistic confidence. I have seen drugged patients take fantastic spills and brag ten minutes later that they never need a walker. I have seen the bruises from breathtaking crashes, and yet these patients never seem to learn wariness or

caution from their experiences. Under the influence of dopamine-enhancing drugs, a person has such confidence that he might not take the simplest precautions to avoid danger.

How many times have I heard the frustrated refrain, "He broke a rib again (or arm, hip, or ankle). He was falling down so much this week. I tell him over and over and over to use his walker. It's always right there. I put the walker right by his chair, but he won't use it. He just won't use it." A person who is using dopamine-enhancing drugs is not capable of really understanding that he is at risk. He may know that logically he might fall down, and that if he falls, he may hurt himself, and that hurt is a bad thing, but somehow, he honestly cannot bring himself to register that this means he should be careful. The caregiver thinks that he is just being defiant, but the truth is that a person is not capable of normal caution when under the influence of mind-altering drugs – and all the antiparkinson's drugs are in this category.[1] The limbic area is the eventual resting place of excess dopamine (after about ten days), and when the limbic area is over-saturated with dopamine, the PDer cannot feel fear. He just can't. This inability to make life-preserving decisions is the very reason that the brain considers dopamine to be the most dangerous neurotransmitter, the reason that so many dopamine-reducing mechanisms are present in the brain, and the reason that the brain performs an addiction response. The addiction response is an effort to save a life, to prevent a person from dying in a foolish act resulting from mindless fearlessness.[2] And so a person who is taking antiparkinson's drugs in excess (a tricky amount to determine) will not take steps to prevent falls or injuries, even though advancing Parkinson's disease worsens balance and motor skills.

The Fear of Reality

This joyous confidence begins to crumble, however, during drug reduction. The opposite of irrational confidence is nameless, primordial dread. This fear that appears during drug reduction may be perceived as a symptom of worsening Parkinson's disease; it is not. The emergence of fear appears as the veil of drugged delusion is pulled aside. Because this terrifying return to reality may come at the same time as physical symptoms of drug decrease, the combination can be overwhelming. In such a circumstance, the patient is certain that he is worse than he has ever been before, and only a rapid increase in drugs can save him.

I remember the week that Margaret was so pleased, she just gushed, saying, "Mark is having real moments of clarity. I guess it was the medication that was fogging his mind. I had thought it was the Parkinson's. He used to have such a dry wit. Very acerbic. A little too acerbic for a lot of our friends…but such a sharp wit. It was what first

---

[1] Every single one of the antiparkinson's drugs can be found in the book *A Primer of Drug Action, A Guide to the Actions, Uses, and Side Effects of Psychoactive Drugs*. Every one of these drugs is a mind-bender. That is what imparts their power to create movement in a brain that is determined not to move.

[2] This is not the fearlessness espoused by great souls such as the Mahatma Gandhi, who said that the first step to a spiritual life was fearlessness. For a spiritual seeker, fearlessness means having unshakeable certainty that God is the one in charge, and that under His divine plan, all events in the universe will eventually bring each person back to God, regardless of the seeming paradoxes and apparent tragedies. Faith in God allows fearlessness to perform right action, unburdened by fears of what the future might bring. Drug-addled fearlessness such as jumping off cliffs is not right action – it is merely an arrogant denial of the laws of the universe, including the laws of cause and effect and the laws of gravity.

attracted me to him – you know, we've been married over forty years…and lately, he's got that wit back. It's amazing. It's all still there! It just showed up again after this most recent drug reduction!"

So I warned Margaret of what was coming. When a person starts having mental clarity, even for fleeting moments, he may also start having clarity, often for the first time since starting the drugs, as to the actual nature of reality, including his health condition.

Sure enough, the next week, Mark was, for the first time in his life, fearful of being left alone at home, fearful of being abandoned, and fearful that he was unloved. He needed constant consolations and reassurances to sooth his sobs and whimpers.

What we have seen time and again is that a person who was falling down every day and not bothering to use his walker can do an about face almost overnight. Following the drug reduction that allows him moments of clear thinking, he may suddenly become aware that it is very possible, no, likely, that his poor balance might cause him to fall down. He may suddenly be aware that falling down is a very bad thing to do. He might become paranoid about falling. It is as if a gauzy bandage had been covering his mind's eye, blinding him to the possibilities of getting hurt from falling. Suddenly, at some point during drug reduction, at lowered drug levels, that gauzy, dopamine-veil will get wiped away. With the dopamine gone, frightening possibilities appear, leering at him from all sides.

The support person's response might be, "Oh good, he's starting to remember to use his walker." The patient's response may be just the opposite: "I am getting much, much worse. I might fall down! When I was taking more medication, I didn't have fear of falling." It is the briefest of steps from "When I was taking more medication, I didn't have fear of falling" to the logical "If I take more medication, I won't have fear of falling." From there to "I am undermedicated! I need more medication so I won't fall down!" is a transition that we see over and over.

If the spouse or friend holds the line at this medication level, the person may start getting very paranoid around his particular fears. It might be fear of spilling food when eating or fear of not being able to express himself verbally. It will be a fear of something that was never considered a problem while under the influence of L-dopa. The drug-addled person might have been spilling his lunch on his shirt front for several years, but suddenly, in the new glimmers of clarity that come with gradual drug reduction, this person might be ashamed and appalled at the soup stains on his shirt.

When a person begins to have awareness of worrisome problems, whether emotional or physical, these problems can inspire an inner voice, a limbic-led voice, to sidle up to his mental resistance and say, "This never used to be a problem when you were taking more medication…"

This serpent-like voice is clever. It will not mention that you were spilling your soup, taking whole body spills, or whatever, when your medication was higher. This compelling voice will only remind you that *it didn't use to be a problem.* It *won't* tell you that it wasn't a problem because nothing could be a problem when you were that stoned.

Lack of Logic

This inner voice is very convincing. Because it is your own voice, coming from your own mind, it alone knows those very arguments to which you will be the most susceptible. Your inner voice knows the guilt-inducing voice, the fear-avoiding voice, all

your intimidating authorities from your deepest psyche, and it can pull them out and your logical mind doesn't have a chance. There is a reason that all the twelve-step programs that help with addiction insist that a person rely on a higher authority than their own logic. Your own subconscious can outwit your own logic every time. Your subconscious knows where all the fear and guilt is hidden, where your weaknesses are. Your logic is relying on your frontal lobe, on the other hand, and when your dopamine levels drop, there isn't much action in the frontal lobe. Logic goes out the window. A person who is left to his own devices, without an external guide, will succumb every time. The external guide can be a spouse, caregiver, friend, or a source of spiritual counsel. But a person cannot easily go this route alone. We have found this over and over.

Memory loss while medicated

Although Margaret was able to recognize the patterns of drug withdrawal in Mark, and drew strength from them, Mark was never able to see the big picture. Not only that, he appeared to be incapable of remembering his previous drug reductions. He had no recall of how he had felt during previous drug reductions, and so, every time he slid down into darkness, it was a new and terrifying experience.

For example, during one reduction phase, one that was, oddly enough, relatively pain free, Mark felt scared and helpless, even though he had to admit that he was standing taller and moving in ways that he hadn't moved in years. For example, he could slide in and out of the car gracefully for the first time in years. Friends were remarking on how his posture and walk had improved. But he still wanted more medication. He couldn't sleep at night and he was unhappy about everything in particular and life in general.

His wife decided to override his demands for a drug increase based on his improvements in motor function. They argued heavily. The following week, as his logic returned yet again, he agreed that she had been right, and he was glad he had not increased his medication. But – and follow me closely here – when they were in the midst of the next medication decrease, they went through the entire dance again.

For Margaret, it got easier every time for her to see that his intermittent, hysterical demand for a drug increase was all part of the drill. "First we reduce, his dyskinesia gets better, and his sleep is better. Then, after a few days, he gets scared. Then, after a few days, he gets paranoid and he says he's getting worse. And then he can't move, and he nearly falls down, and then he's really paranoid. He screams for more drugs, and then, after another week, he is doing better: he admits he's glad he decreased the meds, his mind feels clearer. And then the dyskinesia is back and it's time to start all over again. I'm getting used to it. It's always different, it's something new every time, but I can see the pattern in it."

For Mark, however, *it was a new experience every time*. When Mark was in the throes, he was incapable of recognizing that he was repeating a pattern that he had played out a month or two earlier. Every bout of paranoia was new, original. Every longing for the drugs was a first. Each time he felt the need to increase the medication, it was because he had *never* felt these sorts of feelings before – these menacing feelings that everything was bad, hopeless, dangerous and wrong. Every time he felt a yearning to go back up to a higher drug level, he was certain that this craving was a unique experience.

Regarding this same problem, Becky made an illuminating remark. Once, during a time when she was completely off drugs and done with withdrawal, she confessed, "I

don't really remember anything from the entire time I was taking L-dopa. I remember there were good times and bad times, but I don't remember any details. It all seems like a strange dream." I have heard this over and over.

Any lack of memory of previous drug conditions is a good indication that the person was taking drugs at a level far too high. If the drugs were so high that a person was not capable of forming accurate self-assessments, then the drugs were also high enough to be causing addiction-type changes in the brain, possibly including cell suicide and changing baseline or threshold. Therefore, this lack of memory, though galling, can be a good warning that the drugs were too high – a psychological weapon to be used against the allure of increasing the drugs back up again, back to where the PDer was mindlessly happy.

Blows to the ego

The idea that pure logic and an arrogant force of will may not be adequate to overcome drug addiction is an especially difficult concept for many PDers. They may have had such a lifetime of over-developed will power, due to their overactive adrenal responses, that they actually believe that they have more will power than others, that they are different from others.

Well, that's true, of course. As long as they are sick, they will have this unnatural will. But as soon as the recovery begins, the will power disappears. At this point, if they are still medicated, they find that they are addicted to the most powerful of all drugs, with the will power of an infant. Their actual will power may be that of the child that they were at the time of the injury that started the PD.[1] Drug reduction adds to these ego blows the reintroduction of fearful reality and the emergence of the wily limbic seducer. This combination contributes to the strange behaviors that can erupt during drug reduction. How many spouses, during these difficult times, have wailed, "It's as if I'm living with a stranger."

Vacillation

Just so you won't think that every story is one of violence and lying, I am thrilled to be able to tell you about the team of Mark and Margaret. They were wonderful. Margaret always wanted to know what was the worst that might happen from week to

---

[1] "Not my will, but Thine, be done," is not just a statement by Jesus about yielding to His Father's will, it is also a statement of ultimate truth. In the end, after we have had our lifetimes of strutting our arrogant will in gleeful opposition to logic or goodness, we must, if we will ever be in harmony with the universe, acknowledge that our own egoistic will has to die. Eventually, God's will, a will predicated on love and directed by perfect balance, wisdom, and fairness, *will* be done.

Many PDers have said that the beginning of their recovery was the realization that their powerful will power, the strongest motivator of which was self-preservation, was beginning to ebb. They say that acceptance, not to death or pain, but to being a part of the human race, was the beginning of their recovery.

I know that by including so many spiritual references in my work I may be building barriers between my work and a few anti-spiritual western doctors. My work is evidently not written for them. It is written for PDers of every faith and of no faith, and they will very likely know exactly what I am talking about. I also know that most of them, in the beginning of their recovery process, will be saying, "But that won't happen to me – my will power is different, it is already coming from a spiritual basis." But any will power, if it has not yet surrendered utterly to the will of God, if it still sees itself as separate and different (as in saying "My will power is *different*"), is an egoistic will.

week and what to expect, so that she would be prepared for it. She'd tell me the preceding weeks' events in great detail.

"He's arguing with me all day long," she would say. "And sleeping. He naps all day. And his ankles are swelling up. But mainly, he vacillates between gratitude that he's doing much better overall than he has in a long time and terror that he's worse than ever, going to hell in a hand basket."

Margaret was eager to learn, dubious about everything, but willing to listen to alternatives. She was also a brilliantly objective observer.

"The back and forths are just like you said. You said he was going to start getting illogical, and oh, brother! Has he been illogical! People are starting to say how good he looks, his posture is so much better, his stride is longer, and most of all, his face is so much better. He doesn't have that twisted face (grimacing) that I just hated. He hears what everyone is saying, and sometimes he'll suddenly say, 'I'm glad I'm reducing the meds, I feel so much clearer in my head.' And then an hour later he'll say he needs to increase his medication back up. During his negative times he sure wants to fight with me about the meds. I'm just glad you warned me."

Predictability vs. "Strange" Behaviors

As an aside, and to break up this chapter of negativity with a positive slant, let me tell more about Mark's case. Mark started with us shortly after we had assembled our new drug hypotheses and discovered the 10% pattern. I had seen enough cases that I could make an educated guess as to how he might behave in each week to come based on what Margaret told me had happened the week before. As Margaret told me every detail of what he was saying and how he was moving, I found I could predict which of the drug-related scenarios were most likely to occur next based on the experiences of previous patients who had already gone through a similar phase.

Mark did not hear these predictions; Mark was being treated by an intern at our weekly clinic. Every week after his brief intake, Margaret and I would have another intake across the hall while she told me details of the events of the week and I suggested what might be in store for her and shared pertinent case studies with her. The point of this separation of information was that we did not want Mark to overhear my predictions for how he might behave during the upcoming week. Although it is highly probable that Margaret's expectations could be subliminally conveyed to Mark, still, it would have been difficult for him to create the physical changes, such as grossly swollen ankles, screaming for Margaret all night long, and the emotional changes, such as return of wit and lucidity, merely because Margaret had been vaguely forewarned. My suggestions were never about specific symptoms but just a bit of theory, combined with examples of what other patients had been through at a similar stage in recovery or drug reduction.

I also made specific predictions to myself or Mark's acupuncturist when I suspected he was on the verge of becoming maudlin or hostile. This was an intentional experiment. I wanted to see if there was, in fact, some level of predictability in the system. If, after four years of working very closely with PDers who were reducing drugs, I could predict from week to week whether Mark would suddenly bound from undermedicated to dyskinetic, or if I could predict during which reduction he was most likely to try hitting his wife, it meant that these drugs were not unpredictable. Over a period of nearly a year, my predictions were right on. Responses to drug changes were

predictable! I'd had to follow his case like a hawk, and I had to apply every observation I'd made in preceding years, but it was doable.

Weighting the dice

Finally, while some people might say that any sharing with the subject of case studies is "unscientific" and mars the Truth inherent in a double-blind study, I would like to offer this reminder: patients do not live in a vacuum. They do read about other similar cases. They do have expectations based on what they are told by their friends and physicians. If, as these skeptics might insist, Mark only recovered from Parkinson's and was able to stop taking his medication because of my mild suggestions, then maybe what is needed for medicine in general is a more positive attitude from doctors and more time spent telling people how others have fared who have successfully passed this way before.

The meds were not "unstable"

And however important my role was in Mark's recovery, Mark and Margaret's case was also important to me, because it proved that, with enough experience, even the strange ups, downs, and emotional alterations brought on by med changes could become predictable.

The drugs are not acting wrongly, nor are they unpredictable rogues. They are predictable, but only if we understand how they work. There is tremendous benefit in knowing that these drugs are predictable. The difficult times, the dishonesties and illogic, when combined with the moments of lucidity, and added into the data of time frames and predictable drug symptoms, can be used to paint a fairly precise, if encoded, trajectory of just where a person is on his journey with the drugs.

Summary

A PDer who is reducing drugs may protest verbally and physically. The violence and dishonesty may be utterly out of character. The drugs themselves create mental conditions of bravado and irrational self-assessments. The glow of inner love created by the drugs gives way to fearful confrontations with reality when the drugs are decreased.

There may be a thousand reasons why a PDer who is obviously improving and having fewer adverse effects from his drugs will still want to increase his medication on any given day. The most common reason to resume or increase the medications has been, "I'm much worse. I am *not* getting better. Everyone who thinks I am getting better is wrong, and I am torturing myself for no reason." The second most common is, "I will die soon anyway, and so I might as well die happy."

It requires good record keeping on the part of the patient and the caregiver, and good analysis of those records, to battle the onslaught of these compelling arguments. The patient may be unable to logically assess his condition. He may not understand that he is getting better, healing deep inside, despite the physical difficulties – including rigidity and shaking – that occur during drug reduction. He may also feel in his heart that he is getting worse due to the emotional torments coming from his limbic area. During times of physical pain, limbic taunts, and self-doubt, the answer will appear to be drugs, the whole drugs, and nothing but the drugs.

Hopefully, by learning about a few of the possible complications such as personality change and relationship conflicts, you will start to appreciate that the 10% solution is not just a simple matter of mathematics. Although there was uniformity of success (as defined by lack of life-threatening heart and lung symptoms, hallucinations, nausea, and irrational terrors) with the ten percent approach, every path through drug recovery had its own, seemingly unique, pitfalls and detours.

"Farewell! Thou art too dear for my possessing…
My bonds in thee are all determinate…."

Shakespeare's Sonnet 87

17. THE LAST LITTLE BIT

THE PATH FROM "JUST A TAD" TO "NONE AT ALL!"

Let's resume our discussion of reduction amounts and methods. It appeared that when drug-reducing patients reduced down to just a small amount of medication, they tended to fall into two groups for making the jump from "little" down to "none": the all-at-once club or the infinitesimal decrease gang.

Definition of a "small" amount

Here is what we now consider a small amount for a person who still has Parkinson's:

Low levels of antiparkinson's medications

L-dopa (levodopa): 150 mg/day or less
Mirapex: .5 mg/day or less
Requip: 3 mg/day or less
Permax: .5 mg/day or less
Eldepryl: 2.5 mg/day or less
Amantadine: 25 mg/day or less

Most doctors believe that at such low levels these drugs have no effect whatsoever. However, just try giving these drugs, at these very low levels, to people who don't have Parkinson's; the drugs may be intolerable because of the side effects, and they will certainly be very addictive because of their powerful dopamine-enhancing properties. They all pack a lot more punch than two cigarettes, yet two coffin nails deliver enough dopamine-enhancement to set in motion addiction changes and other semipermanent brain damage.[1]

I have heard about doctors who consider anything under the maximum allowable dose to be a "small" amount of medication. In our project, when we say "small" dose, we mean a dose normally considered sub therapeutic. Traditional theory says that doses this small do nothing. However, we consistently found that patients could detect a powerful influence at only 10% of the "therapeutic" dose – if they were patient.

This chapter will present examples from the wide array of patients who reduced or tried to reduce their drugs from low, supposedly non-therapeutic levels down to zero, and also some studies of patients who only took medication for a short time.

[1] "Coffin nails" is archaic slang for cigarettes. -Ed.

Short-term medication

Some of our patients only used anti-PD drugs briefly. Even though some of them took the "full size" dose, their cases are more apropos for this chapter on small doses. We have found that the number of days that a person takes the drugs, if less than two months, may be a factor in drug reduction.[1]

The people who had only been taking the drugs for a few weeks or months usually had no problems at all in reducing their medication quickly. In the long run, however, anyone who had ever used drugs at any time seemed to have a harder time staying off the drugs than people who never took them at all.

Here, then, is an assortment of small dose and short-term drug reduction case studies. I will not make hypotheses and conclusions in this chapter. Instead, I would like you to just peruse these experiences and wonder, as we did, why some people had problems and why others did not. A hypothesis on the apparent differences is offered in chapter 24.

Elaine - Mirapex

Elaine started taking Mirapex shortly after she was diagnosed at age 58. She started getting treatments in our program within a few months of her diagnosis and stopped taking the Mirapex. Mirapex is a drug that one begins in very small doses and builds up to a therapeutic level over several months. It can take several more months for the full benefit of Mirapex to manifest. She had only been taking it six weeks and was only half way to a "therapeutic level" at the time she stopped taking the drug. She did not have any particular difficulties when she stopped taking the drug. It has been over two years since she took the medication, she has not taken it up again, and does not anticipate ever doing so.

Sammy - Eldepryl

Sammy had been taking Eldepryl (two five mg pills per day) for two months when she started working with us. As you will recall, she was 55 years old and recently diagnosed.

She reduced by eliminating one of the two daily pills every other day, and then she took only one pill per day. She then took the one pill every other day only, then one every three days, one every fourth day, and so on. After two months she was only taking one pill a week, on Mondays, to "help face the work week." After a month of these Monday "helpers," she stopped completely – or so she thought. As you have read previously, after she started recovering, Sammy did take Eldepryl twice more, both times to give herself an energy boost for the weekend. Each time she felt more deeply affected by drug withdrawal symptoms in the week that followed. Three years after getting off the medication, even after her Parkinson's symptoms have, for the most part, abated, she

---

[1] We had as many short-term drug users as long-term users in our clinic. This may be because people who have been only recently diagnosed are still exploring, looking for alternatives. Such seekers may stumble across our program with greater frequency than those who were diagnosed many years ago and who have become resigned to their fate. For whatever reason, we have been fortunate enough to have dozens of people in our local program that either never took medication or only took it briefly.

feels a need to "take something" every winter when the cold weather comes and her winter depression returns. She resisted this urge for two winters, and in the following summers she felt OK. The third winter she started taking a very low dose of Requip. When spring arrived she was certain she no longer needed the Requip and quit taking it. She moved easily throughout the spring and summer and was working on her posture and attitude. However, five months after she stopped Requip, just as autumn was coming on, she became *deeply* depressed, could barely rouse herself to move, and resumed taking Requip.

Earle - Sinemet, Mirapex

When Earle started reducing Sinemet, he was only taking 400 mg levodopa/day (four 25/100 Sinemet regular). Earle was 68 years old when he joined our program. He had been diagnosed six months earlier. He decreased abruptly, to three pills a day from four, one month after starting our program. Over the next six months, he reduced down by half-pill increments each month. In the seventh month, he went from half a pill a day to one half pill every other day. Over the next two months, he reduced down to half a pill every third day. Then he reduced again, to half a pill once every four days.

He continued to take half a pill once every four days for one month, and then, when he saw Dr. Grumb (the double-your-dose-on-party-nights doctor) at his annual visit, Dr. G. told him to stop taking the half pills. The doctor said, "A half pill does you no good at all. Stop taking them at once. You need to be taking something or your Parkinson's will get worse. Since you don't want to take Sinemet, you must take Mirapex." Earle took Mirapex for nine days and then stopped cold. Earle did point out to Dr. G. that his main PD symptoms were gone. Dr. Grumb restated that he "must use something!"

When Earle was diagnosed, he had the following symptoms: constant tremor of the left hand, depression, listlessness, cogwheeling of the wrists and ankles, slightly masked facial expression, rigidity causing aches and pains, slow movement, hoarseness, and lack of arm swing. Earle, a brilliant engineer and an excellent man for research and details, was a steady source of obscure and helpful information about the state of PD research and brain research in general for our project.

On the day that Earle gave me his journal, it was two months after he had taken his last Mirapex. He affirmed in his sweet Texas drawl, "Two days this week ah made an effort to try to detect Parkinson's symptoms but ah found nun." I examined him and found, at that time, that his face was very expressive, his sense of humor and motivation had returned, there had been no return of the depression, his arm swung easily, his leg did not drag, the hoarseness was gone, and the tremor, reduced in amplitude, stopped intermittently.

At the time of this writing, two years after his last pill, his left arm that previously had tremor has gone through many permutations. He is just starting to be able to make long, flowing motions with his left arm. For nearly two years his left arm got weaker and weaker, to the point where he could not wash out a glass. And then, in the last few months, he has started noticing that, not only can he wash a glass, he can do it with a graceful motion. He has, however, developed a shaking in both arms. This may be the tardive dyskinesia (late onset, often appearing after stopping the drug) that is an expected side effect of having ever used antiparkinson's medications.

He may have this shaking for the rest of his life due to his medication use. Or, this lifetime shaking may become more controllable through the years as he rebuilds the muscle, especially that of his left arm and hand, that was damaged during his decades with Parkinson's. It does appear as if he is growing stronger in his left arm and also gaining some control over his shakes. However, despite the recovery of strength in the limb, which may aid in the control of the shaking, the semipermanent changes to his brain – those that cause the tardive dyskinesia – may always remain somewhat in effect.

He continues to have a few minutes of powerful shaking every morning when he first awakens and tardive dyskinesia in both arms. Unlike the resting tremor of Parkinson's, this shaking occurs whether he is resting or active. This dyskinesia has a mechanical quality which is very hard to describe but it resembles more the nervous tapping that we associate with people who have been given antipsychotic drugs than it does the tremoring of Parkinson's, all the more so since his walk and overall movement no longer resemble that of Parkinson's. Despite the arm tapping, he is not interested in resuming the medication.

Nat - Requip, Artane, Mirtazipine

Nat was 41 years old when I met him. He had been diagnosed with Parkinson's a year and a half earlier based on his right leg tremor and the increasing rigidity in his right hand, which he had attributed to excess typing at work. Also, he had noticed that if he controlled his right hand tremor while handwriting, his leg tremor would increase.

When I met him, he had just begun to notice that, if he turned to look at something, he would find himself leaning precariously, as if about to lose his balance. He often caught himself in the mirror with an expressionless look on his face. His right foot dragged along the ground a bit, and he noticed that his right foot made a very different sound than the left when he was out running. He was in fabulous shape, very athletic.

He was also a scholar and a gentleman, one of my favorite patients. I think what endeared him to me were his humility, his honesty, and his priorities: his children, wife, extended family, and pets – all of these things came first. When he was diagnosed, he resigned from his high powered, high-paying job in order to spend as much quality time with his family, especially his young children, before the Parkinson's became too severe.

Most of my patients, I must tell you, prefer to keep working. Most people tell me that their goal in life is for no one to know that they have Parkinson's. They want to disguise their situation from family and friends. When they come to see me, very often the first thing I am asked about is whether or not they will be able to recover so that no one need ever know they had Parkinson's.

An aside: the healer who couldn't be sick

I had a massage therapist patient who was adamant that no one should ever know he was ailing. While this is the norm with PDers, his case was especially ironic.

"I need to recover in secret. I work with sick people, and I am their inspiration to get better," he said to me. "I take great care of myself, I have a great body, and I set myself up as an example for how right eating and living can lead to health. I have to show then, when I give them health advice, that it has worked for me. I would be a failure if they ever knew that I was actually sick."

This attitude is so common in many PDers and so opposite to the attitude of gracious acknowledgement that prevails in those patients who recover, that I'm afraid I rather goaded him with what I thought was a biting rhetorical question: "You think it is better to lie to your patients than to let them know that you are mortal?"

"Yes," he answered, my sting falling far wide of the mark. "Absolutely. I am their inspiration. How could I be inspirational if I don't walk the walk?"

(Funny choice of verbs, I thought.) "…You won't be walking much longer, at least not very well." I asked if his patients might appreciate him just as much if he showed that he was human and exemplified a good attitude towards life. I asked if maybe they would even be supportive of him and relate to him better if they knew he was ailing.

"No," he replied. "My patients look up to me because I'm healthier than they are."

"But you aren't healthier – you have Parkinson's disease."

"Yes, but they don't know that."

"So you'd rather lie to your patients so that they will admire you for being healthy, while you recover from Parkinson's disease on the sly?"

"Absolutely. That's the goal."

And back to Nat

You can see why Nat was such a breath of fresh air. He was not looking for sympathy, and I never sensed that he was ashamed of himself or that he saw himself as a failure because he had Parkinson's. Yes, he wanted to recover, but I think he wanted to recover by looking his disease in the eye, not by denying its existence. Nat was noble.

He was taking Requip, 4 mg three times a day (12 mg /day), and Artane, 5 mg two times a day.

When he started the Requip, it helped his rigidity but not the tremor. He added Artane to the mix to treat the tremor, though he was concerned about the warnings for Artane. The Artane didn't help the tremor so he decided to drop it – he hadn't been taking it very long – and he reduced it slowly, dropping one mg every four or five days. When he got down to about one third of his original dose, he experienced what he referred to as withdrawal symptoms: restless legs, skin crawling, 48 hours with no sleep. His doctor recommended he try Amantadine, so he stopped the last bit of Artane and took Amantadine for two months. After two months, the tremor was even worse, so he went back on the Artane. As he pointed out to me, the Artane didn't really help him, but after having taken it, he was much worse if he didn't have it.

He was one of the rare patients who had the clarity of thought to realize that he had addicted himself to a drug that wasn't doing him any good. He didn't chastise himself or make excuses either, he just told me what had happened.

After five weeks of treatment, Nat was feeling searing pain in his right foot, between the second and third toe. He said the pain was not muscular and not a cramp, but a pain from deep within his foot. This pain is a classic symptom that the suppressed foot injury that causes Parkinson's disease symptoms is beginning to be exposed.

Two weeks later, at his regular check up, Dr. Pender told him that, based on the appearance of tremor on the left side of his body, it was time to make another increase in his Requip, up to 5 mg, three times a day.

A few days later, Nat felt severe abdominal pain, as if he was hemorrhaging. This pain increased every day and was at its worst half an hour to an hour after taking the Requip. Dr. Pender told him to reduce the medication back to its original level. Eight days after he started reducing by several pills a day, he was having even worse abdominal cramping. He thought he was dying. Even at the smallest dose of Requip he would go into abdominal spasm; he had become "sensitized" to Requip, and could no longer tolerate it at any level. He decided to just quit the Requip, cold turkey. He did not have much choice – he was doubled over in pain every time he took it.

Several days after stopping the Requip, he felt more rigid, and this rigidity increased over the next few weeks. However, he experienced neither longing for the Requip nor severe insomnia, violent shaking or terrors following this abrupt discontinuation.

We have seen that when people who still have Parkinson's need to make quick changes in their medication, they are usually able to do so, sometimes with minimal discomfort. This is very different from the responses that occur if the patient has clearly started to recover. Even so, all of the anti-PD drugs, including the agonist drugs, can be dangerous if stopped abruptly. Nat was just lucky, and his Requip dose was still very low.

He started taking two Amantadines a day together with his two Artanes a day.

After two months, during which time his wife said that his face was more expressive, and the long-standing dead feeling in his leg, knee and foot was reduced to a dead feeling in the foot only, he saw Dr. Pender again. At this visit, Dr. Pender noticed that his left side appeared to have no symptoms. Nat was sleeping much better; on his best night he got a record twelve hours of sleep instead of the usual six. His feet were never as cold as before even though it was February, and he no longer had restless legs at night.

Because tremor was now his main complaint and his medication appeared to not help with tremor, Dr. Pender wanted him to try Mirtazapine, a drug that had just recently been approved for Parkinson's tremor. This drug is a powerful tetracyclic antidepressant[1] that has recently been found to be helpful in inducing a near-sleep state in which tremor is suppressed. The tremor of Parkinson's typically eases up when the PDer is deeply relaxed and stops during sleep. Mirtazapine keeps a patient heavily sedated, in a pre-sleep condition, similar to the state in which tremor ceases. However, the patient taking Mirtazapine is likely to drop off to sleep in the blink of an eye. The second week on Mirtazapine Nat fell asleep at the wheel twice while driving his daughter to school.

Nat reduced the Artane, another form of sedative, from 5 mg two times a day down to 2 mg in the morning and 1 mg in the afternoon. After a week, he increased the Mirtazapine, from 15 mg/day up to 30 mg/day, and further reduced the Artane down to 1 mg in the morning and 1 mg in the afternoon. With this further cut in the Artane, he again noticed the symptoms that he had noticed before: bugs crawling under the skin, tension under the skin, and a feeling that he wanted to tremor more. However, with this decrease in this sedative drug, he felt slightly less likely to fall into sudden sleep.

He noted, "Even with the Mirtazapine, the tremor is still there. If I am not using my mind, the tremor is suppressed. But if I'm mentally active, the tremor returns. It's as if the tremor is trying to get out, but it can't always do it."

---

[1] *Physician's Drug Handbook*, 9th edition, Springhouse, Penn, p. 695.

The Mirtazapine was suppressing the external expression of the tremor, but deep inside, if he was at all mentally alert, he was tremoring away.

A month later he further reduced the Artane so that he was taking 1 mg in the morning and only half a mg in the afternoon. (He was breaking his 2 mg pills into quarters.) His Amantadine was the same, and the Mirtazapine was still at 30 mg/day.

The last time I saw him he said that the side effects of the Mirtazapine, particularly the narcolepsy, were too strong, and the anti-tremor benefit was not enough to merit continuing the drug. Again he told me that it only somewhat suppressed the physical tremor – the internal tremor was still obviously thrumming away in his head. The medication just kept him so sedated that he could not manifest the ever-present tremor. He was planning to stop the Mirtazapine.

Due to family circumstances, Nat had to return to the midwest, and I never saw him after that. Still, I feel that his case gave an interesting example of how he was able to get off the Requip without suffering much, how he had restless legs and bugs crawling under the skin when he reduced the Artane, and how the Amantadine, the first time around, only provided benefits for two months. The second time he went on Amantadine, it gave no noticeable benefit. He is also a good example of a slow and unremarkable transition from one sedative (Artane) to another (Mirtazipine). It also shows the combined effect when two sedatives were taken together.

Stephanie - Sinemet

Remember Stephanie from chapter three, who took a very low amount of levodopa and allowed it to accumulate over ten weeks? She had compound Parkinson's, with injuries in her feet, ankles, legs, neck, shoulders, and head. Her PD had officially started while she was still in her 40's, although she recalled that even in her high school days her leg tremor would appear if she was standing up to make a presentation.

Over the years that I worked with her, Steph worked out several foot, ankle, hip, and head injuries, including two concussions. When she remembered these during treatment, they left her feeling faint and seeing stars for days, although at the time of injury she had jumped up from her head-first landings on the cement and told her older brother, who had dropped her from a height, head-first, "as a joke," that she hadn't felt a thing. Working with me, she regained feeling and warmth in her extremities, flexibility in her hips, and began having days where she felt perfectly normal. After working several years together, she had started experiencing the extreme fatigue that occurs when the body's adrenaline finally turns off and the healing begins in earnest.

She had started medication to keep her going at work during the difficult days of recovery when she just wanted to sleep.

Her eyes started to have the "Sinemet glow" and I reminded her that she had intended to reduce her medication as soon as possible. She was only taking 150 mg/day, a dosage level that is considered to be inconsequential by most neurologists. Her tremor had changed after she started the Sinemet and looked more like ticcing to me. She was developing toe spasming and facial grimacing, symptoms that, at one time, just a few weeks earlier, *she had known* to be signs of excess.

She admitted that she was having those symptoms, but she didn't think they were a problem. She said that after her vacation in France, two months away, she might consider reducing her meds. Her vacation was canceled due to the air strikes on the Twin

Towers in New York City in the fall of 2001. I asked her if she was planning to reduce her medication, since she wasn't going to France after all. She replied that she was so depressed about postponing her vacation that she couldn't even think about reducing her medication for quite a while.

Prior to her starting her medication, I had described the case studies of patients who had become addicted to the antiparkinson's drugs. She had listened carefully and asked me to work closely with her if I ever detected any signs that she might be heading in that direction. She had been truly afraid of becoming addicted. After wavering for several months, forced to choose between quitting her high powered, high paying job or working another year or so to get early disability retirement, she had chosen to keep her job – by taking the drugs. Her firmly held conviction was that she could wait until she retired and then stop taking the drugs cold, since she would be still taking a very low dose, and then focus her energies on recovering.

She did reduce her medication every now and again, for a few days at a time, by half a pill or so, but invariably went back up to 150 mg/day.

She did try one slightly longer period – nearly three weeks – of reduced drugs, but found that "it just wasn't worth it. I didn't feel good."

By now, her facial grimacing was constant, and she was starting to have an increasing number of body parts that went into spasm within 45 minutes of taking her half-pill dose. She was clearly overmedicated and also very much addicted. Her fear of addiction disappeared. She was able to laugh about it, telling me that addiction wasn't so bad, and she was going to live one day at a time.

I countered that, because of the very nature of addiction, she would soon find that she needed more medication to feel the same way. With more medication, the side effects would increase. As the brain altered itself to accommodate to the higher doses, she would soon need yet higher doses to get an effect, and the spiral would continue until such time as the side effects were more of a concern than the benefits, and that the side effects might be painful, and even life-threatening.

She smiled, and agreed that such was indeed the case. She wasn't going to dwell on it, however, and was going to take it one day at a time. She confided that she shouldn't have been so worried about the medication to begin with; life was going fine. She assured me that it might be decades before the meds were a problem, because she was being so cautious with them and had had such good results so far. However, because I was so concerned, she would decrease her medications by half a pill on two or three of the days in the upcoming week.

At her next week's visit she had increased her medication by two half pills per week. She had not been able to bring herself to reduce her medication even for an afternoon. The very thought of it made her want to increase her medication. Her grimacing was more obvious, and she was making less effort to control it. Her fingers were twitching, her eyelids popped open and shut asymmetrically, and her head weaved about on its stalk. She agreed that this increase in dyskinesia might signal a worrisome level of overmedication. She decided that she would decrease her medication this week, and she even plotted which days would be best, based on her work stresses.

A week later she told me she had increased her medication again, just by half a pill over the course of the week. She was thinking about increasing her medication

despite her upcoming retirement. She was still going to retire, but she didn't see any reason to stop taking the drugs just because she wasn't working.

We had a good long talk. I pointed out to her all the positive changes in her body, sleep patterns, emotions and physical movement, all of which had started even before she began taking the little bits of medication, and that these changes might have suggested that her Parkinson's was gone or going at that time. I pointed out that she didn't have any signs of Parkinson's except her tremor, which, in my eye, was looking more like a tic and less like a tremor every day. I reminded her again that I had promised to stick with her and warn her if I suspected she was becoming addicted, and I did now suspect that had happened. I was especially concerned that she would begin each week determined to decrease her drugs, and by the end of the week, she would have increased them, despite her own expressed desires.

I asked if she would like me to work with her more often, using ear acupuncture to ease the pain of drug withdrawal. What did she want me to do? She told me that I didn't need to worry about it, and we should just do the usual weekly treatment. As she was leaving she assured me that our time working together had been extremely valuable, and that, no matter what she was going through, I had always been supportive and had made more sense than any of her other health practitioners.

We had been working together for four years. She called that evening and said she might not be able to make the next few weeks' appointments. I never saw her again.

Staying on the job

Her case, including the two dilemmas of wanting drugs to get through the weakness stage and needing to take the drugs to keep working, is, sadly, far from unique. I have received many emails from patients asking work-related questions:

"How can I avoid taking the drugs? My doctor will not give me disability pay unless I first try every possible solution. This invariably means the drugs. However, since the drugs appear to accelerate the illness, I will be doing myself a disservice, making myself sicker, faster, by taking the drugs. Also, I understand that if I am able to recover, I will go through a period where I will be extremely weak and, most likely, unable to work. How can I survive without a paycheck? Also, you say that you do not accept medicated patients into your program, but the only way to qualify for disability income is to use the drugs.

"What can I do?"

I have no answer for these people.

Stephanie had been in this identical situation and had chosen to take just a tiny bit of medication, planning on stopping it at the first possible moment. Based on what I have seen over and over, I fear that she will never stop taking it, that she will take it in ever-increasing doses, and that soon she will have the rapid decay into painful spasms, dyskinesias, On-Offs, mental shallowness, and then, most likely, the hallucinations that come with this drug if it is taken when PD is no longer present. Where it will go to from there, I cannot guess. Hopefully her daughter will take care of her.

Although I have had patients who were able to stop taking the medication, I have not had any who were able to stop taking it if they started it after showing symptoms of

recovery. Forgive my redundancy, but I repeat, it appears as if a person who no longer has Parkinson's disease has an extreme susceptibility to drug addiction.

As for the other issue, the problem of people not wanting to take the drugs but being required to take them in order to get disability insurance, I would like to share Mic's story.

Mic

Mic was a doctor. He quit working when his tremor could no longer be hidden. He simply lied to his work disability insurance board and his own neurologist, telling them that he was taking the drugs even though he wasn't. As he explained it to me, "Those drugs would destroy me. I do not want to lie, but I have a family and children still in the home. I would rather lie than destroy my health." *He never took the drugs.*

He continued to see his neurologist every six months. After receiving the Tui Na treatments that we recommend for over a year and a half, his neuro was very impressed with his obviously improving condition. The neuro was very pleased that "the medication is finally starting to work!" The neurologist even wondered if Mic might not be able to return to working, since the medications were doing so well. Mic convinced the neuro that his retirement from work was the factor that was allowing the drugs to do their job, and that, if he went back to work, he would probably sink back down into a non-functional condition again. Also, his tremor (typically the last symptom to go away), was still apparent. The neuro concurred, and my patient was able to continue his recovery at his own pace, without medication.[1]

Both Mic and I strongly suspect that the fraternity of doctors probably made it easier for him to get a clearance to stop working and receive disability benefits: both Mic and his neurologists agreed that it was out of the question for a sick doctor to be a health practitioner. His patients could not be expected to accept healing advice from a person who was tremoring!

Becky - levodopa

Becky was taking 400 mg/day levodopa (two doses of 50/200 Sinemet) when she started working with us. Although she ended up taking the pills again and became addicted, with devastating results, she apparently was not addicted when she stopped her drugs the first time. She reduced her pills by breaking off a bit from the end of each pill. Eventually, she was breaking off thirds and then breaking them in half. Then she went

[1] Sadly, just after I last saw him – at which time he appeared perfectly normal: he was driving again, had radiant facial expression, and his aged mother confided in me that she had cried to see him running up and down stairs once again and even his neurologist was thrilled at how the drugs were working even better than before – he had a severe emotional setback due to health problems affecting his teenage daughter. He was at a very vulnerable stage in recovery: he had no adrenaline whatsoever, and his daily ups and downs of dopamine were extremely susceptible to mood and emotion. He had the emotional fragility of a child, not uncommon in this stage of recovery, and he was completely flattened by his daughter's troubles. I heard from him recently via email from his home in Europe: his daughter's health is still tenuous, and he can barely muster himself to move.

from two doses a day to one, and then a half pill every other day, then every third day, and finally, she was taking half of one pill once every four days. When she finally stopped taking the Sinemet altogether, she felt very tired and napped frequently. The total time spent in getting off the drugs was just over three months. As you know from previous reading, she turned to the drugs for a quick weekend picker-upper two months after getting off the drugs and was not able to stop for nearly a year after that. When she did stop, her withdrawal symptoms were violently traumatic, and lasted more than five months.

Elaine, Sammy, Earle, Nat, Stephanie, Mic, and Becky all have very different stories.

Elaine stopped her medication shortly after starting it and noticed no problems.

Sammy stopped easily the first time, but tried taking it a few times after she started recovering and found it more addictive each time.

Earle slowly, slowly reduced, by a small amount per week, even down to fractional reductions. These tiny reductions, such as a decrease of 50 mg/day on two days of the week, and staying at that level for weeks, were laughable to his doctor. And yet, he could certainly feel the effects of each small reduction. He started reducing even before he started recovering, and there was never a time when he showed any symptoms of overmedication. He now has classic ticcing, possibly, probably, drug induced.

When Nat started showing signs of recovery, he was suddenly unable to tolerate Requip. He stopped immediately and, although he felt very stiff and rigid for some time afterwards, did not descend into drug withdrawal.

Stephanie took up a small level of levodopa even though she felt certain she was recovering. She was never able to stop taking it. Any attempts at decreasing by as little as 50 mg/day would ruin her day, leaving her miserable.

Mic never did start taking it; he just lied that he was.

Becky was able to stop it easily, making very small, steady decreases of a fraction of a pill at a time, similar to what Earle had done. This was easy enough to do when she had Parkinson's. When she tried it again when she didn't have Parkinson's, it was impossible. She tried stopping all at once instead and underwent the severe trauma of full-blown drug withdrawal.

You have already read about Hjalmar and how he reduced his drugs from 300 mg/day of levodopa down to none after he became sensitized to the drug and was hallucinating.

No one died

It is not possible to make a neat, orderly formula for drug reduction from these few cases, or even to make generalizations about these cases from which we can build a new hypothesis. It is important, however, to state that not one person who was taking less than a "small amount" of medication, as defined at the beginning of this chapter, died as a result of subsequent medication reduction, whether the reduction was fast or slow.

Leaping off high cliffs of overmedication with an overfast reduction can be lethal, but it appeared that a person could safely choose between ramping down slowly or stumbling quickly over the last few risers of drug use. The latter was distinctly unpleasant, but no one died.

Some of the above patients had a relatively easy time with the last, small reduction, though most had at least some difficult times. Some were not able to stay off the drugs even though their doses had been very small, and some noticed a change in their susceptibility to addiction even at low doses as they noticed improvements in their PD condition.

There is just enough range of case studies here that, hopefully, the reader can see that the last little bit must be viewed on a case-by-case basis.

Adverse effects can include sudden death if high levels of drugs are reduced abruptly (and "abrupt" can mean over weeks). At lower levels, more subtle adverse effects can occur. These subtle effects, which can include mood changes, changes in mobility, and longing for the drugs, are much less obvious than the physical adverse effects experienced at high levels, and yet they can be just as emotionally compelling, or, because they are so subtle, often more emotional than physical, they can be even more compellingly addictive than the high doses.

At high doses, a person might be able to look at himself writhing and admit that he was a tad overmedicated. At low levels, when a tiny change in medication can only mean the difference between a glorious day and a dismal one, it is easy to convince oneself that the drugs aren't doing any physical harm, and that one should do whatever it takes, including using drugs, to assure that one is performing at one's best.

Fig. 17.1 Depression can create a compelling longing for the discontinued drug.

Summary

From a nub down to nothing

Going into the last reduction of the medication, the recovering PDer may think that he is home free. When a person makes the transition from his lowest possible dose, a dose maybe as small as a fourth of one pill, taken just once a week, down to the long-awaited goal of no medication at all, his limbic system will still be likely to kick. Many people who have gotten off their medication following the ten percent, ten week program, who had nice cycles of drug reduction in which, after every reduction, they were able to accommodate to less medication, have seen that the last reduction, down to nothing, is unlike any of the others. The brain may have learned to be patient, and it may be able to get by with less and less of its brain candy, but when the signal finally gets through to the limbic system that there is *no* more candy in its future, ever again, it starts to squall.

This final slide can be fairly gradual. It may last several weeks. However, the final cycle, during which a person's brain accommodates to zero medication, can last months and even *years*. Even when a person is only taking a fraction of a pill once a week, this weekly dose seems to eventually satisfy the limbic system's desire. However, given the power of these drugs, making a decrease from a mere drop of antiparkinson's meds down to nothing may be a leap much greater than going from ten packs of cigarettes a day down to none.

Following the last decrease, there may be sadness, longing, months of waking up in the night shaking violently, sudden lapses into severe symptoms of Parkinson's disease that can last for several days, and every dystonia, dyskinesia, and spasm that your body learned during its years with the medication. Your brain will try every trick it has ever learned to remind you that it wants more medication.

During this first year of no medication, while hovering on the threshold of adequate dopamine, the forces driving a person to resume the drugs can be overwhelming.

Recovering from Parkinson's, even if there is no drug history, is hard enough. The months and years of physiological lingering right at the dopamine threshold, until the brain is challenged so often that a reservoir finally begins to accumulate, can be a daunting time. If you add to that the deep brain trauma of drug withdrawal, it can be an impossible time. Therefore, we do not recommend a recovery program for PDers who are taking the medication.

Finally, the extreme difficulty in stopping the last crumb of medication is counterintuitive. It would seem as if it would be easier to stop taking a final fraction of a pill, a reduction of possibly 25 mg, once or twice a week, than to reduce drugs from 1000 mg/day down to 900 mg/day. However, this is not the case. The brain can distinguish the subtle difference between a soupcon of drug or no drug at all much more keenly than it can differentiate between a very large dose and an even larger dose.

Counterintuitive: easier to stop drugs if sick; harder to stop drugs if healthy

Your friends might assume that if someone still has Parkinson's disease, he will have a hard time stopping the drugs. Conversely, they may think that if someone does not

have Parkinson's disease, it will be easy enough to stop taking the medication. They will be wrong: if a person still has Parkinson's and reduces the medications that are masking his symptoms, his symptoms will become evident. He may also have a backlash period of up to ten weeks during which his symptoms are exaggerated. When he has come to equilibrium after stopping his drugs, or even decreasing them, he may appear to have more symptoms of Parkinson's disease, but this is misleading; he had these symptoms right along – the drugs were merely masking them.

If, on the other hand, a person no longer has Parkinson's disease but is still taking any antiparkinson's medications, he may have the fearful symptoms of drug withdrawal when he decreases his drugs. The trauma of the drug withdrawal may create unforgettable memories of pain and suffering – the trauma is very real. Undergoing the agonies of drug withdrawal can do lasting damage to the brain; it can be as traumatic and lasting as surviving the horrors of war.

This means that it is actually easier for a person who has Parkinson's to decrease his medications than it is for a person who does not have Parkinson's to decrease his anti-PD drugs.

There will be more in-depth case studies of people taking very low amounts of medication or stopping completely in the chapters that follow. But before reading those, it's time to make good on my promise of providing more proofs for the first three hypotheses and introduce a fourth hypothesis.

"Maximum effectiveness of drug may not occur for several weeks or months after therapy begins. Maintenance therapy must be carefully adjusted based on patient tolerance and desired therapeutic response. Observe and monitor vital signs, especially while dose is being adjusted."

On levodopa-carbidopa, *Physician's Drug Handbook*, 9th edition, 2001, p. 589.

18. SUPPORTING OUR HYPOTHESES

THE BASIS FOR OUR THREE (OR FOUR?) IDEAS

Back in chapter three, when I introduced the key new hypotheses, I promised to give more details to support these ideas. Now that you have a PD vocabulary, an introduction to blood-brain barriers and addiction, and some brief case studies as examples, the time has arrived for the long-promised enrichment. You'll even get an additional hypothesis thrown in gratis. This chapter will present a group of graphs from a recovering patient; it was the graphs from recovering patients that finally cracked open the mystery for us and enabled us to see what the drugs were doing, and over what time period.

Theoretical levodopa timetable – the old theory

If you recall from chapter three, levodopa and all the other anti-PD meds are supposed to be taken at dosage rates that ensure a steady On throughout the day. One to four doses, depending on the drug, over the course of the day will supposedly yield a steady level of dopamine. After the first dose of the day brings a person up into the effective, or On zone, regular dosing should maintain a person in the On zone, with maybe a few unavoidable moments of excess movement.

As you will recall, the chart for this theory showed a series of nice neat curves, corresponding to each dose, which rise smoothly, reach a gradual peak, and then taper off.

Chaos from overmedication – the new theory

However, the actual charts we saw tended toward the chaotic. In the first few years of our research, when most of our medicated patients were dyskinetic, On-Offing, and therefore obviously overmedicated, the charts of daily On-Offs did not support the idea of a uniform period of pill effectiveness as advertised. In fact, the reason I asked patients to start keeping track was because their verbal descriptions of their past week sometimes made no sense according to the descriptions of pill function according to text; I assumed that their reporting was suspect. When they charted their On-Offs, most of them had either unpredictability or, if there was any daily pattern at all, a pattern of daily Build Up. At the time, the Build Up did not look like a "pattern" – it just looked like late-in-the-day pill failure. It was only later, when we hypothesized that drugs could accumulate far beyond their conjectured half-life that the pattern of drug failure late in the day became obvious to us. Prior to that, daily Build Ups were inexplicable.

It was only when some patients started reducing their medication and waiting several months to assess the change that we started seeing charts that had regular,

predictable Ons and Offs as described in the section on daily Deficits. (Deficit days featured pill failure of the morning doses but increasing pill effectiveness later in the day.) Those charts were described in chapter 13, and they started us in the direction of looking for accumulation effects, including freezing (which looks for all the world like pill failure), after which the Build Ups began to make sense.

Finally, the wildest, most unpredictable patterns occurred when *recovering* patients with daily patterns did not, for whatever reason, reduce their medication quickly enough. We started seeing extremely chaotic graphs. These cases of unparalleled unpredictability and adverse effects were the final piece of the puzzle for us, confirming the weakness in the previous theories of drug application and suggesting the new hypotheses that we are presenting here. We were forced to conclude that, despite all the opposing information in the guidebooks, the wildly unpredictable charts of the mildly-dosed, recovering patients must be due to a previously unsuspected *long*-acting effect of the pills. Certainly, none of their charts conformed to the theoretical model.[1]

Because these were the charts that clinched our burgeoning hypotheses and made, most dramatically, the point that dopamine-enhancing drugs taken days before have a huge impact on a following day's pill effectiveness, I will give here an explanation of Sonny's charts.

Sonny's situation

After being in our program 3+ years, Sonny broke his hip. The following charts are from a period five months after his hip replacement surgery. After the surgery, he increased his medication from 350 mg L-dopa/day to 400 mg/day. When Sonny had started with us he had been taking a high of 1200 mg L-dopa/day. He had slowly, over three years, gotten down to 325 mg L-dopa and 1 mg Permax per day from a high of 1200 mg of L-dopa plus 4 mg of Permax per day. During his decreases, he'd had cyclic periods of increased immobility, severe depression and/or anger, but more consistent, predictable Ons, and less dyskinesia. During his Feelin' Good days at the end of each reduction cycle, he would have some days when his new, lower dosage level worked perfectly: these halcyon days would have little or no Off time and no dyskinesia. However, this condition would change quickly as the drugs became once again – due to recovery – excessive. As the dyskinesia returned at the new, lower drug level, his wife would make another reduction. His reductions were always modest, and he never experienced full-blown drug withdrawal. He made new decreases each time the dyskinesia returned, based on his wife's assessments.

Afer several years of working with us, he had recovered the ability to smile. At night, more than 8 to 12 hours after taking his evening pill, he could increasingly move his arms and legs by himself – a level of pill-free movement that he had not been able to do for several years.

However, after breaking his hip, he lost all interest in reducing medication and, at his surgeon's suggestion, decided to keep his medication at a level admittedly higher than

[1] Of course, some patients were still getting very good coverage from their drugs. We weren't looking at their charts; only those of our pioneers who were experiencing Ons and Offs were graphing their results, writing up prose accounts of their day, or giving me oral histories of their drugs' daily effects. Patients who were fairly new to the drugs, who were still in the honeymoon period of drug effectiveness and whose drugs still worked uniformly all day, had no reason to chart their daily ups and downs.

necessary (admitted by both Sonny and his wife). For the first few months, he felt just great with the increased medication. Soon he began to complain about the increasing Offs and dyskinesia, but due to residual pain from the surgery and difficulty in using his repaired hip, he was reluctant to begin a decrease cycle again. During the preceding years of drug decreases, he had noticed predictable, cyclical, slowly-changing patterns of On-Off, Build Ups, and Deficits as his meds slowly cycled from excess to deficient and back to excess over a period of many weeks for each small reduction. Now, at 400 mg/day but recovering, his charts returned to being even more unpredictable than they had been when he was taking (unrecovered) 1200 mg/day – nearly three times as much.

These charts come from his post-surgery period, and will show what happens when a grossly high level of overmedication is maintained.[1] His medication levels were quite low according to modern prescribing practice – only 400 mg/day of L-dopa in a patient who had had Parkinson's for over twelve years. However, prior to the surgery, he had been moving well at 350 mg/day and was on the verge of making another decrease in his medication just before he broke his hip.

The most significant thing to see in his charts is that no two days are the same. This in itself contradicts the old half-life theory used in creating prescription advice. Instead, the Ons and Offs have almost no relationship to the time the pills are taken. At this new phase of his relationship with the medication, he usually takes the pills at the same time every day, only making once-in-a-while decreases for one or two days if he is feeling a severe worsening of adverse effects. Even so, some days the charts are almost opposites of the day before. A person whose medication is creating a consistent pattern can usually tell at what point in the day the medication becomes excessive (creating Offs, ticcing, or dyskinesia). However, when a person is completely over the top, every day's drug response can be a reaction against the *previous* day's or weeks' excesses, and it is anyone's guess how the brain will cope with the relentless onslaught of dopamine.

Sonny's wildly fluctuating charts are typical for a recovering patient who is having "instability" with his medication. We now suspect that any patient with Ons and Offs that are unpredictable from one day to the next, as demonstrated in these charts, is grossly overmedicated. The tidy, repeatable daily patterns, described in the preceding chapters, only occur if a person's medication is somewhat reasonably close to the amount that he needs.[2] When a person starts having patterns such as Sonny's, he is gravely overloaded.

Reading Sonny's charts – the new theory

In the following charts, this key applies:

1) The small black triangle denotes the time of day of the dose.

---

[1] Overmedication in this case refers to the amount needed by the individual, not an absolute amount.

[2] This statement only applies to those who are already having On-Offs. A person who is new to the drugs may not yet be having On-Offs. This absence of On-Offs is NOT proof that their drugs are being taken at the correct level. It can take several months, even years, before the brain of a PDer constructs enough defenses to enable itself to protect itself against excess medication via dyskinesia, On-Offs, and other adverse effects. Non-PDers using these drugs will manifest problems much sooner (see: Oliver Sacks' research). The reason for this difference is explained later in this chapter. Again, a person who is fairly new to the medications can be seriously, invisibly, overmedicated but not yet having On-Offs. By the time On-Offs appear, lasting brain damage has probably occurred.

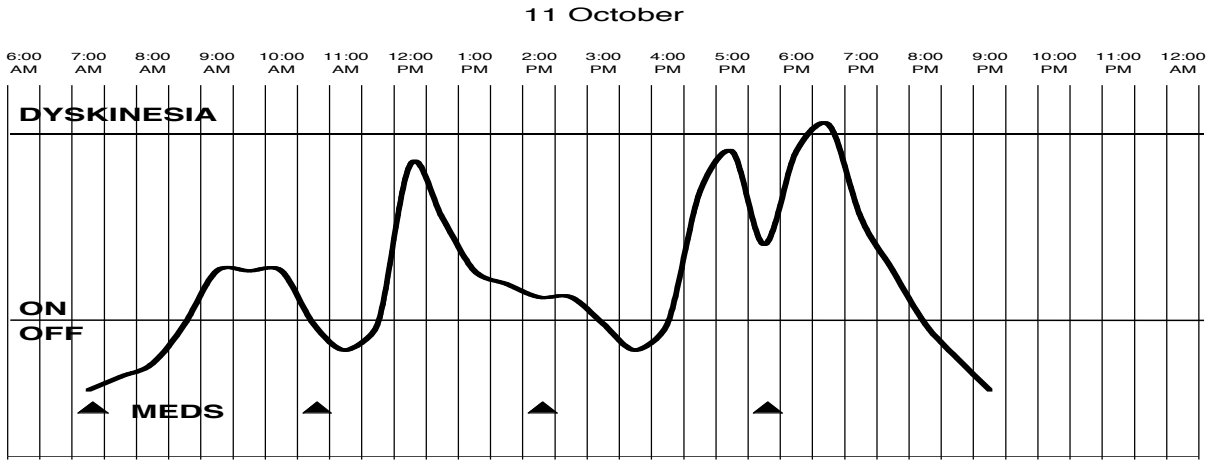
2) The doses were all the same size – a pill of 25/100 Sinemet and .25 mg of Permax.

3) The line denotes motor activity.

4) When the motor activity line drops below the Off line, Sonny could not move at all, nor speak. He was absolutely frozen. When the motor activity line was in the middle part of the On zone, he could move, but not optimally. When the line was in the low part of the On zone, he could shuffle, speak in a whisper, and move if he was given a push from time to time. His posture was hunched, he drooled, and had little small motor function. When the motor line was just below the dyskinesia line, he was moving optimally, smiling (though he had not been able to smile at any drug level prior to starting Tui Na therapy), and his voice was strong.

To collect the data for the charts, his wife asked him every hour how he was doing. If he could talk, he decided where the dots should go on the chart. If he could not talk, she decided. At the end of the day, they connected the hourly dots to create the following charts.

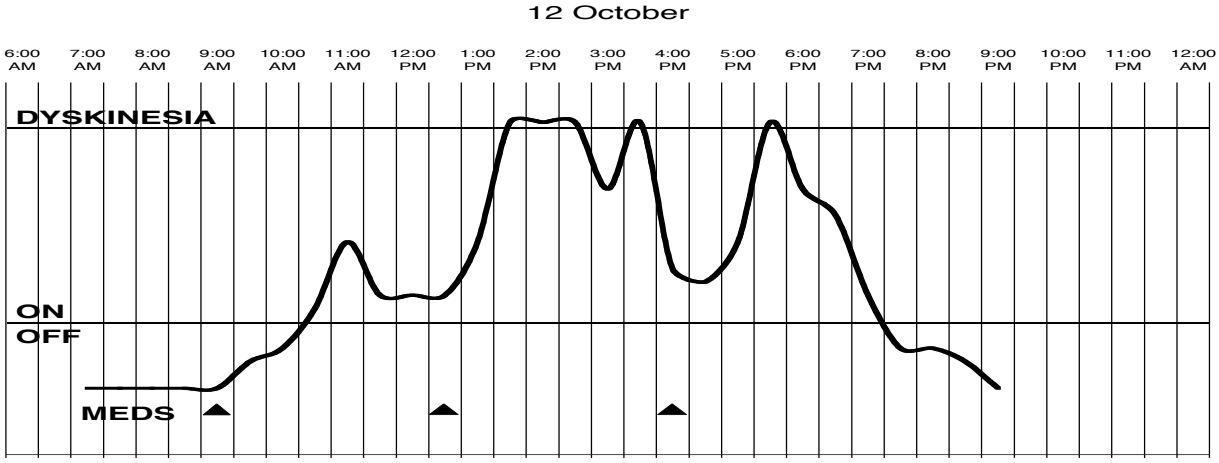
Prior to the hip surgery, Sonny's days began with his movement just barely above the On line, even before his first pill of the day. This meant that more than twelve hours after taking his last pill of the day the evening before, he was moving, albeit very slowly, on his own. After increasing his drugs (following hip surgery), he lost his ability to move in the early morning (pre-pill), and could only move after his first, or sometimes second, pill.



The first dose on October 11 created a nice On, followed by a predictable Off. The second made a spiked On, followed by a rapid decline in functionality.[1] During the decline from the second pill of the day, he took a third pill, after which he was immobile for 2.5 hours. Then, *just before* taking another pill, he shot up to a period of high mobility, with a small amount of dyskinesia. He started to plummet from this dose, but took a pill mid-plummet. Nearly twelve hours after his first pill, when motor receptors that had been shut down by his first dose were just starting to be receptive again, he took his last dose. The resumption of effectiveness in the motor area, combined with the

[1] Review: as noted on the preceding page, Sonny used the low region of the On to indicate that he was able to move, but only very slowly. The Off section was used when he was utterly immobile, unable to initiate any movement, including speech.

accumulation of dopamine throughout the day, caused the fourth pill to appear to be immediately effective: this dose propelled him into half an hour of arm spasming, facial grimacing, and shoulder twitching. Two and a half hours later, he was already Off and crashing. He would be completely immobile until two in the morning, at which time, *without having taken medications since 5:30 the previous evening*, he would again be able to move slowly but steadily, using his arms to adjust his blankets, moving his legs to change position, and to speak softly for several hours. However, this spurt of night-time (drug free) movement never lasted until the first pill of the morning, as it had in the previous year. Instead, it would usually sputter to a halt at around 5 in the morning.[1] He would be completely Off again when it came time to take his first pill of the day and begin the On-Off routine once again.

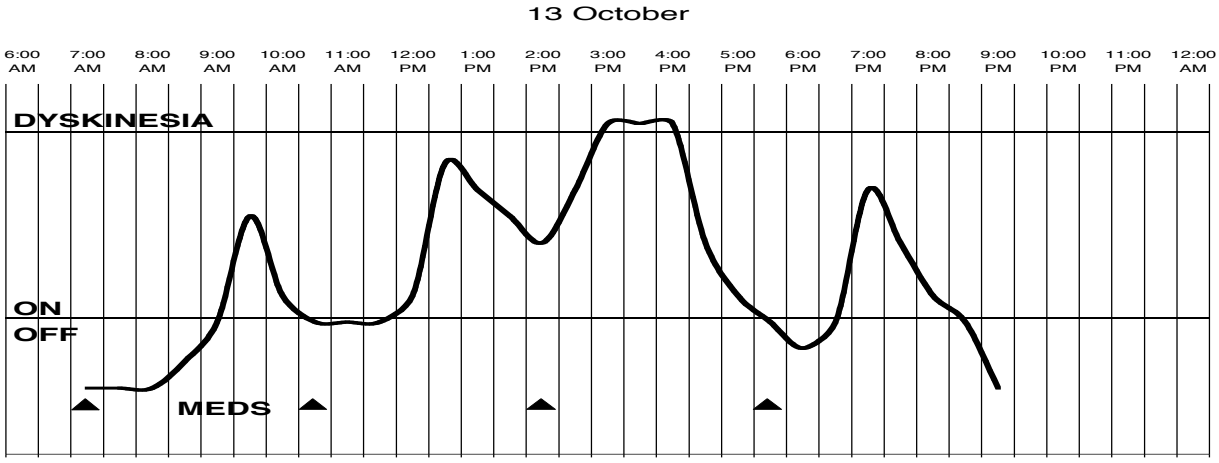


On this chart, Oct. 12, the ups and downs are almost the exact opposite of the day before (Oct. 11). Because of the dyskinesia the day before, on this day he only took three pills, spaced further apart. It took nearly two hours for his first pill to work. He moved well for 20 minutes and then went back to shuffling. Sonny usually took his second pill of the day by 10:30, but his second pill on October 12 was delayed to after noon. However, even with the delay, his second pill led to dyskinesia for most of the dose, except for a brief Roller Coaster down into normalcy and then back up to dyskinesia, again followed by a rapid decline. He was moving well two hours after taking his third dose; however, it soon spiked up into dyskinesia and then slowly climbed down. He was

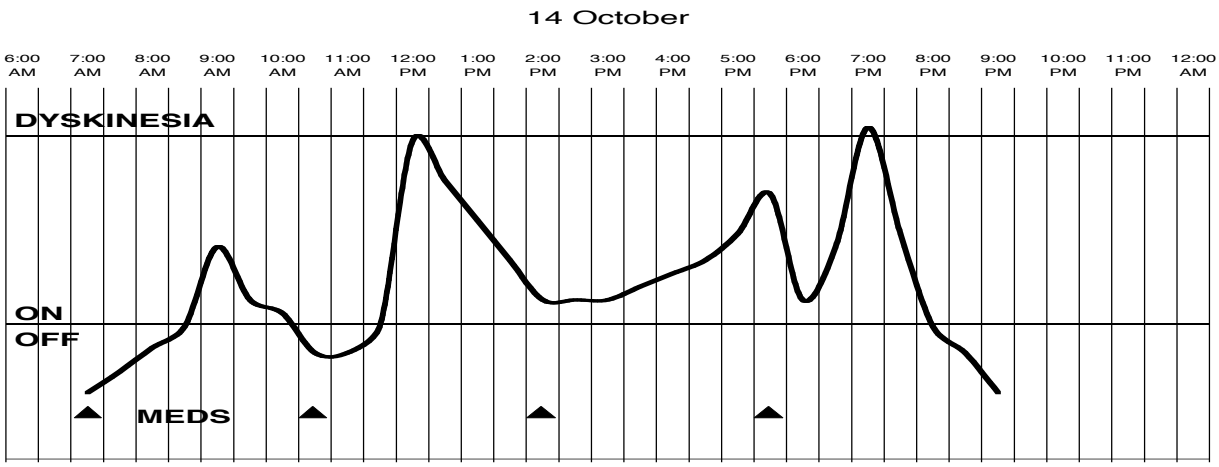
[1] This specific phenomenon will not be discussed in detail in this section, but let me note that this unfortunate decrease in mobility during his 14 hour "unmedicated" span, from 5:30 in the evening until 7:30 the next morning, was most likely due to drug-induced brain damage from overmedication. Prior to this time, during his recovery, before he increased his medication back up, he started having "unmedicated," night-time movement, movement that was very different from his On time. It appeared to be coming from native dopamine. This night-time movement would emerge 8.5 hours after his taking of his last dose, ending his profound, crashing bedtime Off. When it first appeared, he could only move one arm for less than an hour. After four months, he could move an arm and a leg, and the movement lasted for two hours. It took over a year for the night-time movement to include all four limbs and sustain – get this – until he took his first pill of the morning. After taking his morning pill, he would plummet into a severe Off (see: switching), go On half an hour later, and then, two hours after that, crash into a frozen Off. Interestingly, the last limb to finally acquire what we named "Sonny's night-time movement," a slow, rather languorous, weak/gentle movement – very different from his medicated On movement – was his left arm: the very limb in which the Parkinson's symptoms had first appeared.

off for the day within three hours after his last dose. He typically crashed “very hard” by the end of the day.

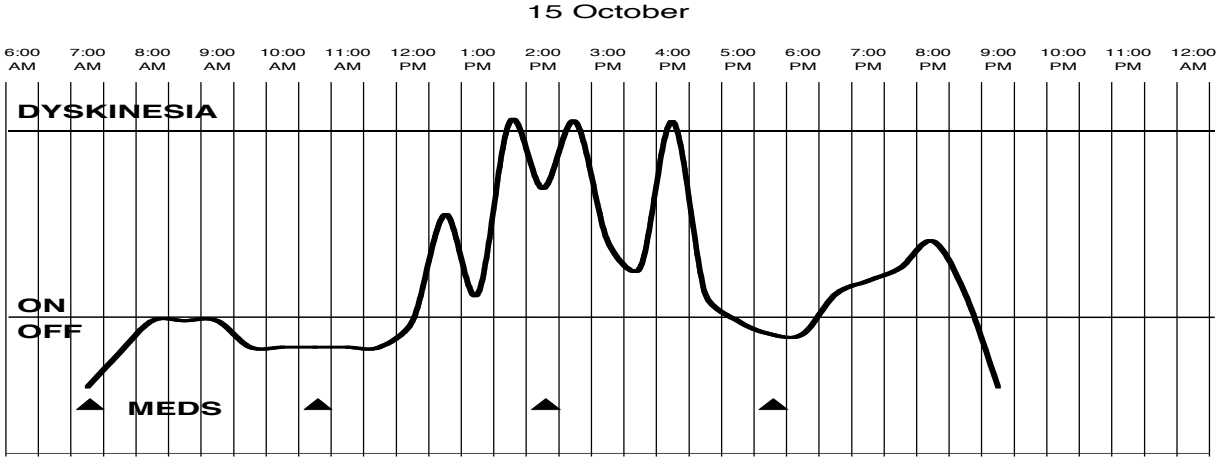
Remember, during a roller coaster type of response to the drugs, the movement line on the chart will go down while the amount of drug in the body, based on time of dose, is still going up.



Unhappy with the slow movement of the day before (Oct. 12), when he had only one hour of good On time up until 1:30 in the afternoon, he went back to four pills (400 mg levodopa) on Oct. 13. Aside from the slow start in the morning, this was a very good day. Due to the decreased dose the day before, he had very little dyskinesia and only a few hours of Off in the morning and a few hours of Off in the late afternoon. This is the sort of several-days pattern that had first led us to suspect that the effects of any given pill were being influenced by pills taken the previous day.



On this day, Oct. 14, he paid the price for having gone back up to four pills a day the day before: his first On of the day was earlier, but the subsequent swings were more abrupt. From 10 to 11, he was moving very slowly and, except for a brief surge at 12:30, continued that way for almost the entire afternoon. (Remember, movement in the lower On zone was shuffling, drooling, and whispering. Normal stride and function was indicated in that part of the On zone just below the dyskinesia line.) His 2:00 p.m. pill didn't work until after 5 p.m., and then it Roller Coastered up, dropped abruptly, and was starting back up when it combined with his evening pill to create dyskinesia.



This day, Oct. 15, is almost the exact opposite of the day before, though he took his pills at the exact same times! On this day, he is clearly overmedicated: even his first pill of the day won't work – his brain is still oversaturated from the day before. His brain builds such a level of resistance when he adds his second pill to the mix that he is able to get On, into the functional zone, only at the tail end of the second dose, as the dopamine levels are slowly forced down by the brain's frantic defenses, down from the immobilized, overmedicated zone.

It appears, from looking at the chart, as if the drugs have finally become sufficient at 1:30 p.m. In fact, they have finally dropped *low* enough that his brain is allowing some

movement. Since he is still somewhat overmedicated, this movement appears as dyskinesia. This is why, on the chart, he shoots up into dyskinesia even before he takes the third pill of the day. When the third pill starts to take effect, about an hour after the dose, he plummets back down into immobility as his brain becomes oversaturated with dopamine.

However, by now, some responses (neuron refresh – see chapter three) to the first pill of the day have been washed out of his system. Also, the Shut down, with its seemingly therapeutic respite and accelerated drug clearance functions, may have been at work. From these influences, his brain's dopamine level has decreased enough that he is able to get back some motor function (On) from the third pill. When this pill begins to wear off, he crashes, of course, after so much excess. After the crash, the last pill of the day works nicely, supporting our ideas that the shut downs seem to serve a therapeutic function and that the refresh rate for overstimulated dopamine receptors seems to be about twelve hours.

Please pause for a moment to look at the two charts on the preceding page. Remember – the pills were being taken at the same time every day, the doses were the same every day. Sonny's minimal physical activities and non-existent social life, due to his injured leg, were the same every day. Nearly all variables were holding in a consistent pattern, but Sonny's On-Off times appeared to be responding to something other than his dosages. **Only if** we theorized a cumulative property for the medication (which meant a greatly longer half-life for his pills than the doctors were using), and different rates of dopamine response in the limbic and motor areas, we could construct a model in which Sonny's drug results were not only explainable, but predictable.

Repeatable randomness

It did seem at first as if my interpretations of these charts were merely specious – random explanations for random events. I feared that I was just making things up to fit the case. However, using our new theories, we found we could predict exactly what would happen with Sonny, or with any other medicated patient, over the next few hours or the next few weeks. For example, every time Sonny tried a lower dose, he had the predictably slow onset with the overall good day. Every time he increased back up, the next day predictably would have more dyskinesia and the day after that would have a long delay before the morning On occurred, followed by predictable spikes of dyskinesia, a crash, and a nice dose at the end of the day.

Although the charts seem erratic and formless on any given day, they actually followed this pattern very nicely over time. Not only that, but all the other patients had similar patterns if they were grossly overmedicated. It was by noticing the similarities in their charts on the first, second, third, fourth, tenth, and fortieth day after a drug change or after becoming overmedicated that we were finally able to make a logical explanation for what was occurring. While there may be reasons other than the ones we are proposing (a certainty!) that more accurately pinpoint the reasons for the On-Offs of our patients, the fact that by using our new hypotheses we were able to predict fairly accurately which "unpredictable" pattern was most likely on any given day, week, or month, and how these patterns would most likely change over the long-term did suggest that our ideas were of value.

Naïve researchers

When we first started this Parkinson's research project, long before we knew the drugs were addictive or cumulative, we were naïvely expecting patient charts to be useful for our questions about two very basic aspects of the pills: onset and duration times. Our earliest questions were very simple: how long did it take for the pills to start working? How long did the effects of the pills linger?

Before we understood that there were changing thresholds and variable baselines, we assumed, as did the patients and their doctors, that we should be able to get somewhat consistent numbers for these easy-to-measure, straightforward events. We assumed, wrongly, that patients who were having Offs and Ons would be convenient subjects for measuring these events; patients who weren't having Offs and Ons usually couldn't tell very accurately when their drugs started to work, but in On-Offers it was obvious.

The common assumption at that time was that it was advancing Parkinson's that was responsible for Ons and Offs: this thinking held that, as Parkinson's disease advanced, the native dopamine further decreased, and this decrease in dopamine was the sole reason that, over time, the pills needed more time or a higher dose to start working. (This view completely ignores the manufacturers' warnings that the medications themselves cause Offs, freezing, bradykinesia and tremor, to name just a few of the adverse effects of pill effectiveness that can *resemble* their opposite: pill "failure.")

We measured the onset and duration times of the drugs in our patients who were having On-Offs. We defined onset time as the time it took from the moment the pill was taken until the time that there was a perceptible improvement in motor function. We defined duration as the time elapsed from the moment that pill-generated motor improvement appeared until the time that it ceased or significantly decreased.

We thought that there should be some consistent relationship between taking the pills, the time until onset, and the time when the pills wore off again. Instead, these are some of the numbers that we frequently observed:

Onset

Almost instant
10 minutes
20 minutes
Half an hour
Forty-five minutes
An hour
An hour and a half
Two hours
Three hours
Four hours
Unexpected Ons more than six hours after dosing
Never

Duration

None
Less than a minute

One minute
Five minutes
Ten minutes
Half an hour
Forty-five minutes
An hour
Two hours
Three hours
Up to eight hours

We had been expecting a nice, six-hour interval pattern. Ha ha ha.

Instability and unpredictability: some examples

One patient had predictable, one-hour onset waits, followed by predictable three-hour On times. Another had predictable half-hour waits followed by predictable three-hour Ons every morning, two-hour Ons every noon, one-hour Ons at 4:30, and no Ons from the evening dose.

Some pioneers were On most of the day, with random periods of freezing that could possibly be related to stress or bad weather. Some patients had utterly unpredictable results; some days they would feel great all day and the next day they could hardly move. Only patients who were pretty new to the drugs had a smooth pattern. Patients who had developed On and Off patterns had decreasing stability/predictability in their daily On-Offs.

This lack of uniformity, when a patient responds differently to each one of his doses, and/or the lack of conformity from one patient to the next is referred to as drug instability and/or unpredictability. Call it what you will, instability or unpredictability, none of the On-Off patients using levodopa had any pattern that seemed to correspond neatly to a three-hour half-life.

As we realized that there was no value in looking for predictable Onset and Duration times, we tried to ascertain just when and why the drugs, in any given person, switched from predictable to unpredictable.

Factors influencing unpredictability of the drugs

The onset of Unpredictability

There appeared to be two key factors influencing the emergence of unpredictability/instability: the number of years a patient had been taking the medication and the dosage level.

We tried to figure exactly how many years it was, after starting the drugs, that the drugs became unstable. However, the responses were so varied that they were statistically meaningless. For patients who had been taking the drugs to the point of On-Offs, or even to the point of feeling the surge and ebb of the drugs, no two patients out of our first group of fifty-nine medicated patients were the same. This lack of similarity made compilation of statistics unreasonable.

But in general, if a patient was fairly new (one to three years) to the medication and dosed cautiously, the Onset time and Duration were predictable for a few years –

namely, Ons and Offs never occurred, and it appeared as if there was a seamless transition from the effects of one pill to the next.

While the number of years before On-Offs occurred appeared to be a key variable, it did seem as if this variable was affected by dosage amount: patients who had been started at 300 mg levodopa/day and told to stay there as long as possible developed the On-Offs much later than patients who had been told after diagnosis to "start at two pills a day and increase by one pill a day until you feel really good, and then stay at that level." Patients in the latter group had much higher starting doses, often getting as high as 800 mg/day levodopa before they "felt really good" and then maintaining that high amount as their "starter dose." This group might have drug-induced dyskinesias and On-Offs within three to six months of starting the medication.

As recently as 1998, the available literature used to suggest that there was a five-year period before the drugs became unpredictable. The more recent literature suggests that a two to three year period is now the norm.[1] I suspect that this changing time frame has to do with the newer, bolder prescribing patterns. These in turn may have to do with our society's increasing demand for quick results from medical intervention, or they may be related to doctor misinformation.

Certainly, in the first few years of dopamine-enhancing drug prescribing, when the drugs were still considered somewhat experimental, most doctors were keenly aware of the risks and adhered closely to the recommended dosages. Possibly, since these drugs have now become accepted tools, breezy nonchalance has replaced cautious trepidation.

For whatever reason, in our experience, the drugs are now being prescribed at much higher rates. My more recently diagnosed patients who were working with younger doctors invariably were prescribed higher starting doses and faster increase rates than my patients who had been diagnosed ten, fifteen, or twenty years earlier and who were working with older doctors.

For example, Old Dr. Rafferty's long-time patients Hjalmar, who had been taking levodopa for over sixteen years, Mark and Honoria, both taking it for nearly ten, had started at 300 mg/day. Honoria was still at that level after ten years (plus two agonists). Mark was still taking 300 mg/day plus an anticholinergic. Hjalmar had increased slowly through the years, but his average increase was a mere 50 mg/day increase each year. They all still had a high amount of predictability in their drugs: as their Parkinson's symptoms had gradually advanced over the years, Dr. Rafferty had added other drugs to their mix or suggested they use a walker. He usually gave them permission to increase slightly, but with a warning that the lower levels were safer, hence the slow increase rate for Hjalmar and the addition of a few other drugs for Honoria.

Dr. Rafferty's long-term patients, in general, had far fewer problems with their medications, even after twenty years on the drugs, than the patients of the other, younger, local neurologists. I did notice that even Dr. Rafferty was starting to be bolder with the drugs, however; his more recently diagnosed patients had been started at much higher levels. He even complained to one patient who was reluctant to go above 500 mg/day that, "All my other patients are taking at least 800 mg/day and there is no problem."[2]

---

[1] A.N. Lieberman, MD, Curing Parkinson's disease in our Lifetime: Part 3, Parkinson Report, Fall 2000, Vol. XI, 3, National Parkinson Foundation, Miami, FL, pp. 10-12.

[2] Considering that I was working with several of his long-term patients, two of whom were taking 300 mg/day, and one having dyskinesia at that low rate, I must question Dr. Rafferty's accuracy.

In summary, it appears that the size of the dosage and the number of years one takes the drugs affect the onset of unpredictability.

Mood

We also noticed a factor that related to short-term drug unpredictability, those unexpected On-Offs that might happen on any given day. Mood, it appears, plays a large role. A visit from a dear, out of town friend might coincide with the drugs working all day, with no Offs. An unexpected diagnosis of early stage, easily treatable melanoma or prostate cancer, on the other hand, was accompanied by little or no On time in the weeks or months that followed.[1]

None of our patients' charts looked like the orderly theoretical one at the start of chapter three, page 37. Most ranged from mildly predictable to completely random. What was the reason for this "instability" of dopamine-enhancing drugs?

Forming the hypotheses

More about half-lives

As related earlier in chapter three, we were assuming, as were the doctors and researchers, that the significant number to keep in mind with regard to dosings of DEDs was the half-life. The half-life of a drug is the amount of time it takes for 50% of any given dosage to be broken down and/or excreted from the body.[2]

---

[1] These examples are drawn from our patients: Hjalmar was diagnosed with prostate cancer. He did hormone treatments, and happily, his PSA levels are now below normal. His drugs did not work well until he got the blood test showing his PSA levels were down. The sores on Mark's face were skin cancer. They were removed and he received a good prognosis. His drugs, however, completely failed him for over two weeks, from the time of diagnosis until the scabs from the cancer removals (the cancer spots were frozen off) started to crumble.

[2] The half-life of a drug is *not* like the half-life of radioactive materials. Radioactive materials have a steady rate of half-life. Here is an example of radioactive half-life: The radioactivity of a hypothetical ore is depleted by 50% over, let's say, 100 years. We would say that the radioactivity in this ore has a 100-year half-life. We can be certain, based on the nature of radioactive decay, that during the second 100 years, 50% of the *remaining* radioactivity will be depleted. In yet another 100 years, 50% of *that* remainder will be gone, and so on. The first span of 100 years is referred to as the first half-life, the next is the second half-life, and so on. In this hypothetical radioactive ore, the half-life rate of 100 years remains constant; in this example, it will always take 100 years for the remaining radioactivity in the ore to degrade yet another 50%.

Half-life in the body is not so steady and predictable. Many factors can alter the rate at which a drug is broken down or excreted. For example, much drug breakdown occurs during the drug's first pass through the liver, when food absorbed from the digestive tract goes to the liver for a first cleaning before entering the main bloodstream. Also, drugs making it through the liver might be stored indefinitely in fatty tissue for later use. Drugs might even be broken down quickly one day and not so quickly the next, because of emotional, hormonal, or even weather-related influences. So many external and internal events can affect the rate of drug processing that it is impossible to accurately predict how long it might take for the second 50% to be broken down halfway. As for the remaining bits in the third or fourth half-life, it is anyone's guess.

Official half-lives of drugs (the first half-life only; the others are ignored) are usually determined using a small test group of college-age students. Researchers are fully aware that drug half-life in an octogenarian can be many times longer than the "official" half-life of that drug. In some cases, a drug that is half removed from a college kid in a few hours might linger in an older person for days. However, since oldsters are so variable, they are rarely used for research. (Footnote continued on next page.)

As noted in chapter three, it had been established decades earlier, based on blood work, that most anti-PD drugs have a fairly short life span in the blood. In the case of L-dopa, for example, half of the medication was gone from the blood within a few hours of ingestion. Mirapex had a half-life of 8 to 12 hours, Permax was 24 hours, and Eldepryl was 48 hours, to name a few. The dosage schedules were built around these numbers. I had sat in on a lecture in 1998 in which a drug representative had assured the audience of PDers and caregivers that the best interval for taking levodopa-containing pills was every six hours, and that this was based on a half-life of approximately 2 to 3 hours.

Build Up questions

It wasn't until two years into our study, after looking at these charts that never reflected anything resembling a two-hour half-life, that it hit me like a ton of pills that the levels in the blood were a side issue. The significant number should have been the level in the brain.

We also realized that no one knew if PDers, whose dopamine levels were low, might also have altered levels of dopamine-related chemistry. For example, were the breakdown mechanisms for brain dopamine *elevated* in PDers? *Diminished?* Did anyone have any idea whatsoever how long or in which part of the brain these drugs lingered after they got inside the blood-brain barrier? No.

What was the reason that, according to the manufacturers, these drugs might take several months to attain a therapeutic effect? Was a slow amassing of dopamine in the brain the cause of the delayed therapeutic effect? In that case, if the drugs *could* accumulate over months, and some people were being prescribed doses so large that the drugs worked within a day or two, they might be amassing nearly thirty five times more drug in their brain than they actually needed. If so, no wonder these drugs rapidly became unpredictable!

Application of this idea to all dopamine-enhancing drugs

The slow accumulation of dopamine, if it was occurring, would not be just an L-dopa problem, but a problem with *any* dopamine-enhancing drug. As an example, Eldepryl has a much longer half-life than L-dopa: two days. Therefore, the suggested dosings of Eldepryl are further apart than the dosings for levodopa. And yet, was this half-life number actually significant?

Some metabolites (breakdown by-products) of Eldepryl pass out through the kidneys. Others get into the brain and are passed out…when? Who knows how much is still in the brain when 50% is still in the blood two days later! The process by which these metabolites are broken down in the brain isn't known. They could be lingering in there for a week, a month, a *year,* and no one would be the wiser.

The theories of medicine, in an attempt to conform to an "objective" model like the still current Cartesian (René Descartes) theory of body = soulless mechanism, discard the evidence that no two people are alike and, instead, use only those healthy humans that have the greatest degree of similarity for their research. They imagine that this makes their work more scientific. Maybe it would be so, if their results were only imposed on those youthful, relatively similar students.

But if *medical* research is done on uniformities and the results imposed on variants, practitioners of such medicine lose their claim of objectivity and "scientific logic." This subject is beyond the scope of this book. But it is worth noting that, although half-lives in biology are NOT similar to half-lives in radioactive ore, the numbers are traditionally applied as if they were.

Because all the dopamine-enhancing drugs work in the brain, and we have no idea how long they last in the brain, we are in the dark about the true effective times of all the dopamine-enhancing drugs, not merely levodopa.[1]

Stabilization theory

The suggested doses of these drugs are usually determined by how a person is feeling on any given day, combined with the half-life information. An outgrowth of this method of calculating drug effects is the current, dangerous theory of psychotropic drug stabilization.

Based on the methods of the three doctors who tried to stabilize Zoe, Birdie, and Viktor when they became dangerously addicted, and on six similar cases reported to us from PDers and health practitioners across the USA, England, and Spain, I can say that 100% of the doctors in this admittedly limited sampling assumed that even extreme cases of drug problems could be "stabilized" within three days.[2] In every case, the results were ghastly or deadly.

We had to conclude that there was something seriously wrong with the current method of assessing drug levels, determining doses, or stabilizing patients with medication problems. But wait! Hadn't government-approved testing shown that these drugs were safe?

Drugs pass tests

Yes, all the drugs had passed through various scientific tests prior to being unleashed on the public.[3] But what these tests showed was that the drugs appeared to

---

[1] Again, the reason we have used levodopa so often in these examples is that it is the most commonly prescribed drug for PDers. However, the principles apply to any drug that increases brain levels of dopamine.

[2] Both Zoe's and Birdie's doctors were Parkinson's specialists, engaged in doing PD drug research. They both currently rely on the blood half-life assumptions.

[3] For an example of the sort of mentality that exists in "scientific drug testing," the *Awakenings* researchers (very brilliant, competent doctors, actually, and Oliver Sacks is a genius), in 1968, assigned drug doses based on the half-life of L-dopa being 1-3 hours. Patients were given different doses once every day in an attempt to figure out what the ideal dosage would be. Knowing that the half-life was a mere 1 to 3 hours, the logical assumption was that the L-dopa was effectively out of the bloodstream within 24 hours. This meant that every day, the researchers could presume that they were starting with a blank slate. If a patient did not respond on day one to 1000 mg of the drug, the next day they might try 2000 mg. Or conversely, if they noticed on day four, at the 4000 mg level, that the patient was showing signs of overmedication, the next day they would try 3000 mg. This makes sense, based on the half-life theory. It also makes sense according to the theory that all brain nerves work at the same speed. A motor response was the "dipstick" test: if a previously immobile person could move, the researchers assumed the whole brain was topped up exactly right.

Those researchers found that, even in patients who responded well at first, after many days had gone by, the same dose behaved unpredictably; sometimes a patient would have worse side effects on a day with a lowered dose, and sometimes a patient wouldn't seem to respond at all to increasing doses, and then, wham! He might erupt with life and vigor on a day with *decreased* medication. They concluded, eventually, that the drug was not stable or predictable, and, as the treacherous side effects appeared, that it was also unsafe for any person.

But the point here is, just because a drug has been tested doesn't mean much. We all know of drugs that make the headlines because they turn out to be toxic, even though they had passed through "rigorous" tests years earlier. The problem, to a large extent, may be that the companies that do the testing

have some benefit, and that people didn't immediately die from them. The tests also established that some of these drugs might not reach full effectiveness for six to eight weeks, a point ignored by all the doctors in our (clinic, travel/lecture, and email) experience. Nearly every doctor that we have heard about has been confident that the PD drugs should reach effectiveness within three days. In the case of Mirapex and Requip, two drugs that are started at low levels and gradually worked up to a higher level (due to these drugs causing fainting and other unpleasant side effects against which the patient must slowly become hardened), the assumption remains that, once the "effective dosage" is attained, the full effect will be obvious within two or three days.[1]

---

have a vested interest in the results. That subject is beyond the scope of this book. But it may be more than financial interest that influences our interpretations. Maybe it is human nature to perceive results through paradigm-colored glasses.

Actually, there is a new field of science devoted to trying to figure out how to make tests be meaningful. But so far, no one really knows how to best interpret the results of all scientific testing, medical or otherwise. Medical testing is, however, recognized as the least scientific kind of testing and the most worthless for predicting results. The entire idea of scientific testing may be a fallacy. The newest theories of physics indicate that, due to the profoundly interrelated nature of all aspects of the universe, in order to correctly determine which event is causing which result, one must either intuit the answer or, in order to make an accurate logical construct, actually know all the initiating events since the beginning of time. Short of using one of these two systems, no "scientific" field of enquiry can actually enable one to make meaningful predictions.

The most basic example of this problem is the old statistics class model: no matter how many times a dropped coin might come up "heads," the odds will remain 50/50 for a result of "heads" with each new drop. The coin may appear to have only one side – the head side – to the foolish observer who imagines that a series of "heads" means that no "tails" exist! The sad truth is this: a given result implies no predictability of the future. Nor does a seeming relationship between two events prove which is cause and which is effect, if any. For example, hundreds of years ago experiments "proved" that a slab of beef wrapped in a dirty shirt and set out in the sun would create living maggots. This appearance of life being generated via two lifeless commodities was "scientific proof" of the theory of Spontaneous Generation, and was frequently used in support of the biblical story of Genesis. Incorrect guesses by centuries of "leading scientists" (see: any good book on St. Thomas Aquinas) should have confirmed the impossibility of "scientific" prediction or proofs by now, but ignorance and ego die hard.

The concept that only intuition and/or knowledge of the origin of the universe can provide truly correct, truly accurate predictions has only recently been demonstrated by western scientific methods in the field of physics, but it is an old, respected Taoist, Vedic, Buddhist, Jewish, Islamic, and Christian principle. In the west, a widely known statement of this, now "scientifically" confirmed (see: any good book on Chaos theory), truth is found in the Hebrew Psalms: "Seek ye first the kingdom of God…and all other things will be added unto you." Wisdom, which includes the knowledge of all forces in the universe, where they are going and what action is precipitating which result, is among those "other things" promised in the psalm. Wisdom, even scientific wisdom, comes to him who seeks not *facts*, facts always incomplete, always perceived through the veil of the current paradigm, but the font itself – the Origin of Wisdom.

Where I am going in this overlong footnote is this: testing does not equal safety, a medical degree does not confer wisdom, and the material taught in medical classes changes as rapidly as the weather. We do scientific experiments to determine whether or not events have some degree of repeatability, and then guess at the meaning. The ultimate conclusions for the experiments, the "why" and the "how will it play out for any given individual," remain mysteries, known only to those who transcend their superstitions, "scientific" or otherwise, and delve into the heart of God.

[1] We have seen that patients who stay at the "sub-therapeutic," starter levels of Mirapex actually do obtain good benefits from the drug, but at these low levels it can require several months before the benefits become obvious.

Two dopamine systems?

But resuming our proposal that these drugs accumulate slowly, this hypothesis created still more questions. For example, if dopamine accumulated slowly, why did so many drug-users, after just a short while on the drugs, have obvious signs of motor function improvement within an hour or so after taking a pill? Was it possible that the motor function, separate from the limbic area, could make a visible flash of response to a flush of incoming drugs and then drop away as blood levels dropped? We were pretty certain that something was going on that took ten weeks to develop, and yet the motor response to drugs was sometimes so quick. Why?

The simplest way to explain this would be the existence of two systems. If we proposed that motor function had a quick response to dopamine as it crossed over the blood-brain barrier, but limbic response was slow, the seeming conflict would evaporate.

More questions

If that was the case, was motor function related to blood levels, but limbic function was not? And if so, how could we tie in the observation that the brain employed the motor area in dyskinesia when attempting to discharge excess levels of limbic build-up?

Did the limbic system act as a reservoir? Could the limbic area continue to slowly stash dopamine into receptors and vesicles even while the motor area appeared to be losing steam as the blood levels receded? Might the motor zone's quick processing of a blood-borne surge of dopamine allow for a visible, short-term burst of movement, while the limbic area was lowly filling up? Did the incoming dosage ride *on top* of the existing limbic amount? Was it easier to get a motor response if the limbic area was already filled, or was the motor area acting completely independently of the limbic?

And what about the very distinct effect of mood on whether or not a drug performed as expected? The frontal lobe is considered the place where mood is regulated. What was the time frame for dopamine retention/processing in the frontal lobe?

Three systems?

What if the limbic level was supposed to serve as the baseline, a baseline that, in a healthy person, would always ride just a hair's breadth below the motor threshold? Next, the mood and thinking area might be the activator, the place that determined whether or not movement should occur. If the brain was perfectly in tune, possibly the dopamine levels were at equilibrium throughout the brain, poised, with almost exactly enough dopamine floating about, but not quite enough to trigger a movement. Then, when a thought of movement occurred, the frontal lobe might initiate a neurotransmitter release that led to the release of exactly enough dopamine to the motor area. This dopamine would stimulate a nerve, thus opening the way for the acetylcholine brigade, which in turn would perform the actual mission of pulling on the muscles.

We in the project now feel strongly that dopamine is not a movement inducer in the same way that acetylcholine is. Dopamine is primarily a health, mood, and will power related neurotransmitter.[1] When a person is relatively pain free, warm, and not under

[1] Not will power in the sense of fear- or panic-driven ability to perform: these forms of drive come from adrenaline. This dopamine-related will power would be the will that is attuned with joyful dynamism: contemplative, intuition- and wisdom-driven will.

stress (limbic zone issues), and the mood/mental condition is healthy (frontal lobe), these two areas are then able to trigger a release of dopamine into the motor initiation zone as needed.

This is actually a new idea (yet another hypothesis!):

**Dopamine does not provide movement; it provides the transition between thought and movement.*

This would explain why, when people begin to recover from Parkinson's, they are often astounded at how they need only to think of doing something, and they find that they have done it.[1]

Dopamine: a multi-purpose neurotransmitter

In order to create a logical system that could account for all the various time frames and the influence of mood, we had to propose this tiered system of dopamine distribution with its variable time frames for processing dopamine, and this idea of equating dopamine with thought-to-movement initiation, rather than movement per se. If this was correct, it meant, conversely, that the old ideas were incorrect.

In the past, researchers might have noted the satisfactory, though brief (three to six hours) motor response to the drugs and assumed that was the whole story. But of course, until just twenty years ago, dopamine was deemed nothing more than a muscle relaxant (see: appendix 5). Now, in the present, researchers are just starting to see that dopamine may have more than one or two parts to play. We applaud these researchers and suggest that there are even more ways in which dopamine plays a part. In fact, we are proposing, by the end of this chapter, that dopamine is a key regulator for multiple systems, so long as a person is not in a condition of emergency.

The usefulness of hypotheses

In science, when we arrive at possible answers to our questions, these "educated guesses" are called hypotheses. They are different from established facts. The hypotheses that we have made about dopamine processing in the motor area, frontal lobe, and limbic system were made in response to our musings about the seeming conflicts in the long- and short-term responses to dopamine.

One of the important tests of a hypothesis is whether or not it has helpful applications. The hypotheses that we made about brain areas were very helpful in helping patients reduce their medication. By assuming that the motor response was only one small part of the story, our pioneers were able to gird themselves for the rigors of drug reduction. By our proposing that the invisible limbic area was making productive changes

[1] One patient, Lynne, a completely recovered patient (who never took the medications), started crying the first time that she realized she had stood up from the sofa the moment that she thought of the action, without having to first summon up any sort of momentum or thinking about why she needed to stand up. What made her cry was that she had never known how easily the "rest of the world" stood up from a sofa. She had just assumed, all her life, that her way of deciding to move and then moving was normal. But when she got up without even having decided to do so, simply by having the thought "maybe I should stand up," she cried in uncharacteristic self-pity, "Is this how easy it has always been for everyone else?"

even when the motor function appeared to be missing in action, the pioneers were able to plot a long, ten-week course of drug reduction through what would have otherwise seemed a pointless exercise in self-denial.

Many pioneers had tried to reduce their drugs in the past to deal with their worsening dyskinesias. Few had ever gotten past the end of the ten-day Slide. When their bodies became much worse than they expected, the pioneers had regretfully assumed that they simply could not reduce their medication: their Parkinson's had advanced too far.

By using these new hypotheses, we were able to see, time and again, that drug reduction was a possibility. Not only was it a possibility, but the cyclical nature of the patterns that emerged during drug reduction gave further support to the hypotheses.

At some point, because of conflicts with doctors and friends of patients, we needed more than just helpful hypotheses; we also had to confront the increasingly obvious fact that all of the officially proposed drug dosing theories were wrong: the blood half-life of a psychoactive drug had very little to do with the true effective period of the drug. It kept coming back to this blindingly obvious but ignored concept: the combined different drug levels in each part of the brain should determine the effective period of a psychotropic drug. Unfortunately, while our research anecdotally supports this premise, we have no hard and fast tallies of actual dopamine molecules in the brain to support our findings.

But, in the end, we have decided to use our new hypotheses as if they have been proven. We needed conclusions that could be used by our patients, regardless of what the actual physiological processes turned out to be. Therefore, while this idea is based on no published research from the old paradigm, nor measured by any machine, it is supported by the tens of thousands of hours of actual drug results reported by our pioneers, and we sponsor it as a working hypothesis.

How can we get concrete proof?

Determining an individual's drug levels mechanically, using an as yet not invented scanner, would still require a tremendous amount of math. All the parts of the brain are probably sharing dopamine back and forth in an ever-changing flow that might vary with the speed of thought. So many events would need to be considered. Just for one example of the floating nature of dopamine, let's consider sleep time, when the movement initiation centers are shut down; the dopamine hasn't disappeared. Does some of this unused dopamine drift down into the limbic area, where it gives us the peaceful feeling and ease that we should experience in sleep? Where does the dopamine go when it moves back and forth between one spot and another? Yes, it gets stored in vesicles, but how much, and in which ones? So many things to ponder! The one-plug-for-one-hole theory of brain science is passé. How can we possibly measure the quantities of these shifting sands?

Due to the complexity of brain factors, together with the absence of appropriate tools, it is understandable that brain lives or half-lives for these drugs have never been measured. Not one drug company is working on determining brain half-lives for these drugs. Because of the blood-brain barrier, which makes dopamine levels inside the brain

different from the levels in the blood, it is simply too difficult at this time to determine the actual amounts of dopamine in the brain or the dopamine half-life.[1]

However, measured or not, this hypothesis was helpful to our patients, and it made sense of otherwise inexplicable unpredictability and instability. As for the accompanying hypotheses suggested in this section regarding specific rates of dopamine retention and prioritization in the limbic, frontal, and motor areas of the brain, these have already been hashed over in chapter seven and will not be discussed further here.

And so, further supported by Sonny's charts and the charts of many others like him, we feel we can, with some degree of confidence, present again our hypotheses, further bolstered.

HYPOTHESIS 1 - THE EFFECTIVE DURATION OF DOPAMINE-ENHANCING DRUGS

Our hypothesis that the significant research number when measuring drug effectiveness of psychotropic drugs should be the amount of time the drug is effective in the *brain*, and not the blood level half-life measurement, is pure common sense. We propose that, in order to determine this number, all aspects of dopamine effectiveness, including a possible accumulation effect, must be considered. Although there is no way to measure these events at the current time, they may be extrapolated from patient data. It appears that there may be a multiplicity of factors involved in determining the lifetime of effectiveness of dopamine-enhancing drugs. The following is then, our first hypothesis.

The effective duration of all dopamine-enhancing drugs is dependent on the rate of transmission over the blood-brain barrier, the rate of breakdown and retention within the brain, and the combined take-up and detachment rates in the various brain areas.

[1] SPECT and PET scans do not show actual dopamine levels: it is suspected that they measure short-term (within a few hours after the dose) dopamine transport activity at certain dopamine receptor sites. You might say that they measure receptor responsiveness, rather than dopamine. The fact that these scans show a decrease in dopamine receptors in PDers is actually further proof that it is not just dopamine, but the entire dopamine system that is being compromised in Parkinson's. (This subject is beyond the scope of this book.) SPECT and PET scans cannot show existing dopamine quantities nor can they measure the relative changes in native DA levels that may occur after weeks or months of dopamine administration. At this point in time, a radiological study of dopamine receptor activity is not able to detect a long-term change in dopamine accumulations in the limbic area.

HYPOTHESIS 2 – DRUG ACCUMULATION

The idea that dopamine can accumulate over months may seem old to you by now, after having read about Build Ups, Daily Deficits, and drug reduction cycles. However, in the world of western medicine, this is a very new idea and constitutes our second hypothesis. We feel that the hundreds of charts of those patients in which dopamine dose effects were clearly being influenced by drugs taken on preceding days, as was demonstrated by the small sampling of Sonny's charts shown in this chapter, supports this hypothesis.

Dopamine-enhancing drugs may accumulate in the brain over the course of a day, so that the drugs of any given day have an additive effect. Dopamine in the limbic area may accumulate or decline over a ten-week period, so that a response to drugs taken (or not taken) on any given day may be affected by drugs taken (or not taken) up to ten weeks earlier.

HYPOTHESIS 3 – CHANGING ADDICTABILITY

PDers are much less addictable than average but a recovering PDer has normal susceptibility to addiction.

The bizarre cases of Zoe, Viktor, Birdie, Coach, Euclid, and Brad, all of whom you will meet over the course of this book,[1] pointed towards this unanticipated conclusion. At least for this third hypothesis, there was no existing, contradictory theory that we had to overcome: no one had even given any thought to the issue!

Our first suspicion that PDers were addiction-resistant was based on the responses our patients gave when we discussed addiction. Fairly early in our program we started warning patients that we suspected their drugs might be addictive. We were stunned by the uniformity of their response: patient after patient told us that they, alone among their friends, had never had any trouble when quitting any addictive substance. Their experiences with addictive drugs ranged from alcohol and cigarettes to cocaine and heroin, and yet none of them, to hear them tell it, had any real problem stopping the drugs.

[1] And this group includes ten others whose cases would be redundant and who have not been included due to space considerations.

A smoke screen

Maybe the best way to convey the significance of this uniformity is to share an unintentionally humorous research report that related Parkinson's disease and cigarette smoking.

A long-term, general survey of the lifestyles and habits of thousands of men showed that only half as many men with Parkinson's were smokers as would be expected, given the percentage of smokers in the general population. The naïve conclusion stated that, therefore, smoking cigarettes possibly prevented Parkinson's disease![1]

Curious about this finding, I discussed the matter with my patients. Many of my older patients told me that, back in the days when smoking was considered beneficial, they had been smokers. Some of them had even taken up the habit at their doctor's suggestion. (In the 1950's, cigarettes were considered a tonic for the nervous system and were widely recommended by physicians.)

None of these patients were still smoking. In the 1970's and 1980's, when the news was coming in that cigarettes were harmful, my "smoker" patients had simply stopped smoking. None of them had any problem quitting. When they were told that cigarettes were good, they had smoked. When they were told that cigarettes were not good, they quit. The fact that fewer people with Parkinson's smoke cigarettes may have much to do with their extreme desire to do what is correct and to avoid what is harmful, aided by their pathological level of self-control. But it may also be related to their decreased susceptibility to addiction.

The researchers in the survey had most likely confused cause and effect – a common error. Because many PDers don't smoke, they had leaped to the conclusion that smoking prevents PD. Instead, it might have been most accurate to say that people with overt or latent Parkinson's disease do not become addicted to cigarettes. Whether they choose to smoke or not may depend on the advice they have been given about health-related issues. Whether they quit or not may simply depend on whether or not they have decided, for whatever reason, to simply stop smoking. The issue of addiction, normally the big stumbling block to quitting smoking, was apparently not a problem with my PD patients.

When queried as to the difficulty of quitting cigarettes, all of my patients made some remark such as, "It wasn't a problem, I just did it," or simply repeated the ironic mantra of so many people with Parkinson's disease, "Mind over Matter."

---

[1] This delightful conclusion was actually published and received quite a bit of press.(D. Morens (University of Hawaii), *Neurology*, June, 1995.)I use this study as a teaching tool: an example of faulty logic. A study about coffee, also done by the University of Hawaii, was actually published in the *Journal of the American Medical Association*. (See A. Nehlig, "Association of coffee and caffeine intake with the risk of Parkinson's disease," *JAMA*, 2000, May 24-31; 283(20): 2647-9.) This study proposed that drinking coffee prevented Parkinson's, because most PDers did not imbibe java. I wrote to the *JAMA*, pointing out that many of my patients had enjoyed coffee at some point in their lives, but that as their internal agitation increased to the point of internal, and then external, shaking, the last thing they needed or wanted was something that would jangle their nerves even further. Considering that most of them were over the top with adrenaline, coffee, a stimulant, was not on their "must have" list. The *JAMA* wrote back and asked if they might print my response. I said yes and indicated with my initials that I was an acupuncturist, not an MD. I did not hear from them again, nor was my response published, to my knowledge.

I have since met PDers who do smoke or drink coffee; they often do it ritualistically rather than obsessively, such as relaxing into a pensive cigarette at the end of the workday. They do not need an ever-increasing amount of cigarettes over the weeks and years to sustain the mildly pleasant effect of the nicotine – one cigarette will suffice: they are not physiologically addicted; they are merely habituated.

With other patients who had overcome drinking problems or cocaine abuse, it was always the same story: "Quitting wasn't a problem," or "I just put my mind to it, decided to quit, and I was done with it."

Their dangerous corollary was that reducing Parkinson's drugs would not be a problem either. They assumed they could apply the same cool, focused will power to getting off the PD drugs as they had used on other addictive substances. Though lesser people might struggle, the PDers – with their superior focus and stick-to-it-iveness – knew they would win through.

As patient after patient expressed this haughty attitude, based on their experiences with wine, tobacco, or drugs, it was borne in on us that we might be seeing something that had never before been studied. It appeared as if most people with idiopathic Parkinson's disease (not drug-induced parkinsonism), people with unexplained low dopamine levels, were less susceptible to addiction than was the general public. There were other puzzling aspects to their relationship with dopamine and addiction, as well.

We listed the following observations:

1) Our PDers often had a history of easily overcoming otherwise addictive substances – or so they said.
2) According to their accounting, if the PDers had used addictive substances, they had not needed to use ever-increasing amounts to obtain the desired result.
3) Our PDers, *even when overmedicated*, and therefore having an excess of dopamine, as evidenced by dyskinesias, did not appear to become *quickly* addicted nor did they need to increase medication quickly, but could usually stay at any given drug level for up to six months before the drugs waned in effectiveness. Also, if they needed to stop taking the drugs for a day or two, they could usually do so without slipping into impassioned cravings.
4) We compared #3 (above) with the fact that highly addictive drugs – in most people – may decrease in effectiveness within a matter of weeks, or even days. Also, cravings for addictive substances can develop quickly, depending on the drug.
5) When people in our Parkinson's Recovery Project began to feel calmer inside and more capable of expressing emotions (as recovery from Parkinson's disease starts up, emotional changes may occur before any physical improvements begin to manifest), their medication sometimes became dramatically more effective, almost overnight. Dyskinesia rates soared. And yet, very often, within as little as 72 hours, these patients expressed a sudden, powerful desire to *increase* their medication.
6) If they continued taking medication after this point, they often had an abrupt turnaround in their goals: they often wanted to quit our program, refrain from contact with former health practitioners, and – most especially – not talk to anyone about medication. They often appeared illogical and/or became extremely defensive and/or secretive about their medications. On the other

hand, some became insanely proud of their new blissful, tireless condition, showing off for friends and neighbors, and wanting to convert us to the School of Infinite Dopamine.

7) When our patients decreased their medication in advance of experiencing symptoms indicative of recovery from Parkinson's, they experienced poorer drug coverage for their symptoms, as expected. But after completing a reduction cycle, their lowered drug doses yielded the same symptom coverage that they had before with the higher drug levels – if they were still taking more than a small, "minimal" amount. On the other hand, if they had not yet recovered and reduced their drugs to a level below the minimum needed to mask Parkinson's, their PD symptoms would be exposed and remain exposed even at the end of a reduction cycle. This minimum level is detailed in chapter 17 (The last little bit). Whether or not their PD symptoms improved back up to the prior (pre-reduction) level at the end of a cycle seemed to depend on whether or not they were taking medications higher than a minimum level. This minimum was much lower than the manufacturers' recommended dosages.
8) Those few patients who did try to reduce medication after exhibiting, for more than 72 hours, symptoms consistent with recovery plus overmedication usually underwent the extreme rigors of classic drug withdrawal. These rigors might include nausea, extremes of insomnia, paranoia, violence, hallucinations, and life-threatening heart or diaphragm arrhythmias. These violent symptoms often lasted ten weeks before they showed any sign of fading. Subsequent drug reductions, if not undertaken immediately after the withdrawal began to ebb, might also repeat this pattern.

Based on these observations, we made the hypothesis and corollary that people with Parkinson's disease are less susceptible to addiction than the general population and people who recover from PD will be more susceptible to addiction than they were before.

We had to look long and hard at the most curious piece of information – item number 3, above. The people referred to in this section had dopamine at levels that were causing their bodies to go into spasm. They clearly had more dopamine than was necessary. They were over the Safety Limit. And yet, they did not behave as if they were particularly addicted. They could often stay at a high, dyskinetic medication level for a long time – some of my patients had been ticcing and thrashing about at the same drug levels for more than a year – without needing an increase. Although overmedicated PDers do eventually have a lowering of their baseline dopamine in response to excessive drugs, it seems that this happens so much more slowly than addiction in healthy people that it can be considered negligible in the short term.

So, despite the most recent research coming from the drug addiction community, which pointed to excess dopamine being the crucial factor in drug addiction, we were seeing that there was something beyond merely the dopamine that was involved.

Also, although those people in situation number 3 above were clearly overmedicated, according to the obvious dyskinesias, if they tried to reduce their medication *before* they started having signs of recovery, they did not undergo drug withdrawal symptoms, but merely suffered from the usual signs of drug decrease: an

extreme decrease in the masking of their Parkinson's disease symptoms which corrected within ten weeks. These decreases did not leave people shell-shocked or traumatized. Following a drug decrease, this group would be merely miserable, possibly immobile, and often in severe pain, but usually after about ten weeks, depending on whether or not their drug levels had been over the minimum, they were having their prior level of movement again, with fewer adverse effects from the drugs.

Many people in the program did begin to reduce their medication prior to seeing any hint of recovery; they initiated the reductions when they learned that their adverse effects were, most likely, coming from their drugs and not from their PD. Many people found that, regardless of recovery issues, they eventually felt much better with much less medication. As described earlier, some of them had tried reducing their drugs in the past, but, because they did not understand the ten-week time frame for brain readjustment, they had assumed after two to ten days at a lower level that they had been wrong to reduce. Armed with more information about how addictive drugs work, and the time frames involved, many patients tried anew to reduce their drugs after entering our program, with beneficial results. And – despite being clearly over the top with dopamine – they did not experience the traumas of drug withdrawal (addiction) if they were not yet manifesting *any*, however subtle, symptoms of recovery.

A person with Parkinson's disease has reduced susceptibility to addiction.

Upon beginning to recover from Parkinson's disease, he will immediately be just as susceptible to addiction as any healthy person.

Medication that was merely somewhat addictive during the PD condition may become rapidly and irreversibly addictive in even very small amounts after recovery begins.

HYPOTHESIS 4 – A SEE-SAW RELATIONSHIP BETWEEN ADRENALINE AND DOPAMINE

Addiction is more likely when the body is in a parasympathetic condition than when in a sympathetic condition.

We created a new hypothesis to explain the normal = addictable versus PD = addiction-resistant hypothesis already stated.

Hypothesis 4 is necessary to explain PDers being overmedicated, oversaturated with dopamine, but not being addicted. If dopamine alone were the sole cause of addiction, as currently hypothesized by the National Institute on Drug Abuse, then these overmedicated patients should have been horribly addicted – but they were not.

This idea has immediate bearing on medication issues. However, in understanding the overall Parkinson's picture, this hypothesis is even more crucial – it is connected to

our realization that Parkinson's disease occurs when the body is locked into a sympathetic system and that recovery begins when the parasympathetic system is reinstated.

The myriad symptoms of Parkinson's, including the decrease in dopamine producing cells over the decades, are merely side effects of this injury-induced sympathetic condition. The fixation that some recovering patients in our program have with finding all their past injuries and blockages that might have contributed to the erratic electrical flow in Parkinson's is, in fact, off the mark. The key factor is returning to a state of relaxation after a lifetime of wariness. Very often, the treatment we provide, which supports the injured areas, allows a PDer to have the emotional recognition of injury, after which he can defuse the sympathetic system. The treatment of the injury and the shutting down of the emergency often go hand in hand. But actually, it was the fearful sense of emergency rather than the injury per se that prevented the injury from healing normally at the time of occurrence.

This idea is the core of our work, and it is supported, in part, by what we saw while working with drugged patients.

Adrenaline vs. dopamine

We propose that a person who is in a condition of sympathetic nervous stimulation will have a body-wide increase in adrenergic function and a corresponding decrease in all dopaminergic functions. This decrease in dopaminergic function applies to the production, transport, reception, and breakdown of dopamine, as well as the addiction response to dopamine. During the sympathetic condition, all brain processes, including motor function, alertness, emotion, and memory, switch from a dopamine-based system to an adrenergic system. While under the influence of the adrenergic system, all dopamine-based systems, including dopamine-induced movement, thoughts, and the addiction response to dopamine are greatly reduced.

Dopamine has helpers

We suspect that the actual molecule of dopamine does not perform all of the neurotransmission that occurs in the dopamine-dominant state. An entire cascade of neurotransmitters can be called into play by either the adrenaline or the dopamine team captains. However, for our purposes, we are going to focus on the two chief NTs of the systems rather than on the torrents of subordinate neurotransmitters. It may be that even the hormonal systems, long considered to be glandular, and therefore falsely thought to be separate from neurotransmitter function, are regulated by these two systems. This would explain why the hormone systems do not function normally in times of high stress; maybe most of the hormones are on the dopamine team.

Dopamine: good for what ails ya

Although most people in western society who have been diagnosed with Parkinson's are directed to focus on the amount of dopamine that they can force onto a brain that is trying to not use dopamine, we are guessing that their real problem lies with every aspect of the dopamine system being turned off, not just the quantity of dopamine available. For those who would counter with the specious argument that declining dopamine levels alone make the Parkinson's apparent, based on the glorious "reversal" of

symptoms when a PDer takes levodopa, I would propose that levodopa at those high levels would make just about anyone feel better, move better, and even dance better, whether they were suffering from chicken pox, a broken leg, or Parkinson's disease.[1]

It is not just a recent dopamine deficiency that is causing Parkinson's disease; most PDers haven't used dopamine in decades and wouldn't know what native dopamine felt like if it bit them in the ankle. The reason that Parkinson's becomes apparent is that the adrenal system, due to a shock, illness, surgery, or exhaustion, begins to be inadequate.

It is the worsening insufficiency of this dynamo, the adrenal system, also known as the sympathetic system, that finally allows the damage from years of irregular electrical patterns – *which includes long-term dormancy in the dopamine-producing cells* – to finally be exposed. The problems of damaged muscle tissue, the disconnected sensory nerves, and the sleeping dopamine branch of motor command centers in the brain have been snowballing for years, concealed in the blizzard of adrenaline. By being in a state of emergency, the PDer has been able to ignore these problems and override them.

PDers can move in an emergency

It is well known that, during the first decade or so after a diagnosis of Parkinson's, an otherwise immobile person can still, when confronted with an extreme emergency, move perfectly normally. In other words, if the wearied adrenal system can be encouraged to rev itself up for a short time, a person can feel exactly like his or her old, dynamic (sub-clinical Parkinson's) self again. As soon as the emergency diminishes, however, the adrenal system slumps back down, exhausted after so many decades of working overtime. Once the emergency is over and the sympathetic system subsides again to an inadequate level, the underlying decay in the body from backwards flowing electrical circuits, the rigidity, poor balance, and inner restlessness/fear are all once again apparent. This decay, falsely attributed to dopamine decrease, is actually the normal consequence of injuries that have failed to heal. The body does not do healing work while in the sympathetic state.

Two separate sets of neurons

Much study has been done on the sympathetic system, but the parasympathetic, falling under the large heading of "Normal," or "default system," has been relatively unstudied. It has been known for nearly a hundred years that adrenaline (epinephrine in England) is the neurotransmitter for emergency, regulating all emergency systems, ranging from the heart, bladder, lungs and blood vessels to the very way in which we

---

[1] Due to deeply depressed dopamine levels and having an autonomic nervous system locked into a permanent, even though depleted, extreme sympathetic mode, people recently diagnosed with idiopathic Parkinson's often have slow response to the antiparkinson's drugs, requiring several weeks or even months on low level antiparkinson's therapy before they begin to notice the result. Many of my classic, idiopathic Parkinson's patients say that they took L-dopa for only a month and quit because they didn't notice much of an effect.

On the other hand, quick, buoyant results from antiparkinson's drugs can sometimes signify misdiagnosis. Recent research (see footnote, p. 159) shows that misdiagnosed patients often notice a dramatic, quick effect, and rapidly begin manifesting addictive behaviors, in addition to feeling just *great* from the medication no matter what their particular problem was. This is particularly ironic because, starting in the 1990's, undereducated MDs began using rapid response to L-dopa as a positive indication of idiopathic Parkinson's.

think and process information. Adrenaline causes a shift in the brain's speed and method of learning.

We have seen events during recoveries from Parkinson's that have directed us to the following new idea:

The neurons (nerves inside the brain) that stimulate muscles, regulate coordination, and integrate left and right brain sides respond so differently to adrenaline than they do to dopamine that there may be *an entirely different set of neurons* used during emergencies. A person may have two distinct sets of physical skills, thinking patterns, perceptions of time, and personalities: one set is run by adrenaline, the other by dopamine. The addiction process is a part of the dopamine-regulated system. The addiction process is dormant, or somewhat modified, when the adrenaline system is dominant.

The existence of two separate systems would explain why, during recovery from Parkinson's, our patients sometimes must relearn coordination for activities that they had mastered during their years on adrenaline, and which they could perform up until they started to recover. Very often, it is as if they have never done the activity before. These activities can range from typing and tying their shoes to reading poetry.

Very often, a recovering PDer feels as if the brain has no way to remember ever having done these activities.[1] This can be combined with sudden bursts of long-forgotten movements that bring to mind memories of childhood: "I just ran my violin bow over the strings and the music came out in the effortless way it did when I was a youth. It wasn't just the physical motion that was different; it was as if the source of the motion, the freed emotion behind it, the fluidity from deep inside – it was all there, all at once. I can remember that that was the way I played the violin long ago. I don't know when I stopped doing it that way. I feel as if I've been trying to recreate that feeling in the bow ever since I was about twenty years old. Suddenly, yesterday, it all came flooding back!"

For activities that were learned subsequent to the injuries that cemented the emergency condition, it can feel as if one is doing the activities for the first time. This could be explained by the existence of two distinct sets of brain responses, which may be only partially integrated.

Just as we now accept that adrenaline runs an entire galaxy of symptoms and commands a phalanx of neurotransmitters, it may be that dopamine runs a separate, parallel, physiological galaxy, equally armed.

This idea is actually more significant to biology in general than any of the other hypotheses. These ideas came to us while observing what happens during recovery from Parkinson's. It was the dramatic brain shift in recovering patients that brought us to this point. Before I can write another word, I take a moment to once again bow to those patients, both medicated and unmedicated, whose brave experiments have brought us to this new understanding.[2]

---

[1] See: "panic attacks" in *Recovery from Parkinson's Disease, A Patient's Handbook*, J. Walton-Hadlock. Free at www.pdrecovery.org.

[2] While this hypothesis does not give particular help to a person who is trying to make sense of his medications, the biologist in me is equally fascinated by this new way of viewing the parasympathetic system, a system I first learned about more than thirty five years ago. If I can make a contribution to our understanding of this system, I dedicate this contribution to my beloved biology professors, especially Mr. Campbell and Miss MacDonald (Santa Monica High School), and my mentor, the late Dr. Kenneth V. Thimann, professor emeritus, University of California, Santa Cruz.

Quick transitions

Another proposal, one that fits in with the above hypothesis, is that the body is capable of moving very quickly between the sympathetic-dominant and parasympathetic-dominant states. The deeper, emotion-based changes that we saw in our patients when they began to recover often occurred within a matter of hours after a breakthrough treatment. The ensuing drug addiction, if they were medicated, might be full blown within three days. Evidently, the switch that allows the body to go back and forth between heightened alertness and normal, between addiction-resistant and addictable, is a quick one.

A graduated scale, rather than an On-Off switch

Although, to make the point, I presented the adrenal versus dopamine systems as one or the other, all-or-nothing, it is more likely that the toggle between sympathetic and parasympathetic is not so much a one-or-the-other switch, but a graduated line.[1] Both systems are ordinarily somewhat active at the same time. When one system predominates, the other decreases. This gradual back and forth, with one system or the other dominant, but both systems active to some extent, allows for healthy people to experience appropriate levels of responses in times of ease and times of concern.

When the adrenaline system is dominant, the dopamine functions are turned down to a very low setting, and vice versa.

Sick people, however, such as those with Parkinson's, those who are in extreme pain, and those who are in a state of constant, unending emergency, may find certain aspects of their dopamine system are turned so low that they are, for all intents and purposes, turned off. This would explain why people in these conditions have almost no addiction response. However, the dopamine system is obviously able to spring back into effect in its entirety as soon as the emergency is removed – even after years of "emergency."

Interestingly, it appears that, in such a case, when dopamine resumes after an unhealthily long period of adrenal hyper vigilance, the adrenal system will give itself a nap and not be rousable by any but the most dire of emergencies. This condition exists in recovering PDers who find that nothing short of calamity can stimulate them into a condition of even mild concern anymore.

---

[1] This idea is developed further in a lengthy footnote in chapter 24.

Further support for our hypothesis

Our hypothesis is helped along by two findings: a change in social status in a primate/addiction study caused a change in both dopamine receptor activity and addictability, and people in extreme pain are less addictable. (See Appendix 7.)

Alpha primates

The sympathetic system in male primates who become alphas may increase due to heightened wariness against challengers and natural enemies. This finding appears to be related to what happens in PDers. (See Appendix 7.)

Pain

People who are in extreme pain, such as post-surgery, may not need regularly to increase their dose of otherwise addictive pain meds in the usual manner of addiction. When their pain later subsides, they can usually stop or decrease their drugs without cravings or drug withdrawal if they stop their drugs in a timely manner. The extreme pain may have triggered the sympathetic system, turning off the addictable parasympathetic.

More on monkeys

Research using cocaine (see appendix 7) has shown that previously addictable primates are no longer addictable after becoming an alpha (leader of the group). What else changes when a primate becomes recognized as the alpha male of his group? A primate that becomes an alpha male takes on responsibility for the safety of the entire clan. He must be awake when others sleep.

He also faces challenges from within his community. He needs not just superior strength but superior cunning and the ability to psychologically keep his potential challengers at bay. He must maintain a heightened sense of alertness. He must be forever watching over his shoulder and may never let his guard down. A single slip up and an entire pack of challengers might be at his throat.

To perform his duties and maintain his position, he must sustain heightened vigilance. He may not slip into a parasympathetic mode – in such a relaxed condition he would soon change species: from an alpha chimp to a sitting duck.

PDers as alphas

Some PDers manifest their extraordinary ability for self-control by being supremely submissive. Some use their unnatural levels of self-control to subtly manipulate or even dominate others. However they choose to use their enhanced state of alertness, intelligence, and speed – all conditions brought about because they are in a state of injury – they are still in a "sympathetic" condition, biologically speaking.

While the sympathetic system is often understood to be a condition of emergency, and is oversimplified for beginning biology students as a condition of "fight or flight," the condition is more complex than mere running or attacking: enhanced mental and diminished emotional/contemplative capability also accompany activation of the sympathetic nervous system.

Enhanced emotional and physical wariness and a tendency to be "stronger, faster, and smarter" than normal people are common attributes of the Parkinson's personality. They also neatly describe the behavior of an alpha primate.

More on pain

In looking around for any sort of comparison with the non-PD community, we have considered the non-addictiveness of people who are in high levels of pain, but who, when the pain is over, return to a perfectly normal level of addictability. Stories are common about those people who abuse pain medication after they are released from hospital and end up becoming addicted: after their pain has receded – if they are still using pain meds – they need ever-increasing doses and their lives revolve around their drug addiction.

However, it is also recognized that some people who are in extreme pain can tolerate extremely high levels of dopamine-enhancing drugs without becoming addled by them, yearning for them, or needing ever-increasing doses. The difference seems to be that people who are in a state of excruciating pain (pain causing a sympathetic system response which includes an extreme dopamine deficiency and/or dopamine override in the limbic area) are not addictable. But I repeat, as soon as the emergency-level pain subsides and becomes background level pain, they are again addictable.

The image of fight or flight

Most people have been taught that adrenaline is the "fight or flight" chemical. The image of a charging rhino is often used to demonstrate what triggers the sympathetic response. But extreme pain, or the perilous potentialities of being an alpha male, can also set the sympathetic system in action. We propose that certain emotional or physiological terrors, and the subsequent perpetual wariness, can also perform this function. This is the most common sympathetic system trigger for PDers. After the sympathetic system is elevated, dopamine levels – and the entire dopamine system, including healing functions and addictability – drop very low.

Why medicated PDers should not attempt recovery

Recovering PDers have almost no sympathetic nervous system. They appear to drop to an almost pathological parasympathetic mode during recovery, during which they virtually cannot be stimulated into an emergency state.[1] We have to wonder if a commonality in recovering PDers and ex-emergency patients (those who have emerged from their crisis) is an overswing into an extreme parasympathetic mode after having been in an extreme sympathetic mode. This change, not dopamine levels per se, may be the reason for ex-PDers and some previously pained patients switching over to an extraordinarily high level of addictability from a previous condition of non- or minimal-addictability.

For purposes of healing PDers, we usually focus on their unhealed injury or, often, multiple injuries. It is the chaotic electrical pattern in the vicinity of their injury that eventually creates the various symptoms of Parkinson's disease. However, the real

[1] Please read J. Walton-Hadlock, *Recovering from Parkinson's Disease: A Patient's Handbook*, available for free at www.pdrecovery.org.

problem has never been the injury. The deeper level problem is that, because the person was in a state of (very often emotional) emergency at the time the injury occurred, the injury did not commence healing. *When a person is in a predominantly sympathetic state*, whether he is conscious of emergency or not, whether using his adrenaline and altered awareness to appear either utterly meek to the point of invisibility or completely ferocious, he cannot heal from injury; nor, we propose, can he become addicted.

What actually allows the healing of Parkinson's to occur is the singular method that we use for healing: we hold the patient's foot. We, the health practitioner, assume a posture towards the patient that has signified, through time immemorial, that the person whose foot is being held can let down his guard and be at peace. The emotional implications of allowing one's foot to be supported, of letting a foot be held peacefully by a neutral fellow human in a safe setting, may provide a signal, long-denied, that the emergency is over. (Fully developing this idea, that the PDer was in a state of emergency, is beyond the scope of this book.[1])

However, if recovery will cause a PDer to revert to the parasympathetic state, the risk of taking antiparkinson's drugs – some of the most addictive drugs known to man – while trying to recover, is apparent.

One of the gravest problems with medicated people trying to recover from Parkinson's is that it may take years for the body to return to full strength. The lingering weakness and slow muscle retraining make it extremely difficult for a PDer to relinquish his medications. And yet, we have seen over and over that, as soon as the person turns off his protective, sympathetic stance so that the electrical disarray can be reconfigured, he is at great risk of becoming addicted to his medications. It has been known since 1969 that these drugs are neither safe nor stable at any level in a non-PDer. You begin to see where the danger lurks in treating a medicated patient.

A patient's extreme level of immobility due to the muscle damage incurred during the decades of Parkinson's may take years to repair. Massive doses of dopamine, such as those used in treating Parkinson's, can provide an emotional high that allows movement despite his technical immobility.

As an example of "being able to move even though technically immobile," let me use a person with a broken leg who, under normal circumstances, is completely unable to support weight with that broken leg. Given enough cocaine or methamphetamine, this person may move about quite happily. He may be shredding his leg inside, further damaging the injured bone and doing himself a great mischief by dancing about with a broken leg, but he can do it, nevertheless.

The dopamine-enhancing drugs impart movement to a PDer in very much the same way. They give the possibility of mobility and the illusion of health in a person who, physiologically, would be better off immobile.

The temptation to continue using the drugs even though the dopamine system has switched back on is so alluring that most people succumb. Their previous years of "iron will" are of no use to them now. Their will power was a part of their training under their sympathetic system, a system which shuts down during recovery. If these drugs are taken when the dopamine system has turned back on, rapid addiction and all the side effects of

[1] Please read J. Walton-Hadlock, *Recovering from Parkinson's Disease: A Practitioner's Handbook*, available for free at www.pdrecovery.org.

addiction – pain, delirium, and permanent brain damage – including parkinsonism – can ensue.

Because of the irreversibility of the brain damage that might occur when a person is, however briefly, addicted, we do not recommend recovery from Parkinson's disease for any person who is taking antiparkinson's medications.

Summary of hypothesis four

The sympathetic system is governed by adrenaline ***and the parasympathetic is regulated by dopamine.***

Addiction is a parasympathetic process.

Resistance to addiction occurs during a heightened sympathetic response.

When in a parasympathetic state, addiction will occur in response to excess dopamine.

Summary

1. The effective duration of all dopamine-enhancing drugs is dependent on the rate of transmission over the blood-brain barrier, the rate of breakdown and retention within the brain, and the combined take-up and detachment rates in the various brain areas.

2. Dopamine-enhancing drugs may accumulate in the brain over the course of a day, so that the drugs of any given day have an additive effect. They may also accumulate or decline over a ten-week period, so that a response to drugs taken or not taken on any given day may be affected by drugs taken or not taken up to ten weeks earlier.

3. Most PDers are much less addictable than average but a recovering PDer may rapidly return to a normal susceptibility to addiction. Medication that was merely somewhat addictive during the PD condition may become rapidly and irreversibly addictive in even very small amounts immediately after recovery begins.

4. The sympathetic system is governed by adrenaline and the parasympathetic is regulated by dopamine. Addiction is a parasympathetic process. Resistance to addiction occurs during a heightened sympathetic response. When in a parasympathetic state, addiction will occur in response to excess dopamine.

"O Captain! my Captain! our fearful trip is done; The ship has weathered every rack, the prize we sought is won;

But O heart! heart! heart! O the bleeding drops of red, Where on the deck my Captain lies..."

"O Captain! My Captain!," Walt Whitman

19. A TWO-YEAR STRUGGLE

A STUDY IN FIRST SLOW, THEN FAST REDUCTION

Coach and levodopa

Coach was mentioned briefly in chapter 12 because he nearly died when he reduced his drugs too quickly. Here is a little more detail on that case study. These details help support our hypothesis that the drugs are more addictive if recovery symptoms have appeared.

Coach was 75 years old when I met him. He had been diagnosed six years earlier during his exam for angina (heart pain). His wife was a doctor and had preferred that he take as little levodopa as possible. That's why he was still taking his prescription of 400 mg levodopa per day (two 50/200 Sinemet CR) six years later.

When he started levodopa, it helped him walk more easily, but within six months that benefit went away, and neither he nor his wife wanted to make a further increase of these dangerous drugs since the benefit seemed to ebb so quickly.

When I started working with him, he moved stiffly, his weak voice was a monotone, he had no facial expression, his arms were bent at the elbows and his posture had the classic stoop. He had a slight tremor in his right hand, excess saliva once in a while, and cold hands and feet.

Coach was a wonderful patient. He had studied massage and Process Acupressure, and he was keenly aware of his own repressed emotions and his responsibility in letting go of them. His symptoms responded quickly to treatment. Four months after we started working together, his charts show the following changes: longer stride, head more jaunty, speech is more fluent, more facial expression, and in his words, "My hands are warm and my voice is back! It's deep and resonant." The only remaining problem was the weakness in his hands causing lack of dexterity: handwriting and using silverware were only slightly improved.

So far, so good

He had decreased his medication by half over the four months that we'd been working together, and although each small, evenly spaced decrease caused him a noticeable drop in energy, he dealt with it by taking long naps.[1]

[1] He started with two pills per day. He made a decrease of half a pill two times per week, making a new reduction each week. Example: 1st and 2nd week, reduce by half a pill on Monday and Thursday (only 1.5 pills on these days, 2.0 pills on all other days). 3rd and 4th week, decrease by half a pill on Tuesday and

After each dose decrease, he felt weaker for a while and needed much, much more sleep, but he never experienced anything approaching what you could describe as drug withdrawal symptoms. As soon as he felt his energy resuming even in the slightest amount, he would reduce again.

In this way, he was down from two pills a day to one pill a day within four months, without ever suffering from drug withdrawal symptoms. He was doing beautifully!

After another month, he reported having days of "torpor," but that despite the extreme lassitude, he noticed that he could rotate his head for the first time in years.

After seven months, he was having difficulty sleeping due to frequent urination and restlessness, and although his stride was returning to normal length, he only had enough vigor to walk the dog once around the block before he wanted to collapse. He continued to reduce his medication, and at month seven reported that he was down to half a pill per day, a mere 100 mg of levodopa – an amount considered to be not worth mentioning, according to the literature. He was increasingly flexible and fluid, but utterly limp and fatigued.

Ominous words

He also reported a curious new symptom that should have caused my ears to prick up and my blood to run cold, but I was still unaware of the addiction danger.

"I'm starting to have moments lasting 5 to 10 minutes, about every third day, when good energy and balance seem to well up within me, emerging naturally from inside of me." While this sort of statement fills me with gratitude when I hear it from an unmedicated patient, I have now learned that, in a patient who is still taking the drugs, this type of "natural feeling of energy welling up from within" is a harbinger of doom.

Over the next month, he noticed that the deep fatigue was, at times, overwhelming. Sometimes he would take a whole pill rather than half a pill but it didn't help. He noticed that if he forgot to take his pill, he did not feel any more draggy than if he did take it. His body's indifference to the drugs showed that his main problems were now due to recovery, not Parkinson's, and therefore were not helped especially by these small amounts of L-dopa.

This also should have set alarms ringing in my head, but it did not. What I might say now, in light of experience, is that, since the fatigue did not seem to be influenced by the medication, the fatigue was coming from the recovery process. This deep fatigue is felt by medicated and unmedicated patients alike, right around the time when they start having more use of their limbs and a return of circulation and sensitivity. Most often, the medication is useless to combat this deep fatigue. The deep fatigue is not caused by a lack of dopamine; it is the result of a body finally shutting off the sympathetic system and initiating tissue healing on a grand scale.

This is NOT a dopamine deficiency problem and therefore is not remedied by dopamine. In fact, very often, the deep fatigue may be accompanied by indications that dopamine levels are rising. It is returning health, not dopamine deficiency, that is causing

---

Friday, as well as Monday and Thursday. 5th and 6th week, reduce by half a pill on Wednesday and Saturday, plus previous reductions. 7th and 8th week, every day of the week he only had 1.5 pills per day. He repeated the process the third and fourth month; at the end of the fourth month he was taking only one pill a day.

the recovery fatigue. At this point, one is sleeping because it is the best possible thing for the recovering body.

Therefore, shoving extra dopamine into a system that has dopamine, or that has no use for it because it intends to nap, is a waste of dopamine pills. Worse, because the dopamine will accumulate in the limbic system whether it is needed or not, this extra dopamine will contribute to addiction. In other words, when the pills don't seem to be doing much good because a person is recovering and no longer needs the pills, the pills make the switch from benign to dangerous.

Coach sometimes felt a lift, albeit a mild one, from his medication. Whereas before he had not noticed abrupt changes when the drugs started to work or when they wore off, he started feeling an abrupt drop off in energy, now and then, in the afternoon. Curiously, if he forgot his pill, he did not feel draggy at all during the day. It was not so much that the pills prevented dragginess, but if he did take a pill, he felt a heaviness when the pill wore off.

At nine months into the program, he got the flu. This hit him very hard; he had to start wearing diapers in bed because he was too weak to control his urination. He increased his medication to one and a half pills a day.

A month later, he was feeling better in general. He was no longer waking up at night to urinate, and two nights in a row he had slept 9 hours. His movement tempo depended very much on the quality of sleep the night before. He was able to roll over in bed finally instead of slowly inching over or asking for help, and he was dressing himself more quickly. If sleep was poor, however, he felt draggy all day and didn't want to exercise, which caused him to feel even more draggy. Whether or not the improvement was due to the resumption of the higher dose of medication was unclear, but he felt he needed the higher dose.

At the end of this month, he said, "Had the best week I've had since I've been in the program. Suddenly, there's 2 or 3 times more body energy than I've been having. It's still cyclic, and sometimes the bottom falls out, but overall, I'm getting better!"

Missed cues

Two months later, almost exactly a year after we started working together, there was trouble. He reported, "My center of gravity is lower; I move youthfully, from the hips, not the waist. No more accidents at night, no more constipation. All movements are faster, more agile. Something has changed. The restlessness is gone, sleep is so deep, I'm no longer right on the edge of the abyss of weakness. This gives me more energy for coordination. My voice is wonderful and rich. I can breathe more deeply down into the gut. The tremors are greatly reduced: I'm quieter inside, there's a much deeper stillness. Everything is better than it's been in years!"

He bounded across my office to show me how well he was moving, and then he did something so uncharacteristic that his wife squeaked. He stuck his arms out to the left side at shoulder level and started wiggling his hips. He fluttered his arms to the right and tried out some side steps. He winked at her and said, "Have you ever seen me do the hula?" Evidently, based on her "Oh mein Gott!" she had not. We all laughed and hugged. These good feelings continued to improve over the next three weeks.

In retrospect, all of the above was good, except for one thing: he did not make a corresponding reduction in his medication.

Three weeks after reporting that "Something has changed!" he started having dyskinesia. Both arms started shaking and flapping, and the insomnia returned, but it was different somehow. He recalled the Robert Frost poem, "Acquainted with the Night"[1] as a way to describe what he was experiencing. He reduced his medication by half a pill a day, so that he was only taking one pill per day (half pill doses twice a day).

The next week was his worst week ever. He reported, "I really look like I have Parkinson's. Can't sleep at night, need help to move." He considered increasing his drugs back up, but decided to try another week with just one pill per day.

Heart pain

The following week his old angina (heart pain) problem resurfaced – with a new twist. He noticed that 45 minutes after taking either dose, he had severe pains in his heart, followed by dyskinetic shaking in his right arm and paralysis and pain shooting down his left arm. It was very painful and very frightening. When he took his nitroglycerin medication, the heart pain would ease up. At the same time, he felt better than ever. In his words, "I am very happy inside. I feel as if I've passed a milestone. Something is different."

He did not associate the "different" feeling with the appearance of dyskinesia in his arm and heart. He only knew that he was much more gleeful and that the heart problem was more frightening than ever. Considering that he had always been very analytical prior to this time, and a body worker to boot, his new inability to form a connection between the strange happiness and the dyskinesia can be considered solid proof, in retrospect, that he was mentally discombobulated by the drugs. Prior to this time, he had been keenly aware of his body and the mind/body/attitude relationship. Now, suddenly, he could not make the connection, and as soon as each heart pain episode passed, he didn't give it another moment's thought.

However, his caregiver was concerned enough that they decided to reduce his medication.[2] I had never met the caregiver, but she started calling me twice a day, each time the severe heart pain started up. I told her that heart arrhythmias and angina could be adverse effects from the medication. She decided to reduce his medication. He took only half a pill a day for two days. This decrease in medication from one pill a day to half a pill may have contributed to his extreme sleepiness and weakness during the next week.

However, he still had increasingly severe heart pain 45 minutes after each dose. The caregiver shifted the pill time to afternoon, thinking that he was usually more tired after lunch, and the boost from the pill would not be so detrimental if his natural energy was depleted. However, no matter when the pill was taken, he would have heart pain, complete with arm pain, paralysis, and a feeling of impending doom, within 45 minutes of taking the pill. They cut the pills into quarters, rather than half, but the reaction was the same. Coach had become "sensitized" to levodopa. It appeared as if his body had learned

---

[1] "I have been acquainted with the night. I have walked out in rain – and back in rain. I have outwalked the furthest city light…"

[2] There was a caregiver in the house because his wife had to leave for Europe to help her dying father. There were two high school aged children in the home, from the wife's first marriage. The caregiver, a live-in, was hired to help run the house, do the driving, and help Coach with bedtime weakness, if any. She became more deeply involved in caring for Coach than anyone had planned, as you will see.

to get rid of the levodopa by instituting powerful dyskinesia in his right arm and his heart, and it would do so no matter how small the amount of medication.

Cold turkey

By the end of the week, he stopped taking the pills altogether, and he felt much more in control of his body. We weren't sure at the time if it was the relief of not needing to worry about the heart pain or whether it was recovery from PD. Most likely it was a drug vacation occurring, but for whatever reason, after two days without any pills, Coach reported that he was sleeping wonderfully, and that he felt high and energetic all day.

I was deeply concerned about his abrupt decrease in medication. He had gone from one and a half pills of 50/200 Sinemet per day (300 mg of levodopa) down to none, over the course of three weeks. The first reduction was just starting to appear, in the form of the increased sleepiness. As for the rest of the reduction, we simply had to sit back and wait to find out what was in store. Taking levodopa was no longer an option; his heart could not handle it.

Coach's wife (his preferred doctor) was still in Europe, attending her father on his deathbed. Coach did not want to work with his neurologist, a doctor who liked patients to find their own comfort level with the drugs and dose accordingly.

I knew from past experience with Becky that a decrease from 400 mg/day to none – in a patient who no longer had PD – could bring on terrors and hallucinations. I had also seen patients who still had Parkinson's who had decreased from the 200 mg/day level down to nothing and survived it quite easily, fatigue, pain, and insomnia notwithstanding. My big question was whether or not it mattered that Coach had been showing signs of recovery for nearly a year. It mattered.

Fighting the Germans

The night terrors began the next week. They first took the form of a repeating hallucination. Coach was in the trenches of the Alsace-Lorraine in 1944. He and his fellow soldiers were under fire from the Germans. Coach's best buddy was hit and was dying in Coach's arms. All night long Coach would scream out for the commander to bring an assist or scream for someone to help him staunch the spurting blood.

Coach's caregiver, and then his wife, recently returned from her father's death, would stand by the bedside, trying to talk Coach down. "You are seventy six years old, you live in California, you are home in bed, I am your wife, my name is Heidi, hold my hand, we're right here," was the sort of soothing chant that they would repeat for hours at a time. Coach never heard a word they said. He would stare at them, eyes wide open in horror, shaking in primal fear, holding onto his dying chum and screaming at them to "Help! Do something! Oh God, please, please help!"

The hallucinations became nearly constant and lasted for over three weeks. He was in a war zone, oblivious to the other reality around him, living in a hellish world of shrapnel, blood, air attacks, and panicked retreats.

They tried giving him sleeping pills with no result. Through the fog of his hallucinations, he would need help getting to the bathroom every two hours, around the clock. His right arm shook violently, shaking the rest of his body like a cat shakes a rat, especially during his times of greatest fears. His wife found that the only non-dopamine

altering medication that appeared to ease the terrors and allow him briefly to sleep was marijuana. She baked it into cookies and fed it to him, a bite at a time.

Post-traumatic stress

After three weeks he began to come out of his darkest confusion. He still had night terrors, but during the day he seemed to know who he was and where. However, he was no longer the same person. It was as if he truly had experienced those three weeks of horror. Whether or not his body had been in the Alsace or in bed, his mind had been shattered by the monstrous, unending experiences of the previous three weeks of torture.

He had no interest in feeding himself, and no ability to dress himself or even move. He was morose, hypersensitive to every sound, and for over two months his chief remark to me was, "The kids are so noisy. They don't understand. I can't tolerate the noise."

They lived away from town, out in the country, and I knew that the kids were moving around on tiptoe, in a house that had good separation between the kids' rooms and his, but he was fixated on how the sounds from the kids were like torture.

He would be brought to my office every week, but I was uncertain what to do for him, or even how to begin. It was as if, aside from the whispered complaints about the noise, he was not actually present, but was floating around in some horrible dream world that no one could behold except himself.

Poetry from the past

Staring at him lying there on my office treatment table, acupuncture needles for drug withdrawal in his ears and forehead, I clobbered my brain looking for some way to get through. I suddenly remembered that he had been a professor of English literature and poetry. I ran into the next room and grabbed a book of American verse. I turned to Walt Whitman and started very softly to read: "O Captain! My Captain! Our fearful trip is done…"[1]

As I read the measured words, slowly and as rhythmically as possible, a smile spread over his face. He turned his head towards me and looked at me, as if seeing me for the first time. He mouthed the words to that famous poem as I said them aloud. It tired him out and he fell asleep, but the poetry had plainly evoked some pleasant memory from the past, successfully transcending the war pains of the moment.

At the end of our hour, I suggested that his wife read some of his favorite poetry every day. She was flummoxed by the request. She had no idea what poetry he liked, and she was not familiar with the American writers. Her beautiful accent and her excellent European education had not prepared her for this new assignment – selecting and reading 19th century American poetry in a voice that could most closely replicate the singsong delivery popular in his youth.

His caregiver took over the job of reading to him now and then, but she didn't know the old poems. I remembered how my father had "sung" these same poems to me. I knew just how a person Coach's age would have recited them in the lilting rhythms of my father's generation. So, for the next few weeks during Coach's visit, I would put in his

[1] "O Captain! My Captain," Walt Whitman.

needles and sit back with a good book of the old poems and read out loud to him. The many hats of the doctor!

During the next few months, his wife made the reports. Sometimes Coach could contribute a whispered appraisal of his condition, but very often he appeared to be in another world. His wife's comments over these next months were encouraging: the nightmares would not begin until after he had gotten four hours of good sleep, and although he still would call out for help, he could be woken out of the nightmares. He had some strange new symptoms: his right arm, the one that had taken on the burden of the dyskinesia, had a new stiffness. Coach associated it with having almost lost it to frostbite in the trenches during the war, where temperatures stayed well below freezing for days at a time. Whether or not this reaction to frostbite was due to his wartime of 1945 or the wartime of 2000 was something we never did figure out.

There were steady signs of improvement after six weeks. Sometimes he could feed himself. Sometimes he slept up to five hours before the night terrors commenced. On some days he felt faint stirrings of energy. One day, 56 days after stopping the medication, he walked a third of a mile. These encouraging signs were intermittent, at best, however. He still spent most of his day listlessly dozing and his nights continued to be rocky. He continued with the marijuana cookies and could not fall asleep without them.

Ten weeks later

Ten weeks after stopping levodopa, he was able, now and then, to right himself; he had been listing hard to the left and was both unaware of the fact and unable to do anything about it. His outlook began to improve, though he said he was "still down in the mouth at times." His right arm was in pain from the frequent spasms that erupted in times of stress or nightmare.

Eighty four days (twelve weeks) after stopping the drugs, here was his slow, whispered report: "An up and down week, with quite a few ups. Walking slightly farther, and able to get in the pool and float around a bit. Actually had some energy this morning, some small reserves of energy. Depression, maybe; Monday was a melancholy day. I'm still leaning to the left, but not as badly, and I can adjust my body position by myself.

"Sleep is much better. I slept through the night a few times. My feet still stick to the floor, and the voice comes and goes."

Thinking of suicide

His wife then added, "He is still very depressed: on Monday he was talking about putting a gun to his head. His right arm seems to be shaking less violently and spasming less."

After sixteen weeks he began to feel a bit independent: he could feed himself breakfast some mornings and even get out of a chair by himself. He said he could see that there was light at the end of the tunnel. He was still subject to depression and had difficulty drumming up interest in the affairs of the people around him.

A month after that, he reported, "My voice is back, and Heidi is giddy with joy that I can actually talk to her again. But I still toss and turn a lot at night. Poor sleep the last two nights, so I nap sometimes. If I'm not pushed, if I'm allowed to go slowly, I can

button my shirt sometimes. Sometimes I still need help with food; it all depends on my tiredness levels."

But the next week, he said, "It's been a bad week, my voice has been gone since Saturday. I wake at 4:30 in the morning and can only move with an extreme effort. I can't talk, and I can't call for help.

"I think of suicide often, when it all comes crushing down on me. I always think of myself as responsible and able to manage, and it's so difficult when the energy runs out and I can't do anything."

A month later, he had some days when he felt as if he was moving to a new stage, that things were about to get better. He had other days when he felt bad, edgy, and less tolerant of others.

A year later

A year after he stopped taking the medication, he was still in this same, vacillating condition. His brain had stabilized, but the limbic system was hovering right at the edge, right at the exact amount of dopamine – no more, no less. It appeared as if his baseline of dopamine was now high enough to maintain life and mind but that his body was reluctant to create anything that might be construed as a dopamine excess. His brain's tolerance for dopamine excess had clearly been permanently reset so that the old dopamine trauma, which had nearly killed him via heart failure, could never again arise. This hovering right at the exact amount of dopamine, with no reserves to speak of, meant that if he did have a few days of feeling good like his old self, they probably would be followed by a few days of feeling edgy and listless.

On the other hand, the fact that he had been able to climb up out of the terrific addiction-induced dopamine vacuum that included the baseline decline in brain-made dopamine did prove an important point. He was able to make dopamine. His brain had restored the baseline and he was again making dopamine at *almost* normal levels. Would he ever make dopamine at normal levels again? We didn't know. He and his wife wanted to wait a little longer, hoping for increasing predictability.

His right arm, the one that had assumed most of the acting out during his times of overmedication and drug withdrawal, appeared to be gravely weakened by its traumas.

At this point, he started taking 20 mg/day of Prozac, an antidepressant.

Assessment

Most of his Parkinson's symptoms were gone. His arms, once bent at the elbow in classic PD fashion, now hung limply at his side. His face, once frozen, was now expressive, so long as he was well rested and in a reasonable mood. Other mood-dependent symptoms included voice, sleep, slowness of movement and walking. Overall, he was limp, rather than rigid, suffering from symptoms typical of recovery, rather than the tightness and stiffness typical of PD.

Comparing recovery and Parkinson's

This similarity of non-functionality in PDers and people recovering from PD makes it difficult to assess changes in the condition. Two factors are very important in deciding the cause of the trouble.

The first is feeling the tone of muscle tissue: limp, heavy limbs are the norm during recovery, and tight, heavy limbs are typical of PD.

The other key factor is the source of the fatigue. In Parkinson's, the weakness and fatigue have been well described as being "exhausted from constantly fighting against a non-responsive body, trying to make it work." In recovery from PD, the fatigue is more like the weakness after a febrile illness. The entire body wants to surrender over to sleep. The desire is not so much to conquer the insolent body and make it obey, but to whimper and curl up in a ball. Attempts to fight this deep, healing fatigue will be met with failure. The old, PD-ish trick of summoning up adrenaline will no longer work – the body, no longer injured, will no longer induce an adrenaline rush simply to put on a show of health and vitality. The body is enjoying a long-earned rest, and the fatigue of the healing body is simply the truth and not the result of a struggle.

Skipping ahead another six months with Coach, the Prozac did prove helpful: he was still feeling regular, albeit very small, improvements. He was not having angina attacks.

He and his wife decided, four months after this, that he should start taking the smallest possible amount of Mirapex, a dopamine agonist. He took Mirapex at .125 mg, three times a day, and noticed a faint acceleration in improvements. He did not feel vigorous or robust, but he found that he vacillated less, dropping down into "bad" days less frequently. After a month, he increased the Mirapex to .25 mg, three times a day, and then again, a month later, to .5 mg, three times a day. At this level he was taking half of the amount suggested by the manufacturers of Mirapex. After a month at this level, he felt a shift. Here are my notes on his visit at this time: "Eating better, feeling so much better. Walk most days, doing Pilates exercises with a trainer. Can read again! Can read for an hour at a time if using eye drops for moisturizing. Voice is always fine, and facial expression is like my old self, pre-Parkinson's. Can move easily in bed, dress myself, pull up my own pants (mostly) and feed myself (mostly). If I'm really tired, I still need help finishing my food.

"Sleep is usually good. Called out for help one night recently while in a nightmare, but quickly came out of it. Still taking the marijuana cookies to keep the nightmares at bay. Shower by myself. Still have round-the-clock caregiver in the house, but mostly to make sure that someone is always there to do the driving and to walk with me when I go out walking on the property.

"Lots of independent time. Most nights don't need any help. Bladder is behaving well. Small motor function is good: can turn pages and sign my name. As for buttons, I avoid them.

"The tremor comes and goes; it's small, and I can stop it consciously. I'm getting out with Heidi again; we go to the beach, the shops, restaurants. I'm not driving yet, but I'm looking forward to driving."

During our visit he looked wonderful and was moving beautifully. He danced a few jig steps for me, showing off how well he could move.

Summary

Possibly the most important point in this case study is that anyone who is starting to feel a deep, natural joy welling up within might want to consider this possibility: if medication is still being taken when this deep joy appears, that joy may be coming from a dangerously high level of medication and not from a sudden shaft of wisdom or sudden evaporation of Parkinson's disease.

For anyone who might say that Coach's Parkinson's had never actually responded to therapy, and that his benefit from Mirapex was proof that nothing had changed (he still had PD), I would like to point out that when I started working with him, he was taking 400 mg of levodopa, and at that level of drugs he was expressionless, slow, and rigid. His head couldn't turn and his arms were bent at the elbows. Even with no antiparkinson's medications, during his long slow recovery from the terrors of drug-withdrawal, on the days when he felt good, he was in much better shape than he had been when we first met: his face was radiant, he could rotate at the neck, waist, and shoulders, and his arms swung loosely from the shoulders.

Now, with the help of a sub-clinical dose of Mirapex, he was relaxed and poised and on most days, to all appearances, perfectly normal. He was logical, making intelligent plans about the future, and engaged with the people and places around him. He did not appear overmedicated; at least he did not yet have the glistening eyes of a person who is taking too much of a dopamine-enhancing drug.

Do not imagine for a moment that the amount of Mirapex he was taking was inconsequential. In fact, even at these "sub-clinical" doses, we have seen recovered PDers have terrible problems. You will read about one of them in the next chapter. Becky, too, tried Mirapex and developed hallucinations and violent shakes at a fraction of Coach's level. So how could we explain why this amount of drug was not affecting Coach as Becky, if he had indeed recovered?

I suspect that, just like with the dopamine researchers who only wait ten days to do analysis of their subjects' brain scans and motor function, not enough time had passed. Coach had increased the Mirapex rapidly. I suspect that when I saw him, what I was seeing was the tiny starter dose. Although he was at a half-way dose level when I saw him doing so well, he had been only four days at this level. I have seen that Mirapex takes much longer than L-dopa to build up or decrease. Benefits from Mirapex continue to build for months after a person starts taking it, and Coach had only just begun.

His wife, justly feeling that life was slipping away, and eager to see what benefit he might get from the drugs, did not want to spend months waiting for him to be functional. She had increased the Mirapex as fast as possible. Based on what we have seen, his excellent movement was more likely due to his first, low doses of Mirapex that were just starting to be effective, and his increased dose may soon begin to cause adverse effects.[1]

---

[1] Breaking news: Just before going to publication I spoke with Coach's wife. Coach further increased his medication, going up to 4 mg/day of Mirapex. At 4 mg/day he developed mental confusion and dizziness, so he dropped back down to 2 mg/day. The first flush of Mirapex benefits did not last; at 2 mg/day he no longer has anywhere near the same level of benefit that he first had with .75 mg/day, one year ago. He is much less mobile now at 2 mg/day than he was a year ago at .75 mg/day. One must wonder if the more-rapid-than-usual decrease in Mirapex effectiveness has to do with his evident recovery from Parkinson's and a subsequent vulnerability to drug-induced parkinsonism from the Mirapex.

"The difficulty in life is the choice."

"The wrong way always seems the more reasonable."

The Bending of the Bough, play by George Moore (1852-1933)

20. TOO MUCH PAIN

THE DRUGS AS SOLACE FROM ERUPTING PAIN

This chapter about Rudyard, like the preceding chapter about Coach, is a single case study. Rudyard's experiences contributed to our growing body of evidence that, once a recovery program has been started, medication may no longer be a safe option.

Rudyard - Eldepryl, Mirapex

A study of "harmless" amounts of Eldepryl and Mirapex

Rudyard was taking Eldepryl and the health store supplement, NADH, when he first came to our clinic. He had been diagnosed one and a half years earlier, at the age of forty-six, but he already had fairly advanced Parkinson's at that time.

He had had symptoms of Parkinson's since age twenty-six: his tremoring was significant at that early age. By the time I met him, his posture was hunched to the point of deformity, including a frighteningly severe reverse curvature of the cervical vertebrae. His feet were grotesque, bluish-grey, gnarled, and utterly numb, with extreme deformities of the toenails.[1] His personal hygiene was appalling due to his inability to wash himself thoroughly. His hands were quite rigid, with all five fingers pressed together, fingers extended. His legs were rigid, as were his torso, shoulders, and neck. He had no arm swing and his left leg dragged badly. He was certain that, because he was only diagnosed a year and a half ago, his was a case of "early" Parkinson's and should respond quickly to treatment.

I did not consider him to be "early Parkinson's." Even with Eldepryl, he had trouble performing small motor tasks, including eating, putting a key in a lock or washing his hair; his balance was very bad and he had had many falls; his whole body was tense and rigid to an extreme degree. It seemed to me that the reason he had only been diagnosed a year and a half ago was that he had only seen the doctor for the first time a year and a half ago. Considering that he had had most of these symptoms, albeit to a lesser extent, for nearly twenty years, and the tremor for a full twenty, and especially in light of the fact that he was to need to move into a care facility in less than a year, I would say that, at the time I met him, he was probably approaching an advanced condition. However, I would have to say that he had done very well, considering that his symptoms had appeared twenty years earlier.

Usually, a person who has symptoms prior to age 55 tends to have a more rapidly progressing form of the illness. I can only attribute much of his success in living for so

[1] Toenail deformities are common in Parkinson's, as is toenail fungus. Happily, this fungus and the deformities go away as a part of the recovery process. As circulation to the feet is restored, the body is able to recognize and kill the fungus.

well for so long with encroaching PD to the facts that he was not oppressed by the knowledge that he had PD and he was not taking any medication.

He had started taking Eldepryl at the recommended dose, 5 mg two times a day, when he was first diagnosed. One month after starting the drug, he started receiving treatments from an east coast practitioner who was using our methods, and, in anticipation of a quick recovery, reduced his Eldepryl to a fourth of a pill, twice a day. He took this dose for a year and a half. He insisted when I met him, after a year and a half of this low dose of Eldepryl, that the drug did nothing for him. He could take it or leave it, he claimed, and only took it out of habit. He became annoyed when I suggested that it might be addictive, and two weeks after starting to work with me, he quit taking his "insignificant" dose of Eldepryl and reduced his NADH to 5 mg a day from 10.

Over the next ten weeks, he went into a tailspin. Five weeks after stopping the Eldepryl, he reported that he was freezing constantly and he couldn't catch his breath. He couldn't sleep, he could not turn to one side or the other, and his balance was completely gone, frequently causing him to crash into walls. His left leg was utterly useless – he had to lift it with his arms to move it – and his left calf and hamstring were painfully cramped. His tremor was "deeper" and dominated his every moment. He could barely eat and he had to spend long minutes calculating how to get up from a chair.

By week six, he said, "I'm not better but not worse, and although mornings are bad, once I start walking I can keep going." After several more weeks, he said he had "turned a corner. I'm doing better, I have to admit it." He was less frequently "paralyzed," and "not needing to lift my feet thirty times to move three feet."

Ten weeks after stopping Eldepryl

After a full ten weeks, he noticed that he was doing much better. He went running twice during the week, and he was sleeping well, only getting up twice at night to urinate. He had only had one episode of hand clenching (dystonia) and the freezing was much less frequent – and when it did appear, he could easily break up the freeze and keep moving. He was angry when I suggested that the ten weeks did correspond to the amount of time that most people need to complete a withdrawal from dopamine-enhancing drugs. He was adamant that he had not been taking enough to make a difference. However, he was surprised when I read to him his weekly notes and pointed out that at five weeks after the reduction, he had been at his very worst, and at eleven weeks he was running again and pretty much behaving as he had been prior to the drug reduction.

Two weeks later, he noticed that his arms were starting to swing, and that he was starting to have sensation in his scalp. After that he started having recovery symptoms, including a sense of tremendous heaviness at nighttime, as if he was sinking into his bed and down into the earth, as if his bed had an enormous gravity and he was of supreme density. This feeling, which he named "being buried alive," understandably terrified him, even though I shared with him similar descriptions from people who were starting to experience the Deep Sleep and extreme weakness that accompany recovery. Even more horrifying, however, was the pain that accompanied the return of sensation in his extremities.

Pain

Foot pain

For over a year and a half, Rudyard took no medication and noticed increasing signs of recovery from Parkinson's. However, over time, as sensitivity returned to many parts of his body, he regularly (daily) considered taking drugs again. When he first regained feeling in his feet, he was so panicked by the extreme burning and the alarming return of pinkness (which he assumed to be an infection) that he went to the emergency room. They offered him L-dopa. He took one dose, felt completely stoned, and decided that he could no longer tolerate this particular drug. (When he had taken L-dopa at the time of his first diagnosis, it had done nothing dramatic for him and so, after a week, he had stopped taking it.) However, concerned by the pain and rosy color appearing in his feet he started toying with the idea of trying some new Parkinson's drugs.

I asked him at his next weekly visit why he had not considered taking aspirin or Tylenol for his foot pain, and he told me that the pain was so severe that these mild remedies could not possibly have helped.

Hand pain

Several months later, after his feet were no longer burning, he started to have restored feeling in his hands. He again went to the emergency room, this time for hand pain. When he told the doctors that he had been diagnosed with Parkinson's, the ER doctor (clearly not a PD specialist) told him that abrupt hand pain and a return of healthy color in the hands was a symptom of Parkinson's disease. He suggested antiparkinson's medications and also told him that the emergency room was not the appropriate venue for getting treated for Parkinson's disease, and to stop coming in.

Rudyard again refused to consider taking mild anti-inflammatory medications such as aspirin. He actually tried taking one aspirin once and, since he still felt pain in his hand, he announced that anything short of anti-PD drugs would be worthless.

After the emergency room asked him not to come back, he asked me every week whether or not he should start taking some antiparkinson's drugs for the pain. I replied every time that I no longer worked with medicated patients. He was certain that only anti-PD drugs would be strong enough to ease his pain. He refused to even try over-the-counter pain pills, massage, meditation, or anything that might possibly have helped his throbbing hands, and instead took nothing for the pain. After several months, the burning in his hands, which was quite severe, preventing sleep and nearly crippling him with pain, began to ebb. The worst was yet to come.

Elbow pain

About two months after the hand pain began to ebb, it appeared in his elbow. On days when the pain was extreme, he felt he was dying, but sometimes, on days when the pain started to ebb, and particularly after nights when he slept well, he felt much better and not at all like he was suffering from PD. Here is a quote from him at this time:

"Interesting week. Yesterday was the worst day of my life, the worst week, the worst everything, but I slept well, and today, I don't feel that way at all. The new thing is my arms are dead, just like my legs used to be dead when the pain first left them. This makes me panic, of course, and the elbow pain continues, but it's less. The problem now

is that the hands are weak and limp. I'm not dizzy this week, but I'm getting a red rash on my feet and torso, and a feeling of bugs all over my skin."

And so it went, alternating between recovery symptoms, which were quite painful, and days when he felt much better, and his increased flexibility made him appear much less like he had PD.

Neck pain

The worst was still yet to come. He started to have feeling in his grossly contorted neck. I can well believe that this pain must have been excruciating. He insisted that what he needed to help with the neck pain were antiparkinson's drugs. Again, he refused to consider any treatment or pain reliever except for anti-PD drugs.

I asked him to try a chiropractor to see if anything could be done about his neck. The chiropractor said that his neck bones had grown into the distortion, and there was nothing that he, the chiropractor, could do about it. This threw Rudyard into an emotional tailspin. He was in great pain. He could not be consoled with the fact that his feet no longer hurt, that they were now flexible and warm and his toenails were growing in normally. He was not consoled with the ebbing of pain and the return of feeling to his hands, or the ability to move his fingers.

Dopamine depletion from pain

Rudyard starting exhibiting postural symptoms of dopamine depletion. Pain is modulated with dopamine, and in times of great pain, one needs either increased adrenaline or dopamine to saturate the limbic zone against the incoming onslaught of pain signals. Rudyard didn't have enough of either.

Rudyard was in extreme pain. This pain had him awake most of the night, curling his body into a protective fetal position, and staggering about, losing his balance and crashing into walls – symptoms of pain-induced dopamine depletion. [1]

He announced that he would take some antiparkinson's drugs for a few months. Having experienced similar burning in his feet and hands, he was certain that he would only need the drugs for a few months until his neck pain also went away. I was concerned that he might become addicted if he started taking the drugs; I had seen this happen several times before. However, he was adamant.

I had shared with Rudyard, just as I shared with everyone, the case studies of his fellow pioneers. Rudyard fixated on Coach's recent success with a low level of Mirapex. He completely disregarded the discrepancies between their cases: Coach was now 76 years old, had been suffering post-traumatic-stress depression and addiction-induced dopamine deficiency; Rudyard was now 47 and suffering from a debilitating level of pain. Their cases did not seem to me to be similar.

---

[1] For that matter, infectious illness also depletes dopamine; I had a high fever and sinus infection only last week, and I was shuffling around the living room, nearly losing my balance several times, and tripping over the furniture. My arms were clenched in a fetal position. I felt incapable of any speech louder than a moan. I mention this merely because I am trying to make the point that there are many syndromes besides Parkinson's disease that can deplete dopamine and cause the sufferer to revert to a hunched over, staggering hulk who is incapable of taking care of himself. However, most people do not treat extreme pain or febrile illness with antiparkinson's drugs. To do so would be, in the phrase I learned in school, "killing a chicken with a buffalo ax."

It did appear as if, when the pain ebbed, Rudyard was able to produce dopamine. He was in fact starting to have more episodes when he would suddenly find himself relaxing down into his chair, and even relaxing emotionally. He was starting, for the first time in his life, to be able to have a circumspect attitude about his condition and about life in general. Prior to this he had always been intense, driven, and demanding. I remember, right around this time, that he amazed the two people who were treating him in the clinic when, in response to their warning that it might take a long time for the neck pain to begin to heal, he shrugged his shoulders, smiled at them, and said, "Whatever!"

Even he was amazed. Immediately after he said it, he sat halfway up from his treatment table and chuckled, "Did I really just say what I think I said? Omigosh! I *am* changing!"

But despite these indications that dopamine was returning, and despite the differences in their ages and case histories, Rudyard compared himself to Coach and asked his neurologist for a prescription for Mirapex.

Mirapex

He started taking Mirapex at the lowest level, .125 mg (an eighth of a milligram), three times a day, for a total of three eighths of a milligram per day.

I did continue working with him, despite my concerns about drugged patients in the recovery program, partly because of legal restrictions on "patient abandonment." I also saw no point in abandoning him as a patient: at this point, he was already getting better and there was no way for me to turn off the recovery. I stopped doing Tui Na, however, and limited my treatments to mostly talking about his week, or very simple acupuncture treatments for reducing dopamine levels if he appeared too overmedicated.[1] I figured that there was a chance that he might be able to use the medication to get through this period of extreme pain and then get off the medication, so I kept meeting with him. At the clinic, the practitioners mostly held his hand or massaged his neck while he talked about his week. We did not want to do anything that might accelerate his recovery.

Despite my misgivings, we were all curious as to the relative addictiveness of the new agonists when compared with levodopa. We were to learn that the agonists are just as damaging.

One eighth the normal dose

Within two weeks, he was somewhat better, having two periods of over an hour when he was perfectly "normal." I pointed out to him that he wasn't supposed to feel this much better at this starter dosage – these drugs are supposed to be increased up to somewhere between 3.0 and 4.5 mg/day before the patient feels better, and here he was feeling greatly relieved at one eighth of the so-called therapeutic dose. He was taking a third as much as Coach. Didn't this suggest to him that he should be trying something a little less powerful?

He felt that I was being melodramatic. He pointed out that he was taking so much less than the therapeutic dose that the drug could not possibly be addictive at this level. I

[1] A needle jabbed deeply and painfully into KI-1 (on the sole of the foot) can waken the sympathetic system, bringing energy down from the head during episodes of mania or overmedication from dopamine-enhancing drugs.

countered that, since the drug was clearly masking the pain at this minute dosage, maybe it was more powerful than he was giving it credit for.

Doubled dose to .75

After two weeks, he decided that, since he was feeling better with the Mirapex, he should increase his dose. He doubled his dose, as recommended by his doctor, up to .25 mg/three times a day, for a daily total of .75 mg. Within two days, he felt much better, with increased range of movement and much less pain.

Ticcing and dyskinesia

However, he noticed that he was having an increase in tremoring, even in his left hip, which had not tremored in many months. Within two weeks, he said, "I feel fantastic, but the tremoring is getting very powerful, more so than ever before." He also had cramping in his legs (dyskinesia) just like he had when had taken Eldepryl. Within four weeks at this level, he told me that moving to alleviate the cramps would cause worse cramping. Also, after doing his jumping jacks, he had cramping in his pectoral muscles. Most frightening was the extreme cramping in the neck and facial spasms, which, in his words, "makes me feel like Frankenstein's monster."

33% reduction

Mirapex is well known to cause cramping in the muscles along the front of the neck. In fact, I should not be surprised to learn that it is this cramping along the front of the sternocleidomastoids that is responsible for the drop in blood pressure in Mirapex patients.[1] He reduced his medication. He started taking the .25 mg pills only twice a day, instead of three times. Now, at this reduced level, he saw the benefits of the medication ebb. Within a week, he had lost most of the benefits that he had gotten when he started the drug. When he had first started, at three eighths of a milligram a day, he perceived benefits. Now, just two months later, he perceived no benefits from the drug at half a milligram per day, a slightly higher dose.

Worse, he continued to have the powerful tremor and the cramping, both of which are listed as adverse effects of the medication. Although the tension in the neck decreased and the facial spasming stopped, he felt weaker than ever, lurching from spot to spot, with a tendency to fall over backwards and, in his own words, "paranoid" about the sudden increase in weakness.

The next week, he felt much better at the lower dose: although his tremor was increased, with greater power, he observed that his sleep was better, he had increased feeling in his low back and could even stretch the muscles of his low back, something he had not done in years. He was lurching less, getting up out of chairs more easily, and his

[1] The pressure along the SCM muscle pushes on the carotid sinus, where the barometer for determining blood pressure is located. The increased pressure on this sinus may be mistaken as a signal of excess pressure by the sinus, which then, appropriately, sends a "Reduce pressure" signal to the body. The corresponding lack of pressure causes the orthostatic hypotension that is particularly severe with this agonist. Conversely, when decreasing this drug, the temporary collapse of this muscle that follows the decrease may send a false "Low blood pressure" signal to the sinus. The sinus then responds with a surge of increased blood pressure via vessel restriction and increased heart rate. This would certainly account for the feeling of pressure and heat in the head experienced by people who reduce this drug quickly, and it may also account for their symptoms that resemble mild stroke.

small motor function was better than it had been in a long, long time: he was now able to floss his teeth and was doing it three times a day.

A slight decrease

At this point, feeling better at .5 mg/day than he had at .75 mg/day, he felt that it had been a mistake to take the Mirapex, and he decreased again by a tiny amount, taking only .25 mg/day on Thursday, though he continued taking .50/day the other days of the week.

Within a week, he was feeling depressed. He reported, "I'm getting weaker, my energy is fading, but a lot of it is depression; I'm laying in bed when I don't need to. I can still get out of chairs and I'm still able to feed myself, but I just don't feel the same about it."

He continued with the above dose: .5 mg/day every day but Thursday, and only .25 mg on Thursdays. He felt "no marked difference on the days with only .25 mg, and the cramping is easing up. I can relax again. I can feel myself sinking my head into my pillow, and it feels so good. When I'm in bed, I feel so relaxed that I think I will be able to be fluid and graceful when I get out of bed, but I can't. Since decreasing the pills, the sleep is continuing to improve. I can stop the tremor now by concentrating on it. I have less choking and tension in the front of the neck, I'm regaining feeling in my stomach and low back, and I can stretch this area. I'm having ants crawling on my face and twitching or shivering in my facial muscles. I was so relaxed that I was able to lie in bed and read a book, and I haven't done any reading in a year. My arm gets tired from holding the book, but my ankles are so relaxed I can lie on my bed with my sneakers on and not get ankle cramps or pain."

The next week, he reported that he felt distinctly "looser" on Friday mornings, after having only .25 mg on Thursdays. However, his throat spasm, though it changed from hour to hour, was starting to frighten him. He reduced his medication again, taking the small dose of only .25 mg on Sundays as well as on Thursdays. After making this extremely small reduction, he felt "a head shift (an attitude shift) this week. I'm seeing each moment as an opportunity to stretch, grow, practice. I am starting to realize now that any improvements that I'll get now will come from me."

He noticed that on Fridays and Mondays (the days that followed his smaller dosage), he had "a clean feeling, with better alertness. But two days after the smaller dose days (on Saturdays and Tuesdays), I have a mild crash." He wanted to stop taking the Mirapex now, as he felt he had made a mistake in starting it, but these mild crashes worried him. He was afraid that if he stopped taking the Mirapex altogether he would be much worse than he had been before starting it. Also, I had warned him that Mirapex has a delayed effect, about three weeks longer than Sinemet. While Sinemet has a ten-day slide, the Mirapex slide is nearly a month before the real withdrawal begins.

Another very small decrease

Nevertheless, he decreased again by reducing to only .25 on a third day of the week. At this point, almost four weeks after his first Mirapex reduction from a "sub therapeutic dose," he started experiencing drug withdrawal symptoms: "Depressed all week. I feel as if I'm lacking serotonin. Balance is worse, freezing is worse. I'm back-pedaling when trying to walk forward, my neck pain is worse, and the throat strangling

feeling is back. It's harder to get in and out of chairs, and I'm feeling weaker every day. I'm still having the ticcing – it hasn't stopped since I started the Mirapex – and I'm starting to have cramping in my legs even at this low dose." After two weeks on this drug regime, his insomnia was back and the depression was very bad. He was experiencing a devastating loss of energy and having dizziness with a horrible new sort of head tremor. In the past, when taking levodopa and later when taking Eldepryl, he had experienced dizziness. Dizziness is listed as an adverse effect of Mirapex. However, from deep in his drug withdrawal haze, he fixated on the dizziness, decided it was the most significant of his Parkinson's symptoms and it could only be helped by the medication.

Return to .75

Over the next several weeks, he went back up to .75 mg/day, his previous highest level. His memory of this level of drug use was that it had felt like a golden time, a time of energy and gracious movement. I pointed out to him that, in fact, it had been a time of severe ticcing, cramping and feeling like Frankenstein's monster, and that was why he had decreased the drugs. He disagreed and said that he had decreased the drugs because he felt so good that he had imagined he was recovered, when, in fact, it was obvious that he was not getting better, as evidenced by how bad he felt with the reduced dose. In fact, he pointed out, he had never felt better than when taking .75 mg Mirapex a day, and he had never felt worse than he did when alternating between .25 and .5 mg per day.

Return of adverse effects

Within two weeks of taking .75 mg/day, he was having strong cramping, increased tremor and dangerous, choking throat spasming again. Worse, he felt none of the benefits of the medication. He could not understand why, only a few months earlier, he had felt on top of the world while taking .75 /day (his faulty memory of those "golden days"), and now he felt only tension and pain. The throat spasms became so severe that he dropped back down to his previous pattern of alternating .5 mg days and .25 mg days.

Decreased dose, withdrawal symptoms

He soon became severely depressed. He could not get out of a chair, feed himself, or sleep. "I'm in bed, tired, but not sleeping well, and every day I'm more weak, with less balance. The depression is *very* bad." He started having leg and throat cramping even at these lowered doses.

The cramping, especially the throat spasms, was so frightening that he decided he needed to increase his medication. He felt that the throat spasms were being caused by the Parkinson's returning, and the only way to make them go away was to increase his medication.

Return again to .75

He increased back up to .75, and the cramping became truly agonizing, but he felt that the increasing dizziness was even more of a concern, as he was starting to crash into things.

After several weeks of frightening dyskinesia he decreased again, down to .5 mg/day, but the tremor and crashing into walls continued to build in intensity.

At this point, I had yet another long talk with him, in which I read to him again from the list of adverse effects of Mirapex. I asked him, "If the drug causes throat spasms, and you do have worsening throat spasms when you increase the drug, what do you think you should do?" He answered, "Increase the Mirapex." Two of my colleagues who had been invited to observe this session exchanged glances.

I asked Rudyard, "If the drug warning says that this drug can cause dizziness, and if you have increasing dizziness when you increase your medication, what do you think you should do?" He answered, after a thoughtful pause, unaware that I was asking him essentially the same question, "Increase the medication." My colleagues cocked their heads and looked at me, puzzled.

"Rudyard, if this drug *increases* tremor, and if your tremoring started up again after you started taking this drug, and if the tremor is worse at higher doses and decreases at lower doses, and you are having worsening tremor at .75 mg/day than you did at .5 mg/day, what do you think you should do to ease your tremor?" This one required no thinking at all. Rudyard smiled and answered quickly, "Increase the Mirapex." He continued, "I feel so bad now, and I remember so clearly, the first time when I went up to .75, I felt so good. I've never felt so good in my life. I could move, I had confidence, and all my pain went away. I just want to recapture that. I know how I felt at .75 and that's how I want to feel."

I suggested, "Within a week or less of being at .75, you have horrible, choking throat spasms and your tremor increases."

Rudyard returned, "No, my tremor is bad now. It was fine at .75. And I never had cramping at those levels"

"Let me read to you from your chart," I said," where I have it in your own words. 'It makes me feel like Frankenstein's monster....'"

"Oh that. Well, that was just because I increased too quickly."

"No, Rudyard, every time you've been at .75, you've had to decrease soon after because you feel you are choking to death, and your tremor gets violently worse."

"What do you mean, 'every time'?" he asked. "I've only been at .75 one time, and it was wonderful, and I only decreased my medication so I could stay in your program."

"Rudyard, you have been at .75/day three times now, and you've had to decrease every time because of the cramping and choking, as well as your tremor getting so violent that it shakes your whole body. I have never threatened to throw you out of the program. We're learning from your experience."

"My tremor is shaking my whole body now, and I'm only at .5." he countered.

"That's because you are now addicted. Your body developed a tic in response to the drugs, it's most powerful when your dose is working, and since you are now addicted, your body is screaming for more medication, and it will tremor until it gets it."

"So then let's give the body what it wants, which is .75…or maybe more!" At this, he smiled hopefully.

"But at those levels adverse effects of the medication develop rapidly: cramping, frantic shaking, and choking."

"Then what I need is more medication, so that I don't have the adverse effects. The whole point of the medication is to overcome the adverse effects, correct?"

I want to make it very clear to the reader that, prior to starting the medication, Rudyard was a very thoughtful person who seemed to understand completely the risks and problems associated with addictive drugs, including the worsening of side effects even as the body becomes inured to the drugs and the beneficial effects fade away. He understood this completely.

Anyone reading this who is living with a medicated patient who is starting to not make sense will understand completely what I am talking about. PDers, for the most part, are extremely intelligent, and very often they are keen analytical thinkers. However, these drugs can curdle their brains so that they no longer think straight. And worse, when it comes to their medication, they can be blinded by love – a love for the drugs. So many of them, especially those who have taken the meds, however briefly or at however small doses, for even a few days, at any time *after* the recovery has begun, only remember that the meds, however fleetingly, made them feel "better than I ever felt before."

As the conference with Rudyard drew to a close, I asked Rudyard a final question: "If your medication makes you feel worse, and not taking it makes you feel better, what should you do?" He thought for a moment and suggested, "Increase the medication more slowly." I turned to my colleagues with a sinking heart. "This is what I mean by drug-addled and addicted," I told them. Rudyard also turned and smiled at them, as if he had just aced a quiz show and it was time to collect the prizes.

I was leaving the country at about this time to write up this book. I left Rudyard in the care of my locums. I have since heard that he is moving better than ever: he has much less pain and newfound agility in his torso and limbs. He does appear to be recovering nicely from Parkinson's.

He never decreased the medication again: he recently increased it to help with his moods and what now appears to be permanent shaking. The increase appears to have increased his ticcing and dyskinetic cramping.

However, the horrible neck pain stage seems to be finished, and he is moving quite well, better than he has in many years, even in the years when he was taking Eldepryl or feeling "great" when he started the Mirapex. We are assuming that he will need ever-increasing amounts of Mirapex to keep his mood up, especially in light of the constant ticcing and spasms, but we are pleased that his previously distorted body has responded to Tui Na treatment by becoming supple, and that his circulation has been restored .

He also appears to be ferociously addicted to the medication. I have also heard that his conversation is highly illogical, and that he has difficulty following a train of thought.[1]

[1] As this book is being published, I just learned that Rudyard has tripled his Mirapex in the last four months, up to 3.0 mg/day. My colleague says that he's "completely nuts." His dyskinetic cramping, choking, and ticcing are severe, but he doesn't seem to notice them. He announced that he is only taking the Mirapex until he begins to have signs of recovery, and then he will quit. He is moving back to New York to show his old friends how well he is doing…

Summary

Very often in our limited experience, a recovering PDer who is taking only a "little bit" becomes extremely addicted and is trapped with the medications for what appears to be a life sentence. If a person has started to recover from Parkinson's, these medications, even "just a little bit," appear to be at least ten times more powerful, and more addictive, than "just a little bit of cocaine" or "just a little bit" of heroin.

I also included this story to make the point that *all of the dopamine-enhancing drugs*, not just levodopa, are capable of quickly setting in motion a very powerful addiction. A colleague who was working with Rudyard after he started on the Mirapex once asked him if he had read the materials that I had been writing up about the dopamine-enhancing drugs, materials that were available to my own patients and also to Internet patients who had specifically inquired. (This was, at the time, a series of short chapters that have now evolved into this book.) Rudyard said, never having cracked it open, that there was no point in him reading it. Despite our assurances to the contrary, he was adamant. "I won't read it. I'm taking Mirapex. Her writing is sure to be about levodopa."

Rudyard probably would have been best served with some sort of pain relieving medication. However, the lure of the medication was always nagging at him. From the time he first got off his Eldepryl, he was always asking me if "just a little" medication might not help. This "looking back with longing" at the happier times with medication – even if those "happier times" were filled with pain, rigidity, and the full spectrum of Parkinson's disease symptoms – is not uncommon in those who have ever, however briefly, taken medication.

Rudyard was starting to experience glimmers of dopamine even before he started taking the Mirapex. We have not really broached this subject yet, the complications that can arise if a person is still taking medication when these first glimmers of native dopamine appear. So in the next chapter we will discuss some complications, such as glimmers of native dopamine, that can arise during recovery or addiction, complications that can add still more layers of confusion and mystery to the subject of drug use in Parkinson's.

"Why didst thou promise such a beauteous day,
And make me travel forth without my cloak,
To let base clouds o'ertake me in my way,
Hiding thy brav'ry in their rotten smoke?"

Shakespeare's Sonnet 34

21. GLIMMERS OF HOPE AND OTHER DANGER SIGNS

CONFUSING MINGLINGS: DRUGS AND NASCENT HEALTH

There are still a few medication complications that we have not discussed yet: the glimmer of returning dopamine, the trio of dopamine depleters, the deep sleep, the tenacity of the On-Off and other drug-induced patterns, the switching phenomenon, and the lingering siren song of the drugs. These phenomena are of interest because they enrich our thinking about dopamine-enhancing drugs as well as presenting challenges to any medicated person who is trying to recover from Parkinson's.

GLIMMER

The glimmer is the name we've given to the first, shy blushes of native dopamine. The glimmer occurs when the body's returning dopamine production is finally large enough to propel a few moments of natural, non-adrenaline movement. The sensation is one that has been long forgotten by PDers, and it seems somehow magical.

The return of dopamine

When patients begin recovering, they often notice glorious, if short-lived, new symptoms occurring now and then. These symptoms most often occur after a period of rest or sleep. The experience may feel like a moment, several minutes, an hour or a day of movement that was perfectly healthy, done to a normal tempo, that went smoothly and easily, and which required no thought.

Most PDers are stunned the first few times that they experience this movement. "I don't know what happened: I thought about standing up from my chair, and the next second I realized I was standing up!" is the sort of description we hear.

There is absolute certainty on the part of the patient that this is a different form of movement than that felt under the influence of the drugs. Patients often struggle to explain how it felt to just move, but "effortless" is the most common description. They often say that they never moved that way even prior to their diagnosis of Parkinson's.

Many people with Parkinson's, hearing this description, protest that they never struggled with movement prior to their diagnosis. However, once they experience this form of dopamine-based movement, they realize that they had no basis for comparison. What they had thought was ease of movement was, in fact, adrenaline-driven, relatively conscious and intentional.

The people who have experienced these glimmers and who have also taken antiparkinson's medication have valuable experiences to relate: they invariably say that the glimmers of native dopamine are unlike the drug-induced movements. The movement that occurs has been described as more "simple" and more "sweet" than the drug-induced movements.

These words may not help the reader. Trying to explain to the reader the different feelings that accompany movement with different neurotransmitters is rather like trying to explain the taste of an orange. But I'm going to try.

How it feels to move

Under the influence of adrenaline, a PDer has been able to move stronger, harder and faster than most people during most of his life, prior to his diagnosis with PD. His movements were not effortless, however. He would create an inner command such as "Now I'm going to stand up!" and then he would make himself stand. Or else "This hill is high, but I'm just going to keep putting one foot in front of the other, and I'll make it to the top." And then, his adrenaline-rich brain would make it happen. A common attitude of the pre-PDer is, "It's just a question of mind over matter; I can tell my body what to do, and it does it."

Then, after the person is diagnosed with Parkinson's disease and starts taking DEDs (dopamine-enhancing drugs), he may notice a new type of movement. (These feelings may not be so obvious in the recently diagnosed PDer, but they become more familiar by the time a person starts having Ons and Offs.) As the drug washes through the brain, it appears that the areas that are altered the most are the limbic and mood areas, not the motor area. There is a feeling of confidence, of the disappearance of pain (somewhat, unless the medication causes painful dystonias or dyskinesias), lengthening of the limbs and then, just before the movement begins, a feeling of power and vigor may pervade the body.

Rigid limbs – like sitting in a cold car

This lengthening of the limbs can best be described this way: short limbs are the feeling that one gets while driving in a cold car on an icy morning. In that freezing car, you are stuck in a sitting position with no way to get your limbs moving, and the cold chill of the car sinks in and you slowly, ever so slowly, tighten up. Without realizing it, you are holding your hands a little too tightly on the steering wheel and leaning forward, hunching your shoulders in, making your body a bit smaller in order to conserve heat. And then, after five or ten minutes, the car heater starts to make a difference, and you feel your body lengthening. Suddenly you realize that you can relax, the shoulders can open up, the death grip on the steering wheel relaxes into a carefree pose, and you just feel more able to move. It is this sort of feeling that is imparted by the DEDs. It is as if the mood and the body temperature both warm up a bit.

Immobility from depression

Another way to understand this type of movement is to remember how you feel when you are deeply depressed. For example, maybe your true love was just reported missing at sea. You sink into despair. You don't want to move. You force yourself to

shuffle along. You have no bounce in your step. Your mood is making you drag. And then, suddenly, you get the phone call from your true love who had been thought lost at sea, and, not only has she been found, but she has discovered a sunken treasure of sixteenth century gold and rubies. At this point, you dance, you sing, you laugh for joy. This is the sort of movement that the antiparkinson drugs provide.[1]

Dopamine-enhancing drugs impart joy, a mental and emotional warmth, and even for many people a physical warmth, which then allows movement as an expression of that joy.

"I don't feel joy from my drugs"

At this point, there will be many readers who will say, "I never feel any joy from my medication. I do not like taking my drugs. You are wrong."

Now, what I am saying is not that the drugs necessarily impart *obvious* joy. No. Although many people have awareness of being bright-eyed and more clever and happier when taking antiparkinson's drugs, many do not. Sometimes, the person taking the drugs does not recognize any mood change at all, but loved ones can detect a brightening in the eyes and a mood shift, sometimes even a goofiness, that herald the beginning of an On.[2]

The drugs effect this change on a subconscious level; maybe this is why so many patients who are clearly undergoing a personality change when their dose kicks in will insist that they have no conscious awareness of any change. Although it *appears* that the dyskinesias and obvious adverse effects are coming from the motor area, most of the improvement in movement may actually come from the limbic center and frontal lobe use of the increased dopamine, both areas that play a role in subconscious movement.

Lightened body, lightened mood

The conscious mind may or may not be aware that it undergoes a change when under the influence of the drugs. But the type of movement, the quality of the movement, and what it feels like inside the body to generate that type of movement are consequences of an uplift in the mood and unthinking animal centers of the brain. When it is easier to move the arms after taking the drugs, it is because the arms feel lighter, less heavy and oppressive. This lightness of being is similar to the lightness one feels when one has a positive attitude and everything is going well.

Due to this lightness and sense of well-being, many people who are thrusting their arms or grimacing imagine that their dyskinesia is not as violent at it looks to their horrified friends. Often the dyskinetic PDer thinks his limbs are merely floating gracefully because they have become so light. *The drugs create emotionally enhanced movement*.[3]

---

[1] The agonist drugs do this to a lesser extent than the other drugs. The dopamine movement imparted by the agonists sometimes feels more natural as long as the doses are minimal. But when the agonists, by decreasing the dopamine need in the motor and mood area, free up some extra dopamine so that there is an excess of dopamine floating around, even though the agonists do not convert into dopamine per se, these symptoms may occur. This is why even an agonist like ergot can cause a person to dance and shout and hallucinate, even though it technically does not provide dopamine.

[2] This unnatural radiance can be quite obvious. I can usually spot an overmedicated PDer from a block away. There's something uniquely strange about the eyes.

[3] I have several patients who will refute this idea that L-dopa makes one feel good. They absolutely hate the way that L-dopa makes them feel. They have described the feeling of moving with L-

Takes one to know one

In response to all you drug-using readers who are going to suggest that you are unique, and that you don't feel this way, let me make this point: most PDers who take the meds are not able to accurately describe what it is the medications feel like until they are off the medication.

Only after they start to experience native dopamine for the first time in their adult lives are they able to realize, objectively, what it felt like to have artificial dopamine enhancers in their brain. It is only when they can compare the two side by side that they realize the difference.

Native dopamine

Native dopamine provides a type of movement that is completely different from the adrenaline or mood-created movements described above. By "native dopamine" I mean natural dopamine, produced in the brain (including that produced in the substantia nigra). This is the movement that most humans take for granted, and it is a type of motion that PDers typically have never felt since childhood. This movement is unplanned. It doesn't require that the mood be good or the body be heated up.

This movement occurs nearly simultaneously with subconscious thinking. It can also direct conscious movement. An example of unconscious movement is swallowing or speaking, or the movements that non-PDers make in their sleep. In sleep one is not supposed to use adrenaline to gently turn over. In our sleep we also don't really care if we are happy or dejected, we can still turn over, regardless of mood.

With PD, swallowing, blinking, and even speech increasingly become processes in which every step must be consciously executed. As many PDers will testify, speech requires a terrific amount of conscious thought if dopamine is absent. From word selection through to sound formation, every step of the way must be thought through and consciously initiated in order to produce speech. In healthy people, dopamine is the neurotransmitter that allows us to speak before we think – an activity we all regret at times!

Native dopamine provides the effortless movement that scratches at an itch and the perfect, harmonious working of the muscles so that one can sit still for a long time in complete comfort. These movements require no conscious initiation or uplifted mood.

Native dopamine coordinates the movement that enables a fast typist to produce a written word as fast as the word occurs in his mind. This movement is not a reflex, but it is not consciously initiated. Nor is such movement affected by mood – it is mood neutral.

This mood-neutral movement is not like the adrenaline drive that a PDer uses to declare, "I *can* get up from the sofa; I just need to make the effort." The native dopamine

---

dopa as being "kidnapped" or having a false energy that feels toxic. The people who have felt this way in my limited experience have all been people who, prior to being diagnosed with Parkinson's disease, spent many years of their life doing meditation or spent an unusually large amount of their daily time in prayer. These people say that the lightness of L-dopa brings with it a film of deceit that prevents them from being able to penetrate into inner peace or higher consciousness in the way that they used to. They recognize that the strange lightness of L-dopa is false, and that it is not coming from wisdom or joy, but from an illusion. On the other hand, many people who have gone through recovery and are able to get off their drugs continue to recall with wistful yearning how much easier, how much happier, how much more carefree they felt when they were on the drugs. These people continue to be at risk for returning to the drugs for the rest of their lives, even though the drugs may now have disastrous effects if they do so.

feeling is so smooth that as soon as the thought occurs "I would like to get up off the sofa," the person finds that they have gotten up off the sofa. It is attitude-neutral, pure thought without the analysis.

"Brain waves taking form as movement"

Rose explained her experience of this phenomenon: "I got out of bed and for some reason, I didn't even know I was getting out of bed. I looked at my clock, saw that it was time to get up, and as soon as my brain registered that it was time to get up, I was sitting on the side of the bed and then standing, and then walking into the bathroom using a perfectly normal stride! I brushed my teeth, and whatever I thought of doing, that's what I did, and I was nearly dressed before the miracle ran out and then I was suddenly moving slowly and everything was slow and difficult again. It was a miracle. It didn't feel like conscious work, it felt like brain waves taking form as movement."

"I have never felt anything like it."

The glimmer can occur even with very small, insignificant motor activities, as in the following description from pre-Mirapex Rudyard: "I was sitting in my chair watching TV last night and all of a sudden, I felt comfortable. I didn't have to think where to put my arm so it wouldn't be tight; I didn't have to think about anything at all; I just sat there in my chair and I felt my body sink into the cushions. I wasn't tired, I wasn't happy or distracted from my problems, I wasn't melting into sleep, I just felt this strange feeling as if things were just, well, sort of OK, and my whole body was relaxed. I wasn't happy or sad, and it didn't feel like I felt with my Eldepryl. I just 'was.' I have never felt anything like it."

Heavier than the earth

Steve was never medicated. During late stage recovery, he had a nearly topped up supply of dopamine. He often went strolling for hours around the local neighborhoods to enjoy the feeling of unpremeditated movement. Now and then he overextended himself and would come to a complete halt. Sometimes he would have as little as half a minute's warning before he was first halting, then shuffling, and then motionless.

During the times of non-moving, as he sat on a neighbor's short retaining wall or at a bus stop, he would analyze how he felt. He described it as feeling as if his body was super-dense, heavier than lead, as if he was heavier than the earth, as if he might sink down into the ground from his intense weight, as if the earth could not support such heaviness. He felt like clay. He was not depressed nor moody, just purely heavy.[1] In fact,

[1] There is a common myth that if the earth were to stop spinning, we would all go flying off into space. Modern science says that the opposite would occur: if the earth stopped spinning, its terrific mass would suck our tiny bodies down into its core. It is the spinning of the earth that keeps us "floating" on the surface. Is it possible that the joy and dynamic of this spinning earth are related to the dynamic in a healthy brain that enables us to defy gravity and entropy and to turn our movement thoughts into physical reality? Is this "lightness" a role of dopamine in the motor-initiation area?

In contradistinction to the utter heaviness that one feels in a dopamine deficiency, as if one's atoms are merging with the atoms of the earth, regard the great saints who cannot help but levitate when their thoughts of God transport them into bliss. Saint Joseph of Cupertino would literally float off the ground every time he walked past the image of the Mother in the Basilica of St. Francis in Assisi, Italy. Saint Theresa of Avila, Spain, was often chided for her inability to stay on the floor when she was

despite the alarm or despondency that usually accompanies immobility when one still has Parkinson's, the immobility that he felt was viewed as something mildly interesting. Certainly there was no panic associated with this experience, nor any particular joy. It was just that he couldn't move. Usually, in three minutes, or as long as ten, he would feel a return of uplift, and he would be able to stand up and walk completely normally again.

Understanding the On-Offs of undrugged recovery

Steve's episodes with immobility were not caused by fatigue or any emotional content. When he stopped moving it was because he had simply dropped just below the dopamine threshold for movement. Because his adrenaline had been turned off during recovery, he had no way to generate movement when the dopamine ran low. Apparently, his dopamine was still high enough to support equanimity of mood and thought at these times, but not high enough for movement. He was right at the threshold.

Most unrecovered PDers (except for those having On-Offs from drugs) do not go back and forth between effortless movement and stone cold motionlessness. Most PDers, including most of those who are taking medication, are able to use dwindling supplies of adrenaline, a neurotransmitter that enables one to keep going at least somewhat, even when the body is physiologically on empty. However, after recovery, the adrenal supply sets itself to near zero for months, if not years. Therefore, when a recovering person runs out of steam, he may be utterly incapable of moving. The adrenaline override is simply not there. And so, without emotional content or fatigue, when the new dopamine supply is temporarily used up, a person who was moving perfectly normally moments earlier may come to a complete halt, and cannot initiate movement again until the supply has been restocked. There will be no emotional override at these times, no sense of urgency to "keep on keeping on." The emotion may be one of bemused reflection.

Many former PDers have remarked on how events that would have been alarming or cause for agitation in the past become emotion neutral after their recovery. "I just don't get all worried about every little thing anymore," is a common, post-

---

overcome by Divine love. Is it possible that these witnessed events of lighter-than-earth movement are the opposite of the denser than earth feelings that are connected to dopamine deficiency?

Is it possible that the spinning of the earth that allows us to move, overcoming the powerful attractive force of gravity, might be a force that can be augmented or diminished by the forces generated by our thoughts as we are attracted towards or away from the earth, towards or away from Joy?

If this is so, then the feeling of density of PDers may be all too real and is not a mere figure of speech. This dopamine deficiency and its heaviness might be the opposites of the lightness and – maybe – a dopamine saturation – that are manifested by many of the great saints. If dopamine is in fact the neurotransmitter of spiritual ecstasy, one begins to see why the body so carefully guards and regulates dopamine levels: too much, and a person goes into ecstasy and cannot perform their earthy jobs; too little, and a person is plunged into the hideous world of pure matter, in which no vibrations of love or higher emotions can penetrate. A delicate balance indeed!

And while we are this far from the subject, I will share with you a favorite story about St. Teresa of Avila. She had an immediate and constant relationship with God – she was always sharing with Him in the language of her heart. Did you know, it was through her work that it became legal for Catholics to pray silently, using their own words, instead of the previous requirement that all prayers be repetitions of the approved catholic prayers? At any rate, one day while she was journeying, making a difficult crossing of a river in the wintertime, she fell into the river, soaking herself through. She stood up and spoke severely to God, "Why did you do that to me?" He chortled, "That's how I treat my friends." To which she snapped back, "That explains why you have so few!"

Parkinson's attitude. Therefore, the On-Off from *native* dopamine deficiency that may occur during recovery is not necessarily an emotion-inducing or an emotion-triggered event.

Slow increase in dopamine

As people recover, they increase from having mere glimpses or glimmers of movement, lasting a few seconds or minutes, to having hours at a time when they can move. But regardless of how much dopamine they have, when the native dopamine supply gets used up, dipping just below the threshold level, they can find themselves abruptly immobile, with a heaviness that feels denser than the earth. Unlike when they had Parkinson's, there is no longer adrenaline flooding the system, and so, when the dopamine runs out, they come to a complete halt. This is different from the Ons and Offs of medication. There is neither dyskinesia nor drug withdrawal trauma. There is just simply an abrupt transition during movement from effortlessness to impossibility.

This can be a challenging stage of recovery – it's impossible to know just how much juice is in your brain reservoir. There's no way to know how long you will be able to walk or drive.

Usually, once a person gets to the point where there is an hour or more worth of dopamine able to be stored up in the brain, they find that they can recharge just by resting or sleeping for a few minutes or a few hours, and then they can move again. However, this early-in-recovery supply is minimal – there are no hidden reserves.

Combining the glimmer with medication

This stage of recovery was particularly treacherous for our medicated patients. Back when we still were taking medicated patients, many of them reached the stage of recovery when they were having glimmers of native dopamine when there was no overt medication in their body, such as first thing in the morning or during the wee hours of the night.

Patients who experienced these glimmers of movement, only to fall back into motionlessness, simply could not refrain from taking their next pill of the day. No matter how often I suggested that these increasing moments of dopamine meant that their body had switched over to making its own dopamine, they were loath to sacrifice any movement time by making decreases in their medication. The typical refrain was, "When the new dopamine can go all day long, then I'll reduce my drugs."

That day never came, of course. If the brain is capable of making dopamine again, but a person insists on shoving dopamine-enhancing drugs into their body, the body appears to learn reliance on the drugs. It appears as if the body will only make as much dopamine as is necessary. In fact, it may be necessary to challenge the brain via dopamine insufficiency for up to ten frustrating, slow-moving weeks before the brain will increase its dopamine producing potential.

We simply don't know what the mechanisms are for regulating dopamine quantities in a person who is recovering from Parkinson's and also taking the medication. In our small sampling, most people who started having these glimmers who were taking the medication were never able to make further reductions in their medication. Those who did, like Becky, went through agonies.

Rose

Although Rose had reduced her medication considerably prior to the appearance of native dopamine, she was never able to make another reduction in her medication after she started to have moments (which slowly extended up to forty-five minutes before she left our program) in the morning of perfectly normal, pre-pill movement.

Sonny

Sonny started having glimmerings of native dopamine. For years he had been unable to move at night. However, less than a year into his treatments, he noticed that at three in the morning, a time when he had previously been the most rigid, he was able to move. He could not move much; at first he could only move his least affected limb, his right arm. Over the course of the next year, while we worked to remove more blockages and restore feeling in his long-numbed left side and right leg, he slowly regained use of these limbs during the night as well. The period during which he could move grew longer. He would wake at two o'clock in the morning and move his legs and arms, slowly and weakly, but with ever-increasing accuracy. Sometimes he was awake for hours, just practicing moving. Sometimes he used his new ability to move at night to readjust his blankets by himself. For years he had needed his wife to move his blankets for him and turn him when he cramped up. Now he was able to lift one leg over the other and back again – but only in the wee hours of the night, when his drug crash was over and his native dopamine was working.

He had slowly eased his medication from over 1200 mg/day of levodopa down to around 300 mg/day. At this level he no longer had the dyskinesia or cramping that he had at higher levels; and his Off times were less painful, but he did still have Ons and Offs. Once he started having these glimmers of nighttime movement, he became more reluctant to further reduce his medication because he did not want to have more Off time than he was having, despite the fact that, following every one of his past reductions, he had recovered all the movement that he had prior to the reduction. When he broke his hip, he had been vacillating on the verge of a drug reduction for nearly two months. He was grateful that the broken hip gave him a reason to stop the reductions. Prior to the glimmers of nighttime dopamine, he did not suffer so much with each drug reduction. After his nighttime movement began, each reduction was more miserable. Strangely, he never had the full-blown drug withdrawal symptoms, but he did appear to be increasingly attached to the drugs as his nighttime movement increased.[1]

The glimmers at night never got any longer than four or five hours, which, coincidentally, was just long enough to enable him to move in the night, but all ability to move ceased about twenty minutes after he took his first pill in the morning. He couldn't move after that until his first pill of the day "kicked in" forty minutes later. It was as if his body was darned if it was going to make any more dopamine than it absolutely needed. If it needed to make enough dopamine to make it through the long, nighttime hours with no drugs, it would do so, but only exactly enough and no more.

---

[1] Sonny's changes in recovery and drug addiction moved along at a slow pace. Some patients made lightening fast transitions from undermedicated to addicted. There will be more about these differences in chapter 24.

After Sonny broke his hip he made no more decreases, but instead, increased his medication. He started having worse Offs and less predictable On times (see Sonny's charts in chapter 18). As indicted by his charts, this increase caused him to become grossly overmedicated (severe adverse effects of On-Off, freezing, and dyskinesia). His case was interesting especially because he had made slow, steady decreases for such a long time, and assumed that he would be able to continue making these decreases until he was off the medication. He was not able to. We now assume that his window of opportunity for getting off the medication was probably the first few days when he started having this strange new ability to move all by himself at night, using this new type of slow, languorous movement during which his arms appeared to "move all by themselves."

Conclusion on glimmers

We have learned that if a person starts having these glimmers of native dopamine and is still taking the medication, it is too late to avoid addiction. Not one person has been able to get off the medication without going through the sort of hell that Becky described in her journal if they are still taking ANY antiparkinson's medications at the time that the first glimmers of native dopamine appear. This creates a dilemma for any medicated PDer who wants to be in a recovery program but is thinking of delaying drug reduction until starting to feel some positive signals of actually recovering; if you can tell that you are recovering, it's already too late. On the other hand, if there is a chance you are not going to recover, why should you go through the bothers of drug reduction?

Therefore, because it is unreasonable to stop taking medication before knowing if a recovery is forthcoming, and because, once recovery becomes apparent, the brain damage due to addiction will have already started, we do not recommend a recovery program for PDers who are taking the medication.

DOPAMINE DEPLETERS

Another challenge to people going through recovery, especially if they have been accustomed to taking drugs, is the dopamine depleters: cold, illness, and social stress. During the time when glimmers of movement are just beginning to appear, and dopamine levels are trembling on the threshold, almost any sort of physiological challenge will cause the dopamine levels to drop just below the threshold level. When this happens, it appears as if there is no dopamine in the system whatsoever. This can be emotionally devastating for a person who has finally started to experience the long-awaited recovery from this "incurable" illness.

A person who has ever taken the medication who starts recovering but who then has any moments of immobility will typically assume that all of his seeming progress was false, merely psychological, and that he actually still has Parkinson's disease. Even if the drugs don't call to him during these times of brain stress, his friends and his doctor may insist that, when he took his drugs like an obedient fellow, he never had any problems. They may use powerful emotional leverage in their attempts to either coerce or cajole him into resuming his medications.

The trio: Cold, illness, and emotional stress

There are three primary factors that can deplete dopamine so that the brain drops below the effective level: getting chilled (or any exposure to extremes of temperature), illness, or emotional stress. If this happens, it will seem exactly as if a person has profound Parkinson's disease. In this stage of recovering from Parkinson's – when there is no adrenaline and just barely enough dopamine – when the movement stops, there is no way of knowing, just from looking, whether or not a person is within a few hairs of normal movement or a thousand miles away. A person in this condition is just like Steve, sitting by the side of the road, as heavy as lead, with this exception – if the immobility is due to one of these three dopamine depleters, it may take days instead of minutes for the dopamine levels to be restored.

Cold

We have seen this repeatedly, and yet it seems very difficult for patients to understand – the effects of these three dopamine depleters can last for several days after the event is over. A full six months after Steve had experienced what he'd imagined was the last of his sudden droppings of dopamine below the threshold, six months of consistently reliable movement, apparently free from any semblance of Parkinson's, he was out on the prow of a boat on a crisp, windy February day. His socks were wet, but everyone was having a good time and he stayed on the deck for six hours. He was cold for the rest of the day and didn't really get warm until he got home and had a hot shower. Three days later, going for an amble, he recognized the warning signs that he was rapidly approaching zero dopamine again. He made it as far as a bus stop and then sat motionless for ten minutes. He was able to start moving normally again after his ten-minute rest, but he was stunned that he should have had a depletion event when he had been doing so well for six months with no signs of dopamine deficiency.

When he asked me about it later, I asked if he had gotten chilled or had a mild flu in the week preceding the event. He recalled the chill on the boat deck but wanted to

dismiss it: “That was three days earlier, and I felt fine afterwards.” But looking back, he had not gone walking or in any way challenged himself in between the chill on the boat deck and the subsequent shutting down. He had probably been just barely at the threshold, and when he went for his walk, he dropped below and experienced, once again, the absolute nature of the dopamine threshold.

Here is the point I am working up to: had he ever taken the medication, he would have been tempted at this point to say, “I had an unexpected episode of immobility after six months of feeling perfectly normal. I guess this is proof that I still have Parkinson’s after all. I will never be completely better; I might as well be taking the drugs.”

We have seen this scenario over and over.

Life sustaining measures have priority over movement

It may seem baffling, at first glance, that a chill or flu can have such a lingering effect on the dopamine levels. But it appears that the crucial life-sustaining functions of temperature regulation and supporting the immune system are high-priority items for the body. It appears that, in the case of these high priority events, the limbic system can sacrifice its dopamine content rapidly, just as it does in the case of an emergency. However, as we have already seen, the restoration of limbic dopamine after the crisis can take two or three months. If a person has recovered from Parkinson’s disease to the point where he has just barely adequate dopamine, he may appear (if his muscle damage has been repaired), for all intents and purposes, completely normal. However, if his dopamine level drops just below the threshold, he will be utterly unable to move.

Any event that decreases his limbic dopamine might create an illusion of profound Parkinson’s that can last for weeks or a few months, until he body restores its dopamine reserves, after which he might appear perfectly normal again.

These events are probably a good thing in the long run: following each of these events, it appears as if the brain cautiously decides to up the dopamine levels just a tiny bit more. The brain, ever efficient and exacting, will not create more dopamine than it needs. However, these crashes, similar to the stunning, complete collapses of a tired infant, seem to be indicators to the brain that the dopamine-making processes need to be increased by just a little bit more.

During the entire time a person hovers at the very threshold of movement, a person is at risk for seemingly inexplicable crashes. If there is a rapid depletion of dopamine during times of either obvious or subtle body emergencies due to the stresses listed above, interludes of immobility, worse than their PD ever used to be, may come and go, or even appear permanent for a period of several months. Due to the slow restoration of dopamine, these seemingly brief, insignificant events may explain the terrific, long-lasting crashes and immobility that we have seen in people who had thought they had recovered but who have appeared to have a Parkinson’s setback following an infectious disease, a chill, or an emotionally stressful event.

Taylor Paul

If you will forgive the inclusion of another case study, I would like to give an example of this. Taylor Paul, age 69, diagnosed five years earlier, had been taking 400 mg/day of Sinemet when he started our program (in summer of 2001). He began reducing his medication and steadily reduced down to 25 mg/day (one fourth of a 25/100 tab). He

then stayed at 25 mg/day for six weeks before making the final reduction – down to no medication – in September of 2002.

When he was at the 25 mg/day level, he fared pretty well; he lived alone, and he was able to perform all the activities of daily living, plus putter around in his workroom and do his beloved projects. Shortly after his reduction to no Sinemet, he went through a period of drug withdrawal that lasted fourteen weeks, during which he felt shaky, had festinating gait, and often woke in the night with horrible, even violent shaking. But after fourteen weeks, he felt he had turned the corner: he was drug free and feeling better every day.

Two months later he started feeling worse. He felt weak and shaky. He just didn't feel right. He had urgent urination throughout the day, and slight burning pain when urinating, but assumed that it was a recovery symptom. His doctor told him that it was the Parkinson's and suggested taking the drugs again. Taylor Paul refused and went home, but he kept feeling worse and worse.

After a month of increasing weakness and shakiness, his son became alarmed and demanded that I do something. I suggested that there might be many causes for the decline and to find out if Taylor Paul had been exposed to colder than usual weather, any illness that was going around, or any unusual stress. The son asked Taylor Paul, but Taylor Paul couldn't think of anything that was unusual.

By the end of the next month, Taylor Paul was profoundly weak, dizzy, confused, and feverish. He was unable to care for himself, and he wasn't eating. Finally, at his son's insistence, he started taking Sinemet again. The MD suggested starting at 500 mg/day (five 25/100 pills), more than Taylor Paul had ever used in his life. After three weeks at this drug level, Taylor Paul was still worsening. Finally, after his daughter came to his house on New Year's eve and found him paralyzed, utterly unable to move, she called the ambulance.

At the hospital, they chided him for having stopped taking his Parkinson's drugs, even though he had been now taking 500 mg/day for three weeks and his condition had continued to worsen. They did a round of blood work, and the blood work confirmed that he was seriously ill – but not with Parkinson's. His white cell count was extremely high. He had a body-wide infection of unknown origin. They put him on intravenous antibiotics. After several days of this, they switched him to pill antibiotics, but they kept giving him the 500 mg/day of Sinemet. He was still in bed, unable to get himself in or out of bed or to walk more than a few steps, when he started having facial grimacing from the medication and feeling stoned and unfocused.

When he told his doctor that he was starting to have spasms from the medication and maybe the med level was too high, the doctor retorted that he obviously needed the drugs – after all, he could barely move.

Taylor Paul tried to point out that he was having symptoms of overmedication, that he had been moving well on only 25 mg/day of his meds a while before, and that possibly the reason that he was still too weak to move was the infection. His face was still gaunt and ashen from his long-term bacterial infection. He had some kidney damage, probably from the months of untreated illness. Though his body had been ravaged by an infectious disease, still, the doctor told him that his main problem had to be his incurable Parkinson's disease, and that the best treatment was Sinemet.

After Taylor Paul's white cell count dropped back down to a healthy level and he was able to walk a few steps, he was discharged to his daughter's house. At this point, because he was no longer so sick, his dopamine levels were starting to climb back up. The extreme levels of Sinemet were starting to accumulate (it had now been six weeks), and he suddenly, for the first time, had full limb dyskinesias. They were frighteningly severe – not only his face and arms but his throat went into spasms: he could not swallow, and for one terrifyingly long night, he feared he was going to choke to death. His doctor should have been curious about the fact that he was having dyskinesia even though he was still too weak to perform normal movements. He could not walk or even get up from a chair without support, and he could barely move his arms. His muscles had deteriorated during his illness. However, despite this immobility of conscious movement, his brain was employing dyskinetic movements both huge and small in response to the drugs. Paradoxically, he couldn't yet move, but his drugs were causing him to thrash about uncontrollably.

He decreased his medication the next day down to 400 mg (four 25/100 pills). He felt less confused by evening. After several days, he decreased further, down to 300/day. He felt better still, though he still felt overmedicated, his arm was still twitching, and his eyes had that characteristic Sinemet glow.

At this point, he was still extremely weak. The doctors had concluded that his infection had probably been a bladder infection that had gone into the kidneys. He had probably been fighting it for months before he collapsed. The pallor in his face was just beginning to ebb, and he was starting to feel as if he had been seriously ill, which he had, but that he was going to recover. He also felt deeply discouraged by the fact that he was going to have to go through drug reduction all over again. He feared that the five weeks at 500 mg/day had probably done him real damage, creating an addiction. He was correct.

His children, watching the whole thing closely, decided that the doctor had been wrong about the cause of immobility during the recent hospitalization being Parkinson's disease. They felt that his immobility had been caused by his infectious illness. They recalled that prior to the illness, Taylor Paul was moving fine on 25 mg of Sinemet. Also, more damning, the 500 mg/day of Sinemet, more than he had ever taken in his life, had not helped his ability to make conscious movement whatsoever, even after five weeks. Certainly, after he started the Sinemet, prior to going to hospital, he had continued going rapidly downhill. The thing that had reversed that downhill trend was the antibiotics.

However, his doctor had completely ignored the possibility of an infectious illness because of Taylor Paul's pre-existing diagnosis of Parkinson's disease. When Taylor Paul was dismissed from the hospital, the doctor insisted that he never wanted to see Taylor Paul playing around with his Parkinson's medications again, as if the medication reduction had been the cause of the illness.

We have seen this over and over. When a person who used to have Parkinson's disease has a new condition, whether it is stroke, infectious illness, grief from a profound loss, or anxiety from social stress, the doctor will tell them that the subsequent heaviness of body and spirit is simply a symptom of Parkinson's disease. The new illness will not be treated. The answer will be, every time, "Increase your antiparkinson's medications."

In Taylor Paul's case, I learned that there had in fact been the entire trio of depleting forces at work. He had started feeling lousy in November – he lived in Canada, where the late autumn nights grow very long and the weather can be abruptly cold as it

shifts into winter. Next, he had just decided to sell the family farm. His wife had died five years earlier, and his adult children were not interested in continuing the family farm. He himself had continued to play an active role in the farm up until they decided to sell. The farm had gone on the market in October, just before he got sick.

When I discussed it with him the following February, he insisted that he wasn't emotional over selling the place, that it was too much work, and that everything changes over time, but there was a set to his chin and a look in his eye that said more than his words.

There you have it: it was growing colder and darker, he was under emotional stress, and then illness struck. With his brain just barely able to make enough dopamine to keep him at the threshold, these three dopamine depleters used up so much dopamine that by New Year's Eve he was literally paralyzed.

Dangerous illness may be ignored

Again, the risk is not so much that a person may, for the rest of his life, be somewhat susceptible to these factors, and that his brain may never be restored to the dopamine excesses of his youth.

The risk is this: anyone who has ever taken the medication who subsequently succumbs to any of these other factors may never be taken seriously by his physician.

If he is no longer taking his medication, even if he has not used it in years, even if he was doing perfectly well without it, at the first sign of any trouble, his doctor may well blame the absence of PD drugs and refuse to look more closely into the matter.

A person who has never even taken medication is much safer. Even though he has the PD diagnosis on his chart, it is highly unlikely that a person who never needed so much as a sniff of PD medication would suddenly become paralyzed. Such a person might point out that he is early stage and doesn't yet need meds. The symptoms of a person with a history of medication who has stopped taking his drugs are not only going to be ascribed to the cessation of meds, but the person will be considered to be mentally incompetent – only a nut case would imagine that he no longer needs his PD drugs.

If Taylor Paul had not had blood work done when he entered the hospital, he might have died of kidney failure due to staph infection. His doctor would have erroneously signed the death certificate, "Cause of death – Parkinson's disease."

Therefore, due to the very real possibility of death, due to misdiagnosis of a life-threatening condition by a doctor who assumes the only problem is a need for PD drugs, we do not recommend a recovery program for PDers who are taking the medication.

THE DEEP SLEEP

The deep sleep phase of recovery is included in this section on dopamine depleters because the effects of this phase on friends, loved ones, and doctors can be the same as in the case with Taylor Paul.

As you may know from reading the *Patient's Handbook*, there comes a time in recovery when a person is absolutely unable to move. This profound inertia usually occurs between 7 and 9 in the morning, although it can occur at other times instead or as well.[1] A person who is undergoing this bizarre daily zombification may decide that he is getting much, much worse, even if he is able to get up and start moving normally every day at 9:15.

This deep stillness in the mornings occurs in nearly all recovering patients, unmedicated or medicated. This makes it hard for a medicated patient who has recently reduced his meds to know if he is having Off time in the morning because of the deep sleep or because the med levels are too low.

It was this stage of recovery that started Zoe on her rapid drug increases. The terror induced by this weird, two hour immobility every morning like clockwork caused her to triple her morning dose, even though I assured her that the deep sleep thing was normal, and even though the increased drugs did nothing to ease the paralysis-like condition. She balked at the prospect of surrendering to the sleep and turned to the drugs instead.

Other patients have also succumbed to the lure of the drugs when they began to have the hours of immobility.

Anyone who has taken the drugs will be terribly drawn to resume taking the medication if the weakness phase stretches into weeks, then months, and possibly even years. This phase is an unpredictable span that may partly depend on how advanced the PD was – a quality that is impossible to measure in a medicated patient.

Of course, when such a person does resume the drugs, "just for a few days," he is most likely lost for good. Becky had been feeling just slightly less vigorous than she liked when she decided to start taking the drugs again, "just for the weekend."

Therefore, because of the heightened risk of returning to drugs during the Deep Sleep or the Profound Weakness stage – the latter has been, for some patients, more painful and debilitating than Parkinson's – we do not recommend a recovery program for PDers who are taking the medication.

[1] The Asian understanding of this is that most repair work to the tissues related to the Stomach channel usually occurs between 7 and 9 in the morning. Each of the channels has a certain two-hour time zone during which the energy in this channel surges. If there is massive repair work to be done in this channel, the body may fall into a motionless paralysis during this time. Surgeons have long known that certain surgeries have better outcomes at specific times of the day. They have ignored this powerful information, for the most part, because it doesn't fit into their theory of anatomy. However, this awareness has been in the records of Asian medicine for over two thousand years.

TREMOR

Tremor is a symptom that merits a special paragraph of its own. Just as long-hidden pain can become exposed during recovery, so can long-suppressed fear, which the body manifests as trembling. When a person with tremor recovers from Parkinson's, his tremor may continue for months or years.

If a person took antiparkinson's drugs for very long, even if he did not have tremor to start with, he may very likely develop tardive dyskinesia, which has a similar appearance to tremor.

As long as there is tremoring or tardive dyskinesia, even if all other Parkinson's symptoms are gone, a person who has once used drugs might be tempted to resume the drugs to "control the tremor." Considering that the tremor may actually be tardive dyskinesia, the drugs may worsen the condition. By the time it becomes apparent that the drug is not helping the ticcing but rather worsening it, the addiction will have begun, and with it, long term damage.

LINGERING ONS AND OFFS

Another difficulty for the person who is trying to decrease his medication is the appearance of Ons and Offs. While some people find that their Ons and Offs decrease when their medication gets down to a non-threatening level, other people find that they have Ons and Offs right up until the time when they take their final pill. This may be a brain habit, but it may also have to do with altered thresholds and baselines.

As you have seen above in the section on dopamine depleters, even a person who never took the medication may have moments of "off" time during recovery, as they dance back and forth across the threshold. While these immobility events are very different from the drug-induced Offs and freezings, it can all become very confusing for a person who anticipated a predictable cycle of drug decreases.

Since unmedicated PDers do not have On-Off cycles, and the only PDers I have seen who have these distinctive, brief periods of off time following an exertion that depletes dopamine are those who are drug-free and have in every other way recovered, it should seem obvious that *dose-related* On-Offs are caused by the medication and are not a symptom of worsening Parkinson's.

If a person does begin to recover and is still taking the drugs, the addiction response and the increased severity of adverse effects may cause drug-induced symptoms such as On-Offs to increase, rather than decrease.

And yet, despite these arguments, those people who are still taking medication after exhibiting signs consistent with recovery who still have On-Offs usually use the Ons and Offs to justify an increase in their medication or else refuse to make any further decreases until such time as the On-Offs have lessened. This maintenance of medication at levels that cause On-Offs or any other adverse effects may lead to worsening of those adverse effects and heightened addiction symptoms.

Therefore, we do not recommend a recovery program for PDers who are taking the medication.

SWITCHING

Many people have noticed that shortly after they take a pill, before the pill begins to work, they go through a brief period during which they feel much, much worse than they did before taking the pill. They may freeze or have dyskinesia or dystonia of such ferocity that they will scream out in agony. This phase is short-lived, and as the medication begins to make its presence felt more, these movements and the unpleasantness may come to an end, and the good On may begin. Then, when the drug begins to wear off, there may be another period during which severe freezing or terrible dystonia or dyskinesia once again occurs. When the benefits of the pill come to a complete halt and the basic Off begins, these symptoms of enhanced freezing, muscle tension, or dyskinesia ebb.

It can feel, in the period just before the pill really takes hold, and again just as the pill begins to wear off, as if the body is more traumatized, less able to move correctly, than it is during a full Off, a full On, or if there is no pill in effect at all.

We named this period the Switching phase because it occurred when the brain was switching from non-pill status to pill-affected status and vice versa. We have heard this phase also referred to as diphasic dyskinesia.

We have no explanation for this very difficult period which usually only lasts for two to twenty minutes. It seemed to many patients as if their brain was struggling with the medication, resisting it; every time the meds went On, they had to endure a struggle for a short while, until their brain's resistance was overcome by the drugs; once the drugged condition was established, there was no more fighting. Then, when the drugs were wearing off, the brain was inspired to take up the fight again, with the resultant difficult movement, either excessive or inadequate, and then, when the drugs were gone and the body was back to its more natural state, the fighting ended – until the next pill was taken.

Sometimes, as with the mouth burning switching that Rose experienced (as described in Chapter One), the switching would be very painful; when the switching ended and the full strength of the medication kicked in, you could almost see her eyes glaze over in relief. A smile spread across her face, and it was obvious that she had no awareness of pain anywhere. As the medication wore off, the mouth pain would resume, and then, when the medication was completely gone, such as when she woke up in the morning and was moving about on her own dopamine for half an hour to an hour, there would be no mouth pain or only a mild version. After she took a pill, as her own dopamine appeared to be turned off and the medication dopamine was just starting to take over, the searing pain would come again. Depending on the particular drug side effects that a person has, they may see that these side effects are at their very worst while the brain is making what appears to be a transition from native dopamine to the medication-induced dopamine.

Incompatible systems?

Observing people making the transition from their own dopamine, however inadequate, to the drug-induced form of dopamine, it really did seem as if the brain was having to make a switch, as if it was moving from one system to another. There appeared to be an incompatibility of the two systems, as if they could not work smoothly side by

side. Most people only noticed this switching phase when their medications levels got high enough to have distinct Ons and Offs.

People who are in the early years of taking these drugs, who are still getting continuous coverage from their medication, do not experience the switching. However, the people who have switching problems during the transitions from their natural dopamine, however inadequate, to the drug form of dopamine, might feel as if the brain is being buffeted between the two systems, creating the extremes of pain and erratic movement.

Unnatural dopamine?

The switching problem suggested that there are two different forms of dopamine or two dopamine systems. It appeared as if the dopamine or the dopamine distribution system that derives from the drugs is somehow different from the dopamine that is produced and distributed in the brain. We have no chemical or radiological evidence to support this, but it did seem almost as if the two dopamine types, native and pharmaceutical, did not work in an additive fashion, but rather in a combative fashion.

Glimmers provide an answer

Our theory of two different types of dopamine, or at least two different dopamine systems, was finally proved to be true: when recovering patients began having noticeable glimmers of native dopamine at levels high enough to breach the threshold to good motor function, after long, Parkinsonian years without it, they invariably noticed that the sensation of the natural dopamine was very different from the dopamine feeling that came from the medications.

This noticeable difference suggests that either the location, receptivity, chemical structure or some other aspect of native dopamine is different from the pill-provided dopamine.

We can only guess at why this is. It may well be that the switching occurs when the brain, flooded by the outside dopamine, shuts down the native dopamine factories, so as to protect against dopamine excess. During this time when the native dopamine is shut down and the pharmaceutical dopamine is not yet positioned, there may be the worsening of symptoms that is noticed during the switch.

Switching rigidity

This idea might explain why the Offs just before a pill starts to work can be much worse than the pre-pill condition. Even if a person with this pattern can move slowly before taking his first pill of the day, or in between pills, he may find that he has utter rigidity after taking each pill, before the pill begins to work. This shut down may be a learned, protective mechanism.

Switching dyskinesia

Other people have just the opposite response: while the switching from one system to the other is happening, they have very powerful and uncontrolled movements during the switching – their arms flail, their legs spasm, and their whole body tenses with painful cramping. We are guessing that those people who move too much are showing a brain pattern where both dopamine systems are functioning at the same time, creating an

excess, and that the brain is trying to rid itself of the excess through the frenzied movements.

In those people who become absolutely motionless or some degree thereof during a switching phase, it may be that the brain is choosing the "Shut Everything Down" method of dealing with the dopamine excess. In those who have violent spasms, their brain may be in a "Get Rid of It" program.

Switching with rigidity and dyskinesia

Some people will have both patterns, unpredictably. The first pill in the morning may be closely followed by a complete cessation of movement, lasting twenty minutes, after which the medication begins, and the slow, unmedicated movement of the early morning is replaced by the drug-induced movements. Then, when the pill begins to wear off, there may be a brief period of frantic movement, or there might be a period of freezing that was even worse than at the beginning of the pill's onset. As the day wears on and the drugs build up, the switching may become more unpredictable with each dose.

People who have painful symptoms, such as the burning tongue and mouth, light-headedness, or hallucinations, may find that all of these side effects are at their very worst while the brain is switching from one system to another. During recovery these switching-related problems may become much, much worse. As the brain makes dopamine in greater amounts, if the person is still taking some amount of dopamine-enhancing drugs, all of the side effects might worsen.

However, if and when there is an end to the switching phase, bringing with it the blissful deliverance from switching symptoms, Parkinson's symptoms, and the nasty reality of life itself, a person may feel just fine – until the drugs begin to wear off. Once a person has started having switching symptoms before and/or after his dose's effective period, these On times might be the only moments of the day when a person feels half human.

One cannot assume that, as drug levels are decreased, the switching will also decrease. In our experience, just the opposite occurs.

Hua To and Laurel both have made steady progress with decreasing their medication, but, probably due to starting recovery symptoms before getting off the drugs, they both have much worse switching now than they did in the past at higher drug levels, but prior to the onset of recovery symptoms.

Hua To

Hua To is now down to .75 Permax, 75 mg levodopa, and Amantadine from a high several years ago of 4 mg Permax, 600 mg levodopa, and Amantadine. His switching worsens with every dose of the day. By evening, his last switching and crash from his 4 p.m. dose can be so severe that he cannot move for hours afterwards. Very often, his wife has to spoon his dinner into his mouth. Finally, by bedtime, the switch and crash are over, and he is able to slowly get into bed by himself. If he needs to use the bathroom at night, he can do it, very slowly, by himself. In the morning, he can usually get up and get dressed and have breakfast – all very slowly – before the morning pill heralds its onset with an attack of freezing, during which he cannot move. Because he must work to help support his family, aging in-laws, and a young child, he cannot stop

taking these seemingly small, "sub-therapeutic" bits of medication which both allow him to keep working and may be, inexorably, destroying his dopamine-producing cells.

Laurel

I recently received an email from Laurel. She has probably the worst pill-induced dystonia I have ever witnessed. (Her consultant – the British word for "medical specialist" – presented a research paper on drug-induced dystonias in 1999: see appendix 10.) When her drugs work, she may be perfectly pain free. While she is switching, a searing pain burns and twists her hip and leg. If she is in a Build Up phase so that her afternoon or evening pill does not work due to excess, the pill that fails sets in motion a ferocious version of this pain – pain that is not touched by even the most powerful anti-pain medications, including apomorphine. As you will read in this email, her consultant, though acknowledging that the pain is due to the drugs, is not suggesting that she decrease the drugs but that she begin taking a powerful anticonvulsant.

Laurel experiences that particularly cruel joke of nature, the phenomenon noted earlier of symptoms of excess and withdrawal being the same. When the brain wants to protest against a drug decrease, it can employ the same vicious spasms and pains that it learned when trying to use up excess dopamine. Both events, a drug deficiency and a drug excess, are seen by the brain as a horrible problem. When either deficiency or excess occurs, the brain may select dyskinesia or dystonia from its learned repertoire of Horrible Problem Solvers. It will then institute these excruciating – but from the brain's perspective, effective – attention-getting methods.

In Laurel's case, her most painful dystonias occur with the afternoon or evening dose, as the drugs build up over the course of the day. If her threshold has risen too quickly, she will not get any On time from her mid-afternoon pill but will spend the entire three hours of maximum pill coverage in a state of scream-level pain. This condition will begin with a feeling as if the pill is just about to come on, but it never makes it into the good On. It suspends in the pre-On condition for a while, and then goes straight into the Excess Zone agony and holds there for several hours. Usually, if this intense pain condition lasts for hours, her evening pill will work better than usual. This may be due to the dispersal of dopamine from the limbic area during times of extreme pain, which is serving to alter the baseline or threshold. We have no idea what is actually happening in this case, but it is not uncommon for one of her agonizing "pill failures" to be followed by a fairly successful pill.

When Laurel is Off, she cannot move well without assistance. When her pills work well, she has a few hours of time during which she is animated and active. She, like Becky, seems to have that rare overview which allows her to observe herself objectively during her Off times and times of extreme pain. She usually gets reasonable (several hours) coverage from her first and second pills of the day, a small amount or none from the third, and, depending on what happens with the third pill, some or no On time from the last pill. However, there is a great deal of instability, and her daily Ons and Offs are affected by all of the other factors of daily life. She has two teenage children and a wonderful husband who have been supportive of her in all of her trials and her decisions.

You will notice in the emails that she has very brief Vacations or none at all. She is addicted; she started having symptoms of recovery over a year ago, when she was still taking 900 mg/day levodopa. Her brain now responds very quickly to any decrease in the

meds. Also note, she uses the words “switch on” or “switch off” to mean the onset or ending of a pill’s effect. She does not use the word “switch” in the meaning that I have defined for this book. She uses the word “tablet” and “carer” where we in the USA would use the words “pill” and “caregiver.” I have added comments and explanations in brackets.

Here are Laurel’s most recent emails:

January 2003

Janice,

Re: my email before Christmas asking about SSRI drugs - in the end I decided against taking them but instead gritted my teeth and decided to try reducing again. To remind you, I have had several attempts to reduce from 350 mg to 325 mg [levodopa in a Madopar format]. (During September, October and November, I tried reducing but always ended up returning to 350 mg.) I wasn't getting any better on 350 mg so on Christmas Eve, I decided I must grit my teeth and reduce again. I just want to explain what happened to see if you can shed any light on it.

Pattern of medication: 7:00 – 100 mg; 11:00 – 75 mg; 15:00 – 75 mg; 19:00 – 75 mg

*For first 4 days reduced by 25 mg/day every other day [and after that every day], - painful and poor 'Ons.'

*5th day good switch Ons with some dyskinesia at 11:00 but shorter length of switch on at 15:00 and 19:00. Slept well - didn't wake up until 5:45 in morning and then fell asleep until 8:00. Unusual.

*6th day less pain and quick switch Ons at 8:00 and 12:00 (doses an hour later because I'd slept in). 16:00 dose didn't switch on until 19:00 and stayed on for one hour.

*Next 6 days less pain - switched on well apart from 15:00. Didn't switch on at 5:00 but felt 'almost on' - during these sessions I had dexterity in my hands but my feet felt icy cold and 'cut off' - my right foot felt worse than my left even though usually my left side is my most affected.

*Next day noticed some dyskinesia at beginning of dose plus loss of balance. Treatment from T. [acupuncturist] She held my right foot first and then left. 5:00 switched On but more pain again when Off. Good night’s sleep.

* Next 3 days switched on each session and noticed feet weren't as cold when off but gradual increase in pain again.

* Another session from T. who felt Gall Bladder channel was clear and Stomach channel more responsive again but not clear.

Now a week after reduction have started having bad days again with extreme pain in my left hip and very poor mobility again. Unbearable.

Can you tell anything from this pattern? Why do my good days turn into such awful ones? Where do I go from here? Stay at 325 mg for awhile and hope things improve or reduce to 300 mg? I know it’s impossible for you to advise but does the pattern described give you any clues or do you need more information?

Love, L

I wrote to her that the poor mobility and extreme pain were probably due to drug reduction and might be unavoidable. I reminded her to keep in mind that she had been having intermittent mobility and extreme pain now and then prior to the decrease as well as after. As for the "good days turning into bad," I thought that might be because of medication building up, even though she had decreased her medication. The first four days, when she had poor Ons every time, were her vacation – she was sleeping well and actually feeling good, mixed in with symptoms of withdrawal – pain and poor Ons. After a person has become addicted, the theoretical separation of drug reduction phases can become a blur, with mingled symptoms of vacations and pills not working well. Also, keep in mind that sometimes people can go through the entire "ten-week" cycle of drug reduction in a matter of days.

Here is her next month's report:

Feb 2003

Dear JJ

Reduced again on 17th January - from 325 mg to 300 mg. For the first 6 days every other day, then every day.

*Day 1: took longer to switch on but only 3:00 pm dose failed. Bad night 10:00 'til 2:00 then slept well until 8:15 - unusual - had also turned myself in the night. [This is new – she had not able to turn herself over in bed while Off.]

*Day 2-7: On for first two sessions of the day, Off 3:00 and variable at 7:00. (Pain when Off almost unbearable: severe cramping in left buttock and the same twisting I was experiencing when you came over. Impossible to move myself at all when Off.)

*Day 8: Reduced every day. Switched on for 1 hour after first dose. Hour and half after second dose and Off rest of day.

*Day 9: 1 hour On after first dose but only On 10 minutes from second dose and Off for rest of day.

*Day 10: 10 mins of On after first dose but then On after 11:00 dose, off 3:00 and On briefly at 7:00. The pain was getting worse as the day went on.

*Day 11: Didn't switch On all day. Horrible!

*Day 12: Not On first dose but then switched On briefly each session.

*Day 13: Pattern changed again: On after pills #1 and 2 but Off rest of day

*Day 14 & 15: On mid morning only.

*Day 16: Off all day.

*Today (day 17) Slightly better day hence I can email you. Life is very difficult at the moment with the pain being almost unbearable. One thing I have noticed is that although the pain is centred in my left buttock and T. has been holding my left foot, my skin on my *right* foot has become very dry and is peeling. Is this significant at all? Over the last few days, by the end of the day, my right knee becomes swollen and has a lot of heat in it. I can't imagine being able to cope if the pain persists at this level for much longer. Do people whose main symptom is rigidity suffer more pain and have you had patients that have come through it? The trouble is that the time On is getting shorter and shorter and when I am Off the pain is excruciating (sometimes it feels as if I've been stabbed in my buttock and I'm sitting on the knife!) and I can't move my lower body at all. The

pain also becomes so bad that I can't move my hands or hold myself upright when sitting. Have you seen people in as much pain as this? Can you reassure me that I can come out the other end? Does the pattern above give you any clues as to what is happening and can you give any guidance as to what to do next?

Possible explanation

In this email she describes many patterns that appear in the drug reduction cycle. The first pattern is her slide, which ended on day eleven. During the slide, her pills became more unpredictable, but she didn't actually become immobile until the eleventh day. During the slide, her first pills of the day sometimes worked, but not the later ones. This is her most common pattern – she was having it prior to her reduction – and it corresponds to a Build Up. She was still having this Build Up pattern even after making a reduction and feeling as if her drugs were not working as well.

In other words, even after making a small reduction she was still overmedicated: by afternoon her pills were "failing" (she was freezing, or Off). However, her addiction threshold had grown so high (due to taking drugs while recovering) that this very small reduction made her feel worse, in general – typical for drug withdrawal. To sum up, she was experiencing a small degree of withdrawal suffering, and still manifesting Build Ups by afternoon. Despite her raised movement threshold, the Safety Zone, the level of drugs that sets in motion adverse effects (including freezing, stabbing, painful dystonia, and pill failure) is a somewhat fixed amount, and does not go up proportionately when the threshold goes up. Her threshold was high; to surmount it, her pills invariably ended up in the Excess Zone, even though she was reducing and experiencing drug withdrawal. She was deficient by habit, and Excess by the numbers – both deficient and excess at the same time. This is not unusual in the early stage of drug reduction, while the brain is still slowly coming to equilibrium with the new, lowered dose. After the slide is done, none of the pills might work, but at least there may be no symptoms of excess – unless this person's body is one of those that exhibits the same sort of symptoms in times of severe dopamine insufficiency as it has learned to do during dopamine excess.

Also contributing to the Build Up, in Laurel's case, is this problem: she has started recovering, and she does have glimmers of native dopamine, especially in the earliest hours of the morning. This is why she is sleeping better, especially in the dawn hours. This extra dopamine may be contributing to her morning Ons, which, in the early days of this reduction cycle, are still good. However, once she has expended this bit of native dopamine – which accrued very slowly during the night – the other doses of the day do not work as well. Possibly, due to the glimmers of morning dopamine, her morning doses are excessive even though she has reduced her medication. If so, that excess dopamine could contribute to a rising threshold, which would prevent the subsequent doses, the afternoon and evening doses, from working. (This is the Build Up.)

This pattern continued, with ever-decreasing amounts of On time, for nearly ten days. By day eleven, the effects of the first reduction and the second reduction were clearly visible. At this point, she became immobile, and the drugs appear to not be working whatsoever. She was now in the darkest part of the drug decrease cycle.

On days 12 through 17, she is having only faint On times. Day 12 had a Deficit in the morning, but by afternoon she had built up her dopamine level so that she was On in the afternoon and evening. This meant that she had, overall, a good day, one that was

hovering on the edge of just right. This nicely balanced day allowed her to build up some native dopamine overnight, so that the next day she went On first thing in the morning; but the glimmer of dopamine combined with the pills pushed her threshold over the top, and her other pills that day did not work.

Bear in mind, the threshold may be going up and down every day, but the baseline is still dropping, due to the decrease in pills set in motion over the preceding weeks. (As the baseline drops, her susceptibility to pain will soar. The stripped limbic area cannot protect against incoming pain signals.)

As her baseline continued to drop (due to dose decrease), her pain increased. She was no longer having On times during the day; these Ons had afforded her mental protection against the pain. Now that she was having no recesses from the pain, it was becoming much more fierce.

She was in a downward spiral, where pain causes a panic or sense of emergency, which may lead in turn to dopamine decrease, and because a dopamine decrease allows more sensation to get through, the decrease causes more pain. This is the classic dilemma of the person in drug withdrawal: damned if you do take drugs, damned if you don't. The animal mind reminds you that if you do keep taking the drugs, you will have intermittent respites from the pain. If you don't take them, you will have the spiraling. Your logical mind, if accessible, may point out that if you do keep taking the drugs, the pain will increase on its own, over time, as the body uses spasms to get rid of the excess dopamine.

I wrote to her most of the above, and also a note that I had no idea what was going on with her right knee, but possibly there was an old injury there, and as her mind was less drug addled, and now that she had recovered to the point where she could feel and heal injuries, her body might be presenting her with yet another case of long-forgotten damage.

Mid-February 2003

I got the following from her in mid February, together with a copy of her consultant's research paper:

> I don't know whether (his paper) has been published but you were correct – the paper was given at the Parkinson's Disease Society's research conference in Coventry in 1998. It is interesting because yesterday I went to see a new consultant in Liverpool (because I can no longer travel down to London) and she openly acknowledged that the long-term use of Levodopa caused the pain. She suggested that I have implants. Needless to say I am ignoring her. I read in a magazine that an herb Macuna Puriens can be used to treat Parkinson's and have found out that it contains natural dopamine. Do you know anything about it? I know I still need to reduce but would this lessen side effects in the mean time? Pain is still my biggest problem. Will be in touch later about my progress - going off!

March 2003

> I thought about quitting (!), but decided I was too 'chicken' to stop taking my L-dopa completely so instead I cut out the first dose of the day to see how I would feel with no drugs in the morning.

*The first day went quite well - it was a day when T. was treating me at 9:15 in the morning. I was obviously off but she said my feet felt much better than when I was medicated. I switched on with my 11:00 dose and was On for one and a half hours. Next dose failed and I was in a lot of pain. 7 p.m. dose worked briefly then to pain again. I slept reasonably and was relatively comfortable when I woke up.

*Day 2 - I noticed that I could move more easily than when I had taken my tablet and it hadn't worked. My carer commented that I felt much lighter to move around and in fact I could just about walk to my chair when I got downstairs. At about 10:00 though I had increasing pain at the base of my spine and down into my feet. It felt different than the pulling/cramping/twisting pain that I get with the meds but it was equally unbearable. The 11:00 dose worked, 3:00 and 7:00 failed and by this time pain was excruciating and instead of easing overnight as it usually does it increased. I felt forced to take my morning dose because I couldn't bear the pain.

*Day 3 - Excruciating pain all day which only eased slightly when my 11:00 dose worked. Strangely though I slept much better last night and felt more comfortable in the morning.

*Day 4 (today) trying again to do without my morning dose but already by 1:45 my pain is at screaming level again.

Any ideas or comments? Is there any other information you need? My GP prescribed clonazepam for the cramping - I know you can't advise about drugs but do you know anything about this?

Look forward to hearing from you

xx L

Pain relief medication

In my response to this email I commiserated with regard to the horrible pain, and asked if she could possibly ask her doctor for a mild pain reliever. She had tried pain relievers in the past for her spasms and they hadn't worked, but this pain seemed to be of a different nature. I also pointed out that T., her carer, and her own observations showed that her body felt lighter and able to move better when she didn't take the morning pill. That should be of significance. I had to wonder if the horrible pain that she was now able to feel – a pain distinctly different from the spasming, twisting pain that she usually got when her drugs built up or failed – was one of the underlying pains that had first started the Parkinson's disease. It might also be sciatic pain, brought about from the years of twisting at the hip – her body's most common form of drug-induced dystonia.

I suggested that she work very aggressively at dealing with this new pain. I suggested that she try counseling, hypnotherapy, massage, Tui Na, acupuncture, craniosacral work, or anything and everything that felt right in her heart, that might make her confrontation with this pain move along most quickly. I felt that a small amount of pain-masking medication might be helpful if she could keep the pain at a level where it was present but not breathtaking.

I made the further point that taking Parkinson's medication to treat hip and leg pain was not appropriate. Parkinson's medications should be used to treat Parkinson's

and pain medication should be used to treat pain. In general, most pain medications seem to disturb the frontal lobe dopamine levels much less than do the antiparkinson's medications.

Meanwhile, pain aside, it was clear from her new experience of a "lightness of movement" that the rest of her body greatly preferred having less levodopa. She seemed to move better than she had moved in a long time when she had *less* drugs in her system – she was now taking less than she had ever taken since she had first been prescribed levodopa. I therefore included in my response that, although I could not make a recommendation, I could suggest that she listen to her body on that count, at any rate.

As for the antispasmodic recommended by her GP, I reminded her that this new pain did not seem to be a spasm.

Her remark that "I could move more easily than when I had taken my tablet and it hadn't worked" refers to the painful Roller Coaster failures and violent freezing which usually included painful switching symptoms before and after the mighty spasms that constituted "failures." As you can see, once there is addiction, along with the painful, drug-induced dystonias, glimmers, and switching, it can become very difficult to negotiate a drug reduction. Even when the body feels better and everyone notices that the body is lighter and more relaxed, the raw pain that the brain experiences when its shroud of dopamine is wrenched away can turn a person away from thoughts of drug decrease. During the hours, days, and weeks of immobility and pain that might occur during drug decrease, the tortured limbic brain, recalling radiant memories of angel-voiced pills, may call on the mind with fervent longing to please, please, just take one more pill.

It is unreasonable to expect a person who is only having a few pain-free hours every day to decrease or stop taking the medication that provides those fleeting interludes. Therefore, we do not recommend a recovery program for PDers who are taking the medication.

"If they make you not then the better answer, you may say they are not the men you took them for."

Shakespeare's Much Ado About Nothing

22. QUESTIONS AND ANSWERS

THE COMMON POSERS FROM OUR WORLD OF PATIENTS

Q. If I stop taking medication will you accept me as a patient?

A. No. If I were to make such a condition it would be the same as me offering you advice about your medication. A promise to work with you if you stopped the medication would carry an implicit suggestion that you stop taking medication. To avoid any such impropriety, I will not work with medicated patients.

Q. Then why did you write this book, if you are not suggesting that I should get off my medication?

A. As stated in the first chapters, this book was written in the hope of making a contribution to this field of study. I also hope our findings may serve as a brake on doctors and patients, both, who hold the simplistic view that, "If one pill is good, two pills are even better."

Q. Since the switching effect occurs from having the brain make a transition, won't I be better off if I just stay evenly medicated throughout the day? That's what my doctor wants me to do. He wants me to take my pills every six hours, even waking up at midnight to take a pill.

A. Well, that seems logical. But it turns out to be incorrect. Research on rats was done to prove that they would have less brain damage if they took a certain total of dopamine-enhancing drugs throughout the day at low doses compared to taking the same total amount of drug in a few large doses each day.[1] This corresponds to what your doctor is recommending.

What the researchers found was surprising. The rats that were given their cocaine at low levels throughout the day sustained much more brain damage. The rats that were given the same amount of daily cocaine, but either all at once or in a just a few doses per day, suffered less brain damage.

The researchers hypothesized that the Off periods provided time between each dose for the medication to completely wear off. They further hypothesized that the damage repair that appeared to be happening in the rat brains was occurring during the Off times.

Again, the total daily amount of cocaine for both sets of rats was the same. The researchers guessed that the rats with the low-level-all-day-long doses – the kind of doses your doctor is trying to give you – were unable to do any sort of repair work or recovery in their brains. It seems as though, as long as there was *any* drug present in the brain whatsoever, the repair work could not commence.

[1] Details on this particular study, and many others that pertain, can be found in Dr. Glenmullen's *Prozac Backlash*. And, unlike me, Glenmullen provides good, thorough citations (reference information).

But if the brain was given a few periods every day when there was no drug present, the brain would go ahead and initiate healing work.[1] The result of these evident healing sessions was that the rats that had the equivalent of Ons and Offs fared much better in the long run than the rats that were constantly medicated.

Problems with the Controlled Release Pills

In another example, in 1997 the makers of Sinemet (carbidopa/levodopa) came out with their Controlled Release pill that would provide L-dopa in a slower, steadier stream throughout the day. The original pills just dumped all the L-dopa into the blood stream at once. With the original type of pill, a person got a nice On, followed by a crash, and these pills had to be taken at least four times a day. The manufacturers assumed that this new and improved pill, taken only two or three times a day, with a long, steady stream of slow-release effectiveness, would be a boon. They were wrong.

A visit to the website of the manufacturer two years after release of the pill showed that they had trouble with this new improved tablet. On the website, the manufacturer suggests that if you have gone from the original form of the pill to the new controlled release form and are having increased dyskinesia and/or all the other problems that are associated with this medication, you might do better by going back to the regular, all-at-once form of the pill.

They explained on the website that many people had experienced an increase in their problems, and that these problems might be due to the CR format and not to the dosage level of the medication. Their finding that many patients fared worse with the new, slow-release pills fits in with the brain repair situation that was seen with the mice.

Stimulants at night

An adjunct to your question is that your doctor has asked you to take your pills at night. The year 2000 edition of *Parkinson's Disease Questions and Answers* pointed out that it appears that people who take dopamine-enhancing medications at nighttime are more at risk for problems with hallucinations.[2]

This makes sense: in healthy people dopamine levels are supposed to be lower at night. And, as rat researchers have learned, by taking medication around the clock (and in the case of humans this might mean waking up at midnight to take a pill), the brain is prevented from having any rest and repair time to heal from the inevitable small bits of damage done during the day. The damage possibly accumulates over time, leading to the increased problems that people often have with the "improved," long-acting version of the pills. While the switching, crashing, and Ons and Offs may be traumatic, they may be signs that the brain is still struggling to maintain its health, at any expense. These valiant efforts by the brain may be signs that the brain is healthy and able to protest what is going on.

---

[1] J. Glenmullen, MD, *Prozac Backlash: Overcoming the Dangers of Prozac, Zoloft, Paxil, and Other Antidepressants with Safe, Effective Alternatives*, Simon & Schuster, NY.

[2] *Parkinson's Disease, Questions and Answers,* Hauser & Zesiewicz, Merit Publishing International, 2000, p. 29. Regarding hallucinations as the most common form of psychosis, the authors state that the "most significant risk factors for developing psychosis …[include] nighttime use of long-acting dopaminergic medications."

This is why some people find that the adverse effects of the drugs can be worse when they use "slow-release" or "long-acting" pills that give their minds relentless barrages of low level drugs from which there is no respite, no peace.

Q. How can I know if my Off is being caused by having my dopamine tank on empty, or if it is being caused by a short-term crash, or if it is being caused by a build up?

A. Chart your daily course. Without keeping track of your symptoms on an hourly basis, you have no idea what is going on.

Q. If people have worse side effects from their medication when they begin to make their own dopamine, why should they keep taking their medication after they start to recover?

A. Because they might die if they stop taking their drugs too abruptly.

Again, in case you missed that, *they might die if they quit too abruptly*.

If it was as simple as saying "if you don't want to take your drugs, then don't," then this book would not need to be written. But it is not that simple. The shock of drug withdrawal from the lightweight drugs such as opiates and cocaine can cause serious trauma. Abruptly stopping antiparkinson's drugs, which are tremendously more powerful, can push the drug withdrawal symptoms over the edge into death or long-term (semipermanent) brain damage.

Also, no matter whether a person is recovering or not, medication may have caused brain changes (parkinsonism). These symptoms, nearly impossible to distinguish from Parkinson's disease, make it hard for any medicated person to be certain that he is, indeed, recovering from Parkinson's.

A good thing to keep in mind is this maxim: any person who decreases antiparkinson's medication will probably display symptoms of parkinsonism when he stops the pills *whether or not he ever had Parkinson's disease in the first place*.

Therefore, a drugged, recovering person is not able to say with confidence, "I am recovering! I shall now stop taking my drugs." It is more likely that he will say, "There is still something wrong with me, I may as well keep taking the drugs."

As for your questions about "just stopping the drugs," even if a person doesn't die from stopping the drugs too abruptly, reducing antiparkinson's drugs too quickly might cause paroxysms of excruciating pain, paralyzing paranoia and hallucinations that can last for months.

Unfortunately, decreasing the drugs must be done slowly. This means that if a patient begins to recover and hasn't already finished slowly titrating down (very slowly reducing) his dosages of medication, he may go through some periods that will be much worse than if he had never recovered. His newly restored native dopamine levels will cause the medication to be excessive. Overmedication may cause the usual exalted moods, spasms, crashes, build-ups, insomnia, dyskinesias and dystonias.

And this same recovering person, who is slowly reducing his drugs, because he is reducing his drugs, will have a simultaneous *under*medication of his limbic system (which is slowly, methodically changing due to decreasing medication levels), which will precipitate the usual symptoms of drug withdrawal, including nausea, insomnia, paranoia and terror. He will not wish to decrease his drugs, his solace, while he is suffering.

Viktor's case study in upcoming chapter 23 is an example of this. His abrupt transition into recovery and relatively quick drug reduction led to a period of weeks in which, immediately following a dose, he exhibited exuberance, joyous hysteria, and up to an hour of uncontrolled dancing followed immediately by severe paranoia, inability to move, sobbing, nausea, and shaking. After several hours, he would settle down and became slightly calmer. Then, after taking his next dose of the medication, he went through the same routine. These bouts of sudden overmedication (due to abrupt recovery) coupled with gradual decline in limbic area levels (due to drug decrease) are a common feature in those patients who have recovered more quickly than they could safely reduce their medication.

Q. Why doesn't the new dopamine from the substantia nigra prevent the decline in the limbic area, thus preventing withdrawal symptoms?

A. A person who is having withdrawal symptoms from heroin, cocaine, or cigarettes who doesn't have Parkinson's has a perfectly normal capacity for making dopamine but still undergoes the pain of withdrawal. A person who had Parkinson's, even if recovering, may have diminished dopamine producing capacity compared to a healthy person. How can he, with only a nascent capacity for making dopamine, hope to avoid the traumas of addiction that are suffered by those who have perfectly normal dopamine-making capability?

As demonstrated by addicts from time immemorial, *drug withdrawal has very little to do with dopamine-making capability*. They are two separate issues.

In theory, if the drugs could be micromanaged perfectly, it might be possible to take just enough medication to keep both the motor area underexposed to the drugs and the limbic area saturated. Such a person would hover at the line below motor function but just above despair. It is nearly impossible for man-made drugs to maintain that perfect balance. Because of the threshold, it is impossible to know if the brain is almost full of dopamine or desperately low. Because most people must use their movement ability to determine whether or not they have enough, they have no way of actually knowing how much dopamine they need to stay right at the threshold, never going over it or under it. Drug titration is an art.

Q. I am taking several types of antiparkinson's medications: an agonist, levodopa, and eldepryl. Which one should I stop first?

A. I cannot answer that: I am not an MD. I cannot give prescriptive advice about your medications. You must work with your doctor. Bear in mind that if your doctor is uninformed about these drugs, what he advises may be dangerous.

Each of your drugs has slightly different side effects. If you are having any problems that you suspect might be related to your medication, read carefully all of the drug information provided on the medication insert that should have come with your pills. If your drug-related problem seems to correspond to the adverse effect of one drug more than the others, you may want to point this out to your doctor. He may or may not suggest that the drug associated with your problem should be the one that you decrease first.

If your doctor tells you that adverse effects don't occur if you actually need the drug, look for another doctor. Adverse effects are the regrettable companions of drugs –

even though they may not occur in everyone. If you are one of the people susceptible to a particular adverse effect, it is of no use for your doctor to point out that most people don't have any problems with the drug. What matters to you is whether or not *you* are having a problem.[1]

If you didn't get a warning insert, ask your nearest pharmacist for a list of adverse effects. You can also look on the Internet; many drug companies list their product information on their company website. You can also look in the appendix of this book, in the section with individual drugs. For example, if you are falling asleep throughout the day on your trio of drugs, you may notice that your agonist is particularly associated with narcolepsy. If you point this out to your doctor, he may suggest that you decrease your agonist first, if he wants you to make any decrease at all.

If, on the other hand, your main problem is On-Offs, he may decide that you should decrease your levodopa drug. Hopefully, if you are taking the above three drugs that you mention, your levodopa is already being taken at a dose lower than the suggested therapeutic dose; the combination of an agonist with levodopa usually decreases significantly the amount of levodopa that is needed.

By the way, if your doctor put you on an agonist when you were already taking levodopa, and never decreased your levodopa accordingly, he was not following the instructions for the agonist. Also, the doctor instructions for levodopa state that levodopa should NOT be combined with Eldepryl; you need to find a better doctor.

Q. I'm not having any particular trouble with my combination of levodopa and my agonist drug. I want to stop taking them both for a while and slowly get back on to see how little I can get by with. Which one should I reduce first?

A. I cannot answer that. I am not an MD. I cannot give prescriptive advice about your medications. You must work with your doctor. Bear in mind that if your doctor is uninformed about these drugs, what he advises may be dangerous.

However, I can point out a few curious items from our patients' patient experiments. Most people taking both choose to reduce their agonist first. They tend to be more emotionally attached to the levodopa. Whether or not they are conscious of the good feeling imparted by levodopa, they are certainly subconsciously aware of it – and addicted to it. Only a few patients, such as Viktor, have boldly decided that, levodopa being the more dangerous drug (in their opinion), they would decrease that one first.

In general, the reduction of levodopa is more dramatic; the mental and emotional traumas and torments are more excruciating. On the other hand, these traumas only last about ten weeks, give or take four months or so. When the final crumb of levodopa is stopped, the yearning for "something missing," coupled with a physical heaviness, as if joy will never again be known, can last for a year or so.

---

[1] I recently saw in the paper (*Times Colonist*, Victoria, BC, Feb. 2003) that Bayer Corp was found "not guilty" of a charge of ignoring research linking the cholesterol-lowering drug Baycol to dozens of deaths. Although Bayer did eventually take the drug off the market, they failed to warn doctors about the possible side effects of the drug even as research was coming in suggesting the link between the drug and a sometimes fatal side effect called rhabdomyolysis. I am merely inserting this footnote to point out that this drug, which had gone through testing and was FDA approved, did kill people – people who had high cholesterol and should therefore have been likely candidates for the drug. The reason I am using this example instead of any other is that this one happened to appear in the morning's paper on the day I was writing this section. These reports show up all the time.

On the other hand, reduction of the agonists is less dramatic and lasts much longer. The depression and confusion that follow decrease of the agonist drugs appear to be more permanent than the withdrawal pain and fear that come with stopping levodopa. Many people who have gotten off the agonist drugs go back on them after six months to two years; they simply cannot go on living with the unending depression.

It is curious that many people who have quit both cocaine and cigarettes insist that stopping cigarettes is harder, in the long run. Cocaine is considered to be a hard drug, and cigarettes a light drug.

Cigarettes are an extremely mild form of dopamine agonist. Some people who thought they had quit cigarettes find themselves propelled in a mad dash for the 24-hour Quik Stop for a pack of smokes following an emotional event – even years after they have quit smoking. The emotional impact of the agonists appears to be more entrenched and long lasting than the emotional effect of levodopa.

People who get off levodopa often hate the drug and vow never to take it again. It can be a love-hate relationship. The agonists, on the other hand, provide neither the same rush of joy nor the same collapse into despair. Their dopamine enhancement is subtler. They work with stealth, just under the surface. They are less associated in the *conscious* mind with love, movement, and self-confidence. In the subconscious, however, the lure of the agonists seems to run deeper and more permanently. The allure of the agonists may be more compelling and harder to combat because it lurks rather than vaunts.

Levodopa is also a difficult adversary. Much of its siren call is heard on the conscious level, but it distorts the deeper consciousness and creates confusion in the logic centers. When one's will power and logic are altered, it is difficult to deny the insistant, arguing voice that requests more brain syrup.

Then again, with the agonist, one may never even hear the voice. Instead, there can be a heaviness of spirit and depression without end. In our experience, if addiction has occurred, six months of this depression is about the most anyone can tolerate before going back on the agonists.

Despite the common association of depression and Parkinson's, our patients who resumed their agonists had not necessarily had a history of depression prior to diagnosis or prior to stopping their agonist drugs. Whether or not they had any lingering Parkinson's symptoms was also not an issue. As one Mirapex quitter put it, even though she was no longer rigid, was having days when her movement and tempo were perfectly normal, and was not depressed about anything in particular, she had to resume her agonist because she "simply couldn't take the subtext of depression anymore."

Q. What about Amantadine? You haven't said much about that one. My doctor put me on that one first because it is the most mild of the antiparkinson's drugs.

A. Good question. I haven't written much about that drug because it is not a dopamine enhancer. It works by a completely different mechanism. It was discovered, quite by chance, that it helps people with Parkinson's disease. The benefit lasts for about three months, after which its benefit ceases. But after even a few weeks of this drug, a reduction of the medication may cause a powerful, lasting backlash of rigidity. Please read more about this drug in Appendix 2.

Q. Have you heard of any supplements or herbs for foot/leg cramps in PWPs [People With Parkinson's]? Is it possible that my leg cramps are being caused by my medications?

A. You need to find out the cause of the leg cramps. They may be coming from structurally-caused dystonia (bone or tissue displacement), low calcium, or the drugs.

Most antiparkinson's medications will augment (increase) the normal cramping and dystonia that often accompany PD. Almost all of these medications can make cramping worse over the long run, not better, so if your neurologist has told you to increase your medication to help with the cramps and they have only gotten worse, then you need to realize that your doctor may not understand how these medications work. In fact, the cramps may have started because your medication levels were too high in the first place.

Cramps in PD are often caused by dystonias rather than traditional muscle cramp problems such as lack of calcium. Mood-altering drugs may relieve some of the painful dystonias by blocking your awareness of them.

If structural damage (illness, injury, or incorrect postural use) is causing the dystonias, you need to address the structural problem. FSR, chiropractic, Alexander work (postural retraining), Pilates exercise, and some forms of massage can all be effective treatments for the bone and tissue displacement that leads to most dystonias and cramping in PD.

Cramps may also be due to low calcium or lack of exercise. If so, they should respond to increasing your calcium and mild exercise. Walking is great exercise. Swimming is good *if the pool is hot*. Some PDers are susceptible to low-grade hypothermia from swimming in normal-temperature swimming pools. Dopamine plays a role in temperature regulation. If one is hovering at a dopamine threshold, immersion in coolish water can cause a dip below the threshold, which may only appear a few hours, or even days, later.

Most antiparkinson's medications can cause tightness and cramping. They can also create a feeling of "snakes crawling under the skin."

Q. A quick note regarding Ernest. He is not doing too well. He has had three cases of pneumonia in a little over two months (not aspiration pneumonia). The last two were walking pneumonia. On New Year's Day we went to the emergency room. They noticed that he had atrial fibrillation, so now he is on Coumidin. The cardiologist is watching his heart and blood.... They say this cannot be improved by a pacemaker, medication, or by shocking the heart...I think all they will do is the Coumidin. His meds are down to one half 25/100 Sinemet each morning and 3 afternoons a week, a total of 500 mg a week. We cut by 10 percent again today. He is still hallucinating all the time, is VERY weak and can hardly do anything for himself. He can still walk; in fact, he's puttering around the house nonstop, driving me crazy because he's not making sense half the time. He can go upstairs. He seems to have lost a lot of the dexterity in his hands. He sleeps more; his breathing is strange the last month. It too is arrhythmic. Does any of this seem like PD or recovery? I was wondering if this new heart arrhythmia could be caused by the Sinemet.

A. Sinemet can cause heart arrhythmias. I have had three patients who had to make a quick exit from L-dopa because of heart problems. In all cases, the heart problems stopped when the L-dopa stopped. L-dopa can also cause hallucinations. The

strange breathing you are noticing can be also caused by L-dopa. I've seen this several times. It can go away if the person is able to get off the drugs, although during the withdrawal period of up to ten weeks, the breathing problem may appear to worsen. Typically, the specific adverse effects set in motion by too much of the drug can be the same or amplified during the drug withdrawal time.

Please point out to your doctor that L-dopa can cause most of the symptoms that Ernest is experiencing.

Considering that Earnest is walking around all the time, and a year ago he couldn't initiate movement without your help, and also considering that two years ago he was taking high levels of three types of antiparkinson's medications and now he's taking much less and moving much more, it does seem as if something has changed. I am especially concerned about the "not making sense." This can definitely be an adverse effect of Sinemet.

Q. My patient was getting so much better, but now each week her dyskinesia, which used to be so mild, is increasingly violent. She hasn't reduced her drugs at all and won't see her neurologist for another few months. She has been injuring herself from the violence of the spasming. What's going wrong? She was nearly recovered, I thought…

Her PD stuff is almost gone, but she seems to be getting worse. I'm thinking of asking her to see her neurologist early; maybe she needs a new diagnosis; this clearly isn't PD anymore. What should I do in the meantime? Should I continue to treat her?

A. We do not recommend the recovery program for medicated PDers. Your treatments, whether acupuncture, massage, or FSR, might create an increase in native dopamine. If a person is already having dyskinesia, the additional dopamine may do damage, not help. I hope that this person can work with her doctor and get her medication adjusted so that she no longer has dyskinesia.

Q. What happens if a person only takes a small amount of dopamine? How long should I wait to find out if I will have a result with a small amount?

A. It can take three months for the optimum effect of most antiparkinson's medications to manifest. The following case study may be helpful in answering your question.

Stephanie's experiment

Stephanie was starting to have recovery symptoms, but due to her need to keep working, she decided to take the L-dopa prescribed by her doctor. Due to our growing suspicions, which we shared with her, that any need for more than 400 mg/day of levodopa appeared to be due to addiction and not to Parkinson's, she decided *not* to take the 600 mg/day starting dose prescribed by her doctor. Instead, she only took 200 mg/day, a third of the suggested dose.

For the first three weeks, Steph felt no improvement at all. The fourth week she was fairly certain that there might be slight signs of improvement in her fatigue. By the sixth week, she had plenty of energy, although she was exhibiting a bit of grimacing. By the tenth week, she was having twitching in her toes and her feet were spasming into a ball now and then. She reduced to 150 mg/day and, over three weeks, the twitching in the toes stopped. She admitted that at 150 mg/day she didn't have the constant good feeling

that her friend, a long-time PDer, got from her meds. Instead, she noted that on days when she did too much, stayed up too late, or had a fight with her boyfriend, she felt lousy. On days when she ate right, exercised, and got to bed on time, she felt absolutely fine. In other words, she was not aiming for the constant, unnatural good feeling that most PDers think is their lost birthright. She was willing to accept the drugs at a level that helped her move, but allowed her to see the repercussions of her chosen daily behaviors.

Had Steph demanded an immediate response from her drugs, she would have needed to take the drugs at a much higher dosage. That higher dosage might have quickly set in motion baseline changes in her brain, causing semipermanent damage to her brain cells, and slowly changing her threshold levels. Instead, because she decided to watch the drugs over ten weeks and see what developed, she was able to quickly reduce her medication when the toe crinkling and other faint signs of dyskinesia hinted that she was overmedicated. When she reduced accordingly, she did not go through traumatic drug withdrawal – she had not been too highly overmedicated for overlong. However, despite her very low dose, she became addicted within four months.

Q. I take Sinemet and Mirapex. I am having violent spasming in the neck muscles. I get feeling hyped up, and then my stress levels just soar, and then my blood pressure goes crazy. I don't know what to do. My doctor has put me on three different blood pressure medications, and I'm still careening from extreme blood pressure lows to extreme highs several times a day.

A. In addition to causing spasms in the muscles of the limbs, medication-induced dyskinesia can affect the muscles of the neck. They can especially cause havoc with the anterior muscles of the sternocleidomastoids, muscles that are already compromised in Parkinson's disease. These neck muscles lay right over the carotid sinus, which is the location of the body's blood pressure regulator. When these neck muscles clench, or, in the case of PD, are continuously pressing on the sinus, they can increase the pressure on the sinus and its blood pressure regulator. When the pressure on the sinus increases due to the external pressure from the spasming or compressed muscle, it may be that the body imagines that this increase in pressure is coming from high blood pressure. It therefore initiates various pressure lowering mechanisms.

The regulating system may have no way to differentiate between the pressure coming from inside the system (from blood in the blood vessels) and pressure building up because of muscle spasm or rigidity from the exterior. When the carotid sinus (pressure regulator in the neck) "thinks" that the pressure is too high, whether from inside the sinus (blood supply) or outside (muscle spasm, external pressure), it will initiate blood pressure lowering techniques, thus dropping your blood pressure for the whole body. When you have a subtle, medication-induced spasm in this area, it may alter your blood pressure.

The opposite can occur: when the neck muscles cease to spasm, the relaxation will cause the carotid sinus area to feel a lack of pressure. This will cause the regulator to issue a warning to the body to jack up the blood pressure.

This neck muscle trick for causing the blood pressure to go up and down uses the same principle as squeezing a person's carotid artery and causing them to pass out – it is the same situation exactly. Most of the antiparkinson's drugs can cause spasming in these muscles. Some antiparkinson's medications have a special affinity for specific receptors that go to the neck. Mirapex, for example, possibly because of its preference for D3

dopamine receptors, has been observed in our study to cause worse pulling of the muscles of the neck than Permax or bromocriptine, both of which prefer D2's. It does not seem a coincidence then that Mirapex has a stronger tendency to lower the blood pressure than some of the other agonists.

Even unmedicated people with Parkinson's disease often have problems with orthostatic hypotension – a fancy name for feeling lightheaded, even dizzy, upon arising from a sitting or lying position. The low blood pressure often seen in PD, even in those who are unmedicated, is probably due to the forward tilt of the head and compression on the carotid sinus. This tilt is caused by the increasingly rigid inflexibility and gradual tightening of the muscles along the anterior face of the sternocleidomastoids of the neck.

So, in answer to your question, if, as seems probable, you are having spasms in your neck muscles from your medications, and the accompanying blood pressure fluctuations, you may want to work closely with your doctor to see if your Parkinson's medications can be adjusted to the point that you do not have dyskinesia, including the neck muscle dyskinesia. At that point, you should then work closely with him again until the blood pressure problem is resolved.

Q. I have seen people go through drug withdrawal and none of them had drooling, but my husband is drooling a lot since he's reduced his medication. The drooling has always been a problem, but now it is much worse. I can handle anything else, but the drooling drives me crazy. I tell him to stop it, but he just ignores me. While I'm at it, just how do the PDers' withdrawal symptoms differ from the symptoms of withdrawal from illegal drugs?

A. The severe PD withdrawal symptoms that I have observed have been more severe than the mere excruciating agonies of the few illegal drug withdrawers that I have seen, but you are asking about generalities in order to get answers for your specific case. Medicine doesn't work that way – every case is unique.

The best way of predicting which symptoms will be dominant during an individual's withdrawal (in addition to the paranoia, nausea, and insomnia) is to take note of the PD symptoms that this person exhibited. These will be exacerbated during withdrawal.

Every PDer shows different symptoms. In all my years working with PDers, no two patients were the same. Some had drooling, others shuffled, some had tremor, and others had no voice. Some had dozens of symptoms; some had just four or so symptoms. It appeared as if the various injuries the PDer's body had received in his life determined which limbs or body parts would be most affected, which abilities and functions would be lost. Over decades, these weak spots become the body areas (or create patterns of weakness that manifest downstream from the injury) where that particular person's PD would most likely manifest. During drug withdrawal, each person's old weak spot might also be the place that the dopamine deficiency, when there was one, was most apparent.

In Hjalmar's case (as described in previous chapters), his drooling worsened as his recovery symptoms increased, and the drooling only began to improve after he was completely off the medication. This proved that, although it was counterintuitive, the drooling was coming from the pills, and not the PD. Drooling (excessive salivation is the way they put it) is an officially recognized adverse effect of Sinemet and some of the other dopamine-enhancing drugs.

Q. I am seeing my doctor tomorrow. He wants to know if there are any benefits to getting off L-dopa completely as opposed to continuing to take a low-level dose. I am only taking 150 mg on some days, and on other days either 100 or 50 mg. He wants to know if it really matters what I do at these extremely low levels.

A. By law I cannot discuss an individual's doses with or offer suggestions about the drugs to a doctor. Pharmaceutical drugs are beyond my scope of practice.

All my low-dose patients can discern acutely the difference between 25 mg/day and 50 mg/day.[1]

There can be a strong mental alteration when completely drug free, as opposed to the mental state at even the very lowest levels of the drugs. Most people who have stopped their drugs (those who have been on them for more than a few months) insist that they feel a difference between no drugs and a very low level of drugs. Upon becoming drug free, they have been able to see in retrospect that their mental state was very altered while taking the drugs.

Several people who have gotten off declare that their years on the drugs seem like a blur, a fantasy, and that it is difficult for them to even remember most of what happened to them during all the long years of their medication. They remember incidents, but the memories have a dream-like, unreal quality to them. Their spouses concur and note that only after getting off the drugs completely do the PDers start taking a practical interest in the future and in others.

As for movement, when people get off altogether, they often go through a period, which can range from several months to over a year, during which time they want to sleep as much as possible. This is very different from the self-driven, mind-over-matter behavior and intensity of purpose that are seen with people who have PD.

People who have recovered and have gotten off their drugs may want to sleep as if making up for the lost sleep of a lifetime. They may have subdued interest in anything around them. They behave like persons who have been assaulted by stimulants, unceasingly, both internal (the drive of the PD) and external (the medications), and are now able to rest. Even when the meds are as low as 50 mg/day, there often is still that feeling of mental-alteration fog and self-centeredness. Although there are hints of the restful, calm state on days when a very low dose such as 50 mg of L-dopa is taken, the real impact of the internal stillness and desire for deep rest does not usually seem to occur until after the person completely stops taking the drugs and goes through at *least* ten weeks of withdrawal.

Q. The only thing I am confused about is parkinsonism. It seems to me that every person with Parkinson's that I know has some parkinsonism. They start out with idiopathic PD, they have at least 4 symptoms, their neurologist convinces them they need drugs and then they develop problems that only PD drugs relieve – the addiction begins with the PD drug that is supposed to help – putting them in PD HELL! So, how can you

[1] I am using numbers that correspond to low L-dopa intake. The same principles apply with all the other antiparkinson's drugs as well. For example, although a therapeutic dose of Mirapex is 3 to 4.5 mg/day, my recovered patients have altered mental states from this drug, including hallucinations, at doses as low as .5 mg/day, an amount that is a mere eighth of the so-called therapeutic dose.

help the medicated person with Parkinson's? I am sure they all have some PD-drug-induced parkinsonism, don't they?

A. Probably yes. Since recovery from Parkinson's disease will cause the drugs to be more dangerous, and since people who are taking meds may already be brain-damaged and will therefore always be needing drugs, we will not accept people into our program who are taking medications.

Still, before we realized that, we did treat medicated patients in our program. Most of the heavily-medicated patients who did get off the meds and recover are in much better mental shape, and in some cases much better physical shape than they were, especially if they were having painful dyskinesias. Also, PDers who did recover and got off their meds timely are at least no longer getting worse from idiopathic PD, even if they do have the permanent (and often slowly worsening) damage of drug-induced parkinsonism.

A two-part question:

Q. I never get any good feelings or a sense of being drugged from my medication. I don't enjoy taking it, and the movement ability that it gives me is a natural feeling. It is nothing like being drugged. Why do you say it is a mind-altering drug? And…

Q. I get a gentle good feeling from dopamine, but consider it to be merely a return to health, and not a condition of being "stoned," or drugged. You are wrong.

A. When the medication begins, it can be a liberating, exhilarating experience for some. For others, the drugs create a drugged, stoned, slightly fogged feeling. Still others say that they notice nothing at all. Those who do feel a sense of strength and confidence from the medication refer to the first few years as a honeymoon period, which should give you some idea of how they relate to the drug. However, the honeymoon is only a few years, and often very subtle. Sometimes the psychological effects are just barely noticeable. Because the drugs make the patient feel good from a deep, naturally occurring neurotransmitter, the patient assumes that any good feelings are merely a return of normal emotions. People taking the drugs only rarely suspect that the feeling of normalcy is actually a psychoactive effect of the medication.

Of course, many people with PD will argue that they always have been very positive in attitude, and that the medication does not contribute to that feeling of positive attitude and competence. These people may be mistaken. If they examine themselves more closely, they might recall that they have always been able to *appear* good natured and positive. They could force themselves to wear a smile even if they didn't feel one. Rather than welling up effortlessly from within, their high-powered intensity of activity and cheerfulness was willed into action in order to combat the slowly encroaching depression that is a part of Parkinson's disease. When they start taking medication and begin to feel good, these people just assume that their good spirits have resumed, without realizing that the mechanism behind them has changed.

The medication is rarely credited for the return of good mood or recognized for the strong psychoactive drug that it is. It does seem that PDers have less of a drugged feeling from their antiparkinson's drugs than do non-PDers or recovering PDers who try to use these medications. To better answer your question, let me share the experience of Rudyard's two brief experiences with L-dopa, one prior to Tui Na treatment, and one after.

Rudyard and L-dopa

Rudyard had tried various antiparkinson's drugs (Sinemet, Permax, and Eldepryl) when he was first diagnosed. He never took any of them for very long – he expected instant relief, didn't get it, and therefore assumed they weren't working. Even L-dopa had not "done anything."

A year after he started our recovery program, when he was in the throes of the extreme weakness and fatigue phase of recovery, he tried taking medication again. He took one 25/100 pill of carbidopa/levodopa and felt, in his words, "really stoned." A thick, honeyed haze flowed over his brain. It was an unmistakable sensation of being deeply drugged. It terrified him. He had used "light" illegal drugs in the past and had never experienced anything like the tempting promises of L-dopa, nor had he experienced this haze from L-dopa when he still had Parkinson's.

For another example, I received an email from a complete stranger, a non-PDer, who said that though he had experimented with all the popular mind-altering drugs, he never took L-dopa more than once. In his words: "I've taken everything, but I could tell that L-dopa was different from all the other drugs. It scared me. It was a high like no other. It was too perfect. I knew that if I took it one more time, it would claim me for its own."

"When the Hare awoke from his nap, he saw the Tortoise just near the winning post."

"The Hare and the Tortoise," Aesop

23. BUZZ AND VIKTOR

A PAIR OF STUDIES: TORTOISE AND THE HARE

In August of 2001 I got an email from a total stranger named Buzz. He had recently recovered from Parkinson's disease, and he wanted to know how he could contribute to our program. I was thrilled to hear from him. The local television station had scheduled a one-hour broadcast about our program and I wanted to feature people who had not been my own patients.

A growing misconception held that my personality, and not our techniques, was responsible for people recovering from Parkinson's disease. To squelch this idea, we chose people for the TV interview who had not been my own patients.[1] None of us had ever heard of Buzz until he contacted me. Perfect! Three weeks after initial contact, he motored down from Oregon for the broadcast.

The evening before the TV interviews, I met Buzz and learned details of his recovery. It turned out that his acupuncturist, Eileen, had visited our clinic and attended a weekend workshop on treating PD two years earlier. She was now working with eight Parkinson's patients at her private practice up in Oregon. Buzz told us about her technique and about himself. The most amazing thing about Buzz's story was his all-at-once decrease in his drugs. He is the "hare" of our chapter. His crazy method of drug reduction was harrowing, but he survived it. His relative youth and his otherwise good health may have helped. His spiritual convictions certainly played a part. He was very lucky to survive; historically, case studies exist for patients who have died from abrupt stoppage of this drug.

We do NOT recommend Buzz's method of drug reduction. However, here is his story.

Buzz

Buzz was 53 years old when he was diagnosed with PD, a diagnosis confirmed by two neurologists. He began recovery therapy when he was 57 years old. At that time his symptoms were already severe. His second doctor considered his case advanced Parkinson's. He was no longer able to work when he started getting treatments from Eileen. He had severe tremor. His balance was poor: he had to hold on to the shower safety bar with one hand while washing his hair with the other hand – there was no way that he could keep his balance without holding on. Several times he had lost his balance

[1] Dr. Fred Jones, a retired professor of medical research, advised our project in its infancy. He said I had two choices: I could be recognized for establishing a scientific protocol that was effective no matter who did the work or I could clamor about my own patients' recovery. If I chose the latter, fifty years down the road, people would say "there used to be a woman who could treat Parkinson's disease, but she's dead now." The choice was obvious: the emphasis must be on the treatment, not the one who wrote about it.

and fallen while walking. His small motor skills were almost gone: picking up a coin was difficult, doing buttons and making meals nearly impossible. He walked with little "baby steps." Neither arm swung, and his face had no expression on the left side. He had tremendous difficulty in getting out of bed – had fallen to the floor trying to get out of bed, in fact – so he had moved his mattress to the floor for safety. He wrapped a towel around his neck to catch the drooling. His posture was hunched forward. His voice was weak.

At the time he started receiving treatments from Eileen, his medications were:

1) Sinemet (carbidopa/levodopa), 25/250 six pills a day. (The dosage is not a typographical error. Although the more common ratio of carbidopa to levodopa is 1/4, the drug is also available in the 1/10 ratio.)

2) Klonopin. (Buzz does not remember the dosage.) This powerful anticonvulsant suppresses limbic system response. It is used to suppress seizures, mania, some symptoms of schizophrenia, and recently, it is used for dyskinesias caused by antiparkinson's medications.

3) He had recently stopped taking BuSpar for anxiety. BuSpar affects serotonin, norepinephrine, and dopamine activities.

After the TV interview, I asked him to jot down details on his drugs. He sent this to me in email format:

Buzz's own story

"I was taking Buspar, don't remember the dose (not in my journal either), but I started taking it for 'anxiety' about six months before I started your program. Took it for two months, but the anxiety was escalating faster than the medication could handle, even when I cheated and took extra tablets (yeah, I know, stupid...but fostered by desperation!!). Stopped taking it when I was switched to Klonopin by my neurologist.

"Klonopin, don't remember the dose (maybe 0.5 something – not in my journal either), but was switched to it four months before starting your program to counter the growing anxiety. At the time I started your program, I was taking 2 pills twice a day by prescription. But I was cheating again...and would sneak more as needed, usually six pills a day. Yeah, cheating on myself! I began reducing the use of Klonopin about 4 weeks into your program...simply because my anxiety was lessening pretty dramatically. I kept taking less and less (only as needed) until New Year's Day...when I flushed 'all of it' down the toilet too.

"Here are my best recollections (plus reviewing my journal)...in response to your questions. It's so interesting – how quickly we "forget" the things that we don't want to remember anymore...

"When I started your program at age 57, I was taking:

"L-dopa, 25/250, two pills three times a day: morning, noon, and early evening. I had been on this higher dose for about a year. Prior, for three years, I was using lower dosage pills, 25/100, two pills three times a day, same times. When my dosage was increased to 25/250, I started to try to periodically skip a dose once in a while, or even for a couple of days, if possible, just to see how long I could endure. I felt like the increased meds were making me deteriorate faster...something was sure screwing me up.

"I was having On/Offs from the L-dopa (it never lasts long enough), but since I was 'almost' living a shut-in existence...I just toughed it out because of my fear that the meds were taking me down faster. I was just 'very careful' and timed my trips outside the house with excruciating care, so that driving, shopping and seeing people were always done while I was "On"...even when I was almost Off. Yeah, my driving was getting kind of scary too!

"I never took extra meds at night, although the temptation was starting to "haunt me" along about the middle of the night. There were lots of nights I just "sweat it out"...until 7:00 a.m. and the first daily fix to get me "On" again.

"Gosh, that first fix of the day was even better than sex...or at least what I remembered about it!

Buzz's drug reduction program

"After I was in your program for two months, I made my first decrease in meds (against the advice of my neurologist) to two in the morning, one at noon, and one in the early evening.[1] That went well[2] (somewhat!), and a couple of months later, on New Year's Day 2001, I decided (against "everyone's" advice...Eileen's too!) to quit all meds totally. It worked for me, but the first 8 weeks was, simply put, a real total absolute bitch!! I made it through luckily...and have never looked back.

"For a year+ now, I have taken zero meds. Even when I get a headache (only on occasion), I practice Qi Gong for 15 minutes or so, until it goes away."

Crazy dangerous

As Buzz recounted his story, I was horrified. He had, against the recommendation of his therapist and his doctors, gone off his medications in two reductions – a rate that approached cold turkey. I asked for details. The verbal account did not match the daily journal entries. He remembered only that it was hard, and that it lasted at least eight weeks. He had forgotten most of the details. The journal was more revealing. He had endured fourteen weeks[3] of incomprehensible motor and mental collapse. As he told me after the TV interview (and I paraphrase),

"I am so glad I live alone. If I hadn't lived alone, I couldn't have done it. I had whole days where I couldn't move and just lay there in my own confusion. Couldn't eat, think, nothing. If anyone had seen me, he would have been scared to death. I would have been put in the hospital and put on drugs against my will. There is no way, absolutely no way, I could have done it if anyone had been trying to help me.

---

[1] If you are following the math, that is a change from six to four, a decrease of 33%. However, he was not addicted, because he still had Parkinson's. Therefore, this reduction, though difficult, was not full-blown withdrawal. It's probably significant that he was taking the 1:10 Carbidopa: Sinemet variant. (See: Sinemet in Appendix 2.)

[2] This is a glossing over of the facts. As recorded in his journal, this was a very difficult time.

[3] In his email, recorded here, he states that it was a period of eight weeks that was so difficult. However, this number is incorrect. His own journal, which he has graciously shared with me, indicates that it was fourteen weeks, not eight, before he began coming out from under the agonies of drug reduction. This is an example of selective memory, no doubt. I have seen this faulty memory very often in other drug-using patients; their after-the-fact summarized memory of their experience does not tally with their daily records. It was fourteen weeks before he *began* to even think that he was beginning to see an improvement, not eight weeks.

"I had arranged to have friends and family bring food and leave it on my doorstep. If they had come in, they would have called an ambulance."

His formidable attitude is reflected in his daily entries. For example, he had a day when his entry read "not too bad today." The next day his only entry was again, "not too bad today." But the following day, it reads, "I was able to move my arms a little bit today and drag myself along the floor for the first time in days, thank God."

In case you've missed the point here, on the days when he was "not too bad," what he evidently meant was, he was still alive and not insane. "Not too bad," does not mean that he had mobility. "Not too bad" meant, "I am still alive and conscious."

I cannot recommend what he did. He is lucky he didn't die. I do not recommend this method. I include it here only because he had the fewest total problems with his drug withdrawal of any of our patients who had been taking such a large amount when starting the program. Also, he never developed a subsequent yearning for the drugs, which has occurred in our patients who took longer to get off their medication. Therefore, in the name of science, his case must be included, but I cannot stress strongly enough, this does not appear to be an ideal method, and it may entail many risks, including death.

The other reason that I am including Buzz in this book is that many people have seen the television broadcast or a video of our subsequent interview with him, and I fear they might admire his methods without appreciating his suffering. Therefore, to buffer his televised insistance that "stopping the drugs quickly was the best thing I could have done," I have included his case study here as an opportunity to point out that he is lucky he didn't die.

Buzz's current condition

He currently takes no medication and no one would ever guess that he had ever had advanced Parkinson's disease. All of his obvious symptoms are gone except for a small tremor in his left hand that occurs when he is nervous – such as when he is being interviewed.

His small motor capabilities are excellent. As a small aside, when I showed the video of him for the local Parkinson's support group, I was surprised that no one seemed particularly moved by his "testimonial" type words. There was a stronger response, surprised shaking of heads and tutting, when he said that he was cleaning rain gutters again. I did not anticipate the gasps of amazement from the group when, in the video, he deftly picked up a penny from the table to demonstrate his new manual dexterity. It was clear, after running the video, that it was not his words of gratitude nor his reflections on "being given a second chance," but the conquering of little motor function problems that most deeply affected the audience. Curious.

As for balance, he is once again getting up on his dad's roof and cleaning the gutters. His energy level is good. He is actually in excellent health. When he came to our town for his interview, I taught him how to dance the leg-kicking bit of the can-can; he picked up the left-right coordination quickly, and within a few measures of singing along with me, kicking his feet in the air, he had all the moves down.[1] He has a radiant smile.[2] He is continuing to improve. He still has a faint degree of rigidity in his left arm and

[1] It is rumored that I have promised to dance the can-can with anyone who recovers from Parkinson's disease…

[2] The immobility of the left side of his face is completely gone.

wrist, and the tremor is continuing to change. The tremor is becoming more delicate, smaller, and more fluttery. He is confident that the shaking hand will go away. I am waiting to see – even the relatively mild antianxiety drugs taken by the general population can produce delayed onset (sometimes years later) permanent tardive dyskinesia.

Attitude

I am going to include a bit from Buzz's continuing journal to show his attitude and incorrigible sense of humor. He was having a particularly virulent shaking in his hand one day, although his other symptoms were long gone. He is certain that the rapid changes in the tremor are due to the changing muscle tone in his arm – as the rigidity left his left arm, the left hand began to tremble uncontrollably. I am uncertain whether it is tardive dyskinesia or residual weakness, but it is the only remaining indication that he ever had advanced PD.

Buzz's journal

> "49 weeks now meds free, and believe me, the best thing I ever did for myself...was getting completely off those PD meds as quickly as possible. My recovery process really started rolling from that point on...and has only accelerated since that was achieved!! This has been a good two-week period. Some of the stiffness is working itself out – I really started loosening up a little about four days ago. The shaking of my left hand, however, continues to increase in intensity (which is good!!), as the last of the PD works itself downward toward the end of my left side limbs.[1] It is such a slow process, but probably only because I am in a hurry to rid my body of the last vestige of this nasty disease. My left hand now vibrates so frantically and unceasingly that it is really getting to be funny!! If my hand is out of my pocket everyone thinks I'm waving at them...if it's in my pocket, they think I'm some kind of pervert!! One lady was staring so hard at me at the grocery store (my hand was in my pocket) that I felt compelled to say, "Sorry, it's just Parkinson's disease." Her response was, "Yeah, sure!!" Who would have ever thought that there could be humor in recovering from Parkinson's. But there is...and each and every day I continue to move a little tiny bit closer toward a complete recovery – it's practically within my grasp now!

Buzz's faith

I have had the honor of reading his whole journal. It is evident that he was helped tremendously by his deep and unwavering faith, not in our program, nor the possibility of recovery from Parkinson's, but in the presence of God and the infallible wisdom of God's mysteries, even the mysteries of illness and suffering. His journal is deeply moving. He has donated it to our project, in the hopes that it will someday be made available for

[1] His symptoms in his torso, including balance problems, had improved first, and then the facial expression, and then manual dexterity. The stiffness in his left neck and arm was the most recent to go. As his arm became more limp, his ability to restrain his tremor decreased. Even though the internal shaking and restlessness were gone, the long-time brain habit of shaking continued. Tremor is usually the last symptom to ebb.

others who are recovering. We have a long list of things that we hope to publish, and Buzz's journal is on that list.

Eileen's thoughts

Eileen, Buzz's acupuncturist, confided that Buzz was her most bold adventurer with the medication. He had also recovered the fastest of all her patients. Eileen felt that Buzz's distinguishing characteristic was gratitude. Eileen said that the patients in her PD program who were having the least benefit were those who were bitter about their illness and resentful that she wasn't doing a better job at healing them faster. The ones who were making the most progress were those who realized that it was their job, not Eileen's, to return to health.

The ones who were making slow progress, if any, would discount their recovery symptoms or express doubt that the improvement would last. If they couldn't deny their obvious improvements, they might insist that they were just caused by the power of suggestion.

She had one patient whom she had been treating every week for free for nearly two years, and he had never yet thanked her for her time. Buzz, in contrast, always managed to bring her a few flowers or some small gift when he came. He was always thanking her and expressing gratitude for this program. He accepted every symptom of improvement as a good thing and was grateful for it.

Buzz could see the logic of the illness. It made sense to him that he had had Parkinson's disease. He also accepted the possibility that, by overcoming the cause of the illness, he could change the course of his life. Buzz, more than any of her other PD patients, was willing to look deeply at himself, his attitudes, and his actions, and see how they might have been contributing to an illness marked by rigidity and disconnection from both his body and from his fellow man. After embarking on the recovery program, he resumed his long-ignored meditation practice. He was grateful for having Parkinson's and for having recovered from it. He often averred that he never wanted to go back to being the person that he used to be before recovering from Parkinson's. He also opined that people who just want to get treatment so they can go back to being the heroic, unbending person that they were before were missing the opportunity of a lifetime. He felt that he had lived two lives – one in which Parkinson's was the obvious conclusion and the other in which humility, hope, and love were the driving forces – conclusion unknown.

Eileen felt that there was a strong correlation between his attitude of humility, acceptance, and faith in an unseen logic behind the mystery and his relatively rapid rate of recovery. Most of all, she kept coming back to Buzz's gratitude and love for others.

VIKTOR

To contrast with Buzz, who stopped his medications precipitously, I would like to share the case of Viktor, who waited a few days too many to reduce his drugs. He planned on reducing his drugs for more than a year before his hand was forced. He is the chapter's tortoise. I will include quotes from his journal. As you read his journal, imagine that you are a researcher, looking for clues about the medication as he recounts his hourly changes. It may seem slow going at first, packed with details and repetitions, but by the end of the chapter an excellent picture of dopamine change will emerge.

At the time he began reducing his drugs, Viktor was 51 years old, had been diagnosed three years earlier, and was taking 500 mg/day L-dopa and 3 mg/day Dostinex (a European dopamine agonist, approximately equivalent to 9 mg Requip). He had been using our PD treatment protocol for just over a year when he first came to our clinic.

Viktor had been mildly overmedicated when I first met him, one year before he became our patient. His symptoms of overmedication were a little ticcing pattern that tilted his head several times a minute, as if he was bringing his ear towards his elbow. When the twitch was at its height, he also had a grin that was a bit forced, as the facial muscles pulled up too hard on the corners of his mouth. His main symptoms of PD were loss of voice, bradykinesia, and shortening of stride, all of which had responded well to the medication.

Years of psychotherapy

Prior to using Tui Na, Viktor had spent years working with philosophies and psychotherapists to get to the root of his lifelong inability to cry or emotionally unbend. He felt that his emotional rigidity was related to the physical rigidity of Parkinson's disease. While he could understand logically that his inner and outer rigidity were related, he found he could not change himself through mere mental exercises or talk treatments. He discovered during his FSR treatment sessions that Yin Tui Na could get through to him in a way that talk and thought could not.

Reluctant to reduce meds

He admitted at our first meeting that he was overmedicated but was reluctant to reduce his medication until convinced his recovery had started in earnest. Since starting our protocol with a practitioner from his hometown, he was sleeping better and had more feeling in his feet, but he doubted the significance of these changes. After starting at our clinic, he became buoyant in mood, almost to the point of effervescence. I warned him at our first meeting, and again a year later when he started attending our clinic, of the symptoms and dangers of overmedication. He thoughtfully considered the warnings and appeared to understand the risks. Viktor was very intelligent and objective. He felt well prepared for the eventuality of drug reduction.

At this time, we had not yet started our present policy of not working with medicated patients. Viktor's disastrous aftermath, which occurred despite our sharing with him in advance everything we knew about the 10% plan, the Slide, the Crashes, the ten weeks, and our experiences with Euclid, Angus, Birdie and all the rest, settled our wavering indecision about patients on drugs. After Viktor, we no longer admitted medicated patients to the program.

Recovering and not reducing

I was especially concerned when I saw Viktor after a one-year hiatus: he appeared more overmedicated even though he had not increased his med levels. In addition to his

previous dyskinesias, his shoulders were making a little upward twitch. And then, after two weeks of working with us, something switched in his brain.[1]

Two days after a treatment session in which he felt that some last, resistant knot had come untied in his leg, he went from a dignified and controlled investment banker to a maniac. He was uncontrollably happy, he could hardly sleep, and he had to run, jump and laugh all day. His eyes glowed with a rare effulgence.

Explosion of motion

Viktor was a rarity in that he had the ability to realize that even if in the best of health, he should not need to run on the beach to keep from bursting. Most medicated patients who burst into ecstasy want to attribute their unnatural buoyancy to recovery and their innate eternal youth, rather than giving credit to their drugs. Viktor, who had never been an athlete, recognized that dancing half the night in his flat and running on the beach for an hour simply to rein in his new exuberance was most likely a drug problem.

He decided to reduce his medication to the point where he could keep both feet on the ground. During this explosive time, I saw him more frequently than once a week, and even spent a night at his home once when he was scared.

Terrible tensions

His biggest problems during drug reduction were his "terrible tensions" that started in conjunction with his euphoria, just after he became wildly addicted, on the fourth day of his cautious drug reductions. At first, these tensions only enveloped him during the highest point of his Ons. Soon they began to occur during his Switching, and then in Roller Coastering high points. Within two weeks, the excess drugs transferred their attentions from his mental area to his motor area. Incoming pills were directed to expend themselves in "terrible tensions," and his euphoric feelings came to an end.

The chest tension was especially frightening. While Becky's spasming centered around her diaphragm and Coach's in his heart, Viktor's left leg and entire torso were gripped in a death hug of muscle spasm.

The tensions drove him to make more rapid reductions than he had planned, with the resultant paranoias and withdrawal symptoms that he had been so keen to avoid.

The tension stopped for a few days during a Vacation phase. Sliding from excess to insufficient limbic stimulation, his drug intake was very low and the tensions disappeared altogether. However, once the drug withdrawal symptoms began, even his brief spurts of On time would often be accompanied by episodes of the returned tensions. This led to accelerated drug decreases and worse withdrawal symptoms, which frightened him into resuming the drugs at an even higher level.

Prior to his sudden, glorious burst of drug-joy, he had never experienced Ons or Offs. The introduction to On-Offs was simultaneous with his explosion into addiction.

---

[1] Viktor was participating in the visitor's program and not the free clinic. To accommodate PDers and their practitioners who travel from long distances to observe and receive treatment or free training, respectively, a group of licensed acupuncturists in Santa Cruz operates a clinical program, the PD TEAM of Santa Cruz, in which visiting PDers may receive an intensified program – up to three Tui Na treatments a day – for a period of up to two weeks.

Viktor was one of these visiting patients. He had received several treatments a day for two weeks just before his severe overmedication erupted.

The On-Off pattern continued ever after that day, regardless of whether he increased or decreased his drugs in his search for a "balanced" dose. The deathly tension also became a permanent part of his repertoire and eventually started appearing during his paranoia attacks, as well as during his Ons.

Not all patients' drugs convert to mental dynamite when they begin to recover. For instance, in the case of Olli from chapter two, his adverse effects began to steadily increase, and, though determined to reduce his drugs, he found himself increasing them. These increases created ticcing and blood pressure trauma, but they did not create anything that compared to Viktor. However, we had seen two cases similar to Viktor's, where the drugs transformed into pharmaceutical grade explosives. Viktor was the third, and we vowed he would be our last.

As you read from his journal, excerpted below, try to pick out the small patterns occurring each day – but do not be blinded by them and miss the larger patterns that occur over the weeks. Bear in mind that a one-time appearance of Deficit- or Build Up-like patterns does not necessarily signify a limbic transition; the Deficits or Build Ups are most meaningful when they are daily, repeating patterns, occurring over several days. If those patterns seem to appear on one day or another at random, they may be merely the result of drugs taken in the previous day or two, and therefore *not* indicative of a place on the larger cycle. As you read about his drug responses over the course of any one day, keep in mind his drug levels from the previous day, the previous ten days, and the previous ten weeks.

As noted earlier, he was taking 500 mg/day of carbidopa/levodopa, and 3 mg Dostinex (a European drug, similar to 9 mg/Requip). The following is transcribed from his notes. In a few places I have corrected or modified his English. (His clear and precise prose in the following journal is all the more proof of his very high level of intelligence and analytic ability when you consider that English is not even his primary language. I point this out for those would-be drug reducers who imagine that their intellect or analytical ability will somehow allow them to oversee drug reduction without being traumatized by it.) Some days he sent long reports; other days the reports were brief or were transcribed from phone calls. My comments are in brackets. I have inserted daily totals for his levodopa next to the date. This is the best example in this book of the time frames needed to understand the drugs. After wading through days of seemingly unimportant events in the notes below you will behold the full power of the delayed limbic response.

Viktor's journal: Look at Me – I'm Dancing!

Day 1 – Reduced L-dopa to 450 mg (down from 500 mg).

[From my notes: still grinning with forced, tight facial muscles, twitching in neck and face, no apparent change in motor function after today's 50 mg drug decrease. He felt such unrestrainable exuberance that he had to go running on the beach for over an hour again. He's finally decided to reduce his medication. He plans to reduce slowly and carefully until he stops having any episodes of spontaneous giddiness and wild energy.]

Day 2 (450 mg)

[Even more exuberant, laughing and jumping around his living room. After his noon dose, he felt euphoria and muscle tension that were only relieved by again running on the beach for an hour in the afternoon.]

Day 3 (450 mg) I got up at 8, a little stiffness all over, feeling a little bit strange but OK. 50 mg L-dopa at 9 a.m. and sleeping till 10:30, then little stabbing pain along the sternum, light nausea, tension all over, strange feeling, tension in hips (pelvis) but no impression of motor weakness, but rather a little bit "braked." By noon I feel almost normal, take 150 mg. A little tension all over, which suddenly at 12:30 disappears completely. I am feeling fine in every way at 2:00 p.m. [Euphoric in afternoon.]

Top of the Slide

Day 4 (400 mg) [Another decrease: a 20% decrease in four days.]

I was a little bit more clumsy this afternoon and slower in motion but OK. I feel my own legs; that's a very new sensation.[1] There are very light cramps in the right leg around 9 p.m. but very short only. Only my fingers are slow on the computer. I feel very good; I do not feel much change in motor capacities. There is a bit more difficulty to move in bed, but that's all. But in the morning after the first dosage, I feel overwhelmed; there is pressure on the chest, tension in arms, pelvis and legs and a kind of tiny nausea. That starts about 20 minutes after taking the meds at 9 a.m. and lasts for about 30-40 minutes. The second and third dosage is without major side effects, but more tension in the legs for an hour or so.

Day 5 (400 mg) I might have less back pain. I sleep between 2 and 4 hours, then I am awake for 1 or 2 hours, then I sleep for the rest of the night. I have had this pattern for over six months. In the morning before standing up, before any medication, I have strong recovery dyskinesia.[2] Today even before taking the first dose I had nausea, tension in the legs, pressure in head and stomach, but not in the chest. In the morning my motor function was weak. After the second dose light cramps in the right leg after 30 minutes and stronger after 1.5 hours. Moving very well in the afternoon. Same pattern after third dose: after 1.5 hours light cramps in right leg. Motor capacities slightly slowed down but acceptable. In the evening a bit slower but still OK.

---

[1] In most cases, return of proprioception and increased sensitivity to temperature and pressure in the extremities and limbs after decades of PD-driven, subtly increasing numbness can be guideposts in the journey through recovery.

[2] Recovery dyskinesia is not the same as tremor. It can occur in body parts that are beginning to experience restoration of nerve function. Viktor never had obvious tremor as one of his PD symptoms until his drug decrease plunged him into withdrawal symptoms: if there had been any tremor, it must have been extremely slight and completely masked by the good feelings brought on by the drugs. In the case of this journal entry, I suspect this morning's movement might have been drug based, and was not recovery spasms – but I might easily be wrong; I was not there to observe. For more about recovery dyskinesia, please see *Recovery from Parkinson's Disease: A Patient's Handbook.*

Day 6 (400 mg) Sometimes I feel weak in motor function, but sometimes I have almost full motor function and at the same time a kind of brake which slows me down. Also, there is in addition this phenomenon: I may almost not be able to turn in bed, but when I have enough of that and stand up, I have full motor function. I am holding more again with my right hip and leg. I feel overmedicated. After the last dose this afternoon at 4 p.m. I had some tension and pain in the legs, which rather increased during the evening. It's now 11 p.m. and the tension has gone. I am still moving fine.

[At this point he still needed to go running on the beach every day to work out the buildup of tension in his legs. He also still had moments of euphoria every day after the tension went away. His tension increased throughout the afternoon and evening. His first dose of the day worked best, and the last dose caused the most problems: the medication was building up over the course of the day. However, this does not mean that his limbic system is raising his baseline dopamine levels already in a healthy rebound due to his dosage decreases; in fact, his limbic system is still in decline and doesn't even suspect yet that its dopamine levels are still in a free fall. The apparent Build Up is more likely due to the motor area's increased sensitivity to the drug. (This could be because of recovery, and switching to an addictive, parasympathetic state, as well as being flush with dopamine – for a short while following each dose – in the motor area.)

[His threshold is rising (his brain is building barriers against the drugs, a process also known as addiction), while his baseline of stored dopamine is slowly dropping (due to both the abrupt drug decrease and to the addiction-based brain changes that are suddenly occurring because of using drugs during recovery). However, due to the apparent Build Up, a person who was not watching the larger picture might think that it was already time to make another drug reduction.

[If he had not become addicted, so that adverse effects had not yet become a part of his repertoire, he would be noticing, at this early stage, only the effects of the drug decrease and not those of increasing adverse effects. However, he has been in the addicted stage for nine days, and he is actually still increasing in addiction symptoms even though he has made a 20% decrease in his medication. Now that he is in recovery, any amount of the drug will be dangerously addictive, and cause adverse effects.

The "show"

[He is still having bouts of euphoria, which he named "the show." His brain is still trying to find ways to get rid of the excess dopamine. It is even excessive in the morning before taking his first pill. As you will discover later, his brain is already starting to make glimmering amounts, which stockpile in the night and are released in the morning. Upon his awakening in the mornings, the glimmer of morning dopamine release into the motor area appears to combine with the excess amounts of medication that are still in his brain, causing his adverse effects even before taking his first pill of the day. He notes that, on most days, he still is "fine" after 2 in the afternoon. This "fine" corresponds to the time when he melts into a euphoria unmarred by excess tension. This latter time is a condition so high above the Safety Level that his brain cannot resist – he is in mindless bliss. See: Superdosing.]

Day 7 (350 mg) [Another decrease: a 30% decrease in seven days]

Morning took 100 mg, after half an hour felt tension in the legs, lasted 5 or 10 minutes. In general, motor function and emotions are fine. At 11:00 I had slowness, difficulty writing, pain in right hip and tension in right ankle. Took a nap until 12:00. At noon took 100 mg, 15 minutes later "the show" began; I almost jumped, I had strong electrical sensation all over the body. It won't let me sit quietly, work, or watch TV. I try to lie down, but immediately get up again because the tension is so strong and unpleasant that I cannot relax. Then a brief cramp in the lower left leg and light pressure on the chest. Then heat along the right leg and light, non-muscular tension. The whole thing lasts for 20 to 30 minutes, and then it's over, I could relax again and move normally. By 2:00 I am fully OK.

Day 8 (350 mg) Morning took 50 mg. Light tension after half an hour, motor function weak. At noon took 150 mg. Strong tension after 1.5 hours, motor function good. Evening took 150 mg. Strong tension after 1.5 hours, motor function variable.

Day 9 (350 mg) Morning took 100 mg. Light tension after 1.5 hours, motor function good for 2 hours. Noon took 100 mg. "Nightmare" (tension and feeling strange) after 20 minutes and strong tension after 1.5 hours, motor function OK (bad between 2 and 3). Evening took 150 mg. Strong tension after 45 min and after 3 hours, motor function OK until tensions began after 3 hours.

Day 10 (300 mg) Morning took 100 mg and noon took 50 mg. Both fine, and without cramps. Evening took 150 mg. Made strong tension in the legs after 2 hours; it lasted half an hour, then fine. A bit slow, but not too much, and I am not afraid at all. I may take only 200 tomorrow.

[On day ten has was taking 300 mg, a 40% decrease from his original 500 mg. Adding up all his drugs over the last ten days, he had *averaged* 390 mg/day. This is an average decrease of 22% for the ten day period.]

Vacation

Day 11 - No Sinemet today! The morning was marvelous. I went walking along the beach, slowly and carefully. Then I went to the bank and after (12 a.m.) I was done with motion. I went home and had to lie down. I could not zip my pants. At 2 p.m. I could move again enough to get up. Motor function resumed in the afternoon and I am (6 p.m.) quite well again although a little slow. I increased the Dostinex by 50% to 3 mg. I don't plan to take Sinemet any more and I put it away. I feel fine.

[This was the first day with no moments of euphoria or tension. He went abruptly from 300 mg levodopa to none because the powerful tension in the chest and legs that followed the medication and which often preceded or followed the periods of euphoria

had become too frightening. He is just starting a little Vacation. How confidently he declares that he no longer needs the medication!]

Day 12 – No funny feelings, but the little bit of rigidity this morning, a little bit of pressure in the head and chest, a lot of recovery dyskinesia, and a bit of tremor. Good sleep, but woke every hour. Motor function good. Today is Tuesday – the last Sinemet was Sunday 4 p.m.

(Later the same day) This morning is very different from yesterday. I am slow and feel (over-) excited in the same time. I would like to lie down but cannot stay in bed because I am too nervous. There is some tension in the whole body. And the body is a little heavy. The agonist: Dostinex, 3mg (equivalent to 9 mg Requip) in the morning, that's all. It's a long lasting substance.

[Viktor was starting to feel nervous though motor function was still good. He was just beginning to feel the effects of decreasing his medication, twelve days earlier. The vacation was winding to a close, the Slide was becoming apparent. The tension that he felt in his whole body now seemed to be anxiety related and different from the powerful muscle clenching of the torso. Also, as he will discover later, the Dostinex creates yet another type of all-body tension which is distinct from the dopa-induced clenching and which will start to be apparent soon after he doubles his dose of Dostinex. Unfortunately, in his journal he uses the word "tension" for all these conditions, though his explanations to me indicated that they are distinct. Note that he is experiencing tremor for the first time. Watch how it quickly increases in intensity.]

Day 13 – During night extended periods of intense shaking. Very slow this morning, difficulty in writing and moving. Quite strong tremor, body feels heavy.

[He was no longer having periods of ups and downs. He was starting to feel heavy throughout the day. The vacation was nearly over…]

Day 14 – I went for a walk and had lunch. I am very slow. The difficulty is that I cannot sit and do something because I am too excited despite immobility. But I cannot lie down as well because I start to get more nervous (panicked?). There is also a lot of tremor.

[As he nears the end of the slide, anxiety and discomfort have begun to appear.]

Day 15 – no entry

Day 16 – Today is worse than yesterday. Difficulty moving. I'll survive. It feels as if the limbs were disconnected from the brain. That's new.

End of the Slide

Day 17 (200 mg) Though yesterday was difficult, last night was quite good: I walked around for an hour or so and then slept for nearly one hour and so on until 6 a.m. this morning. It was then I started to feel weak and could not move. After taking the agonist I was fine and moving well for the whole morning. After 1 p.m. I had a kind of breakdown and did not recover for the whole afternoon. I could

nap but did not move better after. I had an immobility crisis, almost like I was frozen. I almost cannot move, sit or lay down without "jumping" up again. I just move very slowly from one end of the room to the other and then back again without end. There is tremor, too. I slept an hour but it's not better – after I lay down again and almost can't get up at 3:45. I felt close to withdrawal shock. At 5:15 I took 100 mg Sinemet. This afternoon was so different than all before that I am convinced that it is the right thing. I felt normal after 5:45. I changed from almost no motion to normal. That lasted until 8 p.m. and then I had the same experience as in the afternoon.
[He took another 100 mg at 8 p.m.]

Back on the drugs

Day 18 (550 mg) I took **50 mg Sinemet at 1:15 a.m.** with little effect by 2:15 in the morning. It's 4:30 in the morning now and I feel heavy and immobile. Then began almost two hours game of lying down and standing up again shortly after. 'Standing up' means fall out of the bed with a special rolling technique. It was easier with the Sinemet, but still slow and difficult. I finally managed to sleep for half an hour until 4 a.m., but did not move better after. It's 4:30 a.m. now and I feel heavy and immobile, but am still better than without dopa. I took 4 mg Dostinex, [double his usual amount] and the restless down-and-up again game continues until after 6 a.m. There is one position I can stay in bed, on the back, but only briefly. On the sides it is impossible to lay, or on the chest – which I always did before. I finally fell asleep around 6:30 and slept until 8:30. When I woke, I immediately began to shake. I had an impression that came from another world, I was kind of glued with the back to the bed and managed only with difficulty to roll and fall out of bed. Standing up was almost impossible, and moving also. I felt like having the feet glued to the ground, turning around was a big job and I was shaking like hell. Between immobility and shaking I couldn't do almost anything. It was not possible to eat or drink something and I hardly could manage to make a phone call. I managed to take **100 mg Sinemet at 8:45** and felt better at 9:15. It's 9:45 a.m. now and after having written this I feel again a little bit weaker. I have to organize something, maybe a nursing home. I am afraid, and I cannot stay longer alone. The Sinemet is fading already. I have some pressure in the head and the impression that my fingers are a little bit slower.

At 10:45 I am back to freezing again. At **11:00 I take another 100 mg** Sinemet. It takes an hour to get me moving again. But the result is not as good as with the first dose. I have strong tension in the right leg. I keep moving, as apparently the fear stays or even increases. For a moment I have the impression that the effect wore off already (12:15). [Roller coaster.] There is an overall tension which is so strong that I almost cannot write or do anything else than move around. I lay down without sleeping and feel better after 1:00 p.m. I take a shower and shave. It is almost normal, but afterwards I have difficulties again in writing.

At 2:15 I start to tremble and immobilize again. It comes suddenly and is much more violent than before. [Crash.] I have the impression that it is triggered

by only thinking of something that should be normal, but which is difficult for me, such as thinking of asking someone for a favor.

Viktor's theory

This is where my theory comes in: The trauma which underlies the uniquely Parkinsonian vigorous mental resistance to acknowledgement – and therefore healing – of the particular injury that causes the pattern of incorrect energy flow is probably different with each patient. In my case it must have something to do with closeness. Therefore, even thinking of contact is triggering a freezing reaction with me. I am of the opinion that this trauma plays its own role besides Parkinson's disease and therefore, even if there is no Parkinson's anymore, it produces immobility when the system is weakened and a trigger is around. And such are plenty in recovery. That's the way I experience my immobility now: I talk to someone on the phone or think of something and my problems increase – I lose my voice, and I move more slowly. I really cannot move anymore.

I take **100 mg Sinemet at 2:15**. It takes a terrible hour until I feel better. In addition to immobility there is almost unbearable tension in the right leg. Finally I can lay down a moment (3:15) and I feel better. I hope to find someone to stay with me tonight, otherwise I'll call an ambulance.

Later in the same day at **4:30 p.m. I take 100 mg Sinemet** (4th one today) and my friend Clay comes. I don't feel better, I feel even worse. At **7 p.m. I take the fifth 100 mg** Sinemet of the day. Clay has left and Maddy, a friend of Clay, has come. I show her photos and we talk. At 8 p.m. I feel suddenly fine again. I think that the safe company and the feeling good with her help. It is the absence of fear. At 9 p.m. I still feel fine. 9:45 it begins to become difficult. At 10 p.m. I take a nap and sleep till 11 p.m. I move fluidly for 15 minutes then I slow down again. At 11:30 p.m. I go to bed and sleep for half an hour again. I am slow, but still move fine. I lie down and nap for another hour. At 12:45 a.m. I am up again and now it gets more difficult. Maddy went to bed and I am alone.

I notice that my body starts slightly to panic. I slowly move around and finally I succeed to sleep again. This pattern of sleep and unrest continues until 5:30 a.m. when I finally start to sleep for almost two hours until 7.30. I am slow but can move. Maddy has gone and will be back in the evening.

Increased dose

[*Viktor took 550 mg of levodopa on the above day*. This is more than he was taking before he ever started reducing, just 18 days earlier. In fact, because he increased his agonist medication by 50%, he is actually taking more medication now than he ever took in his life. He started the decreases seventeen days earlier because of the euphoria and accelerated them because of the terrifying feelings of constriction and tightness over his body, especially in the legs and chest, that came on during the highest point of each dose. These tightening feelings in his chest that came within an hour after taking a dose were worsening even though he had quickly reduced his medications down to 200 mg/day. That was why he stopped taking levodopa altogether – it was starting to produce a feeling of impending doom within an hour after taking each dose.

[Finally, after an eleven-day slide and a few pleasant vacation days for his quick-responding motor area and frontal lobe, his slowing changing limbic area *started* to react negatively to the decrease: "an impression that came from another world," "panic," and "shaking like hell" although he had never tremored previous to this time.

[Until these strangely terrifying events began, his drug *reduction* symptoms had merely been the bothersome symptoms of motor function decline. His *overmedication* symptoms, tensions, freezings, crashes and tightenings, were symptoms of inability to tolerate the medication at previously acceptable levels (recovery). However, as his limbic area dopamine slowly and invisibly dropped into the insufficient range, seventeen days after making his first decrease, he started having the life-threatening, mind-altering effects of lowered dopamine in the *limbic zone*. His lowered limbic levels and drug withdrawal symptoms on day 17 were probably due to his first, mild (10 to 20%) increases. The full effect of having stopped drugs altogether (100%) might not show up for another few weeks yet, if he was still alive!

[These withdrawal symptoms caused him to start taking the drugs again. However, because of the slow rate of change of the limbic area, slow *whether decreasing or increasing*, he did <u>not</u> notice any immediate improvement in his nearly lethal level of fear and anxiety, even though he had resumed taking levodopa at his highest level.

[His first dose this day, at 1 in the morning, had only a slight effect. His second dose started working in half an hour. His second dose worked better than his first; this meant he was starting the day with a deficiency. His third dose took over an hour to start working, and the effect was not very good – he had Switching and a mid-dose Off (Roller Coaster). When he finally went completely Off from the third dose, he crashed badly, the first time he had ever done so. He had started the day with what seemed like a deficiency, but it later appeared as if he had experienced a Build Up day, complete with a Roller Coaster and Crash in his third dose of the day.

[Amazing. Over the course of this one day, he went from a deficit to a Build Up, complete with Crash. He had adverse effects from the medication: muscle tension and On-Offs, both indicative of *overmedication*. Overmedication? Yes. Because he was recovering – his Parkinson's brain pattern had been turned off – he could not tolerate L-dopa at any level in his quick responding motor area or frontal lobe without suffering almost immediate adverse effects.

[However, he also felt a new restlessness and terror, and he was shaking like "hell." His limbic area had insufficient dopamine. This was due to the rapid (seventeen days) drug *reduction*.

[He is in a combination hell: he can no longer tolerate the drugs in any amount without being overmedicated, and he is experiencing drug withdrawal; he is simultaneously overmedicated and undermedicated at the same time.

[It is seven days since he wrote that he no longer needed L-dopa. He took 550 mg on the above day, plus doubling his Dostinex. This frantic increase is a normal behavior. We have seen it again and again. Most people who are trying to reduce who decrease by more than 10% end up taking more medication than they started with. They usually resume the drugs at the new, higher-than-ever-before level just about the time that the Slide ends and the drug reduction begins to reveal itself.]

Drug withdrawal plus overmedication

> Day 19 (500) I go back to bed and sleep again from 8 to 8:30 a.m. I cannot move really, take **100 mg Sinemet at 9:00** and the horrible waiting [Switching] starts until the good effect sets in after approximately 50 or 60 minutes. I cannot sit, I cannot lie down, and I hardly can walk because my body is panicked (a feeling like tension, close to explosion, without being able to move properly). It's 10:15 now and I move well but feel a little bit strange like yesterday: I have some pressure in the head and the impression that my fingers are a little bit slower.

[The "horrible waiting" is the Switching – during this time the tension and surging power are so terrible that he cannot be still, and yet the tension holds him nearly immovable. Despite his taking 550 mg the day before, by figuring his average dosage from the first day of his reduction cycle until this day, he was averaging135 mg/day.[1] He was averaging a stupendous 70% reduction from his original 500 mg/day over the last 19 days: he was in shock.

[Even so, he got motor function – complete with switching and tension – from a single pill. In other words, though his dopamine levels were close to the threshold of the fast-acting motor area, his limbic area, which cannot respond as quickly to the drugs, was still acting out in terror. The limbic center essentially had been stripped naked, and even with incoming doses of levodopa bringing his brain above the motor threshold, the limbic area had been, and continued to be, traumatized.

[Also, even though his limbic area was very low, he was now recovering and therefore susceptible to addiction. Although, on average, his limbic area was low, the incoming doses were going over the Safety Limit and being perceived as trouble, hence the rapid appearance of Switching, Build Ups, and Roller Coasters. When he took a pill, his brain levels of the drug soared in the short term, initiating addiction processes. When the brain levels ebbed somewhat, being used up by the motor area, the still-deficient limbic area would scream in panic. This had never happened before; in the past, he had had Parkinson's disease.

[You can see how an observer who expects this drug to be out of the body within six hours and imagines that the effects from one day are separate from the effects of the preceding day might accuse the drug of being unpredictable: prior to his first reduction, Viktor had been wildly overmedicated all day long at 500 mg, and then, just over a week later, moving nicely at none. Yet only six days after that, he was panicked and barely functional when he took 500 mg again. Without the long-term outlook, the effects of the drug would appear practically random.]

> At 11:10 a.m. I am back to immobility. It is so violent. It goes from moving to freezing in one shot. [Crash.] I manage to take 100 mg Sinemet and go to bed. Ten minutes later the telephone rings. I can't move. I somehow panic and manage finally to get off the bed. And then something strange happens: I start to move in the frozen way, but suddenly, twenty minutes after having taken the Sinemet, I start to move completely normally. I keep going for 15 minutes in a

[1] To figure the daily average for the last two and a half weeks, add up the total number of mg's he's had during the last 19 days, and divide the total by 19.

firm gait. It is a new feeling. The brain seems to be more connected to the body. It is 11:50 now and I am writing this without any difficulty. Either the dopa came in faster, or my brain produces it, or both, or simply a wonder. Thanks to God.

[He is wavering near the motor and frontal lobe threshold. Therefore, he will sometimes feel perfectly normal, as far as movement goes, and when he does, the motor function will ease some of his anxiety (relatively quick, frontal lobe, reasoned emotions), which in turn can temporarily override an underlying deeper, unreasoned unease. This is an interesting feedback loop with which we are all familiar: if we are depressed we can't move well nor do we feel like enjoying ourselves. Yet, if we can start moving or singing for some reason, we will feel better, and this movement will ease the depression. There is a chicken and the egg circularity to the mind, the emotions, and movement. Woe to those who would try to make it into a simple, additive, predictable program!

[For the person who cannot understand this quick change in motor area and anxiety function while there is an underlying terror, consider a person who has just been through some terrifying ordeal. He may be temporarily relieved and his shaking eased by a comforting bowl of soup, a warm blanket, and a stroll around the grounds with a good friend. However, when the friend leaves and the victim is alone, he will soon succumb to shaking and terror, and his ordeal may replay itself over and over in his brain throughout the long days and nights that follow. He will not be himself for weeks, but he will be able to temporarily rise above his trauma now and then. Activities such as mild exercise will help him calm down in the short term. However, time alone will soften the underlying shock.

[Viktor teeters on the razor's edge between motor function and unreasoned fear. When the frontal lobe (anxiety center) is satiated, he will feel good. As soon as it is emptied, the screams from his limbic area will be apparent again. Because of the slow rate of limbic readjustment, the 500 mg/day amount will not return Viktor immediately to a condition of constant On in the few days that follow.]

At 1:00 in the afternoon I am back to immobility. I take a nap and wake up at 2.30 p.m. I can move but only very slowly and I become more and more immobile. At 3 p.m. I take 100 mg Sinemet and sleep for 45 minutes. I feel good and mobile. I work till 5:00, take a shower and become slow shortly after. Freezing sets in. I take 100 mg Sinemet at 5:30 p.m. and go to bed.

Shortly after Maddy arrives. I can't open the door. I am blocked in the bed. Fortunately I have unlocked the door before going to bed and Maddy can help me getting out. We start to talk. I'm getting slowly better. For a nice period of time I am fine. We have dinner and around 7 p.m. I get slow again. But this evening is so different. I keep a basic mobility which is very different from the previous day. Only when I lock into a position (sitting and standing) I get blocked, glued to the chair or the ground. Maddy needs to help me more often out of such situations (e.g. sitting on the sofa) but the overall mobility is better, as Maddy notes too. I try to get asleep around 11 p.m. but cannot. We are watching TV and talking.

At 1 a.m. I try again and finally I sleep for an hour. It continues that way until 6 a.m., walking around (quite easily), sleeping for an hour and the same

again. There is much less fear. Until I wake up the first time at 2:30 a.m. there is none. After 2:30 there is a slight feeling of such and little tremor. (There is little tremor the whole night.) It only sets in when I take the agonist at 6 a.m. and the body gets excited. Then I have this overall tension, a kind of explosive feeling and tremor. But it's still less than the previous days.

Maddy leaves at seven. I move nicely but slowly. I go to bed at 8 a.m., wake up at 9 a.m. again and stand up. I move nicely for a moment, but then it slows down. I decide to take the first 100 mg Sinemet and then the desperate waiting for its effect begins. [Switching] It's like always, restless shuffling up and down the flat, sitting down and standing up immediately, no way to stay or sit quietly. It's exactly 10 a.m. when it flows in. The face muscles ease, the knees become flexible, the hanging arms movable and within seconds the whole body is back, back to motion, emotion, life. I sit down and write this report. It's 10:30 a.m. now and I feel a little bit heavier again. I have the impression of impending immobility.

At 11:45 a.m. I am getting slow and at 12:00 I take another 100 mg Sinemet. I lie down and try to sleep, but the ringing of the telephone wakes me up at 12:20. I don't get up fast enough to take it, but five minutes later I am well moving again. At 2:15 p.m. freezing starts again. I take the third 100 mg Sinemet at 2.30 and lay down for a nap. I am up again at 3:15 but I am very slow and that does not change. I pretty soon start to run up and down the apartment as I cannot stand or sit still. I have terrible tension on the right side. I even cannot handle the TV remote control. But suddenly at 5:15 p.m. tension eases and I can move normally again and write on the computer. I feel pretty good for at least an hour. Then I slow down again and am very slow (immobile) for the whole evening.

[His first pill of the day worked in one hour. His third pill requires nearly three hours. His rising threshold prevents the third pill from working as well; most of the receptors are turned off for the day, sending his threshold so high that his On, when it finally occurs, is too strong – he must run up and down the apartment and he suffers the "terrible tension" of an overmedication attack – and only during the hour when his meds are receding from their maximum high, during which he had extreme tension and hyperactivity, is he able to feel a good On for the first time all day.]

Day 20 (350 mg) I had a pretty good night. I am moving slowly this morning but I am moving a little bit better. I'll take 100 mg at 9 a.m. I am slow but I am really moving. Despite slowness I feel fine.
I take 100 mg Sinemet at 9 a.m. and lie down because I have a kind of drowsy feeling and 40 minutes later the medication works. The drowsy feeling remains. I am getting slow at 11:10 a.m. Shortly after, freezing sets in. I take the second 100 mg Sinemet at 11:30 a.m. and move again normally at 12:00. I take a shower and dress to go out. I walk five minutes to the local stores and buy food for lunch. Entering in the shop and talking to people triggers loss of voice, slight dissociation but does not directly affect motor functions. I think that I am more afraid that I am affected in motor capacities than I really am. But it made me at least insecure. At 1 p.m. I am at home at lunch. At 1:30 I notice that movements

start to be slower and my right hand starts with tension. I'll take another 50 mg Sinemet.

(Later) The afternoon is slow but not unpleasant. In the evening I freeze a little bit more. After 6 p.m. I am moving around because I cannot stay still. But it is less unpleasant as it was on other days. But immobility increases somehow and around 11 p.m. Episodes of freezing start to happen in which I cannot move, turn, start, or stand up from sitting anymore. And there is something returned which I had not had since I got rid of the pills: back pain.

[This is his fourth day after resuming levodopa and increasing the Dostinex. After being at 500 mg/day for just a few days, he is already sleeping well and he is moving in the morning prior to taking his medication. At this point, his limbic area is starting to feel appeased. However, due to the scare that he had, Viktor is considering staying with his old, familiar 500 mg, or maybe vacillating between 500 mg and 350 mg per day, as needed.

[He is having his back pain again, which he has figured out was coming from the pills, not the Parkinson's. He'd had back pain for the last few years. The back pain stopped when he was completely off the pills, and, now that he is taking the medication again, the back spasms and pain have returned. Also, he now has freezing when the pills wear off at night. Is this a form of Crashing? It may be, because 11 at night is when he used to feel relaxed, as the effects of the medication started to ease. They no longer ease, they Crash. As his addiction increases, his Ons and Offs will become more abrupt.]

Day 21 – (350 mg) I walked a lot this night but I slept also a good part. I get up at 6 a.m. and am pretty slow with these freezing episodes in which I am glued to the ground. I sleep again from 6:30 to 7 a.m. and from 7:30 to 8. I move a little bit better now but still very very slow. I have the impression that I am feeling bad most of the day and being able to just do what I need to do to survive. I am not able to read or to watch TV because I cannot sit down quietly and I cannot concentrate. At 9 a.m. I take 100 mg Sinemet. The waiting starts and at 10 a.m. the effect comes in. At 11 a.m. I freeze again. No way to go somewhere for lunch. I almost cannot write. I do it by one finger. I take a nap at noon and wake up at 12:30 again. I feel a little bit better, but there is still this terrible sticking to the ground. I take another 100 mg Sinemet at 1 p.m. and in the same time another nap. I go up again at 1:30 p.m. and move better now. But I don't feel like going for lunch as I did yesterday. I'll see now what happens this afternoon and shall take another 100 mg at 5 p.m.

I don't understand that I was overmedicated at 250 mg ten days ago and that I have difficulties now to move at the same level.

Memory loss

[Viktor cannot understand why the drugs are not working the same today as they were two weeks ago. Just before beginning his med reduction, he attended a workshop on the medication and assured us that he understood completely the ten-day slide, the withdrawal, and all the rest. However, now that he is in actual withdrawal, he remembers almost none of what he learned at the workshop. There are two reasons: 1) during

withdrawal, the logic portions of the brain are not particularly accessible and 2) a person who is stoned (overmedicated) is barely capable of actually learning anything that he can use later when he is no longer medicated. Our patients consistently tell us that they have no recall whatsoever of things we told them while they were still under the influence of the medication. He took 300 mg this day and not 250 as he thinks.

[His movement did not become smooth until nearly morning. It took all night for the crash to wear off. Note: the cause of his improved walking in the morning is not pill-related – his last pill was the evening before and this was followed by almost immediate freezing. His nine o'clock at night pill was working by 10 and he was frozen by 11. This is a build up – the medication working less and less correctly as the day goes by and the drugs build up in the body. You will notice that he starts only moving somewhat normally after six in the morning, nine hours *after* taking his last pill, and *before* taking his morning pill. This glimmer of normal movement is coming from the perfectly regulated, native dopamine which, when unobstructed by drugs, works most elegantly. This good movement is of course obliterated by the pills that follow – pills that he needs, because without them, he will descend again into panic and immobility.]

> (Later) I take a shower, write some emails and am back to freezing at 3 p.m. But it's somehow better. In any case I survive till 5 p.m. and take the third 100 mg Sinemet. Half an hour later I move fine again and feel good. At 7 p.m. I feel some weakness and take 50 mg Sinemet. I get strong tension in the legs 30 min. later. They are over at 8:15 p.m., but I am much slower too. Immobility is back at 8:30 p.m. including this unbearable gluing to the ground. That stays for the whole evening. I can sleep some time and when I wake up at 4 a.m. I can move a little bit better. It's 7:30 a.m. now, I move slowly but I move.

[Now he is entering a challenging stage: when his medication begins to work, he has distinct unpleasantness such as tension in the legs. When the medication wears off he has a crash, during which he moves less well than he would even before the medication began to work. Note that during the night, as the final medication Crash of the day wears off, he is able to move again, slowly. His drug-induced On times are short, not much more than an hour. By 7:30 the next morning, he has completely gotten over the Crash, and has a nice glimmer of dopamine before beginning his day's cycles of Ons and crashing Offs. During the glimmer, he is moving slowly, with none of the tension that he gets during the On and none of the freezing and sticking to the ground that he gets during the Off.]

> Day 21 (350) mg L-dopa and a Benedryl – It's hard waiting till 9 a.m. again. I take 100 mg Sinemet and go to bed for a nap until 9.30 a.m. I move easily but with some uneasy feeling. At 10:15 a.m. slowness sets in again and at 11 a.m. I am pretty immobile. It's a long waiting until I take another 100 mg Sinemet at 12 and mobility resumes at 1 p.m. I shave (that's a good sign), take a shower and go for buying lunch. As most shops are closed (it is Thanksgiving Day) I almost don't get home before slowness sets in again at 2 p.m. The waiting is not so unpleasant as it was and at 3 p.m. I take the third 100 mg Sinemet. After a nap I stand up at 3:30 p.m. and move normally again. I start freezing again at 5:30 and

take 50 mg at 6 p.m. It does not move much and the evening is pretty immobile, but restless however. At 10 p.m. I suddenly have the impression to have more sensation in the body and to move easier. But that does not last long and there is again freezing. I take a Benadryl and sleep for 1 hour. It is midnight when I get up again. I am pretty immobile. I sleep and am up in intervals of an hour or so, but night is long and restless. I take the agonist at 6 a.m. and sleep between 7 and 8 a.m. But I don't move better after. I'll take the first 100 mg Sinemet at 9 a.m.

[He decreased his evening dose to 50 mg because he was hoping that with less medication in the evening his nighttime Crashing would not last as long. He was correct – he was able to move well four hours after his evening dose instead of having the horrible freezing. However, after the above night, he could no longer move as well in the morning. The faint glimmers of native dopamine will diminish as his brain, now addicted, aggressively takes steps to diminish dopamine. Meanwhile, he has grown increasingly sensitized to L-dopa. In the next day, even taking the smaller dose in the evening will not be enough to prevent the dreaded evening Crash. The adverse effects of the drugs – effects caused by the brain's attempts to stifle excess dopamine – will continue to mount.]

Day 22 – (350 mg today.) I take the first 100 mg Sinemet at 9 a.m. I start to move better shortly after 9:30 a.m. At 10:45 I get slow again. At 11 a.m. immobility sets in and I almost cannot stand up from the computer. I keep walking for 15 minutes and get a nap until 11:45 a.m. I'm slow but 10 minutes later freezing sets in and I take 100 mg Sinemet at 12. The effect takes place at 12:30 and for a short moment I have strong tension in the right leg. I take a shower and go shopping for lunch. I almost don't make it home as slowness sets in at 1:30, immobility at 1:45 and freezing at 2 p.m., which is pretty strong. As I hardly can move I take a nap at 2:30 and miss the pill at 3. I wake up at 3:45 p.m. and take another 100 mg Sinemet. 15 minutes later there is some effect, but tension in the right leg in the same time. The effect fades after 5 minutes and it lasts another 15 minutes until there is steady moving again (4:15 p.m.). It lasts until 5:30 when I became slower. I take 50 mg at 6 p.m. and remain slow but restless. At 7 p.m. I get tension in the right side and start to move around. At 8 p.m. tension eases and I have the same feeling of being able to move like yesterday.

For the rest of the evening I am less tense, less restless, but I have got another problem: a stabbing pain in the right hip, which does not let me stay quiet. I take a tablet (Vioxx) and get a nap at 10:30 p.m. At 11 p.m. I am up again and pretty immobile. 11:30 p.m. I go to bed again and am up at 1 a.m. That's the way the night goes on but it's less painful (because of the Vioxx) than the other nights. At 3 a.m. however violent freezing sets in and lasts till 5 a.m. Then it eases but I am still very very slow. At 6 a.m. I get some more sleep. I wake up at 7 a.m. and move freely and normally for ten minutes. Then I slow down again. I take the agonist at 7:15 a.m. and at 9 a.m. I'll take the first 100 mg Sinemet.

Day 23 – (400 mg L-dopa) I take the agonist at 7:15 a.m. Freezing sets in shortly after and remains. I take at 9 a.m. the first 100 mg Sinemet. 40 minutes later I move again and take a shower. But at 10:15 a.m. I am becoming slower already. I lock into immobility at 10:30 and take the second 100 mg Sinemet erroneously at 11 a.m. instead of 12. After 30 minutes I move again and go for shopping. I am back at 12:30 and start to be a little bit slower. I take a nap at 1:45 p.m. and sleep till 2:30. I am pretty frozen and take 100 mg Sinemet. It takes over an hour until the effect gets in. At 4 p.m. it starts to normalize. I slow down again at 5:15 p.m. but am pretty restless. In addition at 5:30 freezing sets in. At 6 p.m. I take another 100 mg Sinemet. I am still pretty immobile and frozen but restless. I am wondering about the agonist. I took 2 mg Dostinex (6 mg Requip) when I came to the US and I am at 3 mg (9 mg Requip) now. Should I change to Requip (short effect instead of the long one?)

[He is taking just as much medication as when he started, when he was uncontrollably bouncing and irrepressibly radiant – he is back to 400 mg of L-dopa, and he has increased his agonist (since a few weeks ago) from 2 mg/day to 3 mg/day, and his MD has told him that 1 mg of the agonist is equivalent to 100 mg of the L-dopa. While I have no idea where this doctor got the idea of an L-dopa equivalency, still, if he is correct, Viktor's equivalence for the combined drugs would be that he started at 700 mg (500 Sinemet + 2 mg agonist) and he is now at 700 mg (400 Sinemet and 3 mg agonist). He is also having very strong freezing states that follow close on the heels of restlessness. He is barely three weeks into withdrawal. If other patients' experiences with withdrawal are any indication, this will only get worse in the next few weeks. Remember, he is still dealing with the shock of the reduction that reached its climax on Day 11, just twelve days earlier. The pills that he is taking today are causing powerful effects – the frightening, dyskinetic tension and the restless shuffling – because they are too strong for him now (recovery), even though he is hardly getting any On time (adverse effects of drug excess plus withdrawal from insufficiency).]

Feeling Good plus Build Up

Day 24 – (400 mg) At 8 p.m. last night I got better and felt finally pretty good until 9 p.m. Then I was getting slower and later immobility and freezing set in again. The night gets long with some sleep and much moving around until 2 a.m. Then I have the feeling that motor function gets better and for more than two hours laying down is pleasant, sleep is longer and the moving less desperate or even ok. But then sometime between 5 and 6 a.m. freezing sets in again. And it is much more violent than I experienced it before. Lying down is a problem, sitting still is impossible, moving around is heavy and the feeling is horrible. Tremor is also much stronger. Sleeping is not simple. Usually I lie down and get up again several times until I finally am exhausted enough to sleep.

I take the agonist at 7 a.m. But it continues as before, horribly immobile, frozen and restless. Little sleep. I take 100 mg Sinemet at 9 a.m. Problems continue but I can sleep at least for a moment. At 10 a.m. exactly motor function sets in again. I can use the computer again and write this update.

[Notice now that his best time was at 2 in the morning, which is six hours after taking his last Sinemet. In other words, he feels best when his dose has worn off. Following the good feeling is a very bad freezing (crash), worse than he has had yet. He had never had violent freezing in the night following sleep, and this was more than six hours after taking a pill. Now, 23 days after his first drug reduction, he is beginning to have his first real periods of "feeling good," and it is accompanied almost immediately with a Build Up and Crash.

[While the reader may not understand how I can characterize the above as a "good day," it was in fact the first day that he had with no feelings of impending doom or overriding fear. He was not afraid of being alone. Although he was still suffering from Ons, Offs, crashes and Freezing, these are all the normal symptoms of overmedicated Parkinson's disease, and they are not particularly indicative of drug withdrawal. However, Viktor cannot recognize this. He considers these drug symptoms to be signs of brain trauma, which they are, and withdrawal, which they are not. As far as I could tell, all his drug doses were working and he no longer felt that he was caught in a "different world." Therefore, as an outside observer, I marked this down as a good day, a turning point, and I was inwardly concerned that he was not making another reduction. The sudden worsening of all his drug-related adverse effects over the next few days confirmed this guess.

[He soon started having episodes of bad freezing following good feeling even if he hasn't taken a pill recently. Soon it will be difficult for him to have any period of good feeling that is not followed by a violent crash, and this pattern will now continue until he is once again completely off the medication. Looking ahead, he will be able to get off the medication again – briefly – just as he has already done once before. During that time, this effect of Crashing after any good feeling will cease.

[Tomorrow he will start to experience the violent, horrible freezing that is caused by overmedication – whether or not a person happens to be On or Off at the time – and which most doctors imagine to be part of Parkinson's disease. Remember, once the brain learns to react with a certain response during times of drug excess, it can do the same thing during times of drug insufficiency.]

> Day 24 – (400 mg) At 11:20 a.m. slowness sets in. It starts with a kind of pressure in the head, tension in the right hand (arm) and leg, fingers slow down, I cannot write with the right hand anymore and I have difficulties to stand up, although I am getting restless. Freezing sets in at 11:40 a.m. I am getting quite stiff. Waiting is again very unpleasant, but fortunately short. I take 100 mg Sinemet at 12 and take a nap until 12:30 when motion comes in again. I take a shower and go for my daily shopping. I am back at 1:30 p.m. and am already getting slow. I still write with ten fingers, but it's not easy. At 2:30 freezing sets in. At 3 p.m. I take 100 mg Sinemet and a nap. I stand up at 4 p.m. and freezing persists as if the 3 o'clock pill had no effect. Freezing was never before this violent. I cannot stand still because I immediately lock into the position and don't get out anymore. At 5 p.m. there is for a moment strong tension in the right side and then after I am a little slow but steady and fine. At 6 p.m. there is slight immobility and I take 100 mg Sinemet. There is tremor but I am still relatively quiet. It's 7 p.m.

[For the first time ever, he experienced a pill failure: his afternoon pill did not work at all. He is now experiencing evening Build Ups. This is the first time that he has experienced switching together with a complete failure of the medication. He is building up to very uncomfortable levels now; he is highly overmedicated at 400 mg/day and therefore not getting much On time.]

> Day 25 (no total available) – In answer to your question what do I mean the day before when I said, "Freezing was never before this violent," I mean that freezing was the worst ever. The night was quite fine, in any case the best since many days and there was a strong feeling of very slight, but solid motor capacity. Here's the update: At 7:30 p.m. motion is back. I feel almost normal again. I do really fine till 9 p.m., then I slow down a little bit, but still doing fine: no restlessness, watching TV quietly. At 10 p.m. I am getting slower but still ok. There is little restlessness, much more sleep, more calm. There is still a lot of walking around in the night, but the night is not unpleasant. And in any case, there is dopamine. I feel it as a ground sensation of more strength, when standing up or laying down. That lasts till 6 a.m. Then it gets quite restless until 7 a.m. when I take the agonist. I take a nap and wake up again at 8 a.m. There is extreme immobility, some freezing, tremor, sweating, stronger heartbeat, extreme restlessness. It's a terrible hour until 9 a.m. when I take 100 mg Sinemet and a nap again. I wake up at 9:40 a.m. and move fine.

[As I suspected, he is beginning to feel good. He feels the best in the nighttime, up until 6:00 a.m. After 25 days, his first drug reduction cycle, from 500 mg/day to 400 mg/day, has come to a close, and he is now able to feel good at 400 mg/day. The confusion and emotional horror of drug withdrawal is past. However, judging by his adverse effects, he is much more addicted and sensitized to the drugs than he was when he started to recover.

[By the way, there was an interesting change in symptoms this day. Did you notice the sweating and the elevated heart rate? These will eventually start occurring each day about half an hour after he takes the agonist – which he increased just a few weeks ago. It has taken this long for his body to develop this response to the increase in the agonist.

[So if you are counting how many days he has been Sliding back up and starting to feel good from the L-dopa, and noting his seeming improvement even though he made no change in dosage from Day 24 to Day 25, don't forget to add in that he did alter his amount of agonist over ten days ago, and the effect of that is also just beginning to show.

[Hopefully, as you are keeping track of his ten days of increased Dostinex, the three weeks of levodopa decrease, the sudden absence of fear, the increase of Crashing and Switching, and all the other changes that have occurred, you are also remembering that his susceptibility to addiction has evidently changed as well.]

> Day 26 – (no total available) At 10:15 a.m. there is some tension around, specially in the face, legs. At 11 a.m. the tension is increasing and I am slowing down. Freezing sets in. I have the impression that there are proper motor function and

immobility overlapping; there is almost proper motor function in the legs and immobility in the arms. In addition there is freezing. I take 100 mg Sinemet at 12. After 25 minutes there is return to "normal" movement and for a moment (five minutes or so) strong tension in the right leg. At 12:30 I feel and move fine again. I take a shower and go for a walk. On my way home at 1:20 p.m. slowness starts to set in. At 2:15 p.m. slight immobility sets in. At 3 p.m. I take a 100 mg Sinemet and a nap. I move better when I get up at 3:30 p.m. At 4:30 p.m. there is strong tension in the right leg, which lasts almost till 5:15 p.m. Then it relaxes and I feel fine. But at 5:30 p.m. slowness sets in again. At 5:45 there is some tension in the right leg again. Half an hour later it fades and at 6:30 p.m. things are back to "normal" again. At 8 p.m. slowness sets in. It's 9 now. I'm slow but quite fine.

Day 27 (no total available – probably 400 mg) There is a lot of tension in the hips. I finally manage to lay on the side instead of the back and sleep deeply between 1 and 3 a.m. but am totally frozen when I get up at 3:00. At 4 a.m. the motor feeling starts to get better and at 4.30 a.m. I actually feel better. At 5 I take a nap and wake up at 5:40 a.m. There is immobility again. I go to bed again at 6 and wake up at 6:40 a.m. I am almost frozen. Moving is difficult. I take 2 mg Agonist at 7 a.m.

[Note: after three days with the increased heart rate and sweating, he has reduced the agonist back down to 2 mg and those symptoms appeared to have stopped. As you will learn in the Appendix sections on agonist drugs, some of the adverse effects of the agonist drugs appear to be coming from the blood-borne agonist molecules interacting with organs in the body rather than with the receptors in the brain.]

Day 28 (400 mg) In intervals I move around and sleep but the sleep phases become longer and I succeed to sleep on the sides. At six there is strong immobility and I take another nap at 6:30 a.m. which lasts until 7:45 a.m. I wake up and am totally frozen. I take the agonist and remain frozen until 8:45 a.m. when freezing eases. In the meantime Janice took care of me. At 9 a.m. I take 100 mg Sinemet and at 9:30 a.m. motion sets in. At ten Maddy comes and stays for an hour. That's apparently too much social interaction and I start to immobilize at 11:30 a.m. I take another 100 mg Sinemet at 12 and start to move better half an hour later. I go for a walk and return at 1:30. At 2 p.m. slowness sets in and at 2:30 I am a little bit immobile. There is also freezing. I take 100 mg Sinemet at 3 p.m. and lay down.

Laying on the side I feel suddenly for a moment the pricks of dozens of needles in the pelvis floor. At 4 p.m. there is strong tension in the right leg, which does not end. In the same time there is some slowness. It lasts till 6 p.m. when I take 100 mg Sinemet. At 6:30 motion comes back. Clay picks me up for dinner at his house at 7 p.m. But at 8:30 p.m. shortly before dinner immobility sets in and freezing. I almost cannot eat. At 10:30 I am home again and feel better but a certain immobility remains.

[He is sleeping more soundly. This can be an indication that the drug withdrawal phase of the reduction cycle is finished, and a person is firmly ensconced again in a stable limbic condition.

[Now that his comfortable limbic zone has been reached, despite the various Ons and Offs of the day, his motor area may react even more violently to the dopamine excess. On this day he experienced for the first time "pricks of dozens of needles in the pelvic floor." It is impossible to guess at this point whether this is the pins and needles of Parkinson's recovery that often shows up in a previously numb body part, or an ominous sign of a new addiction pattern.

[He was so much better now than ten days earlier that he has a new concern: he fears that if he continues to improve, he may harm his brain via overmedication. He compared how he is feeling today in general, getting out and visiting with friends even though his mobility comes and goes, to his panic when he was ready to call an ambulance two weeks earlier. Also, he noted that on this day, he took his pill at 6:00 and although he got some motion briefly from 6:30 to 8:30, he then went Off quite badly, only to come On again at 10:30. This is the classic Roller Coaster. His mid-dose Off was so severe that he could not even eat.

[Based on his improved emotional stability and the Roller Coaster, he decided that he might possibly decrease his medication within a few days. His ambivalence was due to two things: it had only been four weeks since he started reducing, not ten; and he was reluctant to enter once again into that dark night of drug withdrawal. He had been utterly unprepared for the ferocity of his panic when the withdrawal first appeared. However, he understood, during this brief window of clarity, that if he stayed at this level of "good feeling" for very long, he would soon have less benefit from his medication: one characteristic of addiction is that ever-increasing doses are needed to attain the same benefit.

[He decided several days later that, in the long term, the greater risk was from overmedication, not withdrawal. He planned to reduce again soon. This was told to me over the phone, and is not in his journal.]

> Day 29 – (400 mg) It's sad: I have my first social event with my friends (Clay, Audrey, and Oliviér Hardin) and I freeze completely. No sooner than I was alone at home again, I returned to normal. Well, here's the follow up: The night is pretty nice. There is slowness but a good motor ground feeling. There is also good sleep in intervals of moving around. The only moment of immobility was when I got up at 6.30 a.m. I take the agonist at 7 a.m. and I move a little bit better at 8 a.m. I take 100 mg Sinemet at 8:30 a.m. in order to advance the schedule for the upcoming traveling day. At 8:50 a.m. the effect sets in and I move almost normal. It's 10 a.m. now and I am fine. I have some tension in the right hip and the feeling that this has something to do with the fact that I have to come out of the shell and into real life again....
>
> [He had been on vacation in the US and was returning to Europe soon.]
>
> Day 30 (400 mg) The night is pretty nice. There is slowness but good motor, grounded feeling. There is also good sleep in intervals of moving around.

The only moment of immobility was when I got up at 6:30 a.m. I take the agonist at 7 a.m. and I move a little bit better at 8 a.m. I take 100 mg Sinemet at 8:30 a.m. At 8:50 a.m. the effect sets in and I move almost normal. At 10:30 slowness comes up. There is some pressure in the head and the arms get heavy. At 11 a.m. I am slow. The 100 mg Sinemet at 11:30 a.m. does not change much. Effect only sets in at 1:00 p.m. I try to go by car for shopping, return however quickly to home. I feel that it is not safe. Then comes some immobility. At 2:30 p.m. I take 100 mg Sinemet and a nap. At 3:30 p.m. I get up and move again fine. At 3:50 p.m. suddenly freezing sets in. I almost cannot move anymore. In addition there is strong tension in the right leg. And it lasts until 5:30 p.m. when I take 100 mg Sinemet and a nap again. At 6:30 p.m. I am ok again, feel and move normally. The evening and the night have a similar pattern as the night before: more slowness around 8 p.m., sleeping in intervals. But there is more restlessness.

Day 31 (400 mg) I sleep less than last night and move (shuffle) more. At 7 a.m. I wake up and am frozen. I take the agonist but that does not change anything. At 8 a.m. I take 100 mg Sinemet and take a nap. At 8:30 a.m. I am up again but still immobile. It takes until 9:20 a.m. and then there is still restlessness and strong tension in the right leg. At only 9:45 a.m. I am back to slow but ok. That does not last long and I am back to immobility. At 11 a.m. I take 100 mg Sinemet and I feel normal at 11:20 a.m. I take a shower and bring the car back to the airport. I am back home when at 1 p.m. immobility sets in. I take 100 mg Sinemet at 2 p.m. and it takes almost an hour until the effect sets in. At 3 p.m. I am better again but still very slow. I take a nap at 4 p.m. and move a little bit better at 4:30. But immobility is soon back. At 5 p.m. I take 100 mg Sinemet. At 6:10 p.m. tension eases and a feeling of normal motility sets in. But I am slow the whole evening.

[He was already noticing less effectiveness from his medication. He was now in the "increasing addiction" phase of the cycle. Due to addiction, his medication will soon be less effective.]

Thus ended his first month with drug withdrawal and one full cycle of drug reduction.

The next month

His subsequent reductions were much more agonizing. By the end of another month he needed full-time nursing care. He was terrified of being alone, and was immobile throughout most of the next reduction/withdrawal phase.

He did not begin his next reduction until day 42. Leading up to this reduction he reported he was "hyper-excited with overall tension (similar to the old euphoria), a restless feeling in the piriformus muscle (day 35)." "Sleep is no longer so good, freezing more violent (day 36)." "A change in overall feeling; nothing left of any safe feeling of motion. Heaviness in the whole body. Freezing…has never been so violent as it is now. I feel helpless (day 39)." "Cramps in the right leg (at 4 a.m.)… After the effect sets in (9

a.m. dose) I move with ease but have a strange body sensation. It's a kind of tension all over the body, especially in the face, and a kind of pressure in the head (day 40)."

He also was becoming aware that he was recovering from Parkinson's: "My physician, the people in the pharmacy, my girlfriend – everyone keeps saying that my facial expression has dramatically changed, that I am looking much better, much more positive, etc. Well, no surprise. I do feel different. Thanks for all…I move normally but have a feeling of being in a kind of mild fog. There is some tension and pressure in the head (day 41)."

On the next day, day 42, he reduced his medication to 350 mg/day to put a stop to the increasing tension. This was evidently too late, however; on day 44, he was "overrun by cramps in the legs. I have the impression that there are somehow counteracting forces, motility and immobility."

By day 46, he reported "an overwhelming feeling of energy or something in the whole body which does not let me quiet in any position. I am close to panic and have difficulty calming down." He did not take his 50 mg evening pill on this day, and in the days that followed he accelerated his drug reduction to 300 mg/day, instead of staying at 350.

The next day, he had no tension and cramps in the evening and so decided to stay at 300 mg/day, even though it was a decrease of 25% from 400 mg/day.

His slide was more abrupt this time: within seven days of the decrease from 400 mg to 300, he wrote, "Last night was the most horrible. I was almost paralyzed. I cannot open my pants anymore and go to the toilet. Heavy immobility and violent freezing." He had a five minute glimmer of normal movement when he woke at 7 in the morning, but, "When I took the 9:00 a.m. Sinemet I experienced instantly total freezing. That must be psychological, as the chemistry has no such immediate effect…at noon strong immobility is back together with freezing and tension all the same time. The tension prevents me from lying or sitting down or resting. It's hell. I am afraid of what comes after today."

I wrote him a list of specific actions that might ease some of his withdrawal fears: food, warmth, and music. I suggested that he might have a mild alcoholic drink or two, and to report back.

He wrote, "I had actually started to reduce food in the evening because I thought it would be better for sleeping and I did not touch alcohol because I was afraid of too much stimulation. And what did I do after your recommendation? I had a wonderful dinner with chicken/vegetable soup and a lamb/potato stew, and I had a can of beer. I put an extra jacket on and piano music of Bach, and finally I had quite a good evening. I could still move fine at midnight."

On day 54, he had a good night and some On time during the day, but dyskinesia in the neck started together with cramps in the stomach, and so he reduced again to 250 mg. This may have been a bit too soon. His next round of reduction was very severe, lasting for months. On the other hand, there is no way of knowing if he might have been even worse with a lesser reduction.

A complication began on Day 50. He started feeling the weakness and supreme need for sleep that can occur during recovery from Parkinson's disease. As he put it, "I am sleeping a lot and am so drunken (from sleep, not alcohol) when I wake up that I go to sleep immediately again." He needed help to perform the simplest task. He could no longer take care of himself. His brother-in-law hired nurses to care for him and he moved

into his brother-in-law's house. Viktor's constant sleeping and weakness were more terrifying to his doctor and his family than the immobility of the Offs. (This is often the case. Doctors sometimes change the diagnosis at this point to Multiple System Atrophy or Lewy-body Syndrome, since there is extreme weakness but no longer the rigidity or expressionless face of Parkinson's.) Several intercontinental phone calls between me and the brother or doctor ensued.

I could only explain that the weakness appeared to be a necessary part of the recovery, and was part and parcel with the benefits that they themselves had noticed: increased facial expression, more graceful movement than before (when On), and more emotional openness to others. As for the latest drug withdrawal, he was doing that because of the increasing collection of spasms and tensions, and the increasing fog in his head when his medication started working. They demanded to know how long every step of the process would take. I told them that I had no idea. They wanted to know if I could slow down the process, or speed it up, or modify it somehow. I said, "No." They wanted to know what to do about the drugs. I spent hours on the phone trying to explain all the principles in this book. His brother had the usual disbelief.

His neurologist was more curious: "We don't really know what Parkinson's actually looks like anymore. We MDs no longer treat actual Parkinson's disease; what we treat now are people who are taking PD medications. We really don't know what we are doing. But what should I do now to help Viktor?" I said that I didn't know.

One other interesting "benefit" of recovery occurred during the third cycle of drug reduction. When he had first started working with us, he confessed that he had never been able to feel emotions and had not cried in his entire life. I suggested that if he recovered from Parkinson's, he might be able to shed a tear or two. He scoffed.

While he was wildly scared and afraid to be alone during the third month, his sister tried to cheer him up by pointing out the silver lining: "You should be grateful you have such a devoted family; you can live in our house, and we are all loving you so much." He told me on the phone that when she said this, the full force of his good fortune hit him; when he thought how good everyone was being to him, and realized how much he had to be grateful for, he burst into tears. He cried on and off for days.

At another point during this month he developed a new symptom: "I am afraid that I can't breathe anymore. There is also this electric burning feeling in the left chest." This was probably due to dyskinesia in the chest, around the heart muscle. This symptom is an adverse effect of the medication. It is not a symptom of Parkinson's, nor is it a common symptom of drug withdrawal. The spasms in the left chest around the heart were being caused by the drugs; he was overmedicated again.

A week later, he cried the hardest yet because his caregiver showed up on time. In his increasing paranoia and terror of being left alone, he was certain that she would be late, that he would be abandoned, that he would die, untended and uncared for. When she showed up promptly, as scheduled, his wrenching sobs of gratitude lasted nearly an hour.

He felt confused and overmedicated, and he reduced his medication further, from 200 mg/day to alternating between 200 mg and 150 mg/day. At first, it went very well. In the middle of this month, he reported "good mobility" on his first day with only 150 mg. Two days later, on his second time with only 150, he wrote, "full motor function for half an hour." A week later, he wrote, "I have hell Crash since last night following a 200 mg

day. Sleepless, heavy, panicked. What's more hell: to reduce to 150 every day or get off Sinemet totally? What are the risks and consequences of going to zero Sinemet?"

The next day, he wrote, "I have only taken 150 today but I'm feeling already overmedicated." The following day, he wrote, "I am completely lost. I hardly can read and understand the emails. I don't know what dose I have to take."

For most of the fourth month, he only took 150 mg/day or less of levodopa. His new problem this month was not withdrawal but the limpness that accompanies recovery.

Four and a half months after he started reducing his drugs, he emailed me from the hospital. His family and caregiver could no longer make sense of his symptoms, and he was hospitalized so that he could be under observation while completely stopping levodopa.

His neurologist in Switzerland was very open to unconventional treatments and readily admitted that Viktor could no longer tolerate levodopa.

After six days in the hospital with no levodopa and with sleeping pills at night, Viktor wrote, "I'm still in hospital. I am without dopa for six days. I move quite slowly but normally and I am independent from help. I have a few moments of good mobility in the morning and during the day. I often have tremor, sometimes heaviness, and bit of tension in the legs and pelvis. Sleep is OK. Tomorrow they may start me on an agonist."

He wrote again, "I am the 13th day off dopa without serious consequences and move fairly well. It appears that the withdrawal effect was stopped by the increase of the agonist. That means that the agonist compensates the effect of dopa but in another way. That does not resolve addiction but shifts the problem on another level. But that seems to be the only way traditional medicine can handle the subject. Let's see if it works. In any case we should be able to learn how withdrawal from a high dose of the agonist (Cabergoline/Dostinex) works."

He wrote two days later, in answer to my greedy questions about his response to the dopamine agonist, "With the agonist I still have some "twitching" – contraction – around the eyes similar to the one I had in Santa Cruz, but not in the neck. Am I lightheaded, you ask? Yes, but not especially. I have no nausea and no insomnia – but I take sleeping pills and I wake up quite early. I shall try to sleep without pills this or next week. I move almost normally. My health is still fine."

In the month that followed, Viktor and I exchanged many emails about his experience in the program. He wondered why he needed the agonist medication if he was in fact getting better. He wrote, "I am convinced that if I no longer have Parkinson's, I should not need any medication." He did not accept the possibility that the drugs themselves can inflict permanent damage and cause drug-induced parkinsonism, after which increasing amounts of medication are needed to treat the damage.

He also felt strongly about our program's lack of sufficient warning about the dangers of medication: "You knew the risks of these drugs from experience, and, as I see it, you waited too long to post the warnings." He disliked the tone of our newly posted (at that time) warning that doctors might give bad advice; he thought that our warning wrongly implied that doctors were acting maliciously. He felt that our program was to blame because we should have known better than to have worked with medicated patients.

I replied, "I think you overestimate how much time I have had since I realized what was going on. The first deaths from overmedication just occurred in this last year. It

was only two months ago – after we posted the warning – that research was published that proved addictability of drugs can change as circumstances change. Prior to that, even though we warned patients that they might suddenly become addicted to the drugs if they recovered, they laughed at the idea. We posted the warning that "your doctor's advice may be harmful" on the website just a few months ago – when the third patient was "stabilized" by a doctor who doubled her drugs over a period of three days and sent her home just before her violent spasms began.

"In the year 2000 we were still adhering to the conventional thinking, which was that a medicated patient should work with his doctor, and that it would not be hard for a person to quit taking medication if he started feeling better. Now, less than two years later, we have reluctantly rejected all the conventional wisdom, reversed our position and posted warnings on the Internet.

"When I first met you, in January 2001, I was not yet aware of the real power and dangers of these drugs. I suspected by then that they were bad, but I didn't realize

1) Just how dangerous and addictive they were.
2) A recovered person was more at risk than a PDer.
3) MDs' training was based on incorrect information about the drugs….

"…But you are right. We have an obligation to 'first, do no harm.'"

We took Viktor's criticisms to heart. Two weeks later, the PD team of Santa Cruz decided by consensus that we would no longer work with medicated patients. We changed the warning on the website to state that medicated patients should not follow the protocols that we offered.

One of Viktor's last emails to me said, "Set up a pilot study with newly diagnosed and unmedicated patients. Show the effectiveness of the program in a controlled way. Dream it. Do it. And you will succeed. (I found the last eight words in a book. Nice.)

Take care,

Viktor."

Summary

In summary of this chapter, Buzz and Viktor were both in their fifties, in good health other than their Parkinson's disease, and both had been diagnosed about four years earlier. Both went through approximately three and a half months of drug reductions from 400 mg and 500 mg, respectively.[1] They both suffered from periods of crushing immobility.

In addition to the immobility, Viktor suffered from adverse effects of his pills after nearly every dose after he became addicted: terrible tensions of impending doom in his chest, freezings, Ons, Offs, Switching, Build Ups, Roller Coasters, and, very possibly, long-term addiction-related damage such that he will always need some amount of antiparkinson's medication. Given what we know about drug addiction, we have to suspect that Viktor's suffering during his increasingly excruciating drug decreases was exacerbated due to his week of pre-reduction euphoria and gross overmedication. Buzz had never experienced euphoria or symptoms of overmedication and, because he stopped his drugs abruptly, no dopamine excess-related traumas.

Viktor is still dependent on a dopamine-enhancing drug, albeit a somewhat more moderate one (in terms of side effects) than he was taking originally, and at a much lower dose. He may always need medication now; his brain was certainly affected by the addiction as well as by the genuine traumas that he underwent. Whether or not this brain damage is short term or long term remains to be seen.

The question arises: had Viktor decreased his drugs before he became over sensitized and addicted, might he have avoided the tensions and cramps, the mental fog, the euphorias, and the violent On and Off swings, with their Roller Coasters, freezings, and Crashings? Might his drug decreases instead have produced a simple decrease in drug effectiveness, an unveiling of his underlying PD symptoms? In such a case, his symptoms of drug decrease might have been an increase in slowness, rigidity, poor balance, or tremor – the four characteristic symptoms of Parkinson's disease. These symptoms, though frustrating, humiliating, and often very painful, are nevertheless a far cry from the excruciating physical agonies, panic attacks and night terrors of drug withdrawal. And do not forget, despite Viktor surviving these assaults, at the time of this writing he is still needing a powerful antiparkinson's medication, Cabergoline.

On the other hand, Buzz, with his drastic approach to medication reduction, might easily have gone into limbic shock and died from heart or diaphragm spasms or arrhythmias. He certainly ran the very real risk of being hospitalized against his wishes and having high amounts of medication forced on him. (It is likely, based on our past experiences, that the doctor in charge of such a case would have increased his dosage

---

[1] Actually, although Buzz was taking 600 mg of levodopa a day, he was *absorbing* probably around 400 mg/day. Here's why: for some unknown reason, his doctor had prescribed the Sinemet with the lower percent of carbidopa. Usually, the lower carbidopa amount also causes a decrease in available levodopa. This means that a person who takes a 10/100 pill will have less available levodopa than a person who takes the more common 25/100 pill. (The first of the two numbers is the amount of carbidopa.) It is impossible to guess how much less levodopa was available to Buzz than to Viktor – no two people process the medication in exactly the same way.

daily until he was moving well. In Buzz's traumatized, limbic-depleted condition, this could have taken 10 days, by which point he might have been up to 1000 mg/day.

Questions

While Viktor's route was more painful and possibly more damaging in the long run, was it possibly the safer and saner route to traverse? What lies ahead for Viktor as he continues to work on reducing the agonist? After all, we have seen with Becky, Rudyard, and Sammy that the agonists, while less traumatic than L-dopa and Eldepryl during the short-term (ten weeks) phase of reduction, may possibly do more long-lasting damage in terms of creating long-term depression that will never again be satisfied by anything less than a return to the drug. On the other hand, if Viktor had started reducing his drugs slowly, in advance of recovery, who can say what might have transpired? It would be pure conjecture to offer an opinion.

It is pointless to ponder "what ifs," or to extrapolate from these two cases the ideal style of drug decrease for any other individual. In this particular pair of cases, the rapid hare unexpectedly faired better than the steady tortoise – defying tradition, but not necessarily proving that one method is safer than another. However, though I do not advocate either one of their extremely intense methods of drug decrease, their cases may suggest promising directions for further research, and so I have included them in this book.

Although their cases proceeded differently and had different conclusions, Buzz and Viktor had one factor in common: they were both looking for something more than a return to their "old, healthy" self; they were both looking for the deeper reason behind their illness. They were looking for answers, not just a fix. Buzz and Victor had never met each other, but they had this in common: their greatest hope was that others might learn from their experiences.

"No rule is so general, which admits not some exception."

Anatomy of Melancholy, Robert Burton (1577-1640)

24. VARIATIONS IN ADDICTABILITY

SUSCEPTIBILITY: A SLIDING SCALE

Although some medicated patients flipped out after manifesting signs of recovery, many patients who were still using drugs after starting to recover never exploded into frenzied motion or crazed addiction. Why?

For example, Olli, from chapter two, showed considerable improvement in his Parkinson's symptoms prior to starting any drug reduction. Yet he easily stopped taking Eldepryl during this time. Subsequently, after his frightening fall on the first day that he stopped his 500 mg/day levodopa (Sinemet) cold turkey, he never tried to make any further decreases in his medication. Within a year he started having problems related to adverse effects of the drug, which he, predictably enough, treated by making increases in his Sinemet, but these problems, for the first year, were only some moderate increases in dyskinesia, grimacing, and ticcing. He never went hyperkinetic in the way that Viktor and a few others had done.

We had to wonder why Olli and others like him had such a gradual increase in sensitivity to the medication, when Euclid, Brad, Angus, and Viktor had apparently flipped out overnight.

Secondary injuries

In Olli's case, although his foot responded well to treatment – his foot-dragging walk, feeble voice, and absence of facial expression (all of which symptoms are located on the Stomach channel) recovered completely and his tremor stopped – he also had a significant injury in his shoulder. This shoulder problem had chronologically preceded his foot injury. At the time that the foot-related PD symptoms began to ebb, the shoulder problem had not yet been addressed via our treatment protocol.

It appears in retrospect as if the unhealed shoulder problem helped his body retain a partial condition of injury, which in turn prevented the medication from becoming as addictive as it was in those patients who had completely recovered from all major injuries.[1]

[1] I am not suggesting that every single lingering, unhealed injury must be detected and treated in order for a person to recover – absolutely not! Very often, when the pre-existing fear that prevented the foot injury from healing is assuaged and the foot begins to heal, other injuries that occurred *subsequent* to the initiation of that particular fear/foot injury (either/or) combination may spontaneously surface and self-initiate the healing process. Possibly the brain may store certain associated "Fear: Do Not Heal" signals and injuries all together in the same brain compartment. Once the compartment is unlocked, the body may have healing access to any and all unhealed injuries stored in that particular collection. However, in Olli's case, his neonatal shoulder surgery long preceded the foot/leg injury that he received at age six. They may both have failed to heal at the time that they occurred, but for significantly different reasons. Possibly, the non-healed shoulder injury was based on different fears than the foot and leg injuries that we had treated using FSR.

His shoulder problem appeared to have derived from a huge birth mark/mole that had been removed in his infancy. The doctors had not used anesthetic during the surgery, a typical pediatric practice of the day. He had never used this arm much, even as a child, and the shoulder still bore a deep, five inch by four inch scar.

When Olli decided, in spite of glaring improvements in his Parkinson's symptoms, to not make any more decreases in his medication, I was reluctant to rid him of any more blockages: without telling him my reasons for doing so (which legally might have constituted an opinion about the medication), I redirected my efforts away from his shoulder and focused primarily on maintaining the restored Qi in his feet.

Over the next year he started increasing his medication to address new problems that were arising: dyskinesia, grimacing, and ticcing (problems of overmedication). After he learned from the standard physicians' drug reference books that the meds were probably contributing to, or even causing, his new problematic symptoms, he planned to decrease his drugs. He tried to reduce for over a year and failed to ever maintain a pill-per-day decrease for more than one day.

After almost a year with no success in reducing his drugs, symptoms of drug-induced parkinsonism began to appear, joining his symptoms of overmedication. One of his most noticeable symptoms was partially losing his voice each day. His voice was fine in the mornings, at its worst by the end of the day – a Build Up, obviously, but at that time we hadn't figured out that these drugs had a cumulative effect. He felt that the abrupt loss of voice each evening forty-five minutes after taking his 7:00 p.m. pill was due to increasing PD. In addition to the evening voice loss, his balance became worse, and his walk was more uneven. His facial grimacing worsened (this symptom was also the most severe 45 minutes after taking the pills and worsened in intensity over the course of the day), and the ticcing became non-stop.

By the end of a year of these worsening "symptoms," he succumbed to his MD's suggestion that Mirapex be added to the Sinemet to try to control the increasing dyskinesia, grimacing, and other drug-related problems. Olli knew that these were side effects of medication, and not necessarily an increase in Parkinson's, and he was angry with himself for not having reduced earlier. He began having On-Offs. He dropped out of the program after a few weeks on the Mirapex. He assured me that he was utterly unable, at this point, to reduce his medication, and he also realized that he had probably done himself enough damage with drugs that he would now need ever-increasing medication in order to treat this damage. Sadly, if you will recall from chapter two, Olli was a professional counselor who worked with drug-abusers, and had assured me from the start that he, of all people, would have no problem reducing his medication when the time came.

And yet, my point here is that, although Olli's condition declined over the course of the year following his improvements in his PD symptoms, (and the decline was probably due to taking drugs that he no longer needed) *despite the excess drug usage,* he never flipped out as Viktor had done. Olli's mental clarity remained high,[1] though he became more subject to moods. Most significantly, he only *gradually* declined into drug-induced parkinsonism and worsening adverse effects of the medication.

[1] Mental clarity was still OK the last time I saw him. I have not seen Olli since he dropped out of the program.

Yves

Yves was taking 1000 mg/day levodopa (five 50/200 CR Sinemet) when he started in our program. He was 73 years old and had been diagnosed five years earlier. He had mild dyskinesia from the medication. He was treated, with good results, by one of the student interns; he began to have improved sensation and gait in his affected foot. He correspondingly reduced his medication slightly, by 100 mg/day, down to 900 mg from his starting level of 1000 mg.

As he continued to notice symptoms of improvement, such as improved function in his ankle, improved sleep and a decrease in rigidity, he continued to make gradual decreases, decreasing by approximately 100 mg every month. He was one of our earlier patients, back before we had any theory or experience to fall back on. He was reducing slowly, not because we'd seen any good experiences with slow reduction, but because, though he wanted to save money on the medication and reduce his dyskinesia, he was, by nature, cautious about making changes.

Over the next nine months, he reduced his medication until he was down to 300 mg/day. By then he was moving well and had very little dyskinesia. Yves felt that he was taking "next to nothing." But Sharon, his student intern, could still feel the characteristic "Sinemet snakes" crawling under Yves' skin when doing Tui Na. Although Yves had an old neck injury that troubled him now and then, Sharon was doing Tui Na only on his legs. At this very early stage in our research we were still looking only at the foot/leg Qi situation in our patients.

Yves professed to be very pleased with the program and was grateful to be taking so much less medication. However, he told Sharon that he didn't think Parkinson's was curable and didn't intend to decrease his medication any further. Sharon asked why he had entered the program, if he didn't hope to recover. His reply was that the medication was so expensive, and he wanted some treatment that would help enough so that he could reduce his medication. He surprised us by announcing that he had never intended to "recover," and he had never planned on completely getting off the medication.

At this time, we had no idea that the medication was addictive, could cause parkinsonism, or make any lasting brain changes. We couldn't see any reason why he should not continue with the program, even though he no longer had any apparent Parkinson's symptoms with the "minimum" dose of 300 mg/day. He continued to come in for several months, during which time he expressed satisfaction with the program. Sharon noticed that the snakes under his skin were getting stronger, not weaker, and asked him if he was going to ever make a further reduction in his medication. He replied that he was happy with 300 mg/day – it was an affordable level, and it felt right to him.

Two months later, the snakes were growing more insistent, and Sharon told Yves that she felt he might be overmedicated. In my role as intern supervisor, I had to interfere at this point. I spoke to Yves, while Sharon was standing there, and apologized for her remark, adding that, as an acupuncturist, she could not make any comment that might be construed as offering advice, or even a suggestion, about Yves' medication. He must work with his doctor if he wanted any advice about his medication.

Two months after this, still taking 300 mg/day levodopa and starting to have marked dyskinesia again, Yves dropped out of the program. He thanked Sharon for her time and said he was well pleased with the results. He said he'd attained his primary goal: he'd gotten his pills down to an affordable level.

Because his previously slight dyskinesia had reappeared, and with more force than before even though his drug level was supposedly "next to nothing," and because the snakes under the skin were getting stronger, Sharon suspected that he had actually started increasing his medication and lying to her about it. But possibly he was having more dyskinesia because he no longer needed his drugs. In either case, we would have no way of knowing.

But our question here is this: how was it possible that Yves could be increasing his medication or even staying at the same level without becoming violently addicted, when others who had taken the drugs while recovering had erupted with grotesque dyskinesias, new ticcings, or mental aberrations? Clearly, after PD symptoms began to fade not everyone responded to their medication in the same way. After reviewing the cases, we now suspect that multiple injuries may be a factor.

Multiple injuries

A growing hunch on our part is that people who have multiple injuries may have slightly more time to get off their medication than people who have only the one characteristic foot injury. As we have already suggested, very probably the sympathetic/parasympathetic switch is not an all-or-nothing toggle, but a sliding scale. Certainly, when the foot and leg patterns are restored, patients seem much more susceptible to addiction. *However, not all patients become addictable at the same rate.* In Asian medicine, this difference between humans is perfectly acceptable. But for some reason, maybe due to my training in western sciences, I wanted to know the mechanism that might account for these differences. While the old-fashioned (1950's) "one neurotransmitter = one neural response" theory still adhered to by some greybearded MDs didn't offer any answers, it seemed as if Asian channel theory might provide some clues.

DU CHANNEL THEORY

The Du channel, which runs through the midbrain, is the channel that most directly affects dopamine production in the brain. I must introduce a little channel theory at this point so that by the end of this chapter you will understand the special relationship between the Du channel and the Stomach and Large Intestine channels. The Stomach and Large Intestine channels, due to their intersection with the penultimate point on the Du channel (on the philtrum of the upper lip), an intersection not found among the other of the twelve Primary Channels, may very likely be the strongest drivers of the upper part (head portion) of the Du channel – the portion of the Du channel that regulates the midbrain.[1]

The significance of this intersection may be that it gives the Stomach and Large Intestine channels almost an adjunct role (adjunct to the Du) in susceptibility to addiction. Conversely, other channels, while able to affect the Du, may play a lesser role. Damage to channels other than the Stomach channel may influence addictability, but not to the extent to which a rebellious Stomach channel can. This may help to explain the variations we saw in the rate at which patients became overwhelmed by their medication.

If you will forgive the inclusion of eight pages of Asian medical theory at this point, we can consider a channel theory explanation that might account for the variations in susceptibility to addiction that we observed. Let's go.

Du channel location

The Du, or Governor, channel is one of the extraordinary channels, or Master channels. The Du channel runs from the base of the spine up through the medulla oblongata. The medulla oblongata is the group of tissues that make the connection between the nerves in the spinal column and the neurons in the midbrain. The Du channel divides at the medulla, with one branch running over the top of the head and the other running through the midbrain, below and somewhat parallel to the upper branch. These two branches come together again, meeting and merging on the forehead at the point between the eyebrows.

[1] All channels help to push, pull, or otherwise drive all the others. The anciently known schematics of the body's subtle electrical patterns show that all the currents are interconnected. They cycle in endless, interwoven loops through the body. It is not a closed system: excess static can be discharged at the skin, especially the fingers and toes. The system is also plugged into and constantly recharged by an external source of energy. This life force invigoration is purely energetic, and is different from the energy derived from food. After all, man lives, not by bread alone, but by the external Life Force, or, if you want to get scriptural, "The word (sacred vibratory energy) that proceedeth from the mouth of God." The medulla is the place at which outside energy flows into the body. For a fun aside, note that this is the location at which the bodies in the metaphorical movie *The Matrix* were tapped in order to integrate the character's "real" self with a worldly, world-like delusion.

Fig. 24.1 The upper (over the head) portion and inner-head branch of the Du channel

This meeting point is named Yin Tang, or "Meeting Hall of the Yin." The name Meeting Hall refers to the fact that this Du channel point is also the end of three Yang primary channels and the beginning of three others.[1]

[1] The three Yang *arm* channels terminate at Yin Tang, and the Three Yang *leg* channels commence from this spot. This point is called *Yin* Tang because it is the meeting hall of the Yang channels on the *Yin* (front) side of the body, as opposed to the meeting of the Yang channels at Du 14, which is on the Yang (back) side of the body.

The nomenclature also has a spiritual meaning. Although the Divine energy enters in at the back of the neck (medulla), the Divine energy is most often perceived, not at the back of the neck, but as the light of the third eye, the spot on the forehead. (To pinpoint the location of the entry point, see any good book on embryology and note the hole at the back of the neck out from which all growth in spine-bearing organisms proceeds, and which only begins to fill in after the organism has sufficiently formed.) The light in the forehead is only a mirrored image of the Divine light that enters at the back. It is like the moon – having no light of its own but mirroring the light of the sun. The moon is Yin, and the sun is Yang.

As for looking directly at this source energy, the energy that enters through the back of the neck, most men could not withstand beholding the blinding "light of a million suns," as Krishna describes it to Arjuna in the *Bhagavad Gita*, which pours into them directly from the Source and transforms from Love into light and matter. Instead, most seekers of Truth must satisfy their spiritual questing by focusing on their own small part of divinity, the mirrored reflection in their own forehead of their own small spark of Divine soul. This moon-like reflection, the "Moon of my delight that knows no wane," of Omar Khayamm's *Rubaiyat*, is Yin (further from the sun) in relation to the brilliance that can be perceived by great souls at the Yang (closer to the sun) meeting point of the Yang channels just a few inches above Du 14 on the back of the neck.

The pictogram for Yin in the point name Yin Tang is a character that means Self, Self Identification, or Stamp of Identity. This word "Yin" is also a homonym for that word "Yin" which means opposite of Yang. The word play here is wonderful. Yin, while meaning further from the sun (sun also meaning source, or God) can also refer to the individualized self, as opposed to the greater Self, or Yang identity, that recognizes its oneness with all things. The richness of having all these meanings converging in the point name allows the point name to be simply understood as "forehead, where the person's identity meets with the outside world," more deeply as "the meeting point of the channels on the Yin side of the body," or the most profound, "the meeting of the energies that define this person's identity, that mark him as an individual, delusively separate from other beings." (Footnote continued on next page.)

Yin Meeting Hall of the yang channels

For an example of these Yang channels seamlessly ending and starting at Yin Tang, consider the Large Intestine channel discussed in chapter 5. The Large Intestine channel, one of the six yang channels that commingle at the center of the forehead, travels up the arm and ends its journey at Yin Tang.[1] (See chapter five, figure 5.1.)

(Footnote continued from previous page.) As for the character (pictogram) that is now assigned to this point, one must keep in mind that the point was first named in the oral tradition, and a picture assigned to the point name in later centuries.

For acupuncture channel purposes, the following meaning is most useful: Yin Tang, or Yin Meeting Place, is the meeting of all the Yang (meaning more spiritual and transformational, less material, less dense) channels, on the *Yin* (front) side of the body. This understanding of the point name was effectively banned by the 20th century Chinese government, which decreed that all spiritual meanings should be removed from the literature of Chinese medicine. However, while these meanings have been officially forbidden, they remain obvious to anyone who actually probes these channels to their depths.

[1] The drawing of the Large Intestine channel in chapter 5 (fig. 5.1) only shows the channel up to acupoint LI-20, at the side of the nose. This drawing is based on the modern, government-approved, all-spiritual-references-removed version of the channels. In this sanitized version this channel and the other Yang arm channels do not extend all the way up to Yin Tang, but stop abruptly and disappear into thin air after the last named/numbered acupoint on each channel, an inch or so away from Yin Tang. Each of the arm Yang channels supposedly is followed by its paired Yang leg channel. However, in the modern, official version of channel mapping, these subsequent paired channels begin abruptly, out of nowhere, an inch or so away from Yin Tang. The channels that flow towards the center of the forehead stop a few inches away from Yin Tang and the channels flowing away from this major energetic point begin a few inches away from Yin Tang, thus conveniently (for the sake of politics) bypassing Yin Tang. Amusingly, the Chinese government-approved maps of the channel schematics show that *no channels meet* in the point named "Yin Meeting Hall."

This practice of modifying history willy-nilly for political reasons and then insisting that this new policy or condition has been unchanged since time immemorial is called revisionism, and is part and parcel of the Chinese political system.

For an even more glaring example of the fear that the current Chinese government has about Yin Tang, consider this: although the points were understood to be major vortices along the channels, the points historically were not named or described by their channel location. Their names referred more to the function of the energy at the particular acupoint, and point names did not include channel references. When, in the wave of modernization, the collection of known acupoints were assigned designation by a combination of channel name and number denoting the sequence of the points along the channel, the point Yin Tang was not included in the Du channel group, but listed at the back of the book as an "extra point."

Even though Yin Tang is a major point along the Du channel, and the most famous Du channel point from a spiritual perspective, it was not assigned a number or channel, but floats in a nether zone in between officially numbered Du-24 at the top of the forehead and Du-25 on the tip of the nose.

When I asked several of my China-born and -trained colleagues the reason that Yin Tang is not a numbered point on the Du channel, they all ingenuously suggested that Yin Tang must have been discovered after the numbering of points was done in the 1970's. When I point out that Yin Tang is referred to in texts going back hundreds of years, they say that I must be wrong about Yin Tang being on a channel, any channel. Their proof? Their government-approved medical books list Yin Tang as an "extra point," not related to any channel. When I suggest that the government might not have maintained the medical information to a high degree of historical or biological standard, they are uniformly appalled. I paraphrase their response, "The government cannot change history! Chinese history is the truth. History never changes. Chinese medicine has not changed in two thousand years. We learn the same medicine as our ancestors, only we add to it with new research. Yin Tang must be a modern point. It may function as if it is on the Du channel, and even be located on the path of the Du, but it is not numbered as a Du point in our books, so it obviously cannot be on the channel."

Actually, although the three that flow into Yin Tang are considered to end at this point, in fact, the channels themselves continue on, merely bearing different names, so that we can say that three channels (coming from up the arm) flow into Yin Tang and three channels (moving down the leg) flow out from it.[1] The Large Intestine channel changes name and direction when, after having traveled up the arm and arriving at Yin Tang, it flows out again from Yin Tang, heading down the face towards the foot. This now downward-moving (that is to say, heading for the foot, although previously upward moving and coming from the hand) branch of the unending loops of channel Qi is called the Stomach channel.

The Du channel, continued

The Du channel, the one that flowed over and through the head, continues down the center of the face after its intersection with the six Yang channels at Yin Tang. It goes down the ridge of the nose, the philtrum (groove) on the upper lip, and then dives inside the body via the upper palate, whence it travels through the gastrointestinal tract and reemerges at the anus, before repeating its upward journey through the spine. It forms a complete loop. (By the way, the Ren channel, an Extraordinary channel, like the Du, rather than a Primary channel, does the same sort of loop up the *front* of the body and down through the mouth and digestive tract.)

Because of the power and location of the Du channel, particularly as it courses over and through the head, the Du channel plays the largest role of any of the channels in creating and regulating brain function. The electrical and magnetic forces generated by this particular loop of current are most likely the triggers that stimulate the various genetic expressions of the cells of the spine and brain.[2]

---

[1] Some rivers change names when they cross a border into a different country. The river doesn't necessarily have any awareness that man has renamed it; the river simply flows from place to place regardless of political boundaries or name. In the same manner the body's one primary magnetic field and its corresponding electrical current, or "channel," and that channel's side streams and branchings all flow continuously. For the sake of study and communication, we give each major division (primary channel) its own name as the Qi flows from one area to another.

[2] Very likely, most DNA instructions are generated via current/channel flow. The currents influence the closings and openings of the paired DNA molecules (genetic material), expressions of which vary from one body part to another. From the main currents of energy that flow through the body, smaller currents branch off, and from these, still smaller ones branch again. Subtle magnetic field variations, partners of the smallest iterations of tiny channel currents that bathe every cell of the body, influence the DNA in the various body-part cells over which they flow. These magnetic fields affect the DNA within the cells, and thereby determine genetic expression.

(There is a seeming chicken and the egg interwovenness between the cells, which provide a substrate for the electrical currents, and the currents, which direct the development and function of the cells. However, in pre-communist Asian medicine, there is a distinct causative factor to the "which came first, the chicken or egg" question of this interwoven cycle. The Qi, the energy field, precedes the structure. This would possibly translate, in English, to "the soul precedes and guides the development of the body; the body provides a substrate in which the soul can physically manifest and grow." A deeper understanding might be that Love creates an individualized soul which directs the formation of a physical manifestation, and then, in that manifested form, has the opportunity to perform actions that grow yet more Love. In Chinese the ancient expression is "The Qi is the leader of the blood; the blood is the mother of the Qi.")

While various bits of biochemistry may induce changes in the DNA expression process, it is likely that they do so via the electrical field alterations that their presence sets into motion. To paraphrase the Chinese adage above, the electrical field is the leader, the chemistry is the substrate on which the "leading" takes place. And this leading, in turn, is directed to the building, maintenance, or decay of more substrate!

The following diagrams portray the frontal points of the Du channel, the intersection of the Stomach channel and the Du, and the intersection of the Large Intestine channel with the Du. Although three arm and three leg channels intersect the Du at Yin Tang, only the Large Intestine and Stomach channel intersect at both Yin Tang and *again at Du-26*. The significance of this will be explained later. For clarity of demonstrating the Large Intestine's left-right (and vice versa) cross-over, only the right-hand Large Intestine channel is shown. Both left and right Stomach channels are shown – these channels do *not* cross over.

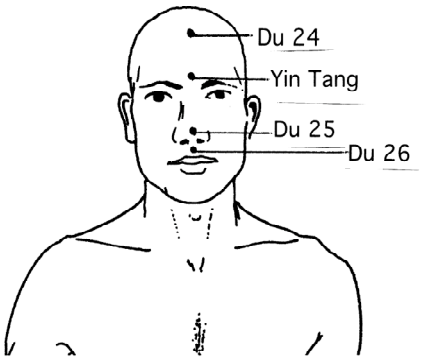


Fig 24.2 Frontal Points of the Du channel

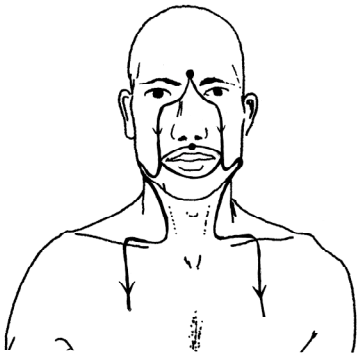


Fig 24.3 Face and neck portion of the Stomach channel
Note: a branch in both the Stomach and Large Intestine channels skirts the mouth.

Fig 24.4 Arm and Face portion of the Large Intestine channel

The Du channel is associated with the parasympathetic system. Meditation is most closely connected with the Du channel. Whether focusing on the third eye (Yin Tang) or the Thousand Petaled Lotus (Du 20), observing spinal energy or the chakras, or controlling the breath, the net result is a diversion of energy out of the Primary channels (arms, legs, and torso) and into the Du channel. When modern doctors tell their patients to meditate for a few minutes every day to decrease their blood pressure and calm their heart, they are actually telling them to increase the energy flow through their Du system. Dopamine, the parasympathetic system, and the Du channel are associated with calmness, even serene fearlessness.

The Kidney channel – opposer of the Du

The adrenal system (the sympathetic nervous system), on the other hand, is regulated by the Kidney channel, one of the "Primary" channels (the Primaries are derived from and subordinate to the "Extraordinary," or "Master" channels). The Kidney channel is the channel most closely related to fear and sexual energy – a juxtaposition that provides much food for thought, but which is, of course, beyond the scope of this book. I will just note here that dopamine is the neurotransmitter of fearlessness and adrenaline is the neurotransmitter of fear.[1]

---

[1] There are many obvious relationships between adrenaline and dopamine. One well-known relationship is this: they both seem to influence our perception of time. During a life-threatening powerful emergency such as being ejected at high speed from a moving car or bicycle, the moments before impact are filled with a vast number of thoughts, far more thoughts than can ordinarily be processed. Time seems to extend forever, and it can seem as if minutes, rather than seconds, transpire before the point of impact. It has been assumed that this is a function of adrenaline. The opposite may also be true – it may be a function of a concomitant decrease in dopamine.

Recently, it has been found that the same extension in the perception of time occurs during drug deprivation. A recent study compared the ability to estimate the passage of time among smokers, non-smokers, and smokers denied cigarettes. After a 45 second interval had passed, both non-smokers and smokers guessed fairly accurately the number of seconds that had passed. When smokers were deprived of their cigarettes for a day, and tested the following morning, they guessed that the 45-second interval was much longer – at least 50% longer, and in some cases up to three minutes! (Laura Cousin Klein, *Psychopharmacology Bulletin*, spring quarter, reported by Santa Cruz Sentinel, May 14, 2003, p. C-5.)

We already know that the neurotransmitter most altered by nicotine is dopamine. In the case of cigarette deprivation, dopamine levels decrease. It may be that the dropping away of dopamine gatekeepers from the incoming sensory nerves allows more sensory information to pass into the brain and creates a sense that more time is passing.

Such an increased sensory input can be helpful in an emergency, when we need as much information as possible in order to make fast decisions. However, this response is historically associated with adrenaline – dopamine was never considered to play a part in emergency neurotransmission. But it may be that the actual change in time perception is not from adrenaline per se, but from a simultaneous decrease in dopamine. If so, this would add weight to the validity of the idea that adrenaline and dopamine are two ends of a seesaw; when one goes up, the other goes down.

A corollary might be that a saint, ever immersed in his dopamine-drenched calm, perceives the timeless aspect of the world. Time can cease to exist for such a one if he so chooses. He may focus, breathless, for hours on a single thought with no impinging awareness of the passage of any time at all. Not only gravity, as discussed in an earlier footnote, but time itself is, to the saint, a relativistic plaything.

Another aspect of the opposing natures of adrenaline and dopamine is the perception of pain. Under the influence of high levels of fear and adrenaline, a person temporarily cannot perceive pain – the pain section of the brain is inactivated. (Footnote continued on next page.)

Du channel relationship with addiction

What does this have to do with addiction? I have already proposed that addiction occurs when the parasympathetic system is dominant over the sympathetic system. I propose further that it is when the Du channel is running optimally that the dopamine-related systems, including the propensity to addiction, are running at full-bore.

On the other hand, when, due to fear or sexual stimulation, the energy is directed towards the animal centers in the lumbar regions (the lumbar chakra, the area around acupoint UB-23, and/or the energetic center of the Kidney channel), there is an increase in adrenal stimulation and a simultaneous decrease in energy in the Du channel and the dopamine system. Fear "scatters" the Qi, as we learn in first semester Asian medical theory.[1] A saint, during his moments of motionless, breathless mystic union with the Divine, has his Qi concentrated in one place – most often, curiously enough, Yin Tang. When a person must interact with the world, moving about and breathing, he must have at least some small, minimal level of fear (he must awaken the adrenal glands), and "scatter the Qi" throughout his body.[2]

(Footnote continued from previous page.) When the body is saturated with dopamine, there may also be a cessation of pain, but it comes from the opposite source: fearless consciousness of the relativistic nature of pain; an accurate assessment of the pain can be calmly made; pain is seen as a mere signal of body inharmony.

When fear is uppermost, pain is blocked via adrenaline. When joy and dopamine are ascendant, pain is acknowledged and then either treated appropriately with effective treatment (a bandage, a stitch, a kiss on the bruised place, or some other appropriate treatment that acknowledges the injury and activates healing in the body) or embraced – even with gratitude or at least spiritual understanding – and observed from a higher realm of Wisdom (transcended).

Most PDers have always imagined that by denying pain they are proving their superiority to it. In fact, they are merely up to their eyebrows in adrenaline and fear. Transcendence (as opposed to suppression) of pain and fear is a facet of spiritual growth. Denial of real and present pain and fear is the key to the Parkinson's personality.

[1] I cannot find this in my translation of the ancient Nei Jing, but it is taught as one of the basic precepts. Part of the difficulty of mixing Asian oral tradition and western footnote tradition is the lack of published references and the lack of uniformity among the translations.

[2] The Qi must be spread throughout the body if one is to engage in breathing and other physiological functions. Inhalation is impelled by the adrenal glands, not merely the lungs, and occurs due to the "fear" of suffocation.

If a person is dominated by fear to an unhealthy level, his Qi will be so utterly scattered that he will even lose his focus, both mentally and physiologically. We would say he acts "like a chicken with his head cut off," and various body functions, including such famous ones as bladder and rectal sphincter control, may become embarrassingly "confused" or unreliable. This is a pathological, excessive scattering of the Qi, as opposed to the healthy level of "Fear scattering the Qi (away from the divine centers at Yin Tang)" which is necessary to engage the adrenals and maintain normal physiological function.

It is far, far removed from the scope of this book, but it may be interesting to note that some practitioners of the self-disciplining arts of Tai Qi, Qi Gong, and some of the martial arts focus their energy on their lower (belly) centers, thus attaining control of their fear and their animal energy.

In higher levels of Tai Qi, Qi Gong, and martial arts practice, and in transcendental practices such as Raja Yoga and original Christianity, energy is focused at Yin Tang or other head/upper spine points. The point that we call Yin Tang in Chinese has been known for millennia in India as the "third eye," or the "single eye, " and even "the star of the east." Riveting the interior focus on these upper spine points, and Yin Tang in particular, rather than on the two (dualistic, delusion-oriented) externally facing eyes, can help one along the path of attaining, not mere physical power and control over the inner animal, but control over the ego and the Self, or soul. The goal in lower Du control is vitality and long life. The goal in upper Du control is timeless peace, intuitive wisdom of universal truths, and eternal communion with God.

Stomach channel regulates the Du channel, and the reverse

The significance of all this in relation to Parkinson's disease is that the Stomach channel, the main channel affected in Parkinson's, plays a major role in setting both the vigor and the shape of the Du channel. It appears that when improvements are made in a long-damaged Stomach channel, the health of the Du channel is simultaneously improved. The PDer, following treatment, may return to a healthier condition in which he is somewhat less dependent on adrenaline and therefore more dopamine oriented – hence, more addictable.

However, if such a person still has some remaining injuries, these remaining glitches, *whatever* channel they may be on, may help to moderate the recovery of the Du channel, preventing full addictability and creating, instead, the conditions that allow for our multi-injury patients to have *mild, creeping* increases in their drug addiction and drug-induced parkinsonism symptoms instead of full-blown, all-or-nothing, bug-eyed addiction and violent adverse effects. Let's look at how this might play out.

First, let's see how the Stomach channel is influenced by the ups and downs in Du channel Qi. In western biological studies, one of the most common tools used in explaining the parasympathetic system to school children is the image of a cow chewing her cud: when Bossy is relaxed, she can make best use of her stomach/gut function. But in times of stress, according to western medical theory, the stomach shuts down to some extent. In times of high stress, the body may even evacuate the stomach and gut by whatever means possible, so as to be better prepared to Fight or Flee. The stomach and the parasympathetic have long been linked in western theory.

This meshes nicely with the Asian theory; the Stomach and Large Intestine channels are doubly (relative to other channels) connected with the Du (parasympathetic-governing) channel. These two are the only channels that meet twice with the Du channel on the face instead of once: not only do they intersect the Du at Yin Tang, but they meet *again* when the Stomach and Large Intestine channels skirt the lips, circling the upper and lower lips on their trek across the face. At this mouth location, they have their second intersection with the Du channel.

Left-Right coordination

The skirting of the lips, as the Large Intestine channel crosses over from one side of the body to the other, is what drives left-right coordination in the body. The *left* Large Intestine channel crosses the philtrum and surges up the *right* side of the face, into Yin Tang, and then becomes the *right* Stomach channel, and vice versa. This rhythmic pulsing of current around the mouth helps do two things. It drives left-right coordination, in which a swing of the left arm activates a subsequent swing in the right leg. In Parkinson's, the decrease in Qi flow over the face plays the major part in the loss of arm swing; this is proven when Qi flow is restored through this area. A more critical role of this second, lower-face junction of the Du channel with the Large Intestine and Stomach channels is the alternating Large Intestine and the Stomach electrical surge that directs the Du channel more vigorously towards the mouth, down from the forehead.

Consciousness

The throbbing of the Large Intestine and Stomach channels as they cross over the upper lip exerts a pull on the Du channel, drawing the Du channel Qi down to the mouth.

The left Large Intestine channel crosses left to right over the midline of the body at the upper lip, and then a moment later the right side Large Intestine channel crosses right to left, and then once again the left side channel crosses left to right, repeating the pattern over and over, driving the right-left balancing of the brain and body. The Stomach channel branch that also skirts the lips at this point is also stimulated by this left-right rhythm. Without this throbbing, electrically driving influence from the Large Intestine and Stomach channels, the Du channel Qi will not descend down the face to Du 26 at full force. If the Large Intestine and the Stomach channels are not flowing or only barely flowing, most of the Du channel can become backed up, slowed, or diverted at Yin Tang. Most often, if the Stomach or Large Intestine channel is not flowing, the Du channel will flow from the back of the neck up through the midbrain to Yin Tang and then disperse to a large extent into the other channels, most likely the Gall Bladder (sleeptime) channel. When the Du channel is in this deep brain, non-gastrointestinal mode, the mind dwells in either the subconscious or superconscious.[1]

This throbbing of Large Intestine and Stomach channel Qi around the mouth pulls the Du channel Qi down to the mouth, both awakening in one a conciousness of the physical body and its needs and activating the gastrointestinal tract. In turn, this additional intersection of the Du channel with the Stomach channel provides the latter with the extra surge of energy needed to activate this most crucial, life-giving channel. Again, the Du flows into the Stomach channel at two points, the upper lip and Yin Tang, rather than at just the one intersection at Yin Tang allotted to the other channels.

Prevention of backwards-flowing Qi from the Stomach into the Du

This powerful mutual influence of the Du and Stomach channels may account for the presence of the unique, Du-protecting, backflow-prevention overflow valve of the Stomach channel. The Stomach channel is the only channel that has, to my knowledge, the protective, channel-altering path that activates when Stomach Qi is running in reverse (Rebellious Qi).

This special path, a backflow prevention channel, runs from ST-6 on the corner of the chin up to ST-8, on the side of the forehead, where it ends right alongside the GB channel.[2] Should the Qi in the lower Stomach channel begin to flow in reverse, this

---

[1] This is the condition of a baby *in utero*. Before birth, the Large Intestine channel is not yet functional; if it were, it would propel meconium into the amniotic fluid, killing the baby. The Stomach channel is also not yet functional; all nutrition comes in through the umbilicus. The gastrointestinal tract of the fetus is unmoving. The GI tract has enough Qi to form itself and stay alive, but not enough energy to move in a peristaltic (gentle squeezing) motion. The baby is at this time, with his Qi focused at the third eye, either subconscious or in a state of superconsciousness (soul awareness), depending on the child's nature.

At birth, it is the abrupt transition to mouth breathing that stimulates the Du channel to drop down to the mouth, where it intersects with and triggers the full-strength flow of the Large Intestine and Stomach channels. These channels in turn pull on the Du channel, tugging it even more completely down from the third eye at Yin Tang, down the face, and into the mouth, thus establishing the newly-awakened *conscious* awareness of both the body and the body's need for food from an outside source.

[2] For you students of Asian medicine who learned that acupoint ST-8 on the forehead is on the normal path of the Stomach channel, I propose you perform this simple test: use your hand to feel where the various channels of face Qi are and which direction the Qi is moving. You will notice that Stomach channel Qi flows from ST-1 to ST-2. From ST-2 it broadens out and flows into ST-3 and ST-7. From here it continues down to ST-4, ST-5, and ST-6, and then into ST-9.

protective channel is activated. If this protective channel were not in place, backwards-flowing current from the Stomach channel would interrupt the orderly movement of the Du channel at both the lip and the forehead. This could possibly deflect or decrease the current in the Du channel so much that a person could lose consciousness. If Qi runs backwards through the head, going from Yin Tang backwards to the neck via the midbrain, consciousness ceases, and death can possibly ensue.

To prevent this calamity, backwards running Qi from the Stomach channel cannot flow into the face portion of the Stomach channel. This deadly possibility is instead deflected; rebellious Stomach channel Qi coming up the torso and into the neck is shunted into the diversion channel at ST-6 (a Stomach channel acupoint on the corner of the mandible), and is redirected to the sides of the forehead.

Backwards-flowing Qi that is coming up the Stomach channel can get as far as the neck before it flows, not across the lips and up the face backwards to Yin Tang, but instead, up the side of the face towards the Gall Bladder channel.

It is activation of this protective circuit, which redirects Qi from the Stomach channel to the GB channel, which sets in motion the substantia nigra changes of PD.[1]

Stomach channel flow powers the Du channel

In healthy times, the Stomach channel helps drive the Du channel. A decrease in Stomach channel Qi will decrease the power of the Du channel. In this case, the *force of the pull* on the Du channel is decreased, though the amount of Qi in the Du system overall remains the same. A parallel example would be a decrease in the power of a waterfall that occurs when the distance of the drop is decreased. The amount of water in the river stays the same, but the power generated by the drop is lessened. A change in Stomach channel Qi alters the power and distribution pattern, though not the amount, of Qi in the Du channel.

The dopamine connection with sleep

In western medicine, as noted, the stomach and the parasympathetic system are connected: we are taught that the parasympathetic system is dominant when an animal is chewing and digesting food. So too, in Asian medicine, the stomach and parasympathetic system are strongly connected. They have a special, double electrical relationship via the second meeting of the Stomach and Du channels at the mouth, after the connection at Yin Tang. And remember, the Du channel regulates dopamine and the parasympathetic system.

The Asian system takes this integration a step further – at night, the stomach very nearly shuts down (never go to bed on a full stomach!) and the consciousness goes deep within, into the subconscious. The food-processing, parasympathetic state ceases; breathing and heart rate settle into a sedated pattern – a pattern much less responsive than those that are active when awake. Western science has not yet named this condition, merely considering it a subset of the parasympathetic state.[2] As the stomach shuts down

---

[1]Please read further explanation in chapter two, *Recovery from Parkinson's Disease: A Practitioner's Handbook.*

[2] Which is wrong! In the parasympathetic, blood flows to the gastrointestinal tract, heart, and lungs in a manner that best digests food and distributes food energy. During sleep, flow of blood and energy decreases in the gut and skeletal muscle and goes deep within, engorging the liver, gall bladder, and

at night, the Du channel power is being decreased due to the increase of Qi in the Gall Bladder channel. The GB channel runs the opposite direction of the Du; an increase in the GB channel causes a diminishing of power in the Du. The gastrointestinal tract becomes stilled and dopamine levels drop steeply, while the subconscious reigns and "knits up the ravelled sleave of care."[1] During this nonsympathetic phase, also known as sleep, the brain dopamine level – and we propose, the entire dopamine system – drops precipitously.[2]

Stomach and Du channel during sleep or Parkinson's

During Parkinson's, the rebellious Qi of the injured Stomach channel, for reasons explained in *Recovery From Parkinson's Disease*, decreases the power of the Du channel and amplifies the nonsympathetic (nighttime) pattern, one that is associated with the subconscious rather than the conscious brain. Anthropomorphically speaking, the injured channel does this to encourage the injured person to go to sleep, to surrender the injured body over to the deeply restful mental state in which healing can occur; healing is a process that does not occur during adrenal-driven, fear-based wakefulness. During sleep, adrenaline and dopamine levels drop.

However, due to the pathological level of fear in a person with Parkinson's, this healthy retreat, this surrender into true, non-adrenal slumber, does not occur. Surrender is scarcely a part of the PDer's vocabulary! Although the PDer may dip every night into a sleep, it will never be the willing, surrendering sleep of the trusting child; it will remain ever the sub-alert sleep of one who is in a state, not of peace, but of emergency. Therefore, healing does not occur.

This means that the injury is, in the decades after the Qi starts flowing backwards from the injury site, sending a perpetual signal to the brain that causes Du channel deflection and a nighttime (no dopamine) signal to the midbrain, and all the while the adrenal system is maintaining hypervigilance. Eventually, the dopamine-producing cells, not having received a "make dopamine" signal for decades, slowly revert back to a more basic form of undifferentiated cell. These cells are not dead, but they are no longer receiving "make dopamine" signals from their surrounding electrical flow. The overworked adrenals slowly dig themselves an early grave. When the adrenaline source finally starts to give out due to decades of overuse, the concomitant absence of dopamine becomes apparent. The damage wreaked by the injury, which for decades has blocked life-giving Qi from flowing through the Stomach channel correctly, can no longer be hidden by an adrenaline override because the adrenals are exhausted. The long-term dopamine dormancy is revealed. Parkinson's "appears."

---

lung tissues. This is not a reference to the arterial or venous blood that flows through the liver passageways or through the aeration portions of the lungs, where it is cleaned or oxygenated, respectively. This is in reference to the blood that supplies the tissues of the liver, gall bladder, and lungs themselves. During this state, very different from the parasympathetic, peristalsis is slowed, as are heart and breathing rates. This condition should more aptly be called the nonsympathetic, if the "sympathetic" nomenclature (poor word choice, but now well-established) is to be followed. A better choice might be to abandon the "sympathetic" system altogether…a subject far beyond the scope of this book!

[1] Shakespeare, *Macbeth*.

[2] As recently as the late 1990's many doctors still held to the theory that dopamine levels are highest during sleep. This outmoded theory has been disproven. Dopamine activity is at the lowest levels during sleep. (See appendix 6.)

Stomach channel and Du channel during sympathetic (adrenaline) excitation

In a healthy person, the positive relationship between the function of the stomach and the parasympathetic system is paralleled by the decrease in both during times of stress. During stress, when the sympathetic system prevails, the stomach processes are stilled or diminished. Again, "Fear scatters the Qi," decreasing Du flow at Yin Tang and sending Du channel Qi surging into the Yang channels via Du 14, at the back of the neck, rather than from the forehead. When this back-of-the-neck source of Du Qi distribution is used, rather than Yin Tang distribution, Qi flows equally through all the primary channels – as opposed to the parasympathetic condition in which the Stomach channel gets a double hit. During non-emergency, a person's Qi is distributed via Yin Tang and the Stomach channel receives twice the energy of all the other channels.

In the fear condition, the Stomach is no longer the favored channel. A person's primary energy source, the Du channel, surges out into the arm and leg (primary) channels from a point on the back of the neck even before the wise frontal lobe is stimulated. A person so activated behaves more like an animal. His primary channels, rather than the extraordinary channels, are predominant as he fights or flees. This is the opposite of how he acts when the Du channel is controlled, unscattered, dominant over all other channels. In a condition of fear, when the Du is "scattered" through the other channels, man is more like an animal. Only when the Du is calmly focused at the frontal lobe can one act as a Man, the highest bridge between heaven and earth.[1]

Addiction and the Du channel

Healing the Stomach channel restores the midbrain

This relationship between the Stomach channel and the Du channel helps explain the differences in addictability that we saw in our patients.

When awake, during parasympathetic time, a well-tuned Stomach channel allows for fullest possible movement of the Du channel through the midbrain, which in turn creates the largest possible amount of dopamine-related activities – including addiction. When the Stomach channel is injured, the parasympathetic system cannot flow very well, and a person is not very addictable. If the Stomach channel is restored, the Du channel springs back to life, and addictability and other parasympathetic functions are simultaneously reactivated.

This could explain why people with Parkinson's disease who have only one major unhealed injury, the classic PD injury on the foot, appear to spring from a condition of non-addictability to one of extreme addictability, practically overnight, once the Stomach channel is restored.

Stomach channel restoration increases addictability

Therefore, any improvement in the flow of Stomach channel Qi will move the marker on the sympathetic/parasympathetic continuum strongly towards the parasympathetic, addiction-prone end.

---

[1] Please, this is not a comment on gender. The word "man" in this sentence is used to signify any elevated being, male or female, who is aware of his relationship with all creation, with the Tao, with Love. From the eastern perspective, it can even be argued that all humans are female. Pure thought, in this usage, is the only male aspect in the universe. Our English vocabulary is sadly lacking…

Injuries on other channels

However, those with complex Parkinson's, those who have injuries in other areas besides the Stomach channel, might not become so immediately addictable. Possibly they might not spring back to full Du function when their foot injury begins to heal. The remaining injury may also suppress the Du, although not via the same mechanism.

Stomach channel rebellious Qi decreases Du flow

The Du decrease stemming from the foot injury seen in idiopathic Parkinson's is due to the constant electrical diminution of the Du, a diminution that should normally occur only from 11:00 p.m. to 1:00 a.m. – when the Gall Bladder channel is scheduled to run at full bore. In the case of Parkinson's, however, the Gall Bladder channel runs at pathologically higher than normal levels twenty-four hours a day. This excess Qi in the Gall Bladder channel results when Stomach channel Qi flows rebelliously up the channel and is shunted into the Gall Bladder channel (from ST-8 to GB-4) to prevent backflow (rebellious Qi) into the Du at points Du 26 and Yin Tang. This constant overload into the Gall Bladder channel creates an unending nighttime/sleeptime (no/very low dopamine activity) condition in the midbrain. This leads to the dormancy of the substantia nigra cells and their eventual reundifferentiation into neutral, embryonic-like cells.

Other injuries decreasing Du flow

All Yang channels connect both with the Du channel at Du 14 (at the back of the neck) and at Yin Tang. Impedance on any of these channels will cause a diminution in the Du channel flow. Therefore, an injury to any part of the body will influence the nearby channels and decrease Du channel flow. In this way, injuries can send signals of distress to the brain and activate the sympathetic system. While these other-channel injuries in and of themselves cannot trigger classic Parkinson's (lacking as they do access to the facial muscles, anteriolateral leg muscles, muscles along the mammary line, and left-right coordination, to name a few zones which are regulated most specifically by the Stomach channel, and which help to characterize Parkinson's), they can certainly raise the sympathetic flag, acting as a drag on the addiction process.

When Qi irregularities from other, non-Stomach channel injuries send distress signals to the brain via the Du connection and also via their own brain connections, these non-Stomach channel injuries may pull the sympathetic/parasympathetic continuum marker slightly in the sympathetic direction. While these latter Qi irregularities would not affect the midbrain portion of the Du channel as directly or for as many hours a day as a Stomach channel back-up, they could still exert a moderating influence. Again, the influence of the Stomach channel over the Du is greater than any other channel because of the driving, pulsing push given mutually to the Du, the Large Intestine, and the Stomach channels via their intersection at Du 26 at the end of the Du channel.

Even the modest stimulation of the sympathetic system from injuries on channels other than the Stomach channel may help to decrease that susceptibility to addiction which is a part of the parasympathetic system. Therefore, a patient with multiple injuries might have more time in which to safely recover from medication. Such a person might be able to notice a decrease in his Parkinson's symptoms as the Stomach channel resumes normal function. Even while taking medication, the decrease in rigidity, increase in sensation, and feeling of internal peace might signal to him that his Parkinson's

symptoms are diminishing. And yet, his other injuries might provide him a somewhat safe shelter from the most rapid (seventy-two hour) addiction processes that we have seen. A person with multiple injuries will still become addicted if he is taking antiparkinson's drugs when his underlying Stomach channel injury is resolved, but the addiction may come on somewhat more slowly, somewhat less violently.

It might be dangerous to assume such a safety net, however – we have seen in many patients that the healing mode, once started, might travel throughout the body, addressing many if not all unhealed injuries. Very often, a person whose foot begins to heal may notice that other injuries in the body also begin to stand up and demand to be noticed – or even start to heal on their own, with no healing techniques being administered. This means that whether or not an auxiliary injury can prevent rapid addiction is still a crapshoot – one with dangerous, mind-altering risks.

Injury on a non-Stomach channel

As an example of how an unhealed injury on a non-Stomach channel might affect the Du, consider Olli's shoulder injury. It traversed the area where the Triple Burner channel and the Small Intestine channels cross the shoulder. The scar tissue in that area could block the Qi of those channels. These channels are supposed to flow into Du 14, on the back of the neck. If they were blocked, the resulting decrease in channel Qi would subsequently decrease the Qi that flows into the Du channel at Du 14. However, it would not disrupt the signal in the same way that the Stomach channel diversion does. This decrease in Qi would merely alter the *amount* of Du channel Qi, not its distribution pattern. To return to the waterfall analogy, the Triple Burner injury would decrease the *amount* of water in the Du stream, and the amount of water (or Qi) in its eventual waterfall drop from Yin Tang to the mouth, but *not* the distance of the drop. From an electrical analogy, the amperage would be decreased, but the voltage would remain the same. The shape of the Du channel would be the same – the Du Qi could still flow down to the lip – but the quantity of Du channel Qi would be less than usual.

Therefore, the existence of non-Stomach channel unhealed injuries might cause a decrease in Du channel flow, but not deflect it. (Deflection of current is the most likely mechanism for the Gall Bladder channel's influence on the Du.) These injuries could not trigger or sustain Parkinson's disease if there was no longer a foot injury – but they might decrease the parasympathetic system, and hence the addiction system, just enough to account for the difference in addictability between patients like Olli and Viktor.

Olli was starting to recover from PD, still had remaining injuries in his arm, and because he had not stopped his medication in time, was lured to slowly increase it. Viktor, with no other injuries that I am aware of, experienced the full bliss and addiction of dopamine saturation almost simultaneously with the unblocking of the injury in his foot.

Summary

Possibly the variations in Qi flow due to individuals' various Qi blockages contribute to the variations of addictiveness in people who are recovering from Parkinson's disease. This would provide a simple, logical reason for the range of addiction responses that we have seen in our clinic. It would also match the patterns that we have seen: rapid, violent addiction response in patients with no secondary injuries and slower, more gradual reversion to addictability in patients whose Qi has other, non-Stomach channel blocks.

In this year, 2003, thinking of addiction in terms of deflected electrical fields may not seem logical to many westerners, but that is because they are not used to considering the electrical and magnetic influences of the body in their study of biology. Radio did not seem logical to people who first tuned in to the airwaves. In the late twentieth century, many an old-timer was heard to whine, "But where *is* the Internet?" Qi flow, to many westerners, is still regarded as a pagan myth. But in this new century, when Qi flow – the vibratory energy and microelectrical circuitry of the body – is given its rightful place in the study of western physiology, these systems will be seen to be the drivers, and not the side effects, of metabolic processes. However, the focus of this book is medication issues, and not Asian medical theory; full development of this idea is, you guessed it, beyond the scope of this book. And yet, if the ideas presented in this book can in any way further the understanding of the scourge of addiction, it is not a moment too soon to be considering this ancient/new way of understanding brain chemistry.

"Honest men esteem and value nothing so much in this world as a real friend."

The Fables of Pilpay, Panchatantra (circa 326 B.C.)

25. BECKY AND MIRAPEX

AN AGONIST FOR BECKY; HELP FROM FRIENDS

When we left Becky in December of 2001, she was frail and gaunt, but completely off the drugs and optimistic about the future. Over the Christmas holidays, her friends were saddened by her inability to participate as tirelessly as she used to in their delightful dinners, theater events, and outings. In February of 2002 they put together a position paper for Becky. They said that they would no longer invite her to their events, provide transportation, or assist her in any way unless she resumed working with Dr. Leslie. They were doing this out of love. They were saddened to see her weakness, and they were certain that all she needed to return to her old self was a hearty dose of good medication.

They had no idea that her drugs had caused her tremor to transform into El Twitcho. They refused to believe that the Breath Monster was a side effect of the medication. They were unaware of the dangers of the medication, but they knew that Becky had slid from the driving force of the group to the most needy member. They felt that our program was probably a health-nut cult, and that Becky had been brainwashed. How else could one explain Becky's preference for weakness and frailty when it would be so easy to be healthy? After all, there are medications that treat Parkinson's disease – why wasn't she taking them?

They delivered an ultimatum: either Becky stopped working with me, attending our clinic, and never saw me again, or else they would never see her again.

Becky was devastated. She asked me to meet with them. They agreed to meet, requested four copies of the planned agenda, and then decided against meeting with me. Becky asked me to write up a small paper listing the adverse effects of the drugs. One of her friends, a nurse, dismissed the list of adverse effects, stating that "adverse effects only occur when the drugs are used incorrectly, or for the wrong illness. Parkinson's drugs don't have adverse effects if they are used for Parkinson's."[1]

---

[1] Strangely enough, while some doctors, particularly European doctors, have been very open to our ideas, American nurses have been, in general, much more opposed to our work than their MD masters. While most nurses will resent this idea that they are subordinate to doctors, the blind defence of MD-approved protocols by most nurses speaks volumes. While doctors themselves often disagree about various processes and procedures, many nurses remain loyal to the idea that, although the doctors may promote opposite theories, the doctors are always correct – whatever correct may be. (My doctor, right or wrong!)

I have friends, ex-nurses, who have quit nursing due to this strange loyalty and illogic that is often required of those who would serve under the doctors' banner. It is due to this prevalent attitude, more than any legal or technical regulation, that I refer to their "MD masters." I don't see this attitude so strongly in Europe or Canada, although I do know people in those countries who have quit nursing rather than be silently obedient to medical attitudes and procedures that they knew to be negligent and dangerous. Fascinating!

At our last "official, goodbye session," Becky demanded rhetorically, "Why does it have to be a fight? Why does it have to be one way or the other? Why can't we all get together and take what is best from each kind of medicine?"

We talked a long time about what was most important for Becky. I stammered a bit and tried to explain that I would love her no matter what she did. My door would always be open to her, and she could call any time. She needed to look into her heart and make a decision. I would support her no matter what she chose.

I had known Becky for over twenty-five years. I had worked closely with her in a doctor-patient relationship for four years. We had met weekly in my office, plus an hour a week at the clinic. My students had provided transportation for her to and from the clinic. One student and one clinic FSR volunteer felt a deep connection to her: during her worst stages of drug withdrawal, they had bathed her, given her massages, put lotion on her dry skin, and brought her food and herbal medicines. These two helped her when she moved into a seniors' complex and helped her decide on the best Feng Shui arrangement of her furniture. They came to her apartment every week to provide massage, ear acupressure, or just help around the house.

It was all done for love – no money changed hands. They, like her friends and myself, simply loved Becky. Her fiery spirit, unquenchable sense of humor and droll self-deprecation emanated love. I always felt uplifted after an hour with Becky. She was, and is, an inspiration to all of us in the clinic.

Cult status

Becky's friends felt that these students and I must be cult members. Why else would we be treating her for free? Why else would students take time from their busy days to dote on an old lady? There was something suspicious about our program. Of that they were certain.

Return to Dr. Leslie

Becky called me later in the week. She had gone to see Dr. Leslie. Dr. Leslie had agreed, finally, that Sinemet might not be the answer for Becky, and put her on Mirapex. Becky was to start at a low dose and work up to a therapeutic level over the next two months. Dr. Leslie's nurses praised her roundly for overcoming our brainwashing.

I told Becky that I was grateful for the information, and, although she wasn't allowed to see me anymore, I would be glad for any information she could give me in the future regarding her responses to the drugs and her general health.

The following Tuesday at 2:00, Becky's old time slot, I walked into the waiting room of my office. There was Becky, grinning away.

"What are you doing here? I thought you couldn't come anymore!"

"Shhh! Ha ha! I'm here on the sly! What they don't know won't hurt them!" She lifted one eyebrow and peered over her shoulder as if looking for spies, and winked at me conspiratorially. I wished that Dr. Leslie could see Becky's so-called "lack of facial expression" at that moment!

Such hugs!

We continued to meet once a week as before. Whether or not her friends knew about it, I have no idea. But she took the medication, as ordered. She was determined to have it both ways.

A therapeutic dose of Mirapex is somewhere between 2 and 4.5 mg. Because this particular drug can cause blood pressure decrease, narcolepsy, and stomach problems, patients are instructed to start with extremely small doses. The drug comes with a starter kit containing doses of one eighth of a milligram, one fourth, one half, and one mg. To start, a person takes one eighth of a milligram three times a day for two weeks. Then the fourth of a milligram pill is taken three times a day for two weeks, and so on. After two months of gradual increases in dosage, a person is supposed to begin to feel the effects of this drug.

What we have seen repeatedly is that patients actually feel the effect within two months whether or not they make the gradual increases. It appears that this drug, if taken at low doses, can be as effective in some patients as the higher doses. However, it can take three weeks to two months before a solid, beneficial result occurs. Time, rather than amount, appears to be the more important factor.

Of course, from a marketing perspective, it may be that patients would be unlikely to take a pill for two months if there is no immediate effect forthcoming. Psychologically, it may be easier to take graduated amounts, knowing that you are building up to an effective dose, and that, as soon as you are ready for the big guns, you will see the effect. Compared to taking the same small dose for two months, hoping that at some point the accumulation will begin to be felt, the "graduated dose" approach is easier to understand, and probably better compliance is obtained. On the other hand, most of my patients were recovering from Parkinson's, so who knows how they differ from PDers. Maybe PDers do need the larger amounts! Becky, at any rate, noticed effects of the drug within days, while taking the three eighth-of-a-millgram pills, an officially "ineffective" dose.

Becky no longer typed her journal. She used her pocket tape recorder and, as often as something of interest occurred to her, she recorded it. When she arrived for each visit, she gave me a verbal recap of the week. Then, after her acupuncture needles were inserted, I sat down and transcribed the week's tape. Her first report after starting Mirapex stated:

> The tremor has suddenly gotten bigger. I'm nodding off to sleep more (narcolepsy). I even fell asleep in the theater!
>
> I'm still having improvements in stride, voice, and vocabulary, but that has been steadily improving ever since I got down to 50 mg/week [not per day] two months ago. I really feel good about not taking the L-dopa anymore. I'm having a bit of eye trouble. I'm wondering if it's the hallucinations that they warn about with Mirapex.

[She opened the session the next week by asking me if I was wearing a clown suit. I assured her I was not. She was still taking the minimum, "non-effective" dosage. Her report was telling:]

> Am I having hallucinations I wonder? Oh yeah, they're there! They aren't bad. For example, if I see someone down the hallway, they look like they're wearing a clown suit. My own clothes are covered with salmon-colored webbing, and there are kittens or other little animals playing just at the corners of my field of vision now and then.

I'm sleeping until 4:00 a.m. now before needing to use the bathroom. Yesterday I had a busy day. Walked two blocks to the drugstore, then took the bus across town for groceries, and that about wore me out.

How's my mood? As well as could be expected! The tremor is getting more annoying, but I can enjoy the hallucinations. The tremor at night is much worse. I suspect it has something to do with the medications.

[My own notes for this visit say that she seemed unnaturally buoyant and slightly illogical. Her actual responses to questions were more rambling than the edited version above.]

[Her third week was downright worrisome:]

I'm having new kind of tension. My symptoms feel more "concentrated" in my hands and feet; there is a lot of tension in the extremities. I feel more stimulated, more "go for it and never mind the consequences!" I am taking baby steps at times, but I don't seem to care as much. Sleep is still good though, and I've gained two pounds. And, if you don't mind my asking, have you dyed your hair bright blue? [I had not dyed my hair. It was still a middle-aged grey-brown.]

Week four:

I was worried about the tension in my hands and feet, and so I decreased the medication. Last week I was taking 3 one-eighth pills, so this week I started only taking two pills, a one-eighth and a one-fourth.

[I tried to explain to her that she was still taking the same amount, except that she was doing it in two pills instead of three. She had an alarmingly difficult time understanding what I was saying. She was giddy, laughing, and illogical.

[She explained more about the hallucinations. They were pleasant visual transformations in which, for example, stepping stones on the lawn turned into gamboling squirrels, cats, or puppies. Fire hydrants turned into small children waiting for the school bus. Stop signs became tall, smiling figures that waved as she walked past. None of them were frightening, and she didn't mind them in the least. She couldn't tell if they were real or not, but they didn't particularly bother her one way or the other.

[I have seen other people who rather enjoy their Mirapex hallucinations. In all the patients in our limited experience, the Mirapex visions are cute, friendly, and harmless.

[Becky greatly preferred the Mirapex group to the set of mental disturbances she had experienced six years prior when taking Eldepryl for a few days. When Dr. Leslie had first diagnosed Becky, he had prescribed Eldepryl, excited by the drug maker's claims that this drug prevented worsening of Parkinson's, a claim they no longer make. Becky had violent nightmares. One night she found herself in her closet struggling to pull everything off the hangers in her attempt to wrestle with the bear in the closet that was trying to kill her. Another time there were monsters in her room. She had promptly stopped the Eldepryl and started Sinemet. The nightmares ceased.

[Different hallucination-inducing drugs cause different types of mental illusion. Specifics are provided in Appendix 2, in the descriptions of the specific drugs.]

Week five:

[She was still taking the same dosage as the week before and thought for sure that by now she had reduced her daily intake. She felt that it had been a good week. She was still growing in manual dexterity, as she had been since December. This week she was having an easier time getting clothes on and off. She had gone for a walk to the burger joint a long block away and had felt just fine.

[The most interesting experience of the week was on the day when she forgot to take her morning pill. She was doing very well until mid-afternoon when she suddenly remembered that she hadn't taken her pill yet. She immediately started having baby steps and shaking, and they didn't stop until she swallowed the forgotten dose.]

Week six:

When I first get up in the morning I'm fine. I have breakfast, and then around nine, I take the first pill. About an hour after taking the pill, the tremor gets worse for a few hours and then slowly ebbs. The baby steps are worse starting about an hour after I take the pills also. They get better again after a few hours. The same thing after the afternoon dose. If I eat a lot at bedtime, I sleep better. And I don't have baby steps at night. I'm fine during the night. Slept until five this morning without waking up! Wonderful!

My son's not worried about me anymore. He's suddenly in a confessional mode: now he's worried about himself. He blames his mood swings on my years of bad parenting. He's having bad mood swings, he says he's an alcoholic – I had no idea – and he's been smoking dope. I can't say anything to him without him blaming me for his problems. Says I should never have had children. Then he cries and wants to take me out to dinner.

Meanwhile, my friend Virginia Lee is plotting with Dr. Leslie. Virginia Lee is a *nurse*, and she loves the idea of Sinemet. Since the Mirapex isn't working the miracle she expected, she's going to try to get Dr. Leslie to change his mind and put me back on the "miracle drug" while I'm still in a compliant mood.

Week seven:

First day of spring! I walked to the library to return the taped books and got some others. I was very shaky all day ever since an hour after taking the first pill. My hands and feet are so tense, Terry came and massaged just my hands and feet and back. [Terry was an FSR volunteer from the clinic.] I'm so shaky from the pills; I only took half a pill instead of a whole one for my afternoon dose."

Week eight:

Shake! Shake! Shake! Frequent urination at night, and now the Breath Monster is back. I'm taking baby steps all the time now, and I really need the walker! I'm going out for a half hour walk every day; I *have* to get out; I'm so agitated. My appetite's reduced. I'm not ravenous like I used to be.

The pills don't seem to work very well. They have very little beneficial effect; I'm more shaky than ever. I had the Breath Monster two days ago and again this morning. It's a gloomy morning. I have even more tension in my legs, arms, and upper body. It all gets worse after the first pill of the day. Maybe from reducing the pills last week? I will stay at this level for a week to stabilize, though I am tempted to increase the dose a bit to get some benefit. On the other hand, everything seems worse after I take the pills.

Week nine:

The pills don't seem to be doing any good at all, and I have a new problem that I've never had before: I feel like I'm always about to tip over.[1] My brain feels always slightly out of whack; I'm even using my cane indoors. Outdoors I have a mad clutch on my walker. The forearm and upper arm muscles are *so* tight. At night I must force myself to relax; I feel my muscles clench in anticipation of the stress of going to bed. The Breath Monster is frequent now, or at least always lurking in the background. My shaking is so much worse; I shake like a madwoman. It's so violent I bruised my own nose putting my glasses on. I wanted sympathy and nearly called you at home. The hallucinations are the same as before. Lotta baby steps. A hard, hard week.

I'm so depressed, I don't know what to do. I'm so down. Earlier today I went for a ride up the coast with the girls. It was absolutely gorgeous. I thought of all the times I hiked there with the Sierra Club, and now I'll never do that again; so sad, so depressing. I'm feeling very down remembering the old days with Hit and Run Theater. Those days are gone; those people have scattered with the wind.[2]

Maybe the depression is from the drug reduction? People are coming up to me and saying I'm looking better, but inside I just feel gloomy.

Week ten:

El Twitcho is back, that strong jerking that shakes my whole body. It just comes an hour after the pill, and then after an hour it goes away. I think the pills cause it. The Breath Monster is still there but less compelling. He may have been the result of the drug decrease. [Actually, it was the return of the Breath Monster that had led her to decrease her dose two weeks earlier.]

Fewer baby steps – those have backed off a bit. Now I can feel when they're about to come on and I just step back and reconnoitre. I walked in the grass without the walker! I'm more confident now. One day I had no baby steps. I'm getting a little better at cutting up my own food at meals; that's been hard for over a year, but slowly it's coming back. I still can't cut meat or peel an apple; I can't exert pressure with my fingers. And the muscle cramps and tension are still

[1] Balance problems are a recognized adverse effect of Mirapex.

[2] As you will read in the appendix on Mirapex, deep, intractable depression, rather than paranoia, is characteristic of withdrawal from this agonist. Although the paranoia from levodopa withdrawal is more ferocious, it does not last as long as the depression from decreasing Mirapex. The former is usually over within ten weeks; the latter can last indefinitely.

there, but less than a week ago. I think the Mirapex reduction is starting to show a benefit.

I did exercise class this morning. There's a lot to do at the seniors' complex, but I don't always feel like doing it.

My mood is up and down. I'm sometimes impatient with people around me. They talk such nonsense! Not the fun kind, but the "Oh! She shouldn't wear that dress," petty stuff with no inherent interest. Funny mood today; world news and chatty neighbors all annoy me. I don't care what we're having for breakfast, but everyone else is so fussy. Who cares! They all want to argue about the breakfast menu! As for me, today I'm walking better, but who knows what tomorrow may bring?!

By the way, look how much better I can get up on the table and position myself!

[This was mid-April. Since December she had slowly and steadily been recovering her strength and motor skills. Whether this was coming from recovery from Parkinson's or simply recovering from her traumatic Xanax/Sinemet reduction is impossible to guess. Added to the mix was the Mirapex, which was undoubtedly having some beneficial effect on motor function, although it was clearly too strong for her even at the lowest possible dose. She was still taking less than a sixteenth of the suggested minimum therapeutic dose. Clearly she was not tolerating the medication well, and yet we must add it into the equation. Whether it was helping or hindering her motor recovery, it is impossible to guess.]

Week eleven:

Not a bad week. The baby steps are bad on alternate days. I'm going for a lot of walks, trying to be outdoors a lot. Went to exercise class three times this week – I do most of the exercises sitting in a chair. I was able to read most of an article in a magazine! It was slow, but I could read it. Maybe my eyes are improving? The only really annoying thing is the tremor – it drives me nuts. Could I decrease my meds again? I almost forgot to take it last night…I wonder what would have happened?

I went to a dinner party last night at the X's, we partied 'til midnight, and last night I slept like a baby.

Good news: the hallucinations were gone for three days in a row. Then they were back briefly, but that's OK. Actually, I miss them. They were fun. The halls looked like mosaics, people were wearing technicolor. Now the halls are plain paint sometimes and people are wearing plain clothes. So many times (with the hallucinations) I was tempted to say 'I love your outfit!' but then realized the outfit wasn't there! Ha!

And so it went. Her friends grew angry with her when she told them, in all honesty, that she was not increasing her medication. There were certain that, if only she would increase the drugs as prescribed, she would return, magically, to her old self, ready to hike, perform on the stage, and be the life of every party. However, they were appeased by her low doses of medication. They didn't abandon her.

She tried a few more times to increase the dose, and every time it was the same: her symptoms worsened. Eventually, in response to these worsenings, she decreased and went through a long period of morosity and agitation. She is still taking one or two of the one-eighth milligram pills per day; she has never been able to tolerate any more than that. She suspects that she might do better if she were to stop her medication altogether, but she has promised her friends that she will take her pills, and she is determined to meet them halfway. Also, if she is taking even just a little of the medication, she figures she is appeasing Dr. Leslie. Should the question arise at the doctor's office as to why she is taking so little, she can explain that she gets worse when she increases, and that the hallucinations are too severe. That answer, she expects, will keep the doctor at bay.

My students discuss Becky

Each week, after the patients leave the clinic, the students and volunteers have an hour-long discussion/seminar during which we discuss the various patients and the topic of the week. One week during the seminar, around the time that Becky started taking Mirapex, one of Becky's intern practitioners struck up an impassioned protest of Becky's decision to resume taking medication. (It had possibly been triggered by Becky's surprised outburst when she entered the clinic, "Oh look, you've redone everything! How nice it all looks!" Of course, nothing in that barren clinic had changed – the only new thing was Becky's Mirapex-driven hallucinations.)

The other seminar students angrily joined into the protest. They accused Becky (in her absence) of weakness and lack of purpose, and worst of all, of giving in to her friends though she knew she was right and she knew they were wrong.

They demanded to know why I hadn't prevented her from starting Mirapex after she had gone through such tortures getting off the Xanax and Sinemet. Why hadn't I forced her to make a decision: her friends or our program? It would have been in her best interest. If only I had pressured her, she would have done the Right Thing! How could I, her doctor, have "let her go" without a fight?

The energy in the room moved up into the ultraviolet zone as clinic interns and acupuncturists protested Becky's medication choices with uncharacteristic warmth. The mood was ugly, and the discussion turned in a dangerous direction: how could they somehow tell Becky what to do without violating the code that forbids an acupuncturist to give prescriptive advice about the medications? The students' circle was foaming with ideas.

No one even noticed as I got up from my seat at the head of the circle and left the room to fill my water cup, hoping that words would come to me to explain to the class just what I was feeling.

My emotions had been so mixed when Becky had first told me she could never see me again. Ever since she made the decision to obey her friends, I had not said one word about her subsequent decisions to increase or decrease her drugs. I had promised that, whatever she did, I would support her, and my only hope was that we might stay in touch. I had never allowed myself to dwell on "might-have-beens" of "should-have dones." I had never really given her decision any thought other than how much I would miss her if she stayed away.

The class was expecting me to lead the discussion on how I might stay within the confines of the law and still manage to convince Becky to get back off the drugs. I lingered by the water cooler, asking my heart for counsel. I knew I couldn't give them what they were expecting.

Slowly I set my cup on the counter and slogged back to the room. I could almost see the heat waves pouring out the classroom door.

"Class, may I have your attention please?" I must have been using my best teacher voice because even though I didn't feel like any kind of authority right then, the tumult stopped in mid-fuss, just like in the movies.

"Becky made the right choice." (They all sat up straight and gawked. I didn't yet know where I was going with this, but it seemed I'd gotten their attention.)

I told them that Becky knew that the medication had side effects and that the drugs made her physical and mental symptoms worse. She had been through hell with the other drugs, and she had no reason to believe that this new drug would be any different. So she knew that by taking these drugs, she would probably be putting her health, and even her mental health, on the line.

She was certain that her increased pain, tension, and shaking were from the drugs. She knew that Mirapex gave her hallucinations – the one thing she had always dreaded. Now that she was hallucinating constantly, she knew her mind was unstable – her brilliant mind, her greatest point of pride.

"She is aware, objectively, of these problems," I said, "and she has chosen to abide by them, even if it kills her."

I explained that health comes and goes. Every body that is cured of an illness must still someday feed the flames of the funeral pyre. But love, especially the love of true friends, is eternal. The joy generated in the company of friends is the reason for our existence. What is the point of health if we have no friends? But true friends will be there for us even when we are sick.

Her choice had boiled down to this: her health or her loved ones. She chose her loved ones instead of her health.

Becky has the great good fortune to be a part of a tight circle of friends that has been together for over thirty years. They do activities together several times a week. They have parties, go to shows, travel, and discuss literature, politics and theater. They have taken in stride Becky's increased need for transportation and her slower pace. Becky is still a full-fledged member of her group, despite her disabilities.

Her friends had not rejected her because she was sick. They were understandably upset with her because they wanted to help, and she wouldn't let them. They were suspicious of me, not because they were jealous of my relationship with Becky, but because they felt I was not looking after her best interests.

"While you may disagree with their choice of medical methods," I suggested, "you should not doubt for a moment their love and sincerity."

Maybe it was saying the word "love" out loud that led my train of thoughts off on a track not usually traversed in the clinic.

I tried to explain that Becky had been in a rare position. She was torn between our program and her friends – but *not* because she didn't know which would be more effective; she knew that her friends were staunchly misinformed. She knew that they disbelieved in any drug adverse effects or dangers. She knew the drugs were harming her

from her own experiences with the drugs. She had seen patients coming and going in our clinic for four years, sharing their drug horror stories. So Becky wasn't torn between them and us because she didn't know which team had the answers. She knew which group had the answers in terms of health. But she also knew that she could never convince her friends of what she'd learned. So her choice was this: her health or her friends.

"Many healthy people are unhappy and friendless," I continued, carried away by my own words. "Health does not imply happiness, or happiness health. Many of the greatest saints were sick or covered with sores, sores that they ignored, while they joyfully imparted physical healing and hope to unhappy souls. St. Francis of Assisi was covered with lesions, and he healed lepers and even raised the dead by the power of his love and faith.

"Becky chose friendship over health. She chose the most noble and lasting of all relationships over the fleeting joys of physical comfort."

I suddenly realized where I had been going with this. I didn't have an answer, I had a question:

"How many of us here, with our obsessions for health and dramatic cures, would be brave enough to do the same?"

There was a long silence. Recognizing a good exit line when I heard one, I dismissed the class.

Walking out to my car, I was conscious of a warm glow, despite the chill of the spring night. While speaking, I had finally understood why I had not minded one way or the other when Becky chose to resume taking drugs.

I try (most unsuccessfully) to stay emotionally detached from my patients, so that I can see them scientifically, objectively. My biggest failure in emotional detachment had been that joyous day when, thinking I would never see Becky again, she had shown up in my office at her regular day and time. She had come even though she had been "forbidden" to ever see me again.

Becky, bless her sweet heart, would do anything, even take make-you-crazy drugs, to maintain her friendships. It had finally dawned on me in this seminar, while defending Becky's choices, that, when Becky had chosen friendship over health, she had included me in her list of friends worth fighting for.

"There are three kinds of lies: lies, damned lies, and statistics."

Mark Twain

26. STATISTICS AND CONCLUSION

ACCOUNTING FOR THE VICTORS AND THE DEAD

There are two frequently asked questions about the Parkinson's medications. This chapter will address both of those questions.

The first is: "What are the numbers, the statistics for your project?"

People understandably want to know exact numbers of patients who have recovered, who have gotten off their meds, had brain implants while medicated or unmedicated, switched from levodopa to an agonist, reduced their drugs and felt better, reduced their drugs and felt worse, and, of course, how many of the people in our program would be in the program again, knowing what they know now.

It is only reasonable that I share these numbers with you, and I will, at the end of this chapter, but first I must make something perfectly clear: we were not instructing our patients to decrease their medication. All patients in this program who did decrease their medication did so on their own initiative. All patients who did not decrease their medication, up until a year ago when we stopped accepting medicated patients, received the same forms of Yin Tui Na treatment as unmedicated and drug-reducing patients for the first two years.[1]

However, our discussions about the drugs undoubtedly did influence some patients to have a go at decreasing their medication. As we learned more about the drugs, we did inform those patients whose symptoms corresponded to the adverse effects of their drugs that their symptoms might be drug related (but they would have to ask their doctor to be sure). We also shared information about what other patients were doing with their drugs and what was happening to them. We operated the clinic with the goal of sharing information and making observations. We were never in the business of trying to run a double-blind experiment in which patients were kept in the dark as to our intents and purposes. Nor did we try to create a situation in which similar patients were compared while doing different drug regimens. We never used placebo medications to determine the psychological effect. We were not running a statistically significant study. Our patients made their own decisions – with or without benefit of a doctor.

In the second two years of our clinic, we did start to modify the treatments that we gave to overmedicated patients; when patients appeared at the clinic with drug-induced dyskinesias, we often chose not to perform treatments that could possibly lead towards recovery or even a temporary increase in dopamine production; we also explained that choice to the patients.[2] When, on the other hand, patients showed up at the clinic

---

[1] About two years into the program, we started a new policy for overmedicated patients.

[2] Some of the traditional acupuncture calming treatments, such as Four Gates or Yin Tang, can cause an increase in dopamine. Therefore, we did not use such treatments on patients that presented with dyskinesia, dystonias that worsened with medication, overly fast speech, brightened eyes, or other symptoms of overmedication. Instead, for these patients, we sometimes used strong stimulation of a needle at KI-1 – the point near the center of the sole of the foot – to bring the Qi away from the midbrain and

manifesting symptoms of drug withdrawal, we often employed the standard ear acupuncture treatments for relief of drug withdrawal symptoms, rather than using Yin Tui Na to address the Parkinson's.[1] We tried to treat, at each session, the situation that was dominant in that particular patient at that time. This meant that, in the second two years of our program, after we knew a bit more about the drugs, medicated patients often received different treatments than the unmedicated patients.

Our clinic operates Asian style, with all the patient tables in a large open room with no dividers, so that all the patients and practitioners are visible and audible to all the other patients in the room. Patients heard each other discussing their drugs, the drug side effects, and their drug reduction experiments. The members of the Santa Cruz PD Team also shared with patients in our private practice the things we were seeing in the free clinic and with the other private patients.

We did not see ourselves as advocates of any particular health-nut, anti-drug program, or as pro-drug proselytizers. We did not start with any ideas in particular about the medications. We actually started this program with the assumption that the local doctors were correct, and that these drugs were harmless and could be stopped easily at any time if a patient was found to have been misdiagnosed. We maintained this position for over a year; the earliest editions of my books, in which I passed along the advice of local neurologists, are testimony to this position.

The number of patients who decreased their drugs while participating in our program is not necessarily indicative of who felt able to reduce drugs: medication reduction was not a tenet of our program – not everyone in our project entered the program with the intention of decreasing the medication. We did not run our program as a drug-reduction program. Therefore, the numbers that came out of our study do not represent patients who all wanted to decrease their medication, and then succeeded or

---

create a mild state of emergency to decrease the dopamine levels in the midbrain. We would not perform Tui Na on overmedicated patients, or do anything that might make them feel peaceful or relaxed.

Even if the medicated patients were not overtly showing adverse effects, performing Tui Na on them was often a waste of time. Their body could not respond to the gentle support of the treatment by calmly dealing with old injury. Instead, the body would use the support of the treatment to work out the excess effects of the current overdose of medication. In my many experiences with practitioners who had been pleased to report that their medicated patient was responding to the FSR with "lots of movements," I would almost always find, when I worked with the patient, that the responses were merely drug releases. The deeper releases of old tensions could not occur when a patient was over the top with drugs.

Also, a beginning practitioner can rarely distinguish between the native Qi flow and the various electrical forces just under the skin that are set in motion by the drugs. Each of the drugs creates a distinctive electromagnetic force that can be felt with a sensitive hand. Most practitioners, feeling this distorted energy field, imagine that they are feeling native Qi. So many people have written to me to say "I felt for Qi in my patient's leg, and you are wrong about it being Rebellious (or deficient): it is surging!" They are dubious when I suggest that what they are feeling is drugs. But if their patient comes to them after making a drug reduction, or more than eight hours after taking a pill, they realize that the Qi in the leg is quite different.

Some of the best learning experiences in this regard are the weekend classes. Each class has up to twenty patients and their practitioners. The students (and caregivers and spouses) are allowed to spend a few minutes simply resting their hands on the legs or feet of each of the patients. They are usually shocked to realize that, even with little training, they can quickly ascertain the difference in the under-the-skin sensation not only between medicated patients and unmedicated, but between the various, specific drugs.

[1] See Appendix 4: NADA protocol (ear acupuncture).

not. The numbers offered below are simply a count of what happened to all the medicated patients during the first few years of our patients' experiments and our observations.

What the numbers do not mean

If the numbers below state that a patient increased his medication, this does not mean that he needed more medication in order to be functional. Zoe, for example, continued to increase her medication even after her doctor told her that she didn't need any at all and that she was dangerously overmedicated. In the numbers below, she is listed only as a person who increased her medication, with no mention of the fact that she was overmedicated to the point of near death.

Many of the people who increased their medication did so although they knew they were overmedicated – they could not resist the lure of the medication. Others increased their medication because their doctor told them to and they had no interest in disagreeing with the doctor.

Conversely, some patients who are taking less medication are not necessarily recovered. A person who is listed as having reduced his medication may or may not be having reduced function as a result of the drug reduction. It would be impossible to say, without a full page report on each patient, what level of functionality a person has at this moment in time, and whether the medication reduction – bearing in mind the extended time-frame for observing the effects of a dose change – has led to a decrease in function.

While this may seem as if we are being coy and withholding information with the intent to obfuscate or even mislead, it is just the opposite. Without a complete recounting of exactly what symptoms a person had and how they have changed and are still changing, any report of drug increases or decreases could be misleading. Unfortunately, increasing interest in our program requires that I publish this drug information now, without full case descriptions of every patient. Hopefully, over the next few decades, every patient from the project will have his story written up. At that time, the individual case studies will provide more meaningful information than this list of numbers.

Difficulty in quantifying changes

For an example of how difficult it is to state whether or not a person is doing better or worse, I heard from a patient who now is consistently better in these areas: his life-long constipation (a frequent companion to Parkinson's disease) has not returned since it began to improve six months ago. His sleep is much better, his facial expression has returned, his arms are no longer held rigid. His energy levels are returning to a satisfactory, high level, after having been very low for nearly a year (since starting treatment). On the other hand, he sometimes has more difficulty than before in turning to the sides – but sometimes less than before – and his tremor is wild. He feels he is in the middle of a profound change – but is he getting better or worse?

Therefore, I am not going to make statements as to whether or not these patients are improving or declining. More on this subject is explained in the book *Recovery From Parkinson's Disease*. This current book is simply a record of some of the remarkable events that we witnessed in our medicated patients while they were members of our Parkinson's project.

Who is included

In choosing which people to include in the count, I have eliminated those patients whom I worked with only briefly, including the hundreds of people I have treated and interviewed at weekend workshops around the world. I have also not included the hundreds of patients that I know only through the Internet. Unless I have had a somewhat lasting relationship with a patient, and been able to observe him or her for some period of change, I have not used him or her in putting together the case studies in this book or in the numbers, with one exception – Buzz.

Buzz was included in this book even though I never worked with him as a health practitioner. I met with him over two days after he was already off his medication and moving easily, with no remaining PD symptoms except for tremor. I knew his acupuncturist, Eileen, only slightly from her attendance in a two-day class. I did communicate with Eileen via the Internet, and, after meeting Buzz, I had very pointed communications with her in which she told me about her other eight patients and the course of their progress. Everything she told me squared so well with what we were seeing in our program that I must credit her experience. Still, I don't know if I would have included his story in this book, especially in light of how dangerous it might be for someone to emulate him, except for the fact that Buzz has already been interviewed on television and has made his story available for many people via the Internet. Since he is already known, and I did want to make the point that his drug reduction was not risk free, I have included his case although he was not my patient.

And now, the numbers please:

63 medicated patients participated in our program over a long enough time frame that I can reasonably use their information for this compilation of statistics. Many more came as one-time visitors or attended weekend classes. These short-term patients as well as those who did not stay in the clinic for more than a few months were helpful, and often demonstrated important principles or drug-related symptoms, but their drug dosage changes would not be meaningful for this list.[1] The project started in February of 1998. This chapter is being written in March of 2003.

Completely stopped taking medication: 18

This number includes patients who were not taking much or had only taken drugs briefly when they joined us, but it also includes Hjalmar, Chris, and Lance, who had used drugs for decades before starting with us. This number includes those, like Earle, who are doing well without the drugs while gaining in strength, and Hjalmar, who is still very weak and sleeping much of the day.

Stopped all medication and then started up again at very low levels: 4

This includes Becky, Coach, Sammy, and Taylor Paul. The Taylor Paul update: he is down to 100 mg/day and doing so well that when his doctor saw him at the store, the doc asked if he had increased his medication again, even going above the 500 mg/day that had been prescribed during Taylor Paul's hospital stay. When Taylor Paul told him

[1] We were also fortunate enough to have 36 unmedicated patients in our program.

that he had in fact decreased significantly, and was feeling stronger every day, the doctor said he couldn't understand why Taylor Paul was so opposed to the medication.

Began an abrupt increase in medication: 5

People in this group have increased medication despite (or because of) doctor's orders, and are considered by family or friends to be completely illogical and grossly overmedicated. This group includes Zoe, Birdie, Angus, Brad, and Euclid. All members of this group began to experience distinct symptoms of recovery before they flipped out. Angus, Brad, and Euclid had all reduced their medication down to a "sub-therapeutic" dose and were doing extremely well with the reductions. They had been confident that they were recovering when they abruptly went into drug mania, and they remain there at the present time.

There are many people that I have learned of over the Internet or have met in classes who would fit into this category. This is the most frightening group, in my opinion. The fact that over 12% of our medicated patients fell into this category should be alarming to anyone who is taking medications and considering embarking in a PD recovery program. Four of the five were specifically warned about the possibility of abrupt addiction after we had seen what happened to Zoe – all four were confident that they knew what they were doing and that it could not possibly happen to them.

Increased their dosage levels: 6

This number includes Olli, Stephanie, Rudyard, and Lila – though there is some debate in the PD team that both Lila and Rudyard should be included in the abrupt increase/illogical group above. However, both Lila and Rudyard still seem able to somewhat understand that the medication may be responsible for their worsening side effects, and so they are included in this group. Included in this group is a patient who got a deep-brain stimulator (battery operated wire implant). He was unable to sleep after getting the implant. After more than fifteen harrowing months without more than twenty minutes sleep every 24 hours, he started taking Sinemet. The levodopa helps calm him down enough so that he can now sleep. Like most implant patients, he was taking Sinemet prior to the implant and continued to take it after the implant, though at a slightly reduced level. After a year of FSR, he no longer needed any Sinemet – his movement was normal. However, the sleep problem was killing him. Although the levodopa did not help him sleep prior to his FSR treatments and recovery, after he regained flexibility and feeling in his legs and had the implant, the levodopa paradoxically acted as a sleep aid.[1]

Decreased their medication by up to 50%: 7

This group includes Nat, Moses, and Maurice.

Decreased their medication by more than 50%: 4

No members of this group were discussed by name in the preceding text.

[1] Other patients who suffer from rigidity find that L-dopa can help them sleep. Insomnia can be a symptom of overmedication and can occur after a crash. Therefore, many overmedicated patients must take L-dopa, a stimulant, to get to sleep after crashing.

Decreased their medication by more than 66%: 8

This group includes Rose, Sonny, Hua To, Yves, and Laurel.

Stayed the same: 5

These people, for the most part, were among our first patients. All of them dropped out of the program within less than a year. They were taking large doses of medication, and as their treatments proceeded, their dyskinesias grew worse. They all assumed that the worsening of dyskinesia and other drug adverse effects were simply a worsening of their Parkinson's disease and concluded that the program was not working.

Lost touch: 5

These were people that we met with or worked with for a considerable time but who moved away. Although they contributed to our general knowledge about the medication, we do not know what they have done with their medications since they left.

More information about the patients

Of the people in the groupings above, five have passed away: Rose, Birdie, Honoria, Eli (described in Appendix 2), and Francis. Eli passed on following a massive stroke, and Francis, not previously described in this book, died of pancreatic cancer.[1]

Four of the group had brain implants: Rufino had decreased his medication from 1000 mg/day to 50 mg/day of levodopa prior to this implant surgery in early 2002, and he has now been completely off antiparkinson's drugs for more than a year. His doctors say that he has responded better to the implants than any of their other patients. They have called him their "implant poster boy." It may be that the reason is this: he is their only patient that has not had to keep increasing his medication, adding drug side effects to the inevitable implant side effects, and he is also their only patient who no longer has Parkinson's disease. When his case was discussed at a recent convention of implant doctors, the docs refused to believe the statement by Rufino's doctor that he was not taking medication. A curious response! Another implant patient, as described earlier, is tortured by an extreme form of insomnia.

The third implant patient moved out of the area shortly after her surgery. She was in the first group of twelve patients, before we knew anything about the medication. She had been reducing her medication and was making remarkable progress in recovering from Parkinson's when her name came up on the implant wait list. She was told that she

[1] Although there is not enough room for every case study in this book, I would like to slip in one more. Restored mental function is one of the most appreciated, and often unexpected, results of getting off the medication. Ten days before Honoria passed on, at age 87, her family took her off all medication. Her liver was failing, and the nurse suggested that the anti-PD drugs were hard on the liver. Her family was stunned to see that, within 24 hours of getting off all the anti-PD drugs, she was mentally focused and keenly, incisively intelligent for the first time since she had started taking bromocryptine at the time of her diagnosis ten years earlier. Four years prior to her passing, her daily drug regimen included Sinemet (three 25/100), Bromocriptine, and Mirapex (2 mg/day). She had been in our program nearly three years and had made great improvements in mobility and decreased her medication. At her last visit with Dr. Rafferty before her passing, he asked her what had happened to her cane; she'd been dependent on a cane for years, ever since she had become nearly house bound with poor balance. She replied honestly, with a twinkle in her eye, "I left it in Reno at one of the casinos."

had to decide – if she didn't have the surgery that month, she would never be put on another wait list.

Despite her conviction that she was recovering, her husband felt she should have the surgery – thus covering all the bases. She had the implant surgery. In the days immediately following her surgery, she noticed that she could move well with no medication. When she started with us, she had been taking five pills a day and had gotten down to where she felt very comfortable with two pills a day, just prior to the implant. Within a week of the implant surgery, she was again taking five pills a day. I asked her why she had resumed taking five pills a day since she insisted she no longer needed them. She replied, "I don't need them to move anymore, but they make me feel so good and they ease my back pain." A month later, they moved away.

The fourth is a long, sad story. When this man first got the implants, he was able to reduce his medication by half, down to 300 mg/day levodopa, as well as decrease his Mirapex. After a year with the implants, he was back at 600 mg/day and full Mirapex. He was having dyskinesia from the medication, and even so, was rapidly losing motor function.

At this point he started in our program. As he started to respond to the FSR, the implant caused increasing internal agitation and feelings of burning static in his skin. He started turning the implants off at times, which incurred the wrath of his doctor. Then, after feeling the return of sensation in his feet and legs after several months in our program, he started a rapid decrease in his medication. This incurred the wrath of the people he was living with. Unfortunately, due to the rapid improvements that he made, his medication became more effective and the side effects much worse. The side effects of the implants also worsened. He was forbidden to work with me ever again. It was a long, drawn-out saga, but he is in much more pain now than he might have been had he not started with our program. We no longer accept patients with brain implants into the recovery project.

This concludes the numbers and statistics on the project.

The Second Question

In closing – and I bet you thought I'd never get to this! – the other frequently asked question is: "Are the antiparkinson's drugs good or bad?"

That is a foolish question. The drugs, used judiciously, can provide tremendous benefit. The drugs, used injudiciously, can cause problems much, much worse than mere Parkinson's disease. Some of the most tragic cases I have heard from were people diagnosed with Parkinson's on the basis of a mild tremor or bothersome draggy foot and immediately placed on very high levels of medication. Within six months, some of these people started suffering the excruciating dystonias and On-Offs that are associated with overmedication. They have then been instructed to further increase their drugs to treat their "worsening" Parkinson's.

On the other hand, the medication, and not our program, has been the best choice for certain patients. Let me give an example.

Lemual

A local woman called me to make an appointment for her husband – he was only 75, and he had just been diagnosed with Parkinson's. I asked her to read our *Patient's Handbook*, available for free on the Internet, and call me back. She called the next day, said she had read the whole thing, and I made the appointment for the following month.

On the day of the appointment, her husband slowly shuffled into my office with an expressionless face and extremely delayed vocal and motor response. He was hunched over, his arms bent at the elbow in the classic PD position. He couldn't move his head to the left or right. He had a very mild tremor. He came through the doorway with difficulty. He crossed into my office with difficulty, and, appearing deeply confused, sat down on the chair very slowly.

I asked him how long he had been moving slowly. His wife answered this and all my following questions. She laughingly said that he had been moving "funny" for over fifteen years. She laughed as she described the "funny" way he used his arms while eating, and how she'd been making fun of him eating that way since before he retired, twenty years ago. He had quit golfing fifteen years ago because he couldn't move right any more. But he had been fine; he never had Parkinson's until a month ago.

After an hour, I finally extracted from her, between gales of laughter, that he had not tremored nonstop until a month ago. Prior to that he had been increasingly hunched over, rigid, slow, and silent for more than fifteen years. However, because our website made the point that recently diagnosed patients seemed to respond the best, the wife kept repeating that he had only been diagnosed a month ago and was therefore a good prospect.

Finally, he spoke – slowly and softly – but with determination: "I am seventy six. I have lived a good life. In my family, we all die by age seventy. My parents died just before they turned seventy. My brothers and sisters all died when they turned seventy or seventy-one. I'm ready to go. I've had a good life. There's nothing left that I want to do. I am ready to go."

His wife, embarrassed, started chattering about how he had been diagnosed only recently, and asked how many weeks I thought it would take for him to recover.

This man was in a condition of advanced Parkinson's disease, but more importantly, he did not want to get better. He was ready to move on. Whether it was because he wanted to shed his unresponsive body or his overly responsive wife, I couldn't begin to guess, but he was in his right mind, and he clearly had no interest in our program. This was going to be awkward.

I had an idea, and crossed my fingers for luck. I turned to his wife and said, "OK. Let's start making sure we've got everything ready for when he recovers. Have you picked a care facility yet?"

I will try to recreate the conversation to the best of my ability:

"What do you mean? If he's going to get better, why would he need a care facility?"

"During the recovery stage when he grows very weak, he may need help being moved to and from the bathroom, help getting dressed, help with feeding. You are no longer young yourself, and I don't want to see you trying to carry him to and from the car during the months when he is recovering."

"What on earth are you talking about?"

"Didn't you read the *Patient's Handbook*? It describes very clearly the stage of recovery when a person is extremely weak and may need help even to move."

"Well, I told *him* to read it – I didn't have time. He forgot to read that part! Lemual! Did you read that part! Why didn't you tell me!" Lemual just sat there, unblinking. She turned to me and said, in conspiratorial honesty, "He doesn't always remember everything he reads."

"Maybe you need to go home and read the book. This program can be very grueling. Also, Lemual has advanced Parkinson's disease."

"That's where you are wrong. He was only diagnosed in the last month."

"That's because you never took him to the doctor for a diagnosis. He has not been moving normally for over fifteen years – the funny way he moved, that made you laugh so hard, that was Parkinson's disease. The tremor started late in him – but he had Parkinson's disease right along. His condition is very advanced."

"Well for goodness sakes! Then what do you think we should do? We can't afford a care facility!"

I told her that she should see another neurologist and I recommended Dr. Rafferty. I warned her that, since he might recommend medications for Lemual, she must know that the medications for Parkinson's disease can take a long time before the effect is obvious. It might be three months after Lemual started the medication before there was any visible change.

I asked her to let Dr. Rafferty know that she and Lemual didn't want a quick result – they wanted a safe one.

I suggested that Lemual might find that the drugs slowly, over several months, might gradually allow him to move better, and that, if used judiciously, the drugs might help him move fairly well for the rest of his life. I also warned her that if the drugs were increased too quickly, hoping thereby to restore him quickly to a level of movement that was unrealistic, these drugs might backfire and worsen the Parkinson's.

I asked her to read the warnings on the drug insert carefully and keep an eye on Lemual for adverse effects, and tell Dr. Rafferty about them right away. I explained that there are many different drugs available; each one has slightly different side effects. I did point out that if Dr. Rafferty suggested changes in the drug amounts over the months or years, to keep in mind that people in our project found that increasing or decreasing them slowly, over months, seemed easier on the body than increasing or decreasing them quickly.

I added that Lemual seemed a little depressed and offered that the medication might help him find more joy in life – but only if the drugs were used correctly.

"Dr Rafferty appreciates the risks and will be willing to work with you to make sure that Lemual gets the best possible results from the drugs. Be careful with the medications – using them is a double-edged sword, but they can also be a Godsend. Let me know how it turns out."

I helped Lemual to his feet, and he slowly thanked me. As I helped him to the door, he paused and said again, "I've had a good life. I'm not depressed, and I'm not afraid. I'm ready to go." This time, his wife didn't say anything, and they walked out into the sunshine, out to their car.

One of the reasons I had recommended Dr. Rafferty was that Mark and Margaret, whose case study is in Appendix 2, had the deepest admiration for him. He was the oldest neurologist in town, as I have stated. He had worked with Hjalmar and many of my long-time PD patients. When Hjalmar had gotten off his drugs, Dr. Rafferty had been sad to see him using a walker, pleased to learn that he had recovered his mind, but, most of all, he had respected his decision.

When Mark started our program, at age 68, he had been taking Sinemet and Artane since Dr. Rafferty had diagnosed him eight years earlier. After Mark got off his drugs, his next visit with Dr. Rafferty showed that the good doctor had been doing some thinking on the subject. By this time, several other patients of his from our project had also reduced or gotten off their medication.

"You know," started Dr. Rafferty, after they broke the "no drugs" news to him, "we used to think that the drugs were the answer. And now, it seems that the drugs were, as often as not, a part of the problem. We were wrong. I was wrong."

Dr. R. listened attentively while Mark and Margaret told him about the harrowing six months of drug reduction and the reemergence of Mark's wit and personality after the drugs were gone. They told him that the dyskinesias – for which Dr. Rafferty had tried adding agonists, digestion inhibitors, and increases in Sinemet – had gone away when the levodopa had gotten down below 100 mg/day. They told Dr. Rafferty how excited they were that Mark, for the first time in years, could get in and out of a car by himself. Even more important, according to Margaret, was that Mark was his old self again. She used the same words as Sophia had used: "I've got my husband back."

Dr. Rafferty shook his head and said again, "I was wrong about the drugs."

For the next six months, at every weekly meeting with Mark and Margaret, Margaret made the point to me anew: "I admire Dr. Rafferty so much. It must be so hard to say what he said. He told us he had been wrong. I have never heard a doctor say that before. I admire him so much!"

So, in answer to the question, "are these drugs bad or good?" I can only answer, "That depends."

Finally, in closing, I would like to dedicate this chapter to you doctors, especially you neurologists, who have lived through these rapidly changing times and have kept your minds and hearts open. Though I am approaching the subject of medicine from the perspective of electricity, and you are approaching from the perspective of chemistry, we are all striving for the patients' greatest good. We drink from the same cup.

APPENDICES

Appendix 1

LISTS OF DOPAMINE-ENHANCING DRUGS

This appendix has two lists. The first lists drugs most frequently used in treating Parkinson's disease as of 2003. The second list is a list of other drugs that also enhance dopamine. We do not accept into our program anyone taking drugs from either list.

List I: The PD drugs and their various names

Explanation of List I

Most drugs have several names: the name of the chemical itself and the drug's various nicknames. These nicknames are usually trademarked names, owned by the company that makes the drug. After a drug's patent has expired, any manufacturer can make the drug and sell it under its chemical name, or the new manufacturer can create a new nickname.

Sometimes the various names designate the form of the drug. For example, Deprenyl is L-deprenyl hydrochloride in liquid form, and Eldepryl is a trademarked name of the same chemical placed in a pill form. The drug mechanism and side effects are still the same, no matter what name is used. However, sometimes the matrix (liquid or pill format and inert fillers) may yield a superficial difference in effect or speed of effect.

In another example, in the case of Sinemet, the company currently making a generic form of the Sinemet CR (controlled release) can use a slightly different mechanism for the slow release mechanism, but both the original manufacturer and the manufacturer of the generic version are using the same *active* ingredients: a blend of levodopa and carbidopa – only the matrix that delivers the slowed release of the payload is different between the trademarked and the generic. In the text of this book and in the following appendices, the antiparkinson's drugs are referred to by the name in the right-hand column below. Other names by which these same drugs are known are listed in the left-hand column.

Drug name	**Name used in this book**
Amantadine	Amantadine
Amantadine hydrochloride	Amantadine
Apo-Trihex	Artane
Artane	Artane
Artane-Sequels	Artane
Atapryl	Eldepryl
Atenolol	Atenolol
Bromocriptine	Bromocriptine
Bromocriptine mesylate	Bromocriptine
Cabergoline (Europe)	Cabergoline
Carbex	Eldepryl

Comtan	Comtan
Deprenyl	Eldepryl
Eldepryl	Eldepryl
Entacapone	Comtan
L-deprenyl hydrochloride	Eldepryl
Levodopa-carbidopa	Sinemet
Madopar (Europe)	Sinemet (equivalant)
Mirapex	Mirapex
Mirtazapine	Mirtazapine
Parlodel	Bromocriptine
Pergolide	Permax
Pergolide mesylate	Permax
Permax	Permax
Pramipexole hydrochloride	Mirapex
Requip	Requip
Remeron	Mirtazapine
Ropinirole	Requip
Ropinirole hydrochloride	Requip
Selegeline	Eldepryl
Selegeline hydrochloride	Eldepryl
Selpak	Eldepryl
Sinemet	Sinemet
Sinemet CR	Sinemet
Symmetrel	Amantadine
Tasmar	Tasmar
Tolcapone	Tasmar
Trihexane	Artane
Trihexy-2 or 5	Artane

List II: Wellbutrin, Valium, Prozac, Xanax, and other dopamine-enhancing drugs

Explanation of list II

Because of the extreme shift in susceptibility to addiction that occurs during recovery from Parkinson's, we no longer accept into our program anyone who is using dopamine-enhancing drugs. While these drugs may or may not be safe for the general population (a widely controversial subject), and may not be very addictive to a person with Parkinson's, they all have the potential to become hideously addictive to a person who is recovering from Parkinson's disease. Therefore, we will not accept into our program any person who is currently taking any of the following drugs, or who, during the preceding six months, has taken them more than fourteen times (two weeks' worth).

The majority of these dopamine-enhancing drugs are antidepressant, antianxiety, pain-killer, or antispasmodic drugs. New variations of these drugs are coming out every year. It would be impossible to list them all by name.

If you are taking a drug that is not on this list and wish to determine whether or not a drug may be a dopamine-enhancing drug, learn the mechanism of the drug. Either read the insert provided with the drug and available on the drug's website or talk with your pharmacist to determine if the drug is a tricyclic, an SSRI, a GABA enhancer (including benzodiazepines), or a drug that in any way enhances, sustains, provides, or elevates levels of serotonin, norepinephrine, or GABA. If the drug does any of these functions, it is also, indirectly, enhancing dopamine and should be considered an addictive drug, and many of the issues discussed in this book will apply.[1]

Not all drug labeling is honest regarding addiction. Words like "accommodate" or "toleration" are tip offs that a drug is addictive. Drug manufacturers know that the label "addictive" is bad for business. Therefore, they will go to great costs, even flat out lies, in their advertising. For example, the manufacturers of the drug Xanax, a highly addictive medication, run ads in publications for the general public touting it as a safe alternative to addictive medications, specifically noting that it is a *non-addictive* drug. The physician's instructions for this drug, however, have long warned that this drug is dangerously addictive. The advertisers get around this by noting in the small print that this drug is not addictive *when* used as directed. The actual directions for this drug state that the drug should not be taken for more than two weeks.

---

[1] In Dec, 2003, researchers published a report that dl-methylphenidate (MPH), the most commonly prescribed drug used in the treatment of attention deficit/hyperactivity disorder (ADHD), a drug that ostensibly increased only norepinephrine, is actually dopaminergic: "Evidence of a dopaminergic basis both for the actions of MPH and for the underlying neuropathy in ADHD continues to mount." (Quote from "Advances in the Pharmacotherapy of Attention-Deficit-Hyperactivity Disorder; Focus on Methylphenidate Formulations," *Pharmacotherapy*, J Markowitz, Pharm D., A. Straughn, Pharm D. K, Patrick, PhD, 10-23-03.

It is increasingly understood that all psychoactive drugs alter more than just one, officially-targeted neurotransmitter. The naïve simplicity of the mid 1980's that held that mind- and mood-altering drugs could affect one neurotransmitter without affecting the others, particularly dopamine, is hopelessly passé. This antiquated view is adhered to now only by those clinical MDs who are very behind in their continuing education classes.

Most doctors do not read the fine print. This drug is usually used to treat long-term anxiety and insomnia, and is prescribed casually, in the manner of, "Fill this prescription and take the pills whenever you are feeling stressed." This means that this drug is nearly always offered for usage of more than two weeks – which constitutes long-term treatment, which is known to be dangerously addictive.

The manufacturers know that most doctors prescribe these drugs for the long term. But rather than emphasize more strongly the two week warning, the opposite is the case; the wording about addictive drugs in the manufacturer's warning to physicians has changed through the years to make the drugs appear as innocuous as possible. All quotes below are from various years of the *Physician's Drug Handbook* with regards to Xanax.[1] In 1992 the warning read, "Use cautiously in individuals prone to addiction or drug abuse" (p. 34). This caution has since been completely removed from the literature! In the 1997 edition, the warning included the phrase "Be sure patient understands potential for physical and psychological dependence with chronic use…" (p. 26). In this case, "chronic" means "daily, or regular." The implication is that this drug is only meant to be taken for very short-term relief from anxiety. In 2002, the warning had again been changed to appear even more general: "Be sure patient understands potential for physical and psychological dependence with *long-term* use" (p. 24). The fine print does not explain that "long-term" means anything more than two weeks. Most patients never originally intend to use a mood-altering drug for the long term, and even many physicians do not suspect that, physiologically speaking, "long-term" can refer to anything more than one to two weeks.

The following list is not complete. New drugs are always being added to the roster, and some of the older ones have doubtless gotten past me. I receive a steady stream of emails asking about the safety of this or that new drug. I simply cannot keep up with all the new drugs. You must be your own researcher; here is a guideline: if a drug creates a general feeling of well-being or helps with sleep, it is probably addictive. This general rule applies to the nicotine, alcohol, methamphetamine, cocaine, and morphine groups, as well as the SSRIs, tricyclics, GABA enhancers, and according to the latest reports, the drugs that are used to treat attention deficit disorder. The only "feel good" drugs that do not belong in the addictive group are some of those drugs that only provide hallucinations but do not alter brain chemistry in the pleasure centers. This latter group includes mescaline and LSD.

I have only placed legal, pharmaceutically approved drugs on the list below. Illegal drugs such as ecstasy, which is a variant of methamphetamine, are not included in this list, but they *are* dopamine-enhancing and must be recognized as addictive. Illegal drugs that alter mood are usually addictive because they elevate dopamine levels, and therefore, the theories in this book regarding dopamine-enhancing medications do apply to them.

[1] I chose Xanax rather randomly, maybe because Becky had used it, and maybe because the flagrant ads touting it as non-addictive are so widely distributed. I could have chosen many other drugs to make the same case; nearly all the drugmakers in this group, especially of the antianxiety and anti-depressant drugs, fit the same pattern of downplaying the risks of the drugs, if not being downright deceptive.

List II

Alprazolam
Ambien
Amitriptyline
Amoxapine
Ativan
Aventyl
Bupropion
BuSpar
Celexa
Citalopram
Darvocet
Desipramine
Diazepam
Doxipin
Clonazepam
Clorazepate
Codeine
Effexor
Elavil
Estazolam
Fluoxetine
Flurazepam
Fluvoxamine
Halcion
Hydrocodone
Imipramine
Impril
Klonopin
Methylphenidate hydrochloride
Morphine
Neurontin
Librium
Lorazepam
Lortab
Luvox
Nortriptyline
Norpramin
Oxazepam
Oxycodone
Paroxetine
Paxil
Percocet
Percodan

Appendix 1

ProSom
Prozac
Restoril
Ritalin
Rivotril
Serax
Sertraline
Sinequan
Surmontil
Tofranil
Triadapin
Triazolam
Trimipramine
Valium
Venlafaxine hydrochloride
Vicodin
Welbutrin
Xanax
Zoloft
Zolpidem
Zyban

Appendix 2

SPECIFICS OF INDIVIDUAL DRUGS

General information

Presentation

The list in the first appendix tells the most common name of the antiparkinson's drugs: the individual drugs are presented in this appendix by those most commonly used names.

This appendix has a short (one- or several-page) section for each of the drugs. The material was culled from the various drug inserts, drug manufacturer's advisory web sites, the *Physician's Drug Handbook* (Springhouse, Springhouse PA 2002), and *A Primer of Drug Action* (Robert Julien, MD, PhD, Henry Holt and Company, New York, NY, 2001), as well as from our patients' experiences.

Each section starts with general information about the drug, anecdotal observations from users of the drug, and then shares the official information provided by the manufacturer regarding how the drug is purported to work, the suggested dosing, and the format (tablet or capsule, and amounts) in which the drug is available. Some of the drug sections have short, demonstrative case studies stuck on at the end. Most of the text of this book applies to dopamine-enhancing drugs in general; the drug-specific information is contained in this appendix. There are interesting side notes included in the drug-specific information that may actually be of general interest. If you have the time or inclination, you may want to read about all of the drugs rather than just the one or two that you are taking or considering. By looking at all of the drug methods being used one gets a fuller understanding of the pharmaceutical approaches being used in the western battle against Parkinson's.

The organization of this section is somewhat alphabetical by drug name or group. In the cases of the several agonist and digestion inhibiting drugs, the drugs are grouped together by category.

The listing order is as follows:

Agonists
- Permax
- Bromocriptine
- Mirapex
- Requip
- Cabergoline

Amantadine
Artane
Atenolol
Digestion inhibitors
- Comtan
- Tasmar

Eldepryl

Mirtazapine
Sinemet (regular and CR)

Drugs not included

People with Parkinson's are often taking other drugs as well, including antidepressants, blood pressure medications, heart drugs, and anti-osteoporosis drugs. Most of these drugs have powerful effects on the body and mind. When combined with antiparkinson's drugs, the effects can be amplified or negated.

Do not expect your doctor to know about all the possible interactions. You will be lucky if he has had the time to study the side effects of the drugs he is giving you, let alone the drugs prescribed by your other doctors. As for the effects of the drugs in combination, most of them are not known unless they are so dangerous that they have caused life-threatening situations. It is likely that moderate problems caused by your drug combinations have never even been reported, or, if reported, they have been lost in the great tide of random information.

Your pharmacist, on the other hand, may be extremely helpful, having more time to work with you and greater depth of information than your doctor. MDs spend a small amount of time learning which drug to use for what ailment. Once they are out of school, they may or may not keep up with the changes. Pharmacists spend almost all their time learning about drugs and must keep up with the new drugs – they are filling orders for them.

If you are taking a drug that is not listed in this section, please find out as much as possible about your drug. Learn how it works. Once you know how it works you might (or might not) be able to anticipate how it might exacerbate or decrease the side effects of your other drugs.

About side effects and overdose information

A partial listing of side effects (adverse effects) of each drug is included in this appendix. Adverse effects should be thoroughly studied by anyone taking that particular drug: they provide a good picture of just what the drug is doing to the body in addition to providing the desired benefit. Please bear in mind that many adverse effects of drugs do not stop after the drug is discontinued; the adverse effect may well become a permanent alteration to one's physiology.

Please study the symptoms of overdose as well. A safe amount for one person may well be an overdose for another. The word "overdose" does *not* mean "took more pills than prescribed." An overdose is what happens to your body if the drug is too strong for *you* or you are taking more than agrees with/is safe for *you*. With this latter meaning we can better interpret a manufacturer's warnings such as "dyskinesia is a symptom of levodopa overdose." The meaning is this: if dyskinesias occur, you are taking too much of the drug, whether or not your doctor prescribed that amount, and whether or not it is the officially recommended dose. If you are having symptoms that correspond to symptoms of overdose, you are in trouble, and you need to work with your doctor to get off the drugs or get your drugs down to a level where the symptoms of overdose are no longer occurring.

Drugs can create unusual or rare side effects that are not listed in the drug inserts or in this book. Everyone will react a little differently to drugs. That's not surprising –

people react differently to mild foods, so it is no wonder that they all react slightly differently to powerful drugs.

Milk is a good example of a food that is presumed safe. Some people cannot digest milk products. Those who cannot digest milk have a variety of symptoms, or side effects, from drinking milk. These adverse effects can include but are not limited to headache, digestive pain, diarrhea, chest pains, asthma, skin rash, and several dozen other reactions. Some of these reactions can be so severe that they require medication or oxygen support. Even so, we do not put warning signs on milk cartons.

In the same way, many drugs can have dangerous or unexpected side effects in individuals. Some patients notice a particular new symptom after taking a drug and refuse to credit their own suspicion that the problem is drug related, simply because the problematic symptom is not included in the list of official adverse effects. Such a patient, finding his symptom is not included in the list of adverse effects, may decide that he is only imagining the problem, or that it is not drug related. This patient may be wrong. If a drug sets in motion a deleterious symptom in an individual, it does not matter one iota if that same symptom does not appear in most other people using the drug.

"If it's for sale, it must be safe" = not true

Let's consider a familiar example: most people can eat peanuts; a peanut-sensitive person can die if he eats a single peanut. Even so, peanuts are not sold with warning inserts. Drugs are nowhere near as well-studied and tested as foods. How drugs work is often not understood. Most people want to believe that the mechanisms of these drugs are understood and that drugs must be proven safe in order to be sold. This is not true. In order to be approved by the US Food and Drug administration (FDA), drugs must not kill the small group of healthy young test people during the short period during which the drugs are being tested. Even drugs that are proven to be dangerous may be approved by the FDA if the risks of the drugs are weighed against the benefits and found to be slightly beneficial. Tasmar is an antiparkinson's drug is this category. It is so dangerous that it is illegal in Canada and the doctor's instructions suggest that it only be used as a last resort, and the patient should sign a note saying he has been warned. However, on the off chance that this drug will be beneficial when no other drugs help, the patient may choose to take this drug despite the liver damage that it causes. I have had many patients who have been prescribed this drug and their doctor had no idea whatsoever that it was considered a high-risk drug of last resort. So the patient must be the one to study up.

Remember, all drugs carry risks. Even drugs that are dangerous can be approved for use.

So study the side effects, learn the signs of overdose and be familiar with the drug warnings. These warnings exist because drugs, by their very nature, are powerful, body-altering substances. Some drugs can improve some aspects of quality of life. Others can be life-preserving. All of them have risks and side effects. Judicious use of drugs requires balancing the benefits against the necessary damage that they might do.

The drug insert

Although the information in this appendix is as up-to-date as possible in 2003, new information comes up all the time. The manufacturers are required to put the new findings and changes into the drug inserts. Please learn to read the drug insert. At least

once a year reread the insert on your drugs to see if anything has changed, or if new warnings have been added. Every doctor should review the drug inserts once a year for every drug that he prescribes. I do not know any that actually do. Even though the insert warnings grow and change constantly and established drugs regularly make headlines when they are discovered to have been too unsafe, I know of doctors who state, "I've been using this drug on several patients for years now – so it must be safe." You cannot assume that your doctor has kept up-to-date or that he understands that not all people are alike. After all, a major premise of modern medicine is that people *are* alike. People's bodies are supposed to work like machines. Differences between machines are flaws according to some doctors, and not manifestations of the individuality of every soul.

Marketing brochures

The glossy sales brochures that accompany some drugs are NOT the drug inserts. The drug insert is usually printed in extremely small letters on a very thin sheet of delicate paper. It may feature a drawing or two of an indecipherable chemical diagram, a chart or two of encoded test results, and is written in technical language. The drug insert is not user-friendly. It looks like something that you are supposed to throw away without reading. In general, if the material given to you by your doctor has photos of smiling people playing the harmonica or enjoying a lemonade with a loved one, that is NOT the drug insert – that is advertising. The sugarcoated advertising version of the drug risks or side effects is what the drug company wants you to know. This reassuring handout may list one or two side effects; it will not tell you the whole story. Even the insert, though going into the test results more deeply, may couch the testing results in the most positive light, even to the point of being misleading.

For example, even if the drug was tested on 1000 people, the drug insert may only show the results of a secondary test that was run on 20 people, in order to minimize the numbers of people with problems. The thinking here is that the patient, and even the doctor, will be less alarmed if the insert says only one person out of 20 died due to the drug than if it stated that 50 out of a thousand died, although the percent is exactly the same.

Unless you note how many people were in the study, you may be deceived into thinking the side effects are less frequent than they really are. It might not occur to you from a casual glance at the statistics on the insert that the study quoted on the insert only reviewed the results of 20 people, all of whom were healthy 20-year-old college students, and not the results of the larger testing that was done prior to final approval. If you read the fine print, however, and see that, although 1000 people were tested, the insert only includes a chart of results based on a group of 20 people, and even in that smallest possible sample, one person died from the drugs, you may be more concerned. So, read the insert, and be smart.

Some companies no longer provide inserts with the drugs – you must ask your pharmacist for a copy of the drug insert information. If you get your drugs by mail, the provider should be able to get you a copy of the drug insert.

Drug insert translations into English

Here are explanations for some terms that might show up on the drug insert:

P.O.: drug is taken by mouth (as opposed to shots, rubbed on the skin, or other method).

b.i.d.: twice a day

t.i.d.: three times a day

q.i.d.: four times a day

q: every

CNS: central nervous system (brain, spinal nerves)

CV: cardiovascular system – pertaining to the heart and blood vessels

EENT: eyes, ears, nose, and throat

GI: gastrointestinal: pertaining to the mouth, stomach, intestines, digestion

GU: kidneys and urinary tract

Hematologic: pertaining to the blood cells

Hepatic: pertaining to liver

Renal: pertaining to kidney

Pulmonary: pertaining to lungs

Contraindication: situations in which this drug should not ever be used.

Interaction: what happens if you combine this particular drug with other drugs or substances.

AGONISTS

PERMAX

Permax is also commonly known as **Pergolide**.

Permax is a dopamine agonist. A dopamine agonist is a dopamine substitute that can imitate dopamine in so far as it stimulates some nerve receptors that ordinarily are only stimulated by dopamine. Permax stimulates D1- and D2-type dopamine receptors. Permax is derived from ergot, a fungus of rye. In the middle ages, the bright eyes and dyskinesias precipitated by eating moldy rye were called St. Vitus's dance, and were considered to be a form of divine ecstasy or witchcraft, depending on one's persuasion.

Permax compared to other agonists

Permax, like all agonists, has many side effects. (Despite the similarities of the agonist side effects, each of the agonists is slightly different and affects individuals differently. Some people do well with one agonist and do not do well with another.) While dyskinesia is recognized as a symptom of dopamine excess, people once thought the ergot from which Permax is derived caused the extreme forms of mental confusion and gastrointestinal pain that can be set in motion by the use of the drug. It was hoped that the new synthetic agonists that contain no ergot would have fewer side effects. However, the newer agonists also have gastrointestinal and hallucination side effects, plus other problems. It may be that the side effects are in fact not caused by the unavoidable ergot proteins but by the dopamine-enhancing properties.

In our experience, the chief difference between the agonists is the type of dopamine receptor that is stimulated. Permax stimulates D1 and D2 dopamine receptors. With Permax, compared to the other agonists, the spine and the low back in particular seem to receive more than their share of the dopamine stimulation.

Permax and back pain

We suspect that D1 and/or D2 receptors may play a role in stimulating the muscles of the low back and abdomen. Several patients have noticed their low back muscles become extremely weak during reduction of Permax and their abdominal pain and spasms decrease. The low back weakness is so extreme that the back may become severely bent, with the head being down between the knees. One patient had to shower by bracing the top of his head against the shower wall and his buttocks against the opposite wall. Another simply walked around with his head facing his knees for several months. This condition can last for more than half a year. Although a few unmedicated PDers have manifested this pattern during recovery, we have seen a disproportionate amount of this severe back weakness in our Permax-reducing patients. It is especially interesting because Mirapex-reducing patients seem to have more problems with their neck muscles becoming limp, so that their heads bob limply on the necks following a reduction. Considering that Permax patients have more back pain and tightness in the back and chest, and Mirapex patients tend to have more problems with severe tension in the neck and low blood pressure (possibly related to the blood pressure-regulating sinus in the neck), it may turn out that the Mirapex-stimulating D2/D3 receptors are directed more to the neck muscles and D1/D2 receptors to the low back and genitals. Of course, both drugs can cause low blood pressure and excess muscle tension, but in general, Permax is

more often associated with back pain and abdominal pain than are the other agonists. Then again, Permax has been out longer, and therefore more side effects have been recognized.

Permax reduction

Permax reduction can cause all the usual symptoms of drug reduction. The drug reduction cycle with Permax is slightly different from the cycle with L-dopa. There is less of a vacation, and the reduction cycle lasts longer, sometimes taking five or six months before there is a noticeable lifting of mood and movement. Also, the long-term yearning for the drug seems to be slightly stronger than with levodopa. This is characteristic of all the agonists.

Although the newer, synthetic agonist drugs take longer to build up in the brain, Permax, derived from a plant, is somewhat fast working. Reductions in Permax are usually noticed within a day or two, and subsequent drug reduction symptoms grow in intensity over a period of months and can last for up to half a year. Long-term, lingering desire for Permax may be permanent. As noted above, Permax reduction can lead to a profound relaxation of the lower back muscles. The subsequent supreme bending of the back is not the same as the hunched posture of Parkinson's disease. The classic PD hunch is tight and rigid, and is primarily driven by the rigid stomach channel muscles of the mammary line pulling down on the shoulders.

The heart valve problem

Currently (2003) at least three law firms on the Internet are hoping to cash in on a guess by the Mayo clinic that Permax may have contributed to heart valve disease in the cases of three women who took Permax daily for three to seven years. These firms are advertising for people who have heart trouble and who took Permax, in hopes of making Permax cough up some money. The law firms will, of course, receive some portion of the cash. These firms may be stymied by the recent finding that people with Parkinson's generally have decreased nerve activity to the heart. This decrease in heart nerves, and not necessarily the Permax, may have contributed to the valve problem, in which case, the law firms will not have a big money case after all. However, it may be that dopamine-enhancing drugs do have a deleterious effect on the heart. Most of them can, after all, cause dyskinesia in the heart (heart arrhythmia), which may have an effect on the health of the heart valves. Much research remains to be done.

Manufacturer's recommendations

The starting dose for Permax is .05 mg/day, for two days. After that, the dose can be increased gradually by .1 to .15 mg/day over the next twelve days. After that, increases of up to .25 can be made every three days until "optimum response occurs." The manufacturer does not state what is meant by "optimum response." The average therapeutic daily dose is 3 mg/day. Neither maximum dose nor advice about drug reduction appears in the *Physician's Drug Handbook.*

Format

The pills are available in .05 mg, .25 mg, and 1 mg doses.

Known side effects

Most side effects of Permax are similar to those created by all dopamine-enhancing drugs. The listed adverse effects include: tremor, twitching, dyskinesia, abnormal gait, muscle tension, dystonia, hallucinations, anxiety, personality disorder, psychosis, depression, confusion, insomnia, breathing irregularity, low blood pressure (even to the point of passing out), abnormal dreams, speech disorder, pounding heart beat (palpitations), sinus problems, double vision, dry mouth, distortion of tastes, abdominal pain, nausea, constipation, diarrhea, anorexia, blood in the urine, urinary tract infections, urinary frequency, anemia, pain in the chest, neck or back, and flu-like symptoms. There are other adverse effects as well.

Please study the insert for this drug, or check the Permax website on the Internet to find the latest listing of adverse effects. You will need to go to the page with "full prescribing instructions" and use the magnifying glass – the warnings for this drug are in impossibly small lettering.

The manufacturer warns that low blood pressure, hallucinations, heart palpitations or arrhythmias, or involuntary movements (dyskinesia) are signs of "toxicity." This means that these are *not* acceptable side effects; these are danger signs, signs of overdose. In case that one got past your doctor, who may think that dyskinesia is "normal" for Parkinson's disease, the manufacturer says right out that dyskinesia from Permax indicates toxic levels of the drug.

Ivy

One of our patients, Ivy, had been taking Permax and Eldepryl for several years. Ivy was 53 years old and had been diagnosed with Parkinson's four years earlier. She was very petite but vigorous, had been very healthy, a hiker and practitioner of yoga. She got off the Eldepryl first, and then very slowly weaned herself from the Permax. Ivy's reduction rate was a follows: over nine months, she reduced from 3 mg/day to none. Her first reduction was from 3 mg down to 2.25 mg/day. After three months, she was down to 18.5 mg/week. After that, she decreased by .5 mg/week until she was completely off the drugs. During the decreases, she became increasingly prone to confusion and hypersensitive skin. Within a few weeks of her last dose, she had hallucinations, nightmares, and a feeling that spiders and rats were crawling over her skin. Three months after the final pill was taken, she was still deeply confused, did not want to wear clothes, and would yell at her husband to take her clothes off her when she was not wearing any.

She could not sleep more than two hours at a time until four months after taking her final Permax. Anxiety during this time was almost unbearable; her husband was by her side constantly. Four months after her last Permax, she started sleeping slightly better. Periods after waking from sleep or naps were the worst. She was immobile and felt that she was dying from the extraordinary heat that seared her from the inside. Several hours after waking in the morning, following her pre-noon meditation, she was often able to walk along the beach using full strides and could go up and down the stairs easily, but the burning and terrors would start up again during her sleep and following any wake up.

One of her more interesting side effects during drug reduction, one that we had not seen with other drugs, was extremely severe hot flashes and an acidic feeling and a "severe, excruciating, dry stretching" in her vaginal area. Tests for urinary tract infection and sexually transmitted disease came back negative.

Fourteen months after her last Permax, the excruciating, paralyzing heat events, during which she felt as if she was being burned alive from the inside, and the fear that she would spontaneously combust, and the frantic nightmares that occurred whether awake or asleep had decreased from nearly constant to intermittent. Some days she only had a few hours of the searing pain and debilitating anxiety. Sixteen months later she was able to recognize, with her patient husband's help, that she had less pain on days when she was surrounded by positive people and close friends. After eighteen months she was able to recognize that terror was the underlying emotion during her episodes of immobilizing heat, in which it felt as if her body's normal heat regulating mechanisms went berserk, and overcome the blind fear that saturated her mind every time she had to make a transition from sleep to wakefulness. Despite her awareness of the emotional component and her avoidance of fear triggers, she was still, eighteen months after stopping Permax, very much at the mercy of her drug withdrawal symptoms.

Permax and sex

Curiously, just before she started decreasing her drugs, her husband mentioned that he'd appreciated her medications: they increased her sex drive. We have to wonder if her many years of false sexual stimulation from the drug played a part in creating the agonizing burning and pain in her genitalia after she reduced and then quit Permax. It would seem to make sense. We learned via the Internet that Permax has been discovered by the party-drug, sex-drug crowd. One site on the Internet advertises Permax as a sexual enhancer/libido raiser, and charges $29.00 per pill (!) for members of the website club. I suspect that this is illegal.

It is probably just a matter of time before all the antiparkinson's drugs are being used for partying and sex-enhancement because of their supreme dopamine-enhancing properties. Consider that the illegal, dangerous drug Ecstasy was first used as a dance-hard party drug and was later found to give a short-term, dose-related decrease in Parkinson's symptoms. Now, in the inevitable switch, the anti-PD drugs are being discovered by the get-stoned-let's-party set. The advantage of Permax is that it is legal if obtained with a prescription, unlike cocaine and methamphetamine, two milder but still popular – but illegal – dopamine-based sex-enhancers. Online drug sellers that provide prescriptions over the Internet by request can mail this perfectly legitimate drug to users.

The Internet site that I found during a search for Permax-for-sex did warn "doses of Permax for sexual enhancement are much lower than those for the treatment of Parkinson's disease." This same website also featured articles on: "Nature, our enemy" and "Neuropharmacology – the new route to happiness."

Sex and the low back

In Asian medicine there is a strong connection with the lumbar vertebrae and the energy that is directed to the genitals. Even in western medicine it is now recognized that the nerves to the genitals emerge from the lumbar spine. Considering the influence that Permax seems to exert on the lower back, causing muscle tension and pain especially in the low back, and extreme failure of the back muscles during Permax reduction, it is very possible that the drugs also are producing a surge of energy to the genitals. During drug reduction, the decrease in vitality in the genitals might be the cause of the disordered vaginal nerves/secretions that Ivy has suffered.

It is recognized that bromocriptine, a semisynthetic ergot used as an antiparkinson's dopamine agonist, causes stimulation of the pituitary gland as well as affecting ovarian function and lactation. It can be helpful in restoring menses in some premenopausal women whose menstrual cycles have stopped. This would also point to a relationship between this close cousin of Permax and sexual function.

Ivy, five and a half months later

Five and a half months after taking her last Permax, Ivy started to have moments of less sensitivity in her skin and less confusion. Her children visited her for a week at this time and, though she was drug-free, she felt "On" for the first time since she started her drug reductions. Her mood and movement were perfectly normal, and her sleep was good. She was On the entire week. When they left, she was Off for three days, and then went On again. She may hover between On and Off for years, remaining highly susceptible to changes in mood, weather, and illness.

BROMOCRIPTINE

Bromocriptine is also known as **Parlodel** and **bromocriptine mesylate**.

Bromocriptine is a dopamine agonist. We have only seen three patients taking this drug. Therefore I do not have much to add beyond the material available from the *Physician's Drug Handbook*.

Bromocriptine is a semisynthetic version of the alkaloid derived from ergot, which is the main ingredient of Permax. Most of the information about Permax in the preceding pages also pertains to bromocriptine. The most prominent exception is that bromocriptine does not have warnings about hallucinations.

However, before jumping to any conclusions, Bromocriptine is associated with "mania, delusions, and depression." It may be simply a choice of words that aligns "hallucinations" with Permax and "delusions" with Bromocriptine.

This drug, like most of the antiparkinson's drugs, may take many weeks, even months, to obtain full effect. It is interesting to note that when this drug is used for hormonal imbalance, it may take up to 24 weeks before the effect is evident.

Our very limited experience with Bromocriptine included one patient who added it to her Sinemet regimen when she started having Offs in the evening. (She had been noticing recovery symptoms for several months before her Sinemet became problematic, building up to powerful shaking by the end of the day.) When she added Bromocriptine to the Sinemet, she started having strong shaking throughout her body about an hour after each dose. The evening dose was the worst – an hour after taking it, the shaking was extremely powerful and would last for nearly two hours. She assumed the Parkinson's had taken a dramatic turn for the worse and was thinking of adding a new drug to the mix until it was pointed out to her that her worst symptoms corresponded to one hour after pill time, and that they might be the result of building up of meds during the day.

Manufacturer's recommendations

This drug should be started at a low dose, 1.25 or 2.5 mg/day, with meals. The dose can be increased by 2.5 mg/day for 14 to 28 days, or until a therapeutic response is attained. The dose should not exceed 100 mg/day.

Format

2.5 mg tablets
5 mg capsules

Known side effects

Side effects are mania, delusions, insomnia, seizures, depression, drowsiness, lightheadedness, low blood pressure, nervousness, stroke, acute heart attack, blurred vision, nausea, abdominal cramps, constipation, diarrhea, and urinary retention. We have seen amplification of tremor occur after starting Bromocriptine; however, this is not an officially noted adverse effect.

Warnings

Do not use with alcohol: alertness and coordination may be impaired.

MIRAPEX

Mirapex is also known as **Pramipexole hydrochloride**.

Mirapex is a synthetic dopamine agonist. The researchers had hoped that by building an agonist molecule in the lab, rather than deriving it from ergot, they would be able to sidestep the gastrointestinal and hallucination problems of Permax. However, those problems remain and would appear to be a part of agonist action in general. Strangely enough, Mirapex actually has a few powerful side effects that, for some people, are worse than the problems of Permax. The most problematic of these is narcolepsy (sudden, randomly timed episodes of going from wide awake to deeply asleep, even while engaged in physical and/or mental activity). Many instances of suddenly falling asleep while under the influence of Mirapex were recorded in the first few years after Mirapex was approved. In my own small practice, I've had three patients who have abruptly fallen asleep at inappropriate times, including while driving. One patient fell asleep three times in one week, each time while driving. After stopping the Mirapex, the narcoleptic attacks ceased.

Insomnia, narcolepsy, and turkey dinners

Mirapex and Requip, another agonist, can cause *both* insomnia and narcolepsy, sometimes on the same day, a mere six hours apart. This effect may be due to the agonist's excess stimulation of the stomach. A Thanksgiving dinner example shows how this seeming conflict between insomnia and narcolepsy is actually a normal response to excess stomach stimulation. Many people fall into a stupor after an overlarge meal such as Thanksgiving dinner. The heavy stimulation of the stomach dulls the mind and lowers the eyelids. A short, inadvertent nap following such a meal may be the rude but unavoidable result of overindulgence. However, that night, due to excess food remaining in the stomach, sleep may be poor, with much tossing and turning, if not outright discomfort. Excess stimulation of the stomach causes both conditions: drowsiness and abrupt nodding off, *and* inability to fall into a deep, restful sleep.

The other aspect of excess stomach stimulation, which is intestinal discomfort, can also be a side effect of Mirapex and Requip.

Low blood pressure

Mirapex often causes problems with hypotension, also known as low blood pressure. Orthostatic hypotension (getting briefly dizzy when standing up after having been sitting or lying down, sometimes called a "head rush") is also a problem. Even though PDers tend to have low blood pressure as a side effect of the PD, Mirapex can exacerbate the problem. I have had several patients who tried Mirapex briefly and then stopped, due to the head rush problem. One patient, defying the instructions, started Mirapex with a high dose ("It's such a tiny pill, it can't really do anything. I take L-dopa, and that's a much larger pill…") and fainted twice due to such dizziness. It is to a large extent due to this "head rush" symptom that people must build up slowly to a therapeutic level with this drug. Then again, as noted earlier, my patients who have increased far more slowly than suggested by the manufacturers, spending two to three months at each

dosage level instead of two or three weeks, have found that they get a nice result from the medication at a fraction of the suggested dose – it just takes time.

Neck tension

Mirapex seems to cause an increase in tension in the muscles along the front-sides of the neck (anterior face of the sternocleidomastoids). This tension may contribute to the head rush. A major component of the blood pressure monitoring system (the carotid sinus) is located right under these muscles. It may be that either a direct effect on the muscles of the sinus or an effect on the muscles that lie atop the sinus creates tension and pressure on the sinus, yielding a false signal of excess blood pressure.

The body's appropriate response to an increase in pressure in this area is to dilate the blood vessels and relax the heart rate in an attempt to reduce blood pressure. This purposeful lowering of the blood pressure is possibly a response to a phony "high pressure" alert caused by Mirapex-related muscle tension. The drop in blood pressure then leads to a head rush, and even passing out, when changing from a sitting position to a standing one.

Neck muscles

Patients who have taken Mirapex for a long time find that the neck muscles can be extremely wobbly after stopping the Mirapex. Though this drug is fairly new, I was fortunate to have a patient who had taken Mirapex for six years when I met her in 1999 – she had been in one of the studies that tested Mirapex prior to its approval. When she slowly weaned herself off the Mirapex, her head wobbled so loosely on her neck that she actually stacked her law books up on her desk at work and propped her chin on the top book; she could not support her head any other way. At home she had to stay lying down or support her head with her hands for many weeks after finally getting off Mirapex. Mirapex's extreme tightening of the front-sides of the neck, exaggerating the forward thrust of the neck seen in Parkinson's, coupled with the collapse of the neck when the drug is withdrawn, suggest that the D2 and D3 receptors stimulated by this agonist play some part in the regulation of the upper spine/neck muscles. This neck effect of Mirapex contrasts with Permax, which as you will recall does the same thing but in a different area, the lower back. Both cause excess tension or, upon reduction, collapse.

Stroke

The first four Mirapex patients that I worked with all decreased their Mirapex very quickly (over a period of two weeks or less). They all experienced symptoms of slight stroke (broken blood vessel in the brain). These symptoms included sudden loss of use of one limb (not necessarily on the Parkinson's side), suddenly impeded speech, personality change, and other symptoms of mild stroke, although none of them had a brain scan done and all of them recovered from their symptoms within a few weeks. These events were preceded by a feeling of excess pressure in the head, heat in the head, or a feeling as if something was going to explode. In the drug literature for Mirapex, there are warnings about over fast reduction causing neuraleptic malignant syndrome (elevated temperature, muscular rigidity, altered consciousness, and blood pressure changes). This syndrome is attributed to something going haywire in the autopilot portion of the brain.

It does seem as if the blood pressure pendulum can swing from a drug-induced Too Low all the way over to Too High when the drug is withdrawn. The good news is the manufacturers of this drug now recommend that this drug be reduced slowly. The bad news is they advise "one week" as being appropriately slow.

My subsequent patients did much better: they reduced over a period of months, not seven days. They all went their own ways, but in general they cut back by about .25 mg per reduction, and then they waited several weeks, minimum, before making another reduction. When they got down to .125 mg three times a day, they reduced by eliminating one dose of the three – taking only two doses per day for several weeks. After stabilizing at this level, they reduced to one dose per day for several weeks, and finally went down to none. Though some doctors scoffed, insisting that these low doses were doing nothing, I saw no more of the stroke-like symptoms after my patients started doing the slow decrease method.

Happy hallucinations

Most of the antiparkinson's drugs can cause hallucinations. Some notoriously cause terrible visions and nightmares. Mirapex visions are refreshingly harmless, even endearing. Most people enjoy, rather than resent, the playful creatures and little smiling children that appear under the influence of Mirapex.

Long-term depression after Mirapex reduction

The happy hallucinations and spirit-lifting effects of Mirapex have, of course, a backlash: when this drug is stopped, the vague sense of depression, irritability, and/or hopelessness can last for more than a year after the last crumb of Mirapex is taken.

Combined with Sinemet

I have seen many patients whose MDs have mistakenly told them that the agonist must be taken at the same time as the levodopa to have any effect. Some doctors actually have their patients taking this drug six or more times a day, simply because the patient is taking levodopa that many times a day. These doctors are confused and wrong. Mirapex is supposed to be dosed three times a day. Mirapex is a slow-working drug. Unlike levodopa, it does not wear off in six hours, or even twelve. My patients who only take agonists have never noticed a distinct On/Off from the drugs, and their improved mobility does not appear to be dose related whatsoever. They do not notice any difference if they forget a pill now and then.

The antiparkinson's effect of Mirapex and Requip seems to be a slow, cumulative effect, not a quick response. It is probably due to the unpleasant side effects that this drug can have on the gastrointestinal tract, including narcolepsy, that this drug is recommended to be taken three times a day rather than all at once.

However, our patients who have recovered and continued to take Mirapex (such as Becky and Rudyard) have found that Mirapex has, for them, a quick onset and wearing-off time. The effect may be felt within an hour and wears off within three hours. This is very different from our unrecovered PDers who took Mirapex; for them, the antiparkinson's effect was slow on and slow off, although the stomach (gastrointestinal) symptoms such as stomach discomfort and narcolepsy were still fast acting.

Manufacturer's recommendations

Start by taking .125 mg three times a day. Every week or so increase each of the three daily doses by .25 mg until the therapeutic level, 1.5 to 4.5 mg/day, is reached. Stay at this maintenance level, and continue to take the pills three times a day.

Format

To accommodate the slow dose escalation, this drug is available in many dosages. Many doctors prescribe the starter kit, complete with all the various dosages and a calendar for tracking drug increases, for patients beginning Mirapex.

Mirapex is available in the following doses:
.125 mg (an eighth of a milligram)
.25 mg
.5 mg
1 mg
1.5 mg.

Known side effects

Known side effects for Mirapex are dyskinesia, twitching, muscle spasm, dystonia, gait abnormalities, tremor, dizziness, low blood pressure, sleepiness, narcolepsy, insomnia, sleep disorders, hallucinations, confusion, thought abnormalities, paranoia, delusions, chest pain, edema (water swelling), double vision, vision abnormalities, dry mouth, sinus problems, anorexia, nausea, constipation, difficulty swallowing, arthritis, muscle heaviness, and dyspnea (breathing problems).

Precaution

Dosing may need adjusting in patients with kidney problems.
Levodopa dosage may need to be reduced.

REQUIP

Requip is also known as **Ropinirole hydrochloride**.

Requip is a dopamine agonist that is known to stimulate D2 receptors. It has much in common with Mirapex, including the problems with narcolepsy, passing out from low blood pressure, and gastrointestinal disturbances.

We have not seen enough patients in our clinic taking or reducing Requip to make generalized statements about this drug, although we have seen people who have the problems listed above even in our limited experience with this drug.

Adverse effects

In general, the information, warnings, interactions and contraindications for Mirapex in the section preceding also apply to Requip. The main difference between the two, as far as what we have seen in clinic, is that the dosings and pill sizes are different. The absorption rate is similar for the two, the half-life in the blood is longer for Mirapex (8 hours, as opposed to 6 for Requip), but, overall, the two drugs are very similar.

Manufacturer's recommendations

Starter dose is .25 mg, three times a day. Increases in dose are made weekly, increasing each dose by .25 mg for the first few weeks. After week four, the dosage can be increased by 1.5 mg/day (.5 mg per dose, three times a day) until a dosage of 9 mg/day is obtained. After that, it can be increased by 3 mg/day until the therapeutic level is attained. Maximum dose is 24 mg/day.

Warnings

Like Mirapex, the drug must be reduced slowly or it might produce neuroleptic malignant syndrome (see the section on Mirapex).

As with Mirapex, reduction of levodopa may be needed when Requip is added to the mix.

Mitch

Simply because we haven't mentioned Requip very much in this book, I will include a quick sketch of Mitch. He was taking Requip and starting to exhibit symptoms of recovery. He passed out one morning, about an hour after taking his Requip. His wife found him on the bathroom floor with blood streaming from his nose and from the gash above his eye where he crashed into the toilet on his way down. He had been having dizzy spells and losing his balance for several weeks and had told me that the dizzy spells were getting worse. The second time he passed out he gave me a call. I went over the details of his episodes of syncope (doctor language for fainting – what ladies do, or passing out – what men do). It seemed that his passing out and dizziness were directly related to the timing of his Requip dose: the trouble always occurred about one hour after taking his pills. He reduced his Requip over the course of a month, and the dizziness stopped.

Mitch is a top neurologist, a professor of neurology at a prestigious medical school. It had never occurred to him to track the timing of the side effects compared to the timing of the dosing. He considered reducing his meds even further but was terrified that he might become depressed, and he chose not to reduce his drugs even though he knew he was becoming overmedicated. He hasn't come into the office since his decision not to further reduce his drugs. Mitch is a highly respected neurologist: he confided in me, "We neurologists don't know what we're talking about when it comes to Parkinson's; we're just making things up."

When I asked him if he would tell his fellow neurologists about his experiences with our Asian medicine program he replied, "No. They would think I was crazy if I told them."

CABERGOLINE

Cabergoline is a European agonist drug. We had only one patient who was using this drug. According to the description, it is a slow working agonist. From what we can figure, it is very like Mirapex/Requip. For information about side effects and mechanism, consider using the Mirapex/Requip information.

AMANTADINE

Amantadine is also known as **Amatadine hydrochloride** and **Symmetrel**.

Amantadine is one of the non-dopamine drugs used for Parkinson's. It is a synthetic form of an amine – a very large molecule family that includes the famous vitamines. Amantadine was first used as an antiviral drug when it was noticed that it seemed to inhibit type A influenza (although the mechanism remains unknown). When used within 48 hours of onset of illness from type A flu virus, it can reduce the duration of fever and other flu-related symptoms. It is usually taken only briefly, the drug being stopped within 48 hours after the illness has passed. Amantadine is sometimes used as preventive medicine; when an elderly person or person with a compromised immune system is exposed to type A influenza virus, he may be prescribed Amantadine and told to take it for ten days following the date of exposure. When used in this protective (prophylactic) manner, the drug may be taken for up to 90 days.

By chance it was noticed that PDers who took the drug for viral protection appeared to have improvement in their Parkinson's symptoms. The mechanism for this benefit is utterly unknown. Of course, given the current fashion of Parkinson's disease being attributed solely to dopamine shortage, it was guessed that Amantadine might somehow cause a release of dopamine. Based on our observances of PDers who use Amantadine, and especially those who have tried to stop taking it, we suspect that the helpful mechanism may have to do more with adrenaline than with dopamine.

Our hypothesis has to do with the fact that adrenaline does act as an antiviral, antibacterial agent; when a person is under stress or maintaining a sense of urgency, he is unlikely to become sick. The common scenario in which a very busy person finally gets an overdue vacation and immediately comes down with a bug may be due to the relationships between adrenaline (a.k.a. epinephrine) and norepinephrine (a known immune system booster). In times of intensity of activity and responsibility, a person simply cannot allow himself to get sick. When the pressure is off and the adrenaline drops back to normal, a dormant flu is able to spring forth.

To the above observation we add that Amantadine, unlike all the dopamine-enhancing drugs, does not create mood-enhanced movement or a change in personality. People who take Amantadine notice that they feel like their old familiar, stressed-but-active, pre-PD selves again, almost as if the lagging adrenaline had been turned back on.

Also, when Amantadine is decreased, the patient acts as if "the plug has been pulled." Instead of the emotional jag, insomnia, or shaking that occurs from a levodopa reduction, a reduction in Amantadine usually causes a simple shutdown of motor function, as if the driving force, as opposed to the emotional impetus, has disappeared. In cases of abrupt cessation of Amantadine, the patient may be utterly immobilized, even if he is still taking a full complement of other antiparkinson's medications. One patient, four days after stopping Amantadine cold turkey, described himself as being "frozen stiff, like the Tin Man in *The Wizard of Oz*, before Dorothy brought him the oil can."

Another peculiar difference between Amantadine and the dopamine-enhancing drugs is that the full effect of Amantadine or Amantadine reduction is usually felt within four days. On the first day of reduction there is usually a slight decrease in vitality. This decrease worsens quickly, and by the fourth day, most of the patients in our experience find that they are completely without motivation or power. Typically, they resume the

drug within five to ten days after reducing it. In our experience of those using Amantadine, only 4 people have been able to stop taking it, though all have tried. Even patients who have been able to decrease Sinemet or agonist drugs have been stymied when trying to decrease Amantadine.

For the above reasons – the creation of viral resistance, and a response to decrease that is both non-emotional and very quick – we suspect that dopamine is not particularly influenced by Amantadine. Instead, I propose that this drug is affecting adrenaline or possibly norepinephrine levels.

Accommodation

Although Amantadine is not addictive in the usual sense, the body can accommodate to it. Within about three months after starting Amantadine, the body compensates by reducing its native adrenaline production (or production of whichever neurotransmitter it actually is that is enhanced by the drug) so that the person is right back where he started: the combined amounts of the drug plus the reduced native amount equals the amount of adrenaline that the body had to begin with. At this point, due to the compensating reduction of native adrenaline, it appears as if the Amantadine is no longer effective, and the PDer usually goes back to his neurologist for something "stronger."[1]

Why mild?

The problem with this drug is, once a person has started taking it, it is extremely difficult to stop. Although this is considered a very mild drug by many neurologists, we have never been able to figure out why. Possibly it is because this drug does not cause dyskinesia. I disagree with this "mild" label: this drug has many side effects (it can be especially disruptive to sleep, causing vivid dreaming and insomnia), it is nearly impossible to stop taking it once a person has started it, and the brain responds quickly to counter the drug, so that the benefit only lasts three months, but the side effects never ease up, and in fact, may worsen over time. Those three months of movement come at a high price: a lifetime need for Amantadine after it no longer provides any benefit.

Manufacturer's recommendations

PDers may take up to 200 mg/day. If a person is already taking other antiparkinson's drugs, Amantadine should be started at only 100 mg/day for the first week, and then increased depending on the patient's response. Although patients may have benefit from doses as high as 400 mg/day, patients should be carefully monitored for any dose higher than 200 mg/day.

Anyone with kidney disease or hemodialysis should let their doctor know that this drug must be carefully monitored, such patients receiving only 200 mg per week, not per day.

This drug must be used cautiously in anyone with a history of seizure, heart failure, liver or kidney weakness or disease, mental illness, light-headedness when standing up, cardiovascular disease, edema (water swelling) in the ankles, and in elderly patients. The manufacturer does not state what it means by "elderly."

---

[1] The three month figure is further supported by the manufacturer's note that this drug does not appear to be effective in its antiviral protection capacity beyond three months. Evidently it takes the body about three months to accommodate to this drug, whether it is used for PD or for viral protection.

This drug may impair mental alertness.

Parkinson's patients using this drug are cautioned not to stop this drug abruptly, as this might "precipitate a parkinsonian crisis."[1]

Format

Amantadine is available in 100 mg tablets, 100 mg capsules, and as a syrup (for dosing small children with immune system weakness). All patients in our experience take the gel capsules. None have been able to find the drug in the tablet form. Patients wanting to decrease this drug slowly have been frustrated by the difficulty in reducing by a fraction of the gel cap. Several patients have written to me stating that they have bitten a hole in the gel cap and squeezed out several drops of the drug so that they can reduce this drug slowly, since slow reduction is recommended by the manufacturer. The few patients who have tried switching to the liquid form of the drug to facilitate drug reduction have had difficulty in making the transition; they have all said that the drug behaves differently or doesn't work as well in the liquid form.

Known side effects

Irritability, insomnia, dizziness, light-headedness, nausea, hallucinations, headache, and vivid dreams are the most common side effects. Other side effects, ones that may be related to the brain's compensating mechanisms (a reduction in zip to compensate for the over stimulation of the drug) are, logically, just the opposite of what you might expect from a stimulant: depression, fatigue, confusion, anxiety, constipation, lack of appetite, vomiting, and dry mouth.

Symptoms of overdose

Tremor, nausea, vomiting, anorexia, seizures, heart fibrillations, slurred speech, blurry vision, urine retention, depression, and the movement extremes of agitation or slowness may all indicate overdose. After reviewing the potentially dangerous side effects, and especially the effects of overdose, and bearing in mind that, once accustomed to the drug, one must continue taking it even after the effect wears off, you may wonder, as we do, why this drug is considered to be a mild, starter drug for Parkinson's disease.

Warnings of risk from combination

Artane: may cause an amplification of side effects, especially confusion and hallucinations, if used with Amantadine. With regard to combining Amantadine and Artane, the manufacturers warn, "Use together cautiously." This euphemism means don't use these two together if you can help it. Jimsonweed, an herbal anticholinergic, in combination with Amantadine, can affect the heart/blood supply system. This combination is to be avoided.

Levodopa: Amantadine may cause increased stimulation to the central nervous system if used with stimulants. Curiously, the manufacturer does not name specific stimulants. Bear in mind that no drug manufacturer wants to write down in cold print that his drug should not be used with other specific drugs; this might lead to decreased sales. Without the word "levodopa" serving as a red flag, the Amantadine warning against

[1] *Physician's Drug Handbook*, Springfield, 2002, p. 31.

using in conjunction with “central nervous system stimulants” is more likely to be invisible. Levodopa is a central nervous system stimulant. With regard to central nervous system stimulants, the Amantadine manufacturer recommends, “Use together cautiously.”

Alcohol: Amantadine in combination with alcohol can cause fainting, low blood pressure (dizziness when standing up), confusion, and light-headedness.

ARTANE

Artane is also known as **Trihexyphenidyl hydrochloride**, **Apo-Trihex**, **Artane Sequels**, **Trihexane**, **Trihexy-3** and **Trihexy-5**.

Artane is an anticholinergic: an acetylcholine-suppressing drug. Artane was one of the first drugs used in treating Parkinson's. Back in the days before dopamine was discovered, someone opined that Parkinson's was caused by too much muscle tension. When it was discovered that acetylcholine was the neurotransmitter that triggered muscle tension, it seemed reasonable that an anti-acetylcholine drug could cure the presumed "excess muscle tone" of Parkinson's. Although this theory is now discounted, the sedative powers of anticholinergics do benefit a PDer in this regard: by making the PDer mentally, as well as physically, drowsy and weak, anticholinergics may lull the anxiety-driven tremor and restlessness. The downside is that heaviness of the limbs, a common problem in Parkinson's, is increased by the anticholinergic drugs. Some doctors are now considering that acetylcholine *increasers*, rather than *decreasers*, may be helpful for PDers. Of course, while these drugs may increase muscle tone and vigor, they may also serve to increase the tremor and restlessness.

Side effects following a decrease

Although acetylcholine is not considered one of the addiction neurotransmitters, acetylcholine is one of the neurotransmitters that slowly increase or decrease according to need. Since the drug decreases the amount of acetylcholine present, the brain slowly increases production accordingly. Eventually, the brain can *increase* acetylcholine enough to compensate for the presence of the drug. When this happens, the drug appears to lose its effect.

If this drug is decreased after having been used for more than a few weeks, the compensating increase in acetylcholine will become apparent – side effects that are the opposite of drowsiness and weakness may manifest for several weeks while the brain slowly adjusts the acetylcholine back down. During this time there may be a severe increase in anxiety, insomnia, and nervousness. If tremor is present, the anxiety may exacerbate the tremoring. I have seen intense panic attacks occur in response to a decrease in this drug. The panic attacks ebbed within a month of discontinuing Artane.

Two of our pioneers took Artane (a suppressant) with Sinemet (a stimulant) and had to figure out which drug to reduce when. They both independently came upon the same formula; if the patient slumped weakly in the chair all day, staring vacantly into space, the Artane was decreased. When dyskinesia was foremost, the Sinemet was reduced. Sometimes they would both be reduced at the same time, balancing each other's withdrawal effects.

The manufacturer's recommendations

The recommended dose for *postencephalitic* or *drug-induced* parkinsonism is 5 to 15 mg/day.

Advice from the manufacturer that I have never seen observed by physicians is this: patients with idiopathic Parkinson's (the common type) who use levodopa should take less than the above amount: a mere 3 to 6 mg/day of Artane may be needed if levodopa is also being used.

Older patients (the manufacturers do not specify what they mean by "geriatric") should use lower dosages. The manufacturer does not state what the lowered doses should be.

Artane should be started off slowly, taking only 1 mg on the first day, 2 on the second, and then increases made of 2 mg/day until the desired dose is attained. The pills should be taken at mealtimes. The sustained release pills should be taken once a day, after breakfast.

Users of this drug should not drive or engage in activities that require alertness until the effects of the drug on the individual have been determined.

Format

Artane is available in 2 mg and 5 mg tablets and in 5 mg sustained release capsules.

Known side effects

The side effects of Artane are just what you would expect from a drug that inhibits the neurotransmitter of strength: weakness and drowsiness. Specifically, the research lists weakness, drowsiness, blurred vision, pressure inside the eyes, headache, dizziness, hallucinations, nervousness, dry mouth, nausea, constipation, vomiting, urinary hesitancy or urinary retention.

Some other side effects may be the result of the body's trying to balance things out: nervousness and increased heart rate are also recognized side effects. It may be that, in response to drug-induced weariness, the body increases the heart rate in an attempt to overcome the drug. Likewise with nervousness: the drug sedates anxiety in the short term, but when the brain compensates for the drug by increasing acetlycholine to a higher level than normal, the increased NT may cause anxiety and nervousness. Nervousness and restlessness may also increase due to the panic that can set in when the brain feels as if "something is wrong," a feeling that can be triggered by this powerful muscle sedative.

Recognized symptoms of overdose

As should be expected from a drug that sedates the body and mind, overdose can cause mental symptoms of disorientation, confusion, delusions, anxiety, and restlessness. Physical symptoms can include blurred vision, dilated pupils, difficulty in swallowing, decrease in bowel function or urine release, high blood pressure, racing heart beat, and rapid breathing.

ATENOLOL

Atenolol is also known as Tenormin.

Atenolol is a beta blocker, a type of heart medicine. It prevents the heart rate from speeding up and also reduces blood pressure.

This drug is not a traditional antiparkinson's medication. However, some doctors, recognizing its sedative abilities, have prescribed it to help sedate the anxiety-driven tremor of Parkinson's. Unlike the antihistamines that sedate via drowsiness, a beta blocker sedates by holding the heart rate to a lowered pace. This lowered heart rate in turn reduces one's ability to feel anxiety or excitement. By reducing emotions, this pill can sometimes reduce tremor.

Low blood pressure

The blood pressure reducing properties of Atenolol can be a concern for PDers: many people with Parkinson's have orthostatic hypotension (light-headedness when shifting to a standing position), low blood pressure, or are already taking blood pressure medications. Also, many of the traditional antiparkinson's medications can cause lowered blood pressure. If your doctor prescribes this drug as a way of sedating tremor, stay aware of the low blood pressure side effects.

Other side effects

Other side effects of this drug can include fatigue, lethargy, drowsiness, dizziness, dangerously slowed heart rate, heart failure, high or low blood sugar, nausea, diarrhea, dyspnea (breathing difficulties) and bronchospasm (spasm in the air pipe).

DIGESTION INHIBITORS: COMTAN AND TASMAR

Comtan and Tasmar use different mechanisms to obtain the same type of result. They both block enzymes which otherwise would contribute to the breakdown of levodopa and dopamine in the blood, before the levodopa gets through the blood-brain barrier.

Ordinarily, the body does not want excess levels of blood dopamine floating around and takes great pains to quickly get rid of any excess dopamine by using specific enzymes (COMTs, short for catechol O-methyltransferase). These two drugs prevent these enzymes from doing their job, thus allowing blood dopamine levels to build up to unnaturally high levels. The hope is that more dopamine will make it into the brain if the enzymes are unable to do their job and break down the levodopa or dopamine.

Dopamine digestion, not food digestion

I refer to them as digestion inhibitors, but they do not inhibit digestion in general – they only inhibit the breakdown of dopamine and those foods or chemicals that are similar in structure to dopamine. The idea here is that by inhibiting the enzymes (COMTs) that are supposed to regulate blood (serum) levels of dopamine, the blood can achieve super-high dopamine levels. Then, as this dopamine-rich blood cruises past the brain, the extra dopamine can get sucked inside the blood-brain barrier and help make up the presumed dopamine deficiency in the brain.

These drugs are typically added to the drug regimen when the brain has already started rebelling against the excessive levels of dopamine being shoveled in by other antiparkinson's drugs. Signs of this rebellion range from dyskinesia to freezing and include all the other side effects of excess dopamine. The assumption on the part of the drug industry appears to be as follows: when the brain starts rebelling against excess dopamine, what it needs is even more dopamine. Therefore, these digestion-inhibiting drugs are added to Sinemet to force even more dopamine into an already reluctant and resisting brain.

Increased adverse effects of levodopa

After you finish reading about the digestion inhibiting drugs, you may realize that no one in his right mind will use them – they simply increase the side effects of levodopa. The main reason most doctors do not want to increase levodopa indefinitely is that the side effects become too severe. Doctors who have not read about the mechanism for these digestion-inhibiting drugs think that they are a way to increase dopamine in the brain without increasing the levodopa dose. However, the way that these drugs work is to increase the *effective* amount of levodopa, whether or not the actual dosage of levodopa has been increased. The very effect that the doctors are trying to avoid – too much levodopa – is exactly what happens when taking these drugs. Not one of my patients who have taken the digestion-inhibiting drugs has been able to continue them for any significant length of time – their levodopa side effects became intolerable. Combining the levodopa side effects with the side effects of these digestion-inhibiting drugs makes for a very unpleasant experience. Comtan tends to cause more gastrointestinal problems, stomach pain, and permanent diarrhea. Tasmar causes more liver damage and fatal liver

failure. Neither one is the answer to the question of how to get more effectiveness from one's dopamine without also getting more adverse effects.

COMTAN

Comtan inhibits (blocks) an enzyme (COMT) that is supposed to break down catecholamines. Catecholamines are a member of the amine family. (Amines are a specific type of protein, as noted in the section above on Amantadine.) Familiar (discussed earlier in the text) members of the catecholamine branch of the amine family are epinephrine (adrenaline), norepinephrine (a frontal lobe mood regulator), and dopamine. Ordinarily, the body wants to break down the bloodstream levels of these chemicals very quickly: they are mood and movement regulators whose levels need to change quickly in response to life activities. Comtan prevents the breakdown of these and other catecholamines.

Some medications are also broken down by the anti-catecholamine enzymes. Using Comtan will prevent these medications from being broken down, and the medications may build up to dangerous levels. Therefore, a person using central nervous system depressants, some asthma drugs, the non-selective MAO inhibitors (which may include the antiparkinson's drug Eldepryl, as this drug becomes non-selective if taken at doses higher than 10 mg/day – an approximate number, and all people are different), and other drugs listed on the Comtan drug insert may not be a suitable candidate for Comtan.

Manufacturer's recommendations

This drug is only effective when used simultaneously with a levodopa product such as Sinemet or Madopar (as it is known in Europe). This drug has no dopamine-enhancing properties when used by itself. Therefore, this drug is supposed to be taken at the same time as the levodopa drug is administered. Also, adverse effects such as hallucination, dyskinesia, and other problems of dopamine excess are due to the levodopa drug and not to the Comtan, per se.

Dosage

200 mg taken with each dose of levodopa, but no more than eight doses per day.

Format

200 mg tablets

Adverse effects

Adverse effects of Comtan can include dyskinesia, extreme excess of movement, extreme absence of movement (slowness, freezing), dizziness, anxiety, agitation, fatigue, extreme sleepiness, extreme weakness (asthenia), hallucinations, nausea, diarrhea, abdominal pain, constipation, vomiting, dry mouth, indigestion, taste perversion, bruising on the skin, back pain, dyspnea (breathing irregularities), and sweating.

Warnings

Levodopa dosage should be lowered to avoid adverse effects.

This drug may cause or worsen side effects of levodopa, including dyskinesia or hallucinations, *even if the levodopa dose is decreased* (italics are mine).

Diarrhea usually begins within 4 to 12 weeks after starting Comtan, but it may begin as soon as the first week or not start until after many months of treatment.

Rapid decrease of this drug can cause the same sort of problems as rapid decrease of levodopa (please read chapters 3 through 24), including neuroleptic malignant syndrome.

Keep an eye on blood pressure, be cautious when shifting from a sitting to a standing position, don't drive until you know how this drug will affect your reflexes and alertness, and avoid alcohol.

TASMAR

Tasmar works along the same lines as Comtan, but with this difference: the enzyme inhibited is one also that breaks down red blood cells. It turns out that by inhibiting the breakdown of red blood cells, the rate of levodopa breakdown is also inhibited. The reason remains unclear.

Liver failure

Canada has banned the use of this highly dangerous drug. Logically, no one should use this drug. Even the manufacturers state that "because of the risk of potentially fatal, acute fulminant [rapid, full blown] liver failure, use drug only in patients on levodopa-carbidopa therapy who don't respond to or who aren't suitable for other adjunctive therapy." In common English, this drug should be used only when no other drug is useful, and even then, the user is risking rapid liver failure. The doctor is supposed to discuss the dangers with any potential patient and the patient must give a signed consent form.

Despite this warning, which is one of the strongest ones in the antiparkinson's armory, this drug is blithely prescribed by some doctors because it is included in the antiparkinson's list.

Within a month after this drug was approved as safe by the FDA and released to the public with great fanfare, heralded as the drug to use when levodopa was no longer effective, three patients died of acute liver failure. Rather than taking the drug off the market, the manufacturers continued to offer it with this warning: patients using this drug should have a complete liver panel (blood work lab tests) done every two weeks for as long as they are using the drug or at least the first year. After the first year, if the patient is still alive, the liver panel may be done every eight weeks.

Considering how dangerous this drug is, I am not even going to list the manufacturer's suggestions for usage or the recommended dosage. If your neurologist has told you to take this drug, consider getting a new doctor. If you can't change doctors, please show your doctor the drug insert and gently inform him that even the manufacturer of this drug presents it only as a drug of last resort and suggests "written informed consent" from the patient.

Adverse effects

The adverse effects are the same as Comtan, with these additional known effects: tremor, excessive dreaming, headache, falling, fainting, loss of balance, speech disorder, chest pain, chest discomfort, palpitation, low blood pressure, ear ringing, swollen throat, anorexia, urinary tract infection, urinary incontinence, impotence, muscle cramps, stiffness, arthritis, neck pain, bronchitis, and upper respiratory tract infections, to say nothing of the fatal liver failures.

ELDEPRYL

Eldepryl is also known as **Selegiline hydrochloride**, **L-Deprenyl hydrochloride**, **Atapryl**, **Carbex**, and **Selpak**.

Eldepryl is a powerful mood enhancer. One newly diagnosed PD patient started Eldepryl therapy and within a few weeks announced, "If this is normal, then I've been depressed my whole life and I never knew it."

I can often detect if a patient has been taking Eldepryl for more than a few months, in this way: a tremulous vocal quality often develops, as if the user is always on the verge of tears. This vocal trait goes away after the medication is stopped.

Manufacturer's information

Eldepryl "probably"[1] enhances brain dopamine by blocking the enzyme (MAO[2]) that normally breaks down certain chemicals, including brain dopamine. At low doses it is thought that this drug selectively (only) blocks the type of MAOs that work in the brain (type B). However, there is reason to believe that it also affects the MAOs that work on stomach chemicals. The manufacturer notes that, since stomach side effects worsen as the dose increases, possibly this drug only affects type B MAOs in low doses and affects other MAOs in high doses. This specious logic is a nice attempt to avoid the larger issue, which is that no one actually knows why this drug helps people with Parkinson's. It is also a way to avoid being classified as a non-specific MAO inhibitor, a classification to be avoided, because certain other drugs are specifically contraindicated for use with non-specific MAO inhibitors.

Going off on a completely different tack, the manufacturer also volunteers that maybe this drug works by decreasing dopamine reuptake, similar to the cocaine-type mechanism.

Finally, although much attention is drawn to the MAO theory, it is mentioned in the drug books, almost as a shy afterthought, that one reason this drug may help people with Parkinson's is that it breaks down in the body into methamphetamine and amphetamine. Because meth is a well-known, highly illegal drug, one with many negative connotations (addiction, crime, etc.), it is understandable that the manufacturers might want to play down this fact about Eldepryl. However, even they admit that Eldepryl "has pharmacologically active [understatement] metabolites (amphetamine and methamphetamine) that may contribute to this [antiparkinson's] effect."[3]

Humbugs

The manufacturers of Sinemet (carbidopa-levodopa), the most commonly used antiparkinson's medication, state specifically that when levodopa is combined with MAO inhibitors, a "possible hypertensive crisis"[4] may result. Therefore, patients must "stop

---

[1] *Physician's Drug Handbook*, Springfield, 2002, p. 940.

[2] MAO stands for monoamine oxidase. This chemical inactivates catecholamines such as dopamine, adrenaline and norepinephrine. When MAO is suppressed, these catecholamines can exceed safety levels.

[3] *Physician's Drug Handbook*, Springfield, 2002, p. 940.

[4] Ibid, p. 596

MAO inhibitors for 2 to 4 weeks before starting levodopa-carbidopa."[1] In other words, L-Dopa should not ever be combined in any way with MAO inhibitors, including Eldepryl.

The manufacturers of Eldepryl, a drug whose only listed use is "antiparkinsonian," fail to come right out and say that their drug should not be combined with levodopa. In fact, they imply that it is safe when they suggest that a person taking levodopa can start taking Eldepry and then, after a few days, slowly decrease his levodopa. However, deep in the Eldepryl drug warning portion of the drug insert, one can find that Eldepryl should not be used with "adrenergic drugs." Dopamine hydrocholide is adrenergic, and stimulates dopamine receptors of the adrenergic system. Certainly, despite the addition of carbidopa, some portion of levodopa probably also stimulates these receptors, making Eldepryl, by its own admission, unsuitable for use with Sinemet.

Dosages

10 mg per day is recommended, and never more than 10 mg/day. 5 mg should be taken with breakfast, and the other 5 mg with lunch. If a person is already taking levodopa, patients should be told to gradually decrease their levodopa dose (no suggestions as to how gradually) after 2 or 3 days of Eldepryl therapy. If, after starting Eldepryl therapy, there is an increase in adverse effects associated with levodopa, "most of these patients need a levodopa-carbidopa dose reduction of 10% to 30%."[2]

Format

Eldepryl is available in 5 mg tablets and 5 mg capsules.

Adverse Effects

Eldepryl side effects can include the following long list: tremor, dyskinesia, twitching (including twitching eyelids), restlessness, loss of balance, increased bradykinesia (slowness, freezing), facial grimacing, stiffness, stiff neck, behavioral changes, loss of coordination, fatigue, headache, confusion, hallucinations, vivid dreams [including violent nightmares], anxiety, insomnia, lethargy, sleepiness, low blood pressure, high blood pressure, heart arrhythmias, palpitations, heart pain, chest pain, dry mouth, nausea, vomiting, diarrhea, constipation, abdominal pain, anorexia, weight loss, difficulty swallowing, heartburn, urinary problems (including frequency, hesitation, or retention), sexual dysfunction, skin rash, sweating, and hair loss.

Warnings

Overdose may cause symptoms such as drowsiness, seizures, coma, dangerously high or dangerously low blood pressure, and dangerous heartbeat irregularities, including heartbeat collapse. There may be a delay of up to 12 hours after the drug is taken before the symptoms of overdose appear.

Use caution if combining with alcohol, cacao (chocolate), or foods high in tyramine. These tyramine-rich foods are: cheeses, including cream cheese, bananas, meat, poultry or fish, Marmite (an English yeast product), sauerkraut, soy sauce or other

[1] *Physician's Drug Handbook*, Springfield, 2002, p. 596
[2] Ibid, p. 940.

soybean products including soy milk, beer (including non-alcoholic varieties), red or white wine, avocados, monosodium glutamate, peanuts, and raspberries.

Never use within 14 days of using Demerol. (Fatal reactions have occurred.)

Do not use with Prozac (fluoxitine). Don't start Eldepryl until at least 5 weeks after stopping Prozac, and do not start Prozac until at least 2 weeks after stopping Eldepryl.

Do not combine with adrenergic drugs. [Adrenergic drugs include Dopamine chloride, some asthma medications and epinephrine- (adrenaline-) enhancing drugs. *The Physician's Drug Handbook* has a very long list of adrenergic drugs. Please see a good drug book if you are not sure whether or not some of your medications are adrenergic.]

Never take more than 10 mg.

DEPRENYL

Deprenyl, a liquid form of Eldepryl, is advertised by its manufacturers as the "safe" alternative to Eldepryl, although the active ingredients are exactly the same. Deprenyl is illegal in the USA.

My patients who used Deprenyl had a curious effect when they stopped taking it. Both of my Deprenyl-using patients went through the typical dopamine-related drug reduction symptoms while decreasing, but when they finally got off the drug completely, they both had a highly unusual sensation as if their joints were on fire. The agonizing sensation of flame in all the joints of the body was so severe as to prevent sleep or movement. Neither patient knew the other patient, or had any information about the burning joints phenomenon. In both cases it began within two weeks of stopping Deprenyl and lasted nearly three months. This burning in the joints was unique in my experience, and occurred in addition to all of the usual depression, weakness, paranoia, insomnia and other symptoms that normally can accompany reduction of any dopamine-enhancing drug.

Deprenyl patients' background

Both patients in the above paragraph had been taking Deprenyl for several years. Both had been exhibiting symptoms of recovery for over a year before they tried stopping the Deprenyl. One had been taking a maximum of 14 drops per day, the other a maximum of 10. They both decreased slowly, over a period of two years and half a year, respectively. Neither one had ever used levodopa or a dopamine agonist, although the latter had been using Macuna and had only stopped the Macuna in the previous year. I fully expect to get strong letters stating, "Dear Madam, I quit Deprenyl and never had a moment's trouble," but please bear in mind, I am only reporting what I saw.

MIRTAZAPINE

Mirtazapine is also known as **Remeron**.

Mirtazapine was originally sold as an antidepressant. It is a member of the SSRI (selective serotonin reuptake inhibitor) family, of which Prozac is probably the most famous member. Mirtazapine is more sophisticated than Prozac: it is a dual action antidepressant. In addition to having SSRI-like properties, Mirtazapine also blocks some seratonin receptors in a manner that seems to reduce the usual SSRI side effects, and is effective over a longer time period. However, it turns out that, inadvertently, histamine receptors are also blocked. It is the blocking of the histamine receptors that causes the anti-tremor effect of Mirtazapine.

Most people who have taken antihistamines (anti-allergy, anti-runny nose drugs) will be familiar with the unfortunate side effect of supreme drowsiness. Mirtazapine, possibly due to its antihistamine (histamine receptor blocking) properties, also creates extreme drowsiness and sedation. It is this sedation that allows an anxious PDer to stop tremoring while the drug is at its strongest. Mirtazapine works the same way as a nap: a nap will also stop the tremor – as long as the napper remains asleep. Mirtazapine stops tremor – as long as the user is so sedated that he is on the verge of falling asleep.

Nat, my only patient to try Mirtazapine, had to stop soon after starting it. The powerful sleepiness was effective in slowing his tremor, but he kept falling asleep while driving, and he felt mentally and physically sluggish.

Manufacturer's suggestions

As of the 2002 edition of the *Physician's Drug Handbook*, no antiparkinson's classification was yet available for this drug, and the suggested dosing was for its use as an antidepressant. When used as an antidepressant, the manufacturer suggests a starting dose of 15 mg, and a maintenance dose of 15 to 45 mg/day. Dosage adjustments should be made at least two weeks apart.

Format

15 mg and 30 mg tablets

Adverse effects

Adverse effects of Mirtazapine include severe sleepiness, dizziness, weakness, tremor, abnormal thinking, confusion, water retention, increased appetite, dry mouth, constipation, weight gain, dyspnea (breathing problems), and increased susceptibility to flu-like syndromes (due to potential for decreasing white cell count).

Warnings

Do not use together with Prozac.

Potentially fatal interactions with MAO inhibitors – do not use within 14 days of each other.

Use cautiously with people taking blood pressure lowering drugs.

Concomitant alcohol use is discouraged.

Overdose symptoms include disorientation, drowsiness, impaired memory, and slowed heart rate.

SINEMET

Sinemet is also known as **Carbidopa-levodopa**.

Sinemet is a dopamine precursor-type drug. It is made up of two parts: levodopa and carbidopa. The levodopa easily crosses the blood-brain barrier and converts quickly into dopamine. The carbidopa slows the rate at which the oral dose of levodopa is broken down by normal digestive enzymes while en route to the brain.

Manufacturer's recommendations

According to the manufacturer, "Most patients respond to 25/100, t.i.d."

A translation into English means that a pill with 25 mg of carbidopa combined with 100 mg of levodopa (referred to as 25/100), taken three times a day (t.i.d.), will give a satisfactory therapeutic response. It doesn't seem to matter whether the patient has early or advanced Parkinson's when he starts taking the medication. As long as the person has never before taken dopamine-enhancing drugs (which include many antianxiety and antidepression meds), most people with Parkinson's, even advanced Parkinson's, will have a satisfactory response from 300 mg per day of levodopa combined with the 25% carbidopa. It may take several months to see the full benefit of the drug.

The manufacturer goes on to state that the dose may be increased every one or two days, if necessary. If using the less commonly prescribed 10/100 pills, the dosage may be one or two pills taken three or four times a day. If using the Sinemet CR (Controlled Released formula), in either the 25/100 or the 50/200 formats, the dose is two pills per day, at least six hours apart. These are merely general suggestions, however, as "maintenance therapy must be carefully adjusted based on patient tolerance and desired therapeutic response."[1]

The published time frame for determining tolerance or response is three days, and yet, in the same publication, the manufacturer states that the pill can be increased every one or two days. (In case you are only reading the appendix of this book, please take a moment to read chapter three of the main text. Both of the manufacturer's thoughts on this subject may be grossly wrong.)

The Sinemet website

Sinemet is manufactured by Merck & Co., Inc., Whitehouse Station, NJ, 08889, USA, and marketed by Briston-Myers Squibb Company, Princeton, NJ, 08543, USA. Bristol-Myers Squibb provides a website of helpful information, including "scientific studies" that are favorable to their levodopa products. Most of their scientific studies seem to have been carefully selected to encourage use of levodopa as soon as possible after a patient is diagnosed. They also seem to contradict the overwhelming majority of recent levodopa studies, mentioned nowhere on this website, that show a clear advantage to delayed use of levodopa when compared to starting levodopa therapy as soon as possible.

Website (www.sinemetcr.com/cross_site/CurrentSinemetCRPI.pdf)[2]

---

[1] *Physician's Drug Handbook*, Springfield, 2002, p. 596.

[2] Their website supposedly gives information about either the regular Sinemet, or the Sinemet CR. However, whether you click on the website to request the regular Sinemet information or Sinemet CR info, only the Sinemet CR page appears. I was not able to actually find a regular Sinemet page, only the CR

Website points of interest

"Sinemet CR may cause more dyskinesias than Sinemet. The occurrence of dyskinesias may require dosage reduction…

"A dose of Sinemet 25-100 or 10-100 (one half or a whole tablet) can be added to the dosage regimen of Sinemet CR in selected patients with advanced disease who need additional immediate-release levodopa for a brief time during daylight hours…

"When doses are not equal size during the day, it is recommended that the smaller doses be given at the end of the day…

"An interval of at least 3 days between dosage adjustments is recommended.

"Sinemet CR should not be chewed or crushed…

"Carbidopa inhibits vitamin B6."

Website tips on mixing levodopa with other drugs

"There have been rare reports of adverse reactions, including hypertension [high blood pressure] and dyskinesia, resulting from the concomitant use of tricyclic antidepressants and carbidopa-levodopa preparations…

"Postural hypotension has occurred when carbidopa-levodopa preparations were added to the treatment of patients receiving some antihypertensive drugs. Therefore, when therapy with Sinemet is started, dosage adjustment of the antihypertensive drug may be required." A translation into English might read: dizziness or passing out when changing from a sitting to a standing position has happened with patients taking blood pressure medications together with Sinemet. Because Sinemet can lower the blood pressure, sometimes the blood pressure medication needs to be decreased to prevent passing out…

"Concomitant therapy with selegiline [Eldepryl] and carbidopa-levodopa may be associated with severe othostatic hypotension not attributable to carbidopa-levodopa alone." (A translation of this one might be: combining Eldepryl and Sinemet can cause severe passing out when changing from sitting to standing. If this very bad result occurs, don't blame the levodopa, blame the other drug.) Again, this information is from the levodopa website…

"Anticholinergic agents, dopamine agonists, and amantadine can be given with Sinemet. Dosage adjustment may be necessary when these agents are added."

Format

Regular Sinemet, also known as quick release or fast acting Sinemet, is available in 10/100, 25/100, and 25/250 tablets. (The smaller number tells how many milligrams of carbidopa are in the pill, and the larger number is the measure of levodopa. In calculating how much levodopa one is taking, it is the larger number that should be used.)

A slow-release tablet is available in CR 25/100 and CR 50/200. The letters CR merely refer to the slow release (Controlled Release) construction of the tablet; the active

---

page. Some of the Sinemet CR page is oriented towards teaching doctors how to do a conversion from regular Sinemet to Sinemet CR. It is interesting to note that the patent has expired on regular Sinemet. Some cynics have noted that, although the CR technology was available years earlier, the CR pills were not released on the market until the patent on the regular Sinemet was nearly ready to expire.

ingredients of the pill are exactly the same in the CRs as in the regular pills. The same side effects and warnings apply to both the regular and the CR.

Known adverse effects

A *partial* list of the adverse effects from levodopa includes: tremor, dyskinesia, dystonia, choreiform [jerky, spasmodic movements, another way of saying "dyskinesia"], facial grimacing, head movements, muscle twitching, body jerks, ataxia [inability to coordinate muscle movements], bradykinetic episodes [slowness of movement, freezing], psychiatric disturbances, anxiety, euphoria, excessive salivation [drooling], choking, malaise, fatigue, severe depression, suicidal tendencies, dementia, delirium, hallucinations, confusion, echolalia [repeating a word (usually rapidly), particularly a word that someone else just said, over and over], agitation, heartbeat irregularities, lowered blood pressure, double vision, blurry vision, dry mouth, bitter taste in the mouth, nausea, vomiting, anorexia, constipation, diarrhea, abdominal pain, urinary frequency, urinary retention, incontinence, darkened urine, priapism [persistent, often painful erection in the absence of sexual interest], decrease in red and/or white blood cells, liver problems, weight loss, hyperventilation, hiccups, unusually fast speech.

Symptoms of overdose

"Muscle twitching, including twitching of the eyelids, and heart arrhythmias [irregular heart rate, angina, heart pain] may be signs of overdose."[1] Treatment of overdose includes stomach pumping and treatment for the heart arrhythmia, if needed.

Warnings for patients

"Maximum effectiveness of drug may not occur for several weeks or months after therapy begins."[2]

(The instructions for pure levodopa, unmixed with a buffer such as carbidopa (trade names Dopar and Larodopa), state that it may take six months for the full therapeutic response to appear.[3])

Monitor carefully any patient who is also taking medicine to regulate blood pressure or blood sugar.

Patients on long-term therapy should be tested regularly for diabetes, liver and kidney function, and acromegaly.

"Tell patient to take food shortly after taking drug…" to relieve stomach irritation.

"Patients should take a missed dose as soon as possible, but should skip a missed dose if the next scheduled dose is within 2 hours, and never double the dose.

"Elderly patients are especially vulnerable to central nervous system adverse effects such as anxiety, confusion or nervousness; those with preexisting heart disease are more susceptible to cardiac [heart] effects."[4]

Levodopa should be stopped 6 to 8 hours before administration of anesthetic drugs or hydrocarbon inhalation.

---

[1] *Physician's Drug Handbook*, Springfield, 2002, p. 597.
[2] Ibid p. 597.
[3] Ibid p. 595.
[4] Ibid p. 597.

MAO inhibiting drugs (these include Eldepryl) should be stopped 2 weeks before starting levodopa-carbidopa therapy.

Contraindications

Patients with acute angle-closure glaucoma, melanoma, undiagnosed skin lesions, or those who have used MAO inhibitor drugs within the last 14 days should not use this drug.

I have not been able to locate the study that led to the contraindication of Sinemet in the presence of melanoma (a deadly skin cancer). It is interesting to note that several of my patients have had skin cancers removed from their face after starting with Sinemet. However, since Parkinson's disease involves an absence of Qi in the face, and cancers can grow where there is an absence of Qi, it may be that the Parkinson's, and not the Sinemet, is responsible for the melanoma. It is possible that, since western MDs assume PD to be only an illness of brain cells, and most PDers of the last few decades are on Sinemet, their incidence of skin cancer has been wrongly attributed to their medication.

A Sinemet case study

Mark was diagnosed with Parkinson's disease at age 58. He started our program ten years later. At that time he was taking Sinemet 25/100 three times a day and Artane twice a day. He had tried adding Mirapex when the Sinemet decreased in effectiveness, but the combination of Mirapex and Sinemet caused dyskinesia. Mark's wife of thirty-six years, Margaret, was more alarmed by the facial grimacing and dyskinesia than she was by Mark's increasing immobility.

Dr. Rafferty, assuming that the dyskinesia was due to levodopa going into the body and not the brain, added Comptan to the mix. The Comptan increased the dyskinesia. They tried stopping the Mirapex while continuing with Comptan, but the dyskinesia continued. Just before joining our project, they stopped the Comptan as well. After his experiments with Mirapex and Comptan, Mark was no longer getting good results from his Sinemet, but he now had facial grimacing and mild dyskinesia within an hour of each dose; he had the side effects but not much benefit from his Sinemet.

Immediately upon entering our program, Mark started reducing his medication. Margaret took charge of his drug doses. Her plan, which Mark agreed to, was a 10% reduction every two weeks. When the daily amounts were so small that it was unreasonable to reduce by a daily 10%, she reduced by a weekly 10%. When Mark got down to 100 mg/day, she started reducing by 50 mg/*week*. She made a reduction every two weeks, come hell or high water.

A plan with flexibility

Sometimes she had to accelerate her plan; Mark responded well to the PD recovery treatments and his dyskinesia worsened, despite the reductions. She used his tendency toward facial grimacing or dyskinesia about an hour after his doses as her litmus test for accelerated drug reduction: whenever the dyskinesia reappeared, Margaret made another slight reduction, even if it was ahead of schedule. Within a few days or a week of a drug decrease, the dyskinesia would usually stop and Mark would go into a slump. If this was the case, Margaret still held to her 10% plan: sometimes it seemed as if he was still declining into drug reduction symptoms when it was time to make the next

reduction. Even if he was in a phase of feeling worse every day, Margaret stayed the course and Mark made another reduction. Other times he rebounded within days from a drug reduction: he was moving well or even having dyskinesia again less than two weeks after making a reduction. In these latter cases, Margaret made another reduction, ahead of schedule.

If Mark became too listless, Margaret reduced the Artane (anticholinergic). If the dyskinesia reappeared, the Sinemet was decreased.

There was no obvious pattern as to which reductions would send Mark into a tailspin and which ones would go easily. However, Mark's behavior followed predictable patterns. When he started having feeling in his feet, he became clinging and needy. When he started urinating every ten minutes throughout the night, he also became fearful of being alone. About two and a half months after the first decrease, he became angry and threatened violence unless Margaret allowed him to increase his pills.

The week before his first violent outburst, I had suggested to Margaret (out of Mark's hearing) that he was approaching the time when he might get hostile. When he did become angry and threatening, she was emotionally prepared. She laughed him off, told him that he was behaving according to prediction, and kept with her drug program. Shortly after this, I warned her that Mark seemed ready to collapse into infantilism. During the following week, he started crying, whimpering, and – for the first time in his life – talking about his grandmother, the only person who had ever really nurtured him.[1]

A temporary increase

At one point, Mark became adamant that he needed to increase his medication – his legs were turning limp and would not support him. He was severely disappointed when the weakness appeared – the week before he had been able to get in and out of the car easily, even gracefully, for the first time in years. He had hoped that this increased mobility had meant he was recovering, which it probably did, but when the subsequent limpness and weakness appeared, he went into a profound emotional slump. I suggested that since limpness was a symptom of recovery, an increase in his medication might cause an increase in dyskinesia with no corresponding increase in mobility. However, his despondency over his immobility was so great that Margaret did increase his medication. Within two days his dyskinesia was back with a vengeance and appearing sooner than

[1] While it is impossible to predict on paper just when a person will go through various phases of recovery and drug withdrawal, it was enormously gratifying to work with Mark and Margaret and see that my years of working with patients had indeed given me a reservoir of subtle cues so that I could almost sense when a person was going to have a change. While this may not be of help to the reader, it did help confirm to me that there is some sort of sequence and predictability, even in the timing and patterning of these complex stages. While I cannot yet categorize them or give written prediction/instruction to others in this book format, by working closely with these people it became evident to me that people in conditions of drug reduction or recovery do behave in a highly predictable pattern, if one can mentally integrate all of the incoming behavioral data. Just as a child living with a "crazy" parent can learn just when and under what conditions "mom is going to blow," even though to an observer the behavior seems random, I found that after four years of working with people reducing their drugs, there were dozens of subtle signals that allowed me to guess fairly accurately whether or not a reduction might be difficult or easy, and to predict just which emotional aspects might go haywire during the upcoming week. Try as I might, I still cannot find a way to take the basis for these successful "guesses," built on years of personal experience and learning, and convert it into written information.

before, a mere 45 minutes after the dose. He was still unable to get up out of a chair, even when he was grimacing. He then agreed to continue with the reduction.

Off the drugs

They started the drug reductions in early April and Mark was off his medication on the 4th of July, Independence Day.

Back on, two weeks later

Mark declined rapidly after he stopped the Sinemet. After two weeks of increasing shakiness, utter insomnia, terror at being alone, needing to be helped out of a chair every five minutes and then needing to be returned to the chair, mental and physical agitation combined with moments of utter physical helplessness, long periods of freezing, terrific stabbing pain and cramping in his legs that did not respond to heat, massage, or his nightly rum, he resumed Sinemet, with Margaret's blessing and mixed emotions.

Margaret felt like a failure because he was taking Sinemet again. I pointed out to her that most people who completely stop taking Sinemet have two or three bouts during which they get back on it again, have worse side effects than before, stop taking it, feel horrible, get back on, have even worse side effects, and then, sometimes, get off for good. Getting completely off the meds is a glowing goal, but after the goal is attained there is a post-victory crash, and the looming years of recovery are so daunting that resuming the medication, maybe temporarily, is pure routine: most people do it. My job at this time was to remind them both that they needed to do whatever was best for them and never to feel like failures.

Getting outside help

Within a month of starting the Sinemet reductions, Margaret would repeat to me nearly every week in the clinic, "You told me this would be the hardest thing I've ever done, and I believed you, I was ready for it. But I never knew just how hard "hard" could be."

Margaret asked for and received help. She asked Mark's brother to come over and sit with Mark so that she could get out of the house. This was particularly helpful; Mark's brother refused to yield to Mark's every request and gently made fun of Mark when he slid into self-pity. Mark quickly learned, even in his drug-reduction confusion, that he could be demanding of Margaret but not of his brother. It was very helpful for Margaret to learn that. After that, she started discriminating between the help that Mark wanted and the help that Mark needed. She did what she needed to do to keep her sanity. She got out of the house, she gave Mark rum and big meals to make him sleep a few hours at night, and whenever he slept, she slept. Margaret reported that caring for Mark during this time was more like having an infant in the house than a spouse.

Off the drugs, again

After two weeks of taking either 50 or 100 mg/day of Sinemet, during which time he continued to agonize due to recovery symptoms, Mark stopped taking Sinemet altogether. Margaret felt that since he was just as agitated with the medication as he was without it, they would see what happened after a few months with no medication. Mark

continued to go into a decline. Margaret referred to the next few months as "hell. Pure hell. I'm exhausted. I'm ready to kill him. Seriously."

Skin cancer

During this time he had a serious setback. He was just starting to feel mildly better, now and then, and his old, pre-PD sense of humor and quick wit was starting to shine through, when his doctor diagnosed the sores on his nose and face as skin cancer. When he received this diagnosis, he became completely immobilized, losing any mobility progress he had made. His mood collapsed and he was utterly helpless. The cancers turned out to be superficial. The dermatologist burned them off and announced that they were completely gone: the removal had been a complete success. However, whenever Mark looked in the mirror during the next few weeks, he saw a face disfigured with seven angry red scabs, and his depression deepened.

However, as the scabs started to fall away, he started to feel hopeful again. On October 31, exactly three months after taking his last Sinemet, his mood brightened considerably and his personality returned. Margaret wrote, "The addiction hell is over."

Sleeping through the night

Mark remained prone to restlessness and agitation through November. At the end of the first week of December, 18 weeks after taking his last Sinemet, Margaret announced that he had turned a corner: he could sleep through the night, and he was no longer freezing.

His personality had returned, enriched by the newfound emotional sensitivity that many recovering PDers say is "worth the price of having had Parkinson's." At this time his Tui Na practitioner said, "Lately I feel like I'm treating Mark; I used to feel as if I was treating a mound of twitchy drugs or else the drug withdrawal, and that Mark was buried somewhere inside." Margaret said, "I've got my husband back." Mark said he could not have done it without Margaret.

A few more Odds and Ends about Sinemet

Breaking pills

The regular pills and the Sinemet CR 50/200 can be broken in half with no alteration in speed of effectiveness. The CR pills lose their slow-release property and become very fast acting if they are chewed or crushed. Some patients know almost nothing about their Sinemet CRs except that the pills should *never* be crushed or broken. Based on their misinterpretation of the drug brochure, most patients assume that dire side effects or utter pill failure will occur should they ever accidentally ingest a pill that has been broken. This is incorrect.

What happens if the pill is carefully broken (as opposed to smashed) is this: the *slow-release coating* and internal structure of the pill is slightly compromised. When this happens the CR pill works slightly more quickly than usual. The broken CR doesn't work as quickly as a regular pill, of course, because even if the pill is broken a considerable portion of it still maintains the slow-dissolving structure. The breaking of a CR pill does not effect the properties of the drug or in any way alter the active ingredients; breaking a

CR merely compromises the slow-release feature so that the CR dissolves at a rate somewhere between that of a regular Sinemet and an unbroken Sinemet CR.

Crushing the pill, on the other hand, makes the entire contents of the pills available at once; the result is an overfast surge of drug. Neither the regular nor the CRs should be crushed.

Vitamin confusion

Conflicting evidence about vitamin B6 abounds. According to the Sinemet manufacturer's website, carbidopa prevents inhibition of levodopa digestion by inhibiting vitamin B6 from digesting levodopa. Products that contain vitamin B6 can reduce the effectiveness of levodopa, according to the makers of pure (non-buffered) levodopa. However, the *Physician's Drug Handbook*, based on information provided by the manufacturer, states that vitamin B6 does not reduce the effectiveness of carbidopa-levodopa (Sinemet). It states, "Multivitamins can be taken without fear of losing control of symptoms." Given the conflicting evidence of the authorities, and my lack of experience in this area, you are on your own with this one.

Comparing the 10/100 and the 25/100 formulation

Although the 25% ratio of carbidopa to levodopa is the one most frequently used by patients in our experience (25/100 or 50/200), we have seen one person using the 10% carbidopa (10/100 or 25/250) formulation. Because some people do not tolerate carbidopa well, this pill may possibly be made for those few individuals. However, the one person we knew who was taking the 10% pill was taking it because his doctor chose it at random. The 10% pills have less carbidopa and therefore offer less protection to the levodopa molecule as it tries to make its way past the digestive engines and into the blood. Therefore, the amount of levodopa that makes it into the bloodstream may be less with the 10% pills, depending on the total amount of carbidopa ingested during the day. It is impossible to know just how much less dopamine is available when comparing the 10% pills and the 25% pills: everyone's digestion is different.

The manufacturers suggest that the 25% buffered pills yield 4 times more levodopa to the brain than the completely non-buffered pills. No information is readily available about the relative dopamine yield of the 10% (partially buffered) pills.

Another factor that must also be borne in mind is that the body is supposedly saturated with carbidopa after 70 mg of carbidopa per day. This means that after the first 70 mg of carbidopa of the day have been taken, the rest is simply excess. Therefore, at doses higher than seven pills of 10/100 carbidopa/levodopa, the effectiveness of the levodopa portion of the pill is comparable to that attained after taking three 25/100 pills. At doses lower than seven pills a day, the effectiveness of the carbidopa in the 10% pills may be significantly lower in the 10% pills than in the 25% pills.

Don't forget: although the manufacturers throw the "70 mg" number about as if it were etched in stone, it is only an average number; individuals might vary widely in their response to carbidopa.

One has to wonder about the accuracy of the statement that 70 mg per day provides full saturation of carbidopa. For starters, if this is the case, and if the starting dose of Sinemet CR is 50/200, twice a day, this means that the starting dose is more than the body can absorb. Any increase would be pure excess, pure waste, in terms of the

carbidopa. One has to wonder why the manufacturers are shoving such high levels of carbidopa into the pills if anything over 70 mg/day is wasted. It is a mystery.

Buzz

Throughout this book, except in the case study of Buzz (chapter 23), all dopamine numbers are based on the 25% carbidopa/levodopa pills. Buzz was taking the 10/100's. Therefore, if you are trying to understand what happened with Buzz when he abruptly quit his medication, you must bear in mind that his brain may have been receiving, post-digestion, only a fraction (probably somewhere between a fourth and a half) as much levodopa as a person would have been getting from the more common 25% formulation. Actually, although the manufacturers state (based on what level of testing I do not know) that 70 mg/day of carbidopa is the saturation point, I have to look at Buzz and wonder if possibly he was receiving quite a bit less levodopa than a person taking the 25% format. If so, this might explain his "ease" during drug reduction, compared with all the other drug users: he was possibly absorbing much less levodopa than most.

Final report on Mark and Margaret

Recently (May 10, 2003), I received an emailed notice that Mark had passed on. I had been out of the area since mid-December when he "turned the corner." I had not seen him since starting work on this book in January; my only information on him was in emails from his acupuncturist.

Briefly, the end of his story is this:

In late December, Mark's basal cell skin cancer reappeared. The reappearance of the cancer was an emotional blow and he took it very hard: his father had died of cancer.

He began what Margaret called "massive" radiation treatments several times a week. Margaret said that the radiation seemed to take a lot out of him; he grew weaker in response to each treatment. In mid-April, he started having uncontrolled diarrhea. After more than a week of constant diarrhea and the beginnings of fever, Mark went into hospital. The diarrhea continued, the fever worsened and he was diagnosed with pneumonia. He was barely aware of his surroundings. After twelve days in hospital, Margaret insisted that he be brought home. He was aware that he was home, and seemed grateful to be there. His heart rate and breathing gradually slowed. He peacefully passed on, eight hours after returning home. His son and Margaret were by his side.

Margaret volunteered to me that, despite the hellish hardships during Mark's emancipation from drugs, they were glad to have been in the clinic program. She told me, "We had no regrets. We did it out of hope and optimism. We never gave in to Parkinson's."

Mark's brother, who had often sat with him during the worst of the drug withdrawal, was grateful that Mark had won through the challenge of drug reduction. After Mark's passing, the brother thanked Margaret for her valiant struggles during Mark's drug reduction the previous year. He said, "You gave him this time, this last year, of not being stoned on drugs."

Appendix 3

Appendix 3

SYMPTOMS OF DRUG REDUCTION AND WITHDRAWAL

The first part of this appendix has two lists of symptoms: those caused by drug withdrawal and those set in motion during recovery. The latter are listed because many of them might easily be confused with drug withdrawal. The second part of this appendix explains why the symptoms of drug reduction/withdrawal and the symptoms of Parkinson's are so similar. Please keep in mind that the following symptoms may occur during reduction of dopamine-enhancing drugs whether or not a person has ever had Parkinson's disease. A person might have similar symptoms to those listed below even if he only used dopamine-enhancing drugs recreationally. Therefore, although many of the symptoms below are easily confused with Parkinson's disease, they are actually drug reduction symptoms. It may be impossible in any given moment for a PDer to be able to determine whether or not his drug reduction symptoms are due to his Parkinson's or his drug reduction; both can cause the exact same symptoms of dopamine deficiency. The only way to know if the symptoms are due to drug reduction or Parkinson's is to wait about ten weeks and see if they start to go away. The symptoms of drug reduction may not be completely gone in ten weeks, but they should be starting to modify somewhat. It may take months and even years before the symptoms of drug reduction are completely gone, and even then there may be residual, semipermanent brain damage.

How can one know if one's difficulties are stemming from drug reduction/withdrawal? If the following symptoms do begin to change, vacillate, or modify within a few months, it is likely that that they are drug reduction symptoms and not Parkinson's symptoms.

These lists are not complete by any means. Every person will create his own unique repertoire of symptoms. The following information is merely to give you some ideas of how and why these symptoms manifest so that you can logically interpret your own behaviors, which may be similar or may be superficially different.

Symptoms of drug withdrawal

Tremor

The tremor of drug withdrawal can be mild or ferocious. It can affect the hands, the face, or any body part. It can resemble the tremoring of Parkinson's or it can consist of the whole upper torso jerking back and forth. The back and forth motions are more common than side-to-side motions. The head may bob back and forth.

Whole body shaking

The whole body may shake violently, rattling the teeth and vibrating as if the body parts are going to go flying off, as if the body is a rat being shaken by an invisible cat. The whole body may also be consumed with small shakes, as if it's being vibrated.

Insomnia

This symptom is particularly onerous, as it contributes to all the others. How many times have I heard, "If only I could get a night's sleep, none of the other symptoms would be as bad." The insomnia is not simply a matter of lying in bed, unable to sleep. It is usually quite panicked and resistant to fatigue. It is as if the body has forgotten how to sleep and instead remains on full alert during the night. It is normal for people to pace the darkened house endlessly during the months of drug withdrawal. This is an extreme version of the restless pacing that occurs in Parkinson's and is one of the most common symptoms of withdrawal.

Urination

There may be an increased sensitivity in the bladder, creating a sense of almost constant urgency. Drug reducers often complain about their frequency of urination. During the night, you may need to urinate every hour, if not more.

NOTE: If you are having this symptom, do visit your doctor and find out if you have a bladder infection or some other medically treatable situation. Not all bladder infections are painful. We have had two patients who assumed that their urgency was part of their drug withdrawal. Only when it continued after all their other withdrawal symptoms were gone did they realize that they had infections. There is no need to suffer needlessly, and a bladder infection gone wrong can rapidly damage a kidney.

Bizarre dreams, nightmares

It may be a side effect of sleep deprivation, and it may not. In any case, your dreams during this time, if you ever do fall asleep, may be terrifying. One patient had recurring dreams of his grandmother's death; his grandmother had been the only person in his childhood who had ever shown him any tenderness.

Hallucinations

Wide-awake versions of the bizarre dreams and nightmares can appear and disappear. A common hallucination is a voice speaking to you, telling you that you are getting worse, that no one understands what you are going through, that you need the medication. This voice can create a thousand reasons why you need more medication and how to justify taking it. It will hound you. You may not be able to tell if this is a voice from outside your body or if it is your own inner voice talking to you. I have had patients who scream at the voice, "Shut up!" but to no avail. The voice may tell you that it is your only friend, that you are being used, that you cannot trust the people around you, that you should listen to the voice. A common point that the voice likes to make is this: "You are old, and you're going to die anyway. Who cares if you die an addict? Better to die an addict and feel good than to live and be in pain for the rest of your life. What if you die tomorrow? You will have done all this suffering for no reason. You will die soon anyway, so take more pills."

Nausea

This can range from distaste for food, to fear of food, to vomiting and dry heaves. Curiously, sometimes the powerful distaste for food will not prevent you being able to put away a large meal. Some patients manage to keep eating their regular meals during

the months when they are nauseated. If excessive weight loss is a problem, bear in mind that sometimes a beer or glass of wine, by elevating dopamine, can invoke the return of a healthier appetite.

Weight loss

Most people lose a lot of weight during drug withdrawal. This is due to the nausea, the pacing, the shaking, and the insomnia. It is a serious concern for many patients. The loss of muscle mass is particularly troublesome for a recovering PDer who is trying to re-grow muscle.

Pacing

This restless pacing can be accompanied by shaking, head wagging, clutching at the gut, and a tendency to walk in straight lines, turning corners only with difficulty.

Repeating words

A person might latch onto one phrase, or one word, and repeat it endlessly. It can be logical, or it can be a response to a hallucination, making no apparent sense. A common one is "Help me, help me, help me, help me, help me, help me, help me."

Heightened sensitivity

Lights may seem blinding. Sounds may seem deafening. Smells may be nauseating. Tastes may be too powerful. Ambient temperatures may seem too hot or too cold. Clothing might feel too heavy. Internal sensations may also be heightened, so that the actions of the gastrointestinal tract are painful, and the cramping of muscles is excruciating. This heightened sensitivity can be severe. I have seen people curled up in a ball, eyes squinched shut, hands over their ears, repeating, "I can't take it, I can't take it."

Sense of doom

There may be a certainty that death is hovering. One person felt Death sitting on her shoulder for weeks.

Paranoia

Not so severe as the sense of doom, the paranoia is extremely common. It can manifest as fear of being left alone, fear of the night, fear of the telephone, or a certainty that people are plotting. I had one patient, an English chap who had worked in the merchant marines, who, during the worst of every drug reduction, kept warning us that Tahitians were trying to beat him up. If he had a fall that left a bruise, he would show us the bruise as proof that the Tahitians had shown up while he was sleeping and beaten him.

Temperature regulation

The body may be unable to regulate temperature. If the person feels cold, he may be curled up in a tight ball, shivering, unable to relax or even move his limbs. If he is too hot, he may be sweating and flushed.

Muscle cramping

There may be powerful cramping, or there can be pain from a muscle being held in one position, immobile, for hours. Some people are in such pain from their immobility that they cry out to their caregiver every few minutes, requesting help to move to a better position. I had one caregiver who kept notes. Her husband needed to be moved more than twenty times in one hour. Each time, the pain from immobility was so bad that her previously stoic husband was whimpering.

Dystonia

Dystonia, muscle cramping, and tension anywhere in the body may be worse during drug withdrawal. It can be very painful.

Loss of balance

The slowness of response during drug withdrawal can make it impossible for a person to perform the necessary balance corrections. Falls can occur. Also, uncontrolled walking backwards or sideways is not unusual.

Dizziness

A common side effect of many of the dopamine-enhancing drugs is dizziness. It can also occur during withdrawal. This contributes to the loss of balance.

Violent behavior

The survival mechanisms of the limbic area can cause unexpected, uncharacteristic behavior. These behaviors can range from mere surliness to violence. In our experience, the spousal violence is most likely to occur during a drug withdrawal phase when the caregiving spouse refuses to tell the PDer where the drugs are hidden.

Lying

Uncharacteristic lying about everything in general, and about drug doses in particular, can occur during drug withdrawal. This one seems particularly devastating to the spouses. After being lied to about sneaking some medication, one wife sobbed, “I can handle anything. I don’t mind being up all night, I don’t mind helping him with everything, but I can’t stand it that he is starting to lie to me.” I had to assure her that this is pure routine during drug withdrawal and not to take it personally. It was not her husband’s mind that was doing this – it was his limbic system.

Illogic

A person can become fixated on one thought or caught up in a world of illogic. This is particularly common in their illogic about their medications. A person in a condition of drug withdrawal might easily declare that, “I originally decreased my drugs because they were making me tic, but now, if I increase my drugs, they won’t do that, because I’ve figured out a system of counting to ten backwards to trick them so that I won’t tic anymore.”

In our clinic in Santa Cruz, we used to listen in amazement to some of the brazen illogic that patients would present as their justifications for resuming their previous med

levels, or even, despite worsening side effects, increasing them beyond their previous levels.

"It's never been so bad"

A subset of the illogic of this period is that the person going through it has absolutely no ability to assess his own situation. The withdrawal hell is hell, and every time it is the worst. The person in a state of brain trauma cannot say, "This time is less severe than last time," or "I'm no longer hallucinating, I must be doing a bit better."

Instead, every single day during which the limbic area is fairly dopamine deficient will be equally bad. Patients nearly always insist, during every single withdrawal, "This is the worst withdrawal I've had yet." I may point out to them that in previous times, they were unable to sleep or walk, and this time they are merely hypersensitive to sound or sleeping poorly but still sleeping. They just stare at me with a look of incomprehension and repeat, "This is the worst it's ever been. The reductions have never hit me so hard before."

The days and months of drug reduction are a cloudy mist in the mind. It is pointless to expect a person to remember objectively how he felt during any given drug reduction. Just assume that every single one will be, without question, "The worst reduction yet."

Emotions

A person might manifest any sort of emotion, from sobbing with self-pity to raging to hysteria. These behaviors may be frightening, as they may be uncharacteristic. For example, a previously stoic woman may blubber, and a previously sedate man may lash out violently, spittle hanging on his lips and a wild glare in his eye.

Inability to initiate movement, and freezing

A person may find himself stuck in a chair or on the toilet, unable to figure out how to stand up. The eyelids may seem stuck together, or the blink reflex may be greatly slowed. The fingers may not grip well. Movement in general may be slow and clumsy, or it may vacillate between rapid movement and freezing.

Foot sticking

A subset of the above, there may be a tendency to clumsy foot sticking, foot dragging and shuffling during drug withdrawal. Just picture the classic alcoholic who is having DTs. He may be tripping over himself and failing to lift his feet. There may also be an inability to make the feet, or any limb, behave as directed. Festinating gait, a variant of the foot sticking and difficulty in initiating movement, may manifest.

Inability to talk

Also a subset of the inability to initiate movement, the speech may be slow or mumbling.

Hunched posture

The shoulders may be pulled forward, the head thrust forward, and the torso bent forward from the waist. This can be so extreme that the head is literally between the

knees when standing. This is always terrifying to behold. We actually see it fairly often both in recovering PDers and in people who are reducing their drugs. This extreme caricature of hunched posture was described beautifully in Oliver Sacks' book, *Awakenings*, when they abruptly stopped giving L-dopa to one of the subjects in his experiment. Remember, this subject did NOT have Parkinson's disease or stooped posture previous to her brief fling with the drug. But after taking L-dopa for a short time, and then having the L-dopa withdrawn, she not only carried her back bent over so far that her head was on her knees, but she looked for all the world like someone suffering from a severe caricature of Parkinson's.

Drooling

Salivary problems can range from a bit of spray during rapid speech or a bit of spittle lurking in the corner of the mouth, all the way to long streamers of drool hanging from useless lips. Drooling can also be caused by most of the anti-PD medications as well. Just picture, for a moment, a person who is completely stoned on heroin or sloshing with alcohol. Picture the bleary eyes, the staggering steps, and yes, the drool hanging out of the mouth. This word picture is just a reminder that dopamine-enhancing drugs can cause drooling as well as inhibit it. In any case, during drug withdrawal, drooling can also occur.

Symptoms of recovery from Parkinson's

In addition to the symptoms listed above, which can occur during drug withdrawal, the following symptoms may also be occurring. These symptoms are normal during recovery from Parkinson's, whether or not a person ever took the medication. When these symptoms are occurring in a person who is also going through drug withdrawal, they can add to the confusion of trying to figure out what is causing what.

Extreme emotionalism

While this may or may not occur during drug withdrawal, it is certainly the norm for people recovering from Parkinson's. Self-pity, extreme neediness, fear of being left alone, bleating, whining, and crying, are all common during this time. Many caregivers and spouses feel that they are being tested by their PDers. It is as if, after a lifetime of never trusting themselves or their care to another person, the recovering PDers are trying to see just how needy they can be before the caregiver announces his resignation. Many caregivers say that their charge acts infantile from time to time. It has been proposed that for many of these PDers, this is the first time in their life when they felt that it was safe to cry for help or be needy, and that they are acting out the infancy or childhood that they never had. During this time, there may be instances of name calling or cruel teasing – behaviors that the PDer had never indulged in, even as a child. This behavior often responds very quickly to an adult discussion as to what will and will not be accepted. However, sometimes the PDer is incapable of doing overnight maturation. This infantilism is much easier to tolerate if the childhood history of the PDer is well understood.

Pain

While oversensitivity to sight, sound, taste and touch are all normal during drug withdrawal, there is also significant pain associated with recovering from PD. Although most of the pain is in the extremities (the hands and feet), there can also be tremendous pain throughout the body, from the skin down into the bones, and in the gastrointestinal tract as well. The pains of recovering from Parkinson's can be fleeting or last for weeks. They can be acute or they can be a mild tingling under the skin. Please read more about this in the book, *Recovering from Parkinson's Disease, A Patient's Handbook*.[1]

Deep Sleep

During recovery, most people will have two-hour periods during the day during which they fall, helplessly, into a state of utter immobility, regardless of their physical condition during the rest of the day. Most PDers have this experience between the hours of seven a.m. and nine a.m. All the dopamine in the world will not provide mobility during these hours; this is a time that deep healing occurs along the Stomach channel. This time frame is approximate; some people may sleep from 6:30 to 8, and others from 7:30 to 10. But it is still the same syndrome, regardless. If there are injuries in addition to those of the Stomach channel, other times of day may also be subject to these complete body shut downs. These periods of unrousable stillness may last for a few weeks, occurring at the same time every day, or they may extend over a period of more than a year and come and go somewhat.

If a person is having Offs from their drugs, and trying to decide if they are from overmedication or undermedication, these (approximately) two hour spans will just add one more layer of confusion to the fun.

Taking more drugs during this time is a waste of drugs and a nuisance to the brain – the body has shut down to make repairs, and it will remain shut down until the repair time has passed.

It is quite astonishing to behold when a person wakes up in the early morning, has a perfectly normal start to the day, and then falls motionless, as if anaesthetized, into a rigid heaviness, unable to speak or even blink. Two hours later, the spell is broken, and the person gets up as if nothing had happened, and continues on his day, moving at whatever is his normal tempo. The next day, it happens again. This extreme level of immobilization does not occur with everyone, but it can happen.

Weakness

A person who is recovering from Parkinson's may have muscles that are turning to pudding. The previously rigid, steely, and diseased muscles along the line from the chin to the foot will be dissolving, prior to the formation of new muscle. During this time, it may feel as though there is no muscle whatsoever along these lines. This is different from the inability to initiate movement that occurs in Parkinson's. This is pure weakness. The limbs may be limp. They may even flop helplessly. They may be so weak that it will be impossible to get up from the toilet or a chair, or to even roll over in bed. It *may* be distinguishable from the helplessness of Parkinson's, however. As an example of the

[1] J. Walton-Hadlock, *Recovering from Parkinson's Disease, A Patient's Handbook*, 2003. This book is available online for free at www.pdrecovery.org

difference, here's a description of a simple activity from the Parkinson's patient's viewpoint and from the viewpoint of someone who has recovered from Parkinson's.

Rolling over in bed

A person with advancing Parkinson's may roll over in bed in this characteristic manner: the knees are brought up to the chest. The shoulders are pushed forward. Then, with a rocking motion generated in the hips or shoulders, or by using an arm to pull the whole body over, the body shifts position with no twisting movement occurring at the waist. The spine is fairly inflexible throughout the rotation.

A person who is turning to a jelly because of being in the middle of recovery from PD will be unable to do any of the above motions simply because he doesn't even have the strength to pull his knees up to his chest. When he does turn over, if he can, he may find that the top half of his body has flopped over, and the lower half is still laying in the former position, heavy, inert, unable to move. He may need to be rolled over by a spouse or caregiver. If so, the spouse or caregiver may find him much harder to move than in his PD days; his limp passivity will be much harder to shove around than his previously rigid frame.

A person who doesn't take medications will have an advantage in assessing whether or not the movements, as described above, are more rigid or more limp. A person who was taking medication will not actually know how he used to move, because his motions and his mind will have been masked by the medications. He will not be able to make an astute comparison.

Some of each

Of course, none of this happens overnight. There is a gradual transition from the immobility and tension of Parkinson's to the immobility and mushy muscles of recovery. It is extremely frustrating, during the long months of increasing weakness, trying to decide what is going on.

A nice landmark is facial expression. The facial muscles recover their strength more quickly than the large muscles of the legs and torso. If a person who previously wore the mask has now recovered facial expression, it is a good guess that his increasing immobility is due, at least in part, to his muscles dissolving prior to reconstruction.

This is all well and good for the person who never used any medications. They must simply wait it out. The person who is trying to reduce medication during this time will be in a more difficult position. He and his loved ones will be plagued with doubts as to whether or not the immobility is an indication of overfast drug reduction. These doubts will be augmented by the fact that the drug reductions will reduce the will power center in his brain to a crumb of cheese, and his inner voice will be making the case to him that nothing matters anymore anyway, so why not take more drugs. This is a difficult time.

DRUG WITHDRAWAL UNCERTAINTIES

During drug withdrawal, our patients were baffled as to which symptoms were being caused by the drug reduction and which ones were remaining from their Parkinson's. The distinction between drug reduction symptoms and Parkinson's disease symptoms is a grey zone, without distinct lines of black or white. *All the symptoms, regardless of cause, are related to changing dopamine levels.*

The symptoms of drug withdrawal listed above can occur in nearly any addict, not just a person easing off his anti-PD meds. And yet, when a PDer's body starts showing some of these symptoms, they will have an uncanny resemblance to his old Parkinson's symptoms, filling the PDer with uncertainty as to his recovery.

Many symptoms of drug withdrawal are interchangeable with the symptoms of Parkinson's. They may manifest only slightly differently in the PDer than in the drug user, and that is the result of habit, and nothing inherent in the drugs. But these differences, however slight, may be worrisome to the recovering PDer.

When a drug user develops a tremor during drug withdrawal, he may tremor in any body part. When the PDer manifests tremor during withdrawal, his body will naturally find it easiest to use those body parts that already have a pattern of tremor established. In other words, if the PDer used to have a left foot tremor, then during drug withdrawal he might be likely to manifest a left foot tremor. Thus, the PDer may conclude, incorrectly, that his tremor has returned. The addict who never had PD doesn't make any such assumption. He just concludes that the tremoring is a part of his "cranking"[1] and doesn't give Parkinson's disease a thought.

As another example, a person experiencing drug withdrawal may tend to hunch over in a fetal, defensive posture. This curling up may involve bringing the knees up or pulling the shoulders forwards. When the PDer, who used to be hunched forward, starts to assume a protective posture during his trouncing from drug withdrawal, it is most likely that his body will select his old tried and true fetal form. He will pull his shoulders forward in his remembered position from his PD days.

This ex-PDer is likely to demonstrate his old, hunched, PD posture during withdrawal. This is quite similar to the classic withdrawal/fetal position posture. However, this patient's MD, seeing the hunching, may disregard the possibility of drug withdrawal, and wrongly assert that his Parkinson's is still very much in place.

This reverting to the established PD pattern during times of dopamine deficiency and stress can make it very difficult to determine whether or not the person still has Parkinson's or if he is merely in the agonies of drug withdrawal. Here is an example of the tendency for the body to revert to familiar habits in times of stress:

A bee sting remembrance

Lynne had completely recovered from Parkinson's disease nearly two years earlier and no longer had any symptoms whatsoever. Her doctor had told her that she had been misdiagnosed – which is as close as one can get to being told that one has recovered. But one terrifying night she thought that it had all returned.

She was highly allergic to bee sting. She had had an anaphylactic shock reaction more than twenty years earlier, so she was always wary in case of bee sting and knew how to respond if one occurred.

On the night in question, she had just rubbed a new brand of salve onto a cut on her hand. She'd cut her finger that morning in the garden. As the cut suddenly started itching and then throbbing violently, and a flash of heat started traveling up her arm, she read the label on the salve – it was made with beeswax.

[1] Cranking is a slang term for drug withdrawal symptoms.

She immediately took the appropriate steps: a dose of Benedryl (an antihistamine), together with a dose of epinephrine, a lung and heart stimulant. Together, these two drugs act to keep the airways open and sedate the histamine response which can, in highly allergic people, be fatal. All went as expected: her heart rate increased, and she felt alternately groggy and hyper alert for the next two hours, which is the correct response to the medicines, and her body stopped reacting to the bee sting.

But the most fascinating part of the response was this: during these two hours, when her body was being stressed by the allergen and the drugs, and no doubt, the fear inherent in having an anaphylactic reaction, she assumed every single one of her old Parkinson's symptoms. Her right hand started a pill-rolling tremor, her right arm up to her elbow was shaking, and she carried her arms bent at the elbow, pulled up tightly to her body. Her shoulders rolled forward, her ears practically resting on her shoulders. The tension in the front muscles of her neck pulled her chin down towards her chest. Her steps became a shuffle, and her legs moved with great rigidity as she paced back and forth in the hallway in her house. She felt her facial expression disappearing. Her mouth hung slightly open, with spittle suspended in the corner of her mouth. Her voice was weak and shaky.

She didn't even realize how she looked until she shuffled past a mirror. She stared with horror. Her Parkinson's disease was back in full force! Her husband helped put her to bed, and she fell into a deep, Benedryl-induced sleep.

In the morning, as she lay in bed before rising, she felt fine, but she was frightened of what she might see in the mirror. Had the Parkinson's returned?

The body that looked back at her in the mirror was perfectly normal, not one sign of Parkinson's. She tried her handwriting; it was nice and big. She sang in the shower and her voice was perfectly normal. That was three years ago. She has not had any similar episodes since that night.

Her body adapted certain behaviors during her years with Parkinson's. She now suspects that any time she is severely stressed, she might temporarily manifest some of her old PD physiology of excess adrenal stress, with its concomitant scattering and decreasing of dopamine. During these times, her body might use the same postures or behaviors that she adapted to manifest her old PD lack of dopamine. It makes sense that, in future instances of dopamine deficiency, she may revert to these patterns, or you might call them symptoms, for as long as the emergency or the dopamine deficiency exists.

This means that, if she develops hypothermia, she might shuffle. If she finds herself in a traumatic emergency, she may have a right hand tremor in the weeks afterwards, as the body comes to terms with the shock.

Death and sickness

Symptoms of dopamine deficiency are not rare, nor are they the exclusive domain of Parkinson's disease. It is normal for a person with hypothermia to tremor, pull the arms in tightly, and to walk with a shuffling stride. It is normal for a person who has been through a shock to feel shaky and want to curl up tightly. However, for a person who has once had PD, these symptoms may always evoke the fear that Parkinson's has magically, instantaneously, re-erupted. Fortunately, this is not the case.

I have since seen the same phenomenon with patients in response to the death of a spouse, and in response to severe illness, as was the case with Taylor Paul. In the case of

the spousal death, the patient had never taken medications for PD. He had recovered to the point that he was no longer manifesting any Parkinson's symptoms when his wife was diagnosed with kidney cancer. She quickly switched from caregiver to care *needer*. She died five months after her diagnosis. During her time of illness, he didn't manifest his old Parkinson's symptoms. But immediately after she died, his old symptoms reappeared. He was tremoring and hunched over, and his micrographia reappeared. He was extremely weak. Happily, his voice never went back to a whisper, and he continued to have facial expression and initiation of movement. However, to the casual observer, it looked rather like the Parkinson's had somewhat returned. It was nearly a year before all his PDish symptoms faded away again.

In the case of Taylor Paul, with the severe fever and infectious disease, it was nearly two months before he was even starting to feel like his recovered self again. Again, Taylor Paul's doctor assumed that all weakness must be due to Parkinson's and only Parkinson's, even though Taylor Paul's infectious disease was so severe it nearly killed him.

In other words, as a response to stress, the body might choose to revert back to its known defensive, PDish posture until the stress is gone. All bodies can create dopamine deficient symptoms during times of stress, but it would be the height of madness to insist that they all have temporary Parkinson's disease. But all recovering PDers may have a tendency to re-robe themselves in their own, particular PD symptoms when they are stressed. This short term resumption of old, remembered symptoms invariably raises the question, for themselves and loved ones, as to whether or not the Parkinson's has abruptly returned, in full battle dress. In our experience, this has not been the case.

It will be a matter of removing or getting over the stress, the illness, the shock, or the cold. When the stressor is gone, the PDish symptoms will cease as quickly as they came.

So keeping all this in mind, the symptoms listed here can occur in anyone, not just a PDer or a recovering PDer who makes a decrease in his dopamine enhancing drugs. All of these symptoms have been mentioned already in the course of the book. This is only a list of the most commonly seen symptoms of drug withdrawal. There is an infinitude of potential variations on this list. Each person walks this path alone, however, and during the course of drug withdrawal, one may invent nearly any behavior that serves as balm to the tormented limbic area.

Summary

In summary, most of the symptoms of drug withdrawal look similar to patterns seen in Parkinson's disease. This is because the symptoms of drug withdrawal are symptoms of dopamine deficiency. Those aspects of Parkinson's disease that are caused by dopamine deficiency will therefore resemble the symptoms of drug withdrawal.

However, in Parkinson's disease, these symptoms develop slowly, over years. During drug withdrawal, these symptoms can develop over a matter of days. They can also resolve abruptly, or vary over the course of days, or even months. The two sets of symptoms are so similar that an MD *cannot* recognize whether or not symptoms are coming from drug withdrawal or from Parkinson's disease. However, if you have been following your patient's case closely, you may be able to tell the difference.

A health practitioner or caregiver will be well served by making a keen study of the personality and exact movement patterns of the PDer in their care. Then, when this same person starts manifesting personality changes and irrational behavior, together with some new physical symptoms that are subtly different from his symptoms of Parkinson's, you may be able to guess that there is something afoot that may be recovery and not PD or drug related.

Then again, even this is not proof of anything. Every person with Parkinson's has slightly different symptoms. Every person going through drug withdrawal will work through it in a slightly different manner. Everyone recovering from PD will do it in his own, unique way. Guessing which symptom is being caused by what process at any given point in time is nearly impossible for the person in the throes of drug withdrawal, or for an outsider, such as the family doctor. The spouse or care-giving friend, who knows the PDer's history and can compare every symptom with patterns in his past, has the best chance of anyone at guessing what is going on.

Appendix 4

HELPFUL TIPS FOR SURVIVING DRUG WITHDRAWAL

To best counter the effects of low dopamine, stay warm, stay well fed, be touched, listen to classical or familiar, well-loved music, and avoid crowds or anyone with an infectious illness.

Stay warm

It is no coincidence that most PDers are diagnosed in the fall and winter. Dopamine is used in temperature regulation, and getting chilled can bring latent Parkinson's symptoms to the fore. Most PDers are chronically cold, with especially cold feet that simply do not respond to the normal sorts of warming treatments. About 10% go the other way – they tend to be overly hot. Dopamine deficiency can also create overheating, but this is less common. These people will want to stay cool during drug withdrawal.

During drug withdrawal, staying warm (or cool, if you run hot) is crucial. The caregiver may need to attend to this; the person who is in withdrawal will not be able to think clearly. By "stay warm," I do not mean merely staying in a room that is heated to a comfortable temperature. Wear a hat at all times. Wear socks and slippers in the home at all times. Wear several layers of clothing. Wear something wrapped around the neck.

The human body needs to be approximately 98 degrees. The average home is kept at about 70 degrees. This difference is comfortable to a healthy person, but it can deplete dopamine in a person who is not functioning normally. A person who is in the throes of drug withdrawal has the same inability to regulate temperature as a person who is in the early stage of influenza – aches and chills can occur even in a well-heated room. Therefore, keep your drug reducer very warm, as if he was shaking with chills. A home hot tub or sauna can be very beneficial. A public spa is usually not a good idea; there is too much exposure to infection, and the stress of being out in public is counterproductive.

Heat

During heat spells and the height of summer, the opposite may be the case. You may be utterly unable to deal with heat, and this may be more dangerous than the cold. Your body may not be able to figure out that it needs to sweat, and your pacing and shaking may heat your muscles to a dangerous degree when the ambient temperatures get close to body temperature. During heat waves, stay in cool, dark places. Go to an air-conditioned theater. Drink extra fluids, and take tepid baths. Put your feet in ice water. During times of unusually high heat, expect to feel as though you've been hit by a train.

Stay well fed

Eat a lot. Eat starchy foods, filling foods, comfort foods. Keep your stomach topped up. Insomnia sometimes responds well to a completely full stomach. There are

neurons in the limbic system that saturate with neurotransmitters from either food or dopamine. If you keep these neurons filled from food, the scanty dopamine in the limbic area will go farther.

Staying full of food helps with insomnia. Insomnia is one of the worst symptoms, as it exacerbates all the other problems. Through experimenting, we've found that the following dinner menu ideas can be very helpful in allowing the drug withdrawer to get some sleep.

Start with a glass of wine or beer just before dinner. This may help calm you down enough to enjoy your meal. Eat plenty of starchy foods and rich creamy foods. Try starting with a nice cream soup or creamy chowder, followed by a hearty main dish such as stewed beef with potatoes or turkey with stuffing and gravy. Eat plenty of bread or buttered rolls. Be sure to eat dessert. Dessert should be something that makes your eyes roll back into your head – something with lots of butter or cream. Cake with thick frosting or pie a la mode is good.

If you normally drink alcohol with meals, be sure to indulge this habit. An after-dinner snootful, maybe a nice brandy or whatever you have in your locker, seems to add nicely to the mix. After a meal like this, you may feel almost able to lie down and get some rest for a change.

The quality of the sleep may not be perfect; you may need to burp a bit or take a digestive tablet if your stomach complains. However, this method of inducing sleep has been proven very successful in people who are otherwise unable to close the eyelids; somewhere between the salmon mousse and the avocado omelet, the brain starts to let its guard down.

Haggard, desperate reducers who haven't slept for weeks have found the food treatment remarkably effective. Often the idea of treating withdrawal insomnia with mere food is met with pooh-poohs. But when I call the scoffers the next morning and ask how they fared after a large meal, they often say that they slept well, or at least slept better than usual.

If you are able to digest dairy, don't be afraid to have a glass of warm milk at bedtime. If you can't digest dairy, consider a glass of something alcoholic. If neither of these appeals, try the white bread treatment. Several slices of white bread will temporarily convince your limbic system that all is well. You may need to repeat this treatment during the night, so keep a few slices of white bread on your night table, or a turkey sandwich waiting for you in the refrigerator. Don't think that you will have the presence of mind to put together a sandwich in the middle of the night. It will be hard enough for you to even remember that you made a sandwich specially for snacking on; when the frantic insomnia attacks, you can usually think of nothing except how horrible you feel. That's why the task of forcing you to confront the sandwich in the middle of the night may fall to the caregiver.

Turkey can sometimes be good for inducing sleep. Turkey contains sleep-inducing compounds. It works best if you eat plenty of warm turkey, with some starchy foods on the side. Again, this may seem far too simple, but it can be highly effective.

A high percentage of the PDers I've worked with do not drink alcohol. If a nightcap is out of the question, there are always milkshakes, topped with whipped cream. If both dairy and alcohol are forbidden you, try roasted potatoes with lots of olive oil. Keep some on hand, and heat them up at bedtime. Have extras to munch during the night.

They will work the best if they are heated before eating, so shove them in the microwave or quickly refry them.

Do not worry about gaining weight, especially if you have tremor or ticcing patterns. Most people with excess movement have to be concerned with losing too much weight. You can diet after you are done with the drug trauma. Keep your priorities straight. And if you do gain a few pounds, it is a small price to pay to be free of mind-altering drugs. The efficacy of the food method is seen in people who quit smoking – they often gain quite a bit of weight. This is because their body, denied dopamine, will reach out for the next best thing, and the next best thing is food.

However, when a person is getting off of really powerful drugs, such as cocaine, heroin, or the antiparkinson's medications, the mind is so twisted and battered that it cannot even think of food, it can only think of the drug. Also, the nausea and hypersensitivity to taste and smell during drug withdrawal can contribute to the poor appetite. Therefore, every inducement should be used to encourage a drug reducer to eat.

Alcohol

Alcohol, when used to ward off symptoms of drug withdrawal, is usually safe and effective. Check with your doctor and your pharmacist to make sure that the drugs you are still using can be taken with alcohol. Do not use alcohol therapeutically if your doctor says not to, which he may if you have a history of intolerance to alcohol, liver or kidney damage, or any other reason that precludes alcohol use.

Alcohol, when used as a drug reduction therapy, is not addictive. This is because your dopamine levels are so low that the alcohol won't ever take you over the Safety Limit. As an example of this, consider the small drink of alcohol that is traditionally used, in some cultures, as the antidote for "next morning" hangover. Because of the crash from the excesses of the night before, dopamine levels will be quite low. By administering a "morning after" dose, a smallish drink of something alcoholic, the dopamine levels might rise up to where the agonies of the crash are lessened. As long as the morning-after dose is small, it will scarcely bring the dopamine levels up to a functional point; it will not exceed the Safety Limit.

Dopamine is not the only body function that is affected by alcohol; some of the pain of hangover is due to the dehydrating effects of alcohol and the damage that alcohol (a mild poison) does to various organs. I am not advocating alcoholism, or even a drink every night with dinner. I am presenting some information about alcohol because, based on experiences with drug reducing patients, it appears that alcohol can help subdue some of the more violent reactions of drug withdrawal, especially the insomnia. None of our patients became addicted to alcohol because of their alcohol use during withdrawal. Most of them scarcely noticed the effects of the alcohol in the traditional sense; their limbic centers were so stripped of dopamine that they never felt even a slight buzz from the alcohol. They did feel less frantic and paranoid, however.

It appears that alcohol is less addictive than the antiparkinson's drugs, the lesser of two evils, so to speak. I had two patients who felt strongly that they should not indulge in alcohol, even medicinally. I found it ironic that they felt that it was reasonable to take drugs that are much more powerful, mind-altering, and addictive than alcohol, because the drugs were sanctioned by the doctor. However, every person must decide for himself and feel good about his decisions. But I do note, again, that many patients have found

reasonably small amounts of alcohol to be extremely helpful in combating symptoms of drug addiction. None of them have been addicted or even drawn to alcohol after their recovery. Therefore, we do suggest that you might consider alcohol as an effective weapon in your battle with drug withdrawal.

Massage and therapeutic touch

If you are frightened of being touched, then massage will not be calming, so don't force yourself. But if you can feel comfortable getting a massage, do so, and be sure to tell your massage therapist that you need the room very warm and to go easy on the scented oils unless they are selected specifically for their calming effect. Gentle foot massage can be the least threatening of all the forms of massage, but some people prefer a shoulder massage or having their hair washed and trimmed.

Drug withdrawal sometimes responds to the touch of a living being. It can be a dog, cat, horse, or human. Sometimes, the worst paranoias of withdrawal are triggered by a sense of aloneness. Just having someone in the room is good. Having someone hold your hand is even better. Very often, if you are having trouble with freezing, a friend's hand on your shoulder can help you stay moving. It is very hard for most PDers to ask for help. This is your chance to grow up, and join the rest of humanity in admitting that none of us can go it alone. That "I am a rock, I am an island" stuff is what got you into this mess. Try reaching out for help or asking someone to hold your hand. You might be surprised at how willing we all are to help.

Some patients who are open to new ideas have found that New Age therapies such as aromatherapy and chakra balancing are beneficial. Find what works for you and do it.

Mood

Stay busy. Watch comedy videos and DVDs. Get dressed in the morning instead of lounging in your bathrobe. Most importantly, get out of the house.

"I don't want anyone to see me looking like this," is the defensive cry of the junkie who is crashing. But getting out and getting your mind off yourself is the best way to renew the influx of joy that makes the dopamine flow. We all give this advice to others when they are feeling miserable, but when it's a question of doing it ourselves we are more reluctant. Forget that you look haggard and weary, and too bad that you are tremoring – it's how you are. Your true friends will understand and the others can think what they want.

I hear PDers say over and over again, "I want to get better without anyone seeing me struggle, so that no one will ever know I had Parkinson's." This is sheer vanity, but it is especially common with this illness. It goes back to the original fear of letting anyone know that you are hurt. For those of you whose backs go up when reading this, who want to tell me that it is normal to hide weakness, let me say that this is not true. There are millions of people who, when they are hurt or sick, seek sympathy and help. You, if you have Parkinson's, probably dismiss those people (most of the people in the word) as weenies and weaklings. But they don't have Parkinson's now, do they. So, as you would recommend to others, get out of the house. Let people see you having a hard time, tell them about what you are doing, and be surprised at how they respond with compassion.

As for music, it has been shown that classical music and singing can cause a brain override that will allow movement in a person who is frozen in place from low dopamine

levels. I did an experiment with a patient who was frozen. We were at his apartment, and he was stuck in his bedroom doorway when I arrived; he had been there for half an hour. He whispered that he knew he wouldn't be able to walk until I arrived to help him. I didn't want to encourage this line of thinking, so I stood back and suggested that he sing. He pouted and said he didn't know any songs. He had been to a concert the night before: The Messiah. I told him to sing the Alleluia chorus. He laughed at the idea, but I insisted, and started singing it. Within two measures of him joining me in song, he was walking normally. He was adamant that it was my presence in the room, but twenty minutes later he got stuck again, struck up the chorus, and resumed moving. Music really does work to override the panic zone. Studies have shown that rock music is not particularly effective, and that classical or sing-along music is the best. A dark, silent house is a formula for paranoia. Keep the lights on and the music playing, and good food on hand.

Stressors

While getting out of the house and walking down to the mailbox might be a good thing, events that put you in the spotlight are probably a poor choice. Public speaking, running a fund-raiser, and these kinds of stressful activities may make you more edgy. You may wish to avoid this sort of thing. Many people find that they need to take time off work in order to take care of their health.[1]

If your great aunt drives you batty, or the grandchildren are going through a screaming phase, assert yourself. Don't have them over to the house, and don't go visiting even though your daughter wants you to babysit. You simply can't do all the things that you used to do, especially the things that made you grit your teeth.

Acupuncture

The NADA acupuncture protocol for relieving the stress of detox and drug withdrawal is highly effective. It is used in drug reform clinics with a higher rate of success than any other single method.

The protocol consists of needling five ear acupoints. In order of insertion, these points are: sympathetic,[2] Shen men, kidney, liver, and lung (either one of the two lung points). For pregnant women, the protocol is only three points: Shen men, kidney, and lung.

This treatment is done daily for several months or until the withdrawal symptoms begin to ebb. Usually it is done on alternate ears each day. The needles should be left in as long as possible. In some clinics, the needles are left in for an hour. In China, patients are often sent home with needles still in, and the patients can remove the needles several hours later, at their convenience. In the United States, because acupuncture needles are considered a medical device, and because they must be disposed of in special, red, medical waste containers, very few practitioners allow their patients to go home with the

---

[1] Your health plan, disability insurance, and doctor will most likely not support you in this. Their attitude will be that you should just adhere to the current standard for medical care, which means take the drugs and don't make waves. You are on your own on this. Please do not write to me asking for some sort of note that you can give your boss so that he will give you time off or trigger your disability insurance. I do get these requests, and I am not in a position to answer them individually. This book is my statement.

[2] "Sympathetic" refers to the effect on the sympathetic nervous system, the system that pumps up the adrenaline during a time of crisis.

needles in place. Although several hours with the needles in can be helpful, must of the benefit occurs in the first fifteen minutes.

Sometimes, if anxiety or paranoia is present, it can be helpful to be needled at acupoints P-6 and Ht-7 as well. The acupuncturist should choose the arm that is tremoring the least and observe the needles for several minutes to make certain that they are not going to be expelled by the tremoring. These needles are left in for the more conventional half-hour to an hour. If confusion or lack of focus is more of a problem than anxiety, acupoints Yin Tang and Du-15 or Du-16 may be helpful.

Know the nature of your challenge

Know the difference between Parkinson's and drug withdrawal. Do not assume that everything that is bad is a worsening of Parkinson's disease. If you do, it will be easy to justify increasing the medication. After such an increase, there will be a return of all those symptoms that drove you to make a reduction in the first place. For example, suppose you reduced the meds because of ticcing, facial grimacing, or other symptoms of dyskinesia. After a few weeks, the anxiety, immobility, and shaking of drug withdrawal will make you look back with rosy glasses upon the dyskinesias. You may be able to convince yourself that the spasming wasn't so bad. But if you start taking the drugs again, sometime in the next few days or ten weeks, the violent ticcing, inability to swallow, or the facial spasms might reappear, together with the overly bright eyes and inability to think clearly, and your drug withdrawal work will have gone for naught.

Keep clearly before your mind's eye, or your caregiver's mind's eye, why you decided to reduce your medication. Only in this way can you hope to prevent seesawing back and forth between drug reduction and drug resumption. If you keep in mind just why you started the drug withdrawal to begin with, you may be able to keep a clearer head during the hellish withdrawal phases.

Antianxiety drugs and sleeping pills

Our patients have gotten into serious trouble by accidentally abusing prescription antianxiety drugs or sleeping medications. These pills are valuable tools and can be very helpful if used correctly. However, most of them are extremely addictive and are only supposed to be used for a short period of time: a few days, not a few months. Here are examples of each.

Ambien

Ambien, a sleeping aid, is recommended to be taken for no more than seven to ten days. It comes with a warning that patients may start hoarding it or self-overdosing, and it has a high risk of habituation and dependence. (These are more euphemisms for addiction.) If you need chemical help with drug withdrawal, which lasts for months, you might not want to take a drug which is addictive and which should not be taken for more than a week. Please note that the listed side effects of Ambien include depression, lethargy and "sleep disorder." From our experience, we have seen that one "sleep disorder" caused by Ambien is, surprise, insomnia. (Many of these drugs have side effects that are the opposite of the drug's intended purpose. Sleeping pills can, after several uses, cause insomnia; calming pills can eventually cause anxiety.)

Xanax

Xanax, a popular antianxiety drug, is so addictive that, after prolonged therapy, the manufacturers suggest a 2 to 3 month gradual withdrawal period. "Prolonged" is not defined by the manufacturers. Based on our experience, "prolonged" is anything more than two weeks. Be aware that this drug is advertised as non-addictive.

Note these listed side effects of Xanax: tremor, confusion, insomnia, and nervousness. We have seen all of these side effects in patients who were prescribed this drug to combat their drug withdrawal problems. These side effects are not immediately apparent. They showed up after several weeks. By the time the side effects appeared, it was too late for our patients: they were addicted. Also, the listed side effects of tremor and nervousness will not necessarily go away; they are usually semipermanent.[1] If your withdrawal symptoms include tremor, nervousness, and insomnia, you may amplify all of your original problems by taking this drug.

Eli

Eli had debilitating weakness and severe insomnia – he could never sleep longer than 5 minutes at a time. He found that his sleeping time extended to several hours when he weaned himself off Ambien. His doctor had prescribed Ambien because his sleep cycle was only several hours long. This short sleep cycle was probably due to Eli's Sinemet. While Eli was taking the Ambien, his sleep cycle shortened to 5 minutes. He was terrified to reduce the Ambien, thinking that without it, he would never sleep again. When Eli finally got off the Ambien, his sleep cycle improved back to several hours, just where it had been prior to the Ambien. When he got off Sinemet, his sleep cycle lengthened further still. Over the months it lengthened to 5 hours a night and stayed at this level for over half a year until he died one night, in his sleep, of a massive stroke.

Family Counts

Next, family members who do not know what is going on will not be helpful. Although the spouse, caregiver, or friend who is working closely with the patient might understand what is going on, the children may not. They may be furious. They may only want the parent or Old Friend to be "happy" again. When Hjalmar's wife was so grateful to have her thoughtful husband back, the adult children were angry because daddy was looking less (mindlessly) cheerful. An outsider only sees the large motor movements or hears the loud voice. An outside observer cannot see the subtle changes in mental clarity and the subtle changes in motor skills. These outside observers will usually want mommy

---

[1] Oliver Sacks proposed that the term "side effects" should not be used to describe symptoms that are set in motion by a drug which become permanent. He felt that the general understanding of the term "side effect" was an effect that a drug caused, and *which ceased when the drug was no longer taken*. The permanent and semipermanent damage caused by so many of these drugs is not a "side effect" in the traditional sense.

However, for reasons that are only too obvious, the drug industry continues to lump together both the short term and long term consequences of drug use. Actually, to be fair, the wording is changing. Although patients and doctors still speak in terms of "side effects," the official drug company inserts and warnings use a new wording: "adverse effects." I'm not sure what legal decision prompted this change of wording, but it still avoids the issue, which is warning patients that these adverse effects may endure even after the drug use is ended.

or daddy to just keep taking the pills. They will be understandably distraught that their crazy parents are not following the doctor's orders. This is often the most painful part of the process for the person who is trying to reduce medication. Nearly all of the patients who have Concerned Relatives say that the C. R.'s won't read any of the literature, they won't get involved, and they won't take the time to learn a little biology and/or chemistry and try to understand the complexities of what is happening. My feeling is that it is best to get the family on board before you even get involved in recovering. It is very hard to convince a family member that you are doing better if you are staggering through the throes of drug withdrawal. Over and over again with each 10% reduction, your family members will see you going into a pit of despair. And they will see that every time you finally start to feel better and to get your head above water, you will reduce meds again and go back down in the soup. They will fear for you. Help your loved ones to know what you are doing and why.

Summary

Stay warm.

Eat very, very well.

Indulge yourself with massage or body work.

Keep your mood uplifted: get out and about, listen to music, sing out loud.

Avoid stressors.

Get acupuncture or whatever form of physical therapy is available and acceptable to you.

Keep a journal or some record to remind you why you are going through this.

Read carefully the warnings and adverse effects for any prescriptive drug that supposedly eases anxiety and insomnia; many of them will cause worsened anxiety and insomnia in the long term, plus they are highly addictive.

Appendix 5

WHY YOUR DOCTOR THINKS WHAT HE DOES:

FIFTY YEARS OF CHANGING DOPAMINE THEORIES

Why read a history of dopamine?

One of the most difficult challenges for people with Parkinson's disease can be daring to disagree with the medical/scientific world. The same unquestioning reverence that was once given to high priests is now, in these modern times, sometimes accorded to medical doctors. Despite the constant changes in medical theories, doctor knowledge is, to some, High Knowledge. Even though there can be scant conformity of medical theory from one USA MD to another on certain issues, patients can have emotional reluctance to breaking through the "white coat barrier" and thinking independently about their health issues. And yet, so-called Scientific Facts cherished by one doctor may be laughable history or radical unsupported nonsense to another doctor. Still, most non-medical US citizens turn to their MD when they want "the answers."

When PDers realize that they must find their own way amongst conflicting medical theories, they often are confronted with hostile opposition from family members who have blind allegiance to anyone wearing a stethoscope. The inner-family conflicts about the right medical course to pursue when a loved one is diagnosed with an "incurable" illness can be as painful as religious schisms. Therefore, to muddy the waters further still, this appendix will give a quick history of the science of dopamine to explain how the current medical conflicts of opinion arose.

Medical differences of opinion

Many of my patients unfairly expect their doctors to be up to date with the rapidly changing theories of medicine, pharmaceuticals, and chemistry. It is impossible for a clinical (working with patients) doctor to keep up with the crush of new information, much of which is in full opposite, 180º conflict with the theories that he was taught in school. For an example, this education gap leads to our current medical schisms in which some neurologists proclaim that PD drugs are safe and others warn that the drugs are dangerous. Some older neurologists insist that levodopa is the best drug and some of the youngest doctors point to the agonists as the only sensible choice. A few neurologists still tout Eldepryl! Based on our close experiences with the prescribing patterns and the reactions to new ideas of over thirty neurologists, and our long distance experiences via emails regarding prescribing patterns and opinions of hundreds of others, it appears that the weightiest factor in determining a neurologist's opinion about the drugs is this: those drug trends that were popular in the year the doctor graduated from med school. Despite continuing education courses that tout the newest theories and hypotheses, what a doctor learned in med school is most likely to remain as his body of "facts" against which all new information will be warily judged.

Changing facts – a personal confrontation

The history of dopamine provides a nice example of the way in which science anoints mere conjecture as solid fact and then, as these "facts" are found false, creates convoluted logic in a desperate attempt to keep the "facts" from becoming laughing stocks.

In 1998 I was personally involved in the quagmire of false dopamine information. When I tried to publish my first research article – which included as a sidelight an explanation of the electrical circuitry that causes brain dopamine to be abundant when one is awake and to diminish during sleep – the editor balked at printing that small, awake/asleep dopamine-related portion of the paper. At that time, the facts about daytime/nighttime dopamine were the exact opposite of my finding, and had been for over forty years. As an aside, right now, a mere six years later, the scientific research community has done an about face on the dopamine awake/asleep issue, reversing their earlier, "scientific" fact by 180º. Still, for the most part, doctors who went to school prior to 1998 are still adamant adherents to the old dopamine at night "fact" even though teenagers now learn the new, opposite "fact" in high school biology classes.

Respecting the past

It is difficult to write about the history of science without sounding smug. It's easy to smirk at the past, but, though gratifying, it's not actually productive to say, "Look how stupid our ancestors were! We are so much smarter now." The one lesson that history of science has to offer us is that every generation has laughed at the preceding one, and that those scoffers were in turn found to be fools and simpletons by the next generations of truth holders. We have to assume that we are in the same situation; today's science is going to look pretty shabby in another fifty years; most of today's facts will be proven to be glaring falsehoods, most of today's medicine – like last year's medicine – will soon be proven to have been worthless, if not damaging.

The following pages show that the ideas about dopamine have taken a major shift every ten years. The ideas to which your doctor probably adheres will probably depend on which decade he attended school. Note the date on some of the outmoded dopamine research; you may be surprised at how recent some of the now outmoded theories are. For fun, figure which era's dopamine theory corresponds to the year that your neurologist probably graduated from med school. For a real eye opener, calculate what the age of your neurologist's *teachers* might have been when they, the teachers, were in school. You will realize that your neurologist may have been instructed by professors who did their own college learning prior to the discovery of dopamine![1]

---

[1] Do not imagine for a moment that a professor is necessarily up to date. In 2001 one of my students attended a lecture given by an older Harvard psychologist who explained that "the brain is like a switchboard; pathologies are caused by incorrect hook-ups." This idea, popular in the 1950's, is utterly outmoded.

The current (and subject to change) model of the brain is one of an ever-changing mass of interrelated components. The chemicals in the brain create minute electrical fields, and the larger electrical fields running along the neurons alter the shapes of the brain chemicals. Even the molecules themselves have been shown to shapeshift, so that a molecule with a particular role can change its three dimensional shape without actually changing its atomic components, and can, in its new shape, perform a completely different function than it performed in its previous shape. (Footnote continued on next page.)

Very few doctors have the time to keep up with changing theory; they are lucky if they can keep up with the new medication ads in the Sunday newspaper supplement and the changing legal requirements of practicing medicine in a litigious society.

To enable the modern PDer to compassionately understand why his doctor thinks the way that he does about Parkinson's disease and dopamine, even though that doctor may be in conflict with the latest research, I am presenting this appendix on the history of dopamine theory and how it relates to the underlying philosophy of modern science. Biology research is only slowly reflected in medical changes. For example, many of the long-*dis*proven theories of biology that you will read about below are still used today, decades after having been disproven, as the basis for drug treatments. Not only that, but it is not uncommon for doctors, *especially* the top doctors and leaders in their field, to still subscribe to old knowledge that has long since been proved wrong; they are often the ones who, in their early research days, discovered the now-outdated information, and they will very often fight until their death against the newer findings. Also, money is at stake; research grants most often support building upon previous research; even when the old information has been proven to be outmoded, the research grants are still more likely to go to someone experienced, someone who has established his reputation by creating the old theories and who, coincidently, has a vested interest in fighting against the new changes.

It now appears that biological molecules can change their shape in response to changes in their cellular and extracellular environment. And yet, the crude idea of brain as switchboard is still being expounded by hoary profs!

The brain, once thought to be a static mass of unchangeable cells, is now held to be ever changing. The brain of ten minutes ago has been irrevocably altered by the events of the last ten minutes. In the time you have spent reading this book, your brain has already altered to accommodate what you've read, what you've eaten today, and the good and bad news you've heard on the radio. You truly do not have now the same brain that you had when you started reading this book. Not only that, but the way in which the brain integrates new information may be random!

In contrast to the static switchboard model still being taught today by some elderly, highly respected PhDs, here is a current (March, 2002) description of the brain: "At a conference of the British Association for the Advancement of Science last fall, neurophysiologists from the University of Cambridge revealed that nerve cells respond to the same stimuli differently every time. 'It seems the brain has a sort of cerebral roulette wheel,' said researcher Roger Carpenter. Randomness may be a valuable survival tool." (Kathy Balog et al, "Heads We Go, Tails We Stay," *USA Weekend*, Santa Cruz Sentinel, March 1-3, 2002.)

A powerful condemnation of the 19th century "clockwork" approach to brain function still revered by some doctors was offered by Arnold Mandell, the San Diego psychiatrist and dynamicist who pioneered the applications of modern (late 20th century) scientific theories in physiology. In his dissection and condemnation of overly simplistic, linear, and reductionist methods of regarding the brain, he wrote, "The underlying paradigm remains: one gene ➔ one peptide ➔ one enzyme ➔ one neurotransmitter ➔ one receptor ➔ one animal behavior ➔ one clinical syndrome ➔ one drug ➔ one clinical rating scale. It dominates almost all research and treatment in psychopharmacology. More than 50 neurotransmitters, thousands of cell types, complex electromagnetic phenomenology, and continuous instability based on autonomous activity at all levels, from proteins to the electroencephalogram, – and still the brain is thought of as a chemical point-to-point switchboard."

Gleick, an author in the field of chaos theory, in his commentary on the above statement wrote, "To someone exposed to the world of nonlinear dynamic the response could only be: How naïve." (J. Gleick, *Chaos*, Viking/Penguin, New York, 1987, p. 298-299.)

Sacred tradition

The science, philosophy, and religion of any given people or age must be synchronous; they must fit hand in hand. The so-called facts of science are actually descriptions of phenomena that have been carefully phrased to coordinate with the politics and religion of the hour. The corollary is that, once a scientific theory becomes established, it is as difficult to change as a cultural or religious tradition. The Parkinson's/dopamine theory is no exception. Before I jump into the dopamine history, let me give an example of how difficult it is to change a medical tradition. I will share this tidbit from a recent Parkinson's disease convention. A respected MD and PhD researcher from Columbia University wrote up the report on which the following section is based.[1]

A disappointing dopamine conference conclusion

Various papers had been read at the conference, the sixth International Congress of Parkinson's Disease and Movement Disorders, all proving that using dopamine agonists instead of L-dopa as the first course of treatment, or as an adjunct in combination with reduced levels of L-dopa, was much safer, provided better coverage of symptoms, and greatly extended the effective years of the medication (the time period before the drugs developed adverse effects). At the close of the conference, after the reading of all the papers, the doctors in the audience were asked to indicate, by a show of hands, whether or not they would be willing to consider using the safer drugs or modifying their prescriptions of L-dopa, substituting, where possible, the agonist drugs which had been proven, in test after test, to be safer and more effective.

The writer of the article expressed strong dismay as he reported that, surprisingly, even after hearing paper after paper proving that agonists were safer, had fewer side effects, and gave more years of benefit before the adverse effects began, still a majority of doctors in the audience raised hands to indicate preference for continuing to initiate treatment of PD with levodopa – the treatment that had been proven most damaging – rather than start with one of the new agonist drugs.

The author, in his critique of this response, offered up his own suggestions as to why on earth his peers would be so hidebound. He suggested that "[a] possible explanation is that ease of use (some agonists require a much slower titration of dose than levodopa before reaching the same effectiveness)…may play a bigger role in a practicing clinician's choices than the conclusions of a drug study." He also suggested that habit may play a part. Despite his several possible excuses for his peers' reluctance to change, his writing clearly showed he was both surprised and disappointed in the show of hands; the attendees, for the most part, indicated that doing what was familiar or easier was more significant than doing what had been *proven* to be more effective.

In other words, the majority of doctors who attended a conference to learn the most up-to-date information were not willing to change their methods simply because it had been repeatedly proven that their old methods were dangerous and that better methods had been found. In medicine, as in established religion, it can be just too painful

[1] Pietro Mazzoni, MD, PhD, "Conference Highlights from the Sixth International Congress of Parkinson's Disease and Movement Disorders." *Medscape Neurology*, Feb. 2000.

to upset the status quo. The status quo seems to depend on what year the doctor attended medical school.

So let's look at a decade-by-decade quick review of neurotransmitter history. The time frames below refer to discoveries in biology research, *not* to changes in medicine necessarily. The transfer of new information from the realm of biology research into the realm of medicine is notoriously slow.

A BRIEF HISTORY OF DOPAMINE AND OTHER NEUROTRANSMITTERS

Pre-1930's

Acetylcholine, a molecule produced by nerve cells, was found to be stored in motor nerve endings. When a motor nerve is stimulated, its acetylcholine supply is released from storage and sloshes across the gap between its parent nerve and the next nerve in the chain. The receptor nerve, stimulated by the presence of acetylcholine at its receiving end, discharges *its* stored portion of acetylcholine from the far end of the nerve. The process continues from one nerve to the next, thus transmitting a nerve stimulation impulse from one nerve to the next. Via acetylcholine, a long chain of nerves can transmit a motor impulse all the way from the brain down to a muscle, where the acetylcholine from the final nerve in the sequence causes the muscle to contract.

The discovery that nerves could communicate their stimulation signals via tiny chemical compounds stunned the physiologists of the day. Since the days of Galvani's electrical experiments with frog legs up until this time it had been assumed that that electricity was solely responsible for nerve signal conduction.

This new type of chemical was named a neurotransmitter. The Nobel prize was awarded in 1936 to Otto Loewi for showing that acetylcholine was indeed a neurotransmitter, a neural chemical communicator. A fun tidbit: the idea for Loewi's prize-winning work came to him in a dream.

The assumption at this time was that there was only one neurotransmitter. Acetylcholine was *it* – end of story. Most of your older doctors will remember their first lab experiments in biology class when they splashed acetylcholine onto frog legs and watched how the legs twitched, jerking back and forth in response to the molecule. But what does this have to do with dopamine?

1940's and 1950's

In the 1950's, decades after acetylcholine was discovered, a few other "communicator" chemicals were found to be splashing around in the body, dopamine among them.[1] Dopamine, made in the adrenal gland (just above the kidney), was deemed to be of importance in regulating blood pressure and urinary output. It was used in the treatment of shock.

[1] Dopamine research started in the mid 1950's. Nicotinic acid (derived from tobacco) was discovered to be a nerve activator (an acetylcholine agonist) in the 1940's, prior to the discovery of dopamine. As late as the 1970's, acetylcholine, dopamine, epinephrine (adrenaline), and serotonin were considered by most biologists to be the only neurotransmitters, or certainly the only ones of any consequence. Actually, norepinephrine and epinephrine were considered for many years to be hormones, not neurotransmitters, because of the association of epinephrine with the adrenal gland.

Nomenclature

A determination was made that compounds that were made and stored in nerve endings should be called neurotransmitters. The chemicals that were made and stored in glands should be called hormones. Neurotransmitters traveled the merest of distances, from one nerve to an adjacent nerve, and then back again. Glands, on the other hand, could eject chemicals into the bloodstream, whence they could travel throughout the body in search of their particular target organ. Using this new definition, dopamine, which was made in the adrenal gland, was a hormone.

Dopamine, the hormone

Eventually, dopamine, adrenaline, and norepinephrine presented some labeling problems. Even though they were made in the adrenal gland, they appeared to act on nerves as well as on organs such as the bladder and kidney. This was in conflict with the idea that hormones should act solely on organs. An even greater problem arose when it appeared that brain cells were affected by dopamine. (This research was done on mouse and/or rat brains.) The difficulty was this: dopamine was made in the adrenal glands (down by the kidneys), thus dopamine had an effect on kidney and bladder function, *but* dopamine also affected neurons in the brain. Norepinephrine, also made in the adrenal glands, like dopamine, had an expected effect on nearby kidney function but also seemed to affect the brain. A new description was needed for these compounds that were made in glands, like hormones, and which affected organs but which also affected nerves.

Brain messengers

A happy solution was settled on. The chemicals that were made in glands but which affected brain neurons were renamed "brain messengers." This solved the problem, but created a new one. How did a given molecule of dopamine know whether or not it was supposed to be a brain messenger, affecting the brain, or a hormone, affecting organ behaviors? This problem was never resolved, but thousands of college biology majors and pre-meds, this author included, dutifully memorized the terminology. No one knew how or why the chemistry pulled it off, but dopamine was a molecule that performed both as a hormone and brain messenger.

Dopamine and Parkinson's disease

By the late 1950's it was established that the Parkinson's brain was deficient in dopamine.[1] The mechanism remained unclear. As recently as 1989, the wording in *Taber's Medical Dictionary* was a vague note that this secondary (brain) role of the brain messenger dopamine was "implicated" in some forms of psychosis and abnormal movement disorders such as Parkinson's disease.[2]

---

[1] I must assume that this was determined using autopsy, the only method available at that time for assaying dopamine levels in the brain. I have not found definitive information on how this deficiency was discovered. Most of the research of this era was done on rats. I suspect that it was probably in rats that the blood-brain barrier's (BBB's) impermeability to dopamine was first seen, and also the subsequent discovery of L-dopa's permeability across the BBB with its subsequent conversion in the brain into dopamine.

[2] *Taber's Cyclopedic Medical Dictionary*, p. 524.

Failure to respond to dopamine

Despite the proposed shortage of dopamine in Parkinson's brains, administration of dopamine hydrochloride (a form of dopamine usable by the adrenal gland) to PDers did not seem to benefit Parkinson's patients as anticipated. Therefore, although dopamine was implicated in abnormal movement, the relationship was unclear.

When it was later found that dopamine molecules could not cross the blood-brain barrier, this explained why oral or injected dopamine had not alleviated PD symptoms. (In the early research, as now, confusion about levels of blood dopamine relating to amounts of brain dopamine led to wrong conclusions!)

The finding that dopamine could not cross the blood brain barrier meant that, possibly, dopamine made in the adrenal gland was not a brain messenger at all! Evidently, the dopamine that was used in the brain was actually manufactured in the brain! The brain was made of nerve tissue. If dopamine was generated in the brain, this meant that it was made by nerves, for nerves. It began to appear as if dopamine, with its dual role of hormone and brain messenger, was also a neurotransmitter. The finding that brain dopamine was made in the brain rather than being transported there from the adrenal gland was the beginning of the end of brain messenger theory. (Looking ahead, by 1990, the term "brain messenger," would be hopelessly passé in research circles, but as of this writing, in 2003, some MDs still imagine it to be current.)

Two neurotransmitters

A larger problem was looming, one that would have consequences for the next forty years of Parkinson's research. New research indictated that dopamine had an effect on motor function: dopamine appeared to have a motor effect when applied to the brains of rodents. This was alarming news. It was established fact that acetylcholine was the neurotransmitter of movement; how could there be two movement neurotransmitters?

A solution was proposed and immediately accepted: acetylcholine caused muscles to tense up; dopamine caused them to relax. This was not based on any research whatsoever – it was pure hypothesis, and was immediately embraced and added to the facts of neurology. This new decision, that dopamine caused muscles to relax, created a large group of corollaries, and a new (now proven wrong) theory about the cause and treatment of Parkinson's disease, one that would not die quickly.

1960's

The nineteen sixties saw the creation of two new theories about Parkinson's disease. These new theories were based on two new drugs: one that could suppress acetylcholine and another drug that, once inside the brain, could convert into dopamine. Although both of these theories have turned out to be wrong, one can understand why researchers made the guesses that they did. Many doctors still subscribe to one or the other or both of these two outmoded theories.

Levodopa, a variant of dopamine

Some researchers noticed that brain dopamine, the molecule that had been labeled a muscle relaxant, was deficient in people with Parkinson's disease. This theory could not be tested in living patients, however, because dopamine, as noted above, was not able to pass through the blood-brain barrier. How then could one get dopamine into the brain to

test this theory? A solution to this problem came when a precursor molecule for dopamine, levodopa, or L-dopa, was shown to pass through the blood-brain barrier.[1] Once inside the brain, this precursor was rapidly converted into dopamine.

Levodopa experiments were then tried on human subjects with Parkinson's, with poor results. Although it appeared, based on chemical assay of rat brains, that levodopa did convert into dopamine in rodent brains, people with Parkinson's did not respond favorably to levodopa. Because people with Parkinson's did not respond to dopamine therapy, the theory that dopamine was responsible for Parkinson's disease stumbled.

The opposites theory

At this time it was not yet certain by any means that low dopamine levels were actually the cause of Parkinson's disease. This hypothesis was based, in part, on the theory of dopamine being a muscle relaxor, an opposite of acetylcholine. If there *were* two neurotransmitters instead of just one, if both acetylcholine *and* dopamine could affect motor function, the *two neurotransmitters must obviously have equal and opposite functions*. (Dear reader, don't even begin to worry about why they had to be opposites. It was an idea, the idea became published, and even today, now that more than 60 neurotransmitters have been found, there are still adherents to the "opposites" theory of neurotransmitters.)

An entire theory about Parkinson's disease was created to explain how a dopamine shortage must – via opposite theory – create an acetylcholine excess. This new theory required a new interpretation of all the symptoms of Parkinson's disease. According to this theory – one still held by elderly MDs – the symptoms of Parkinson's are caused by excess acetylcholine, acetylcholine no longer held in check by dopamine.

Because acetylcholine causes muscle tone, or muscle tension, the symptoms of Parkinson's were redescribed as being caused by excess muscle tone. Symptoms such as masked facial expression, which looks for all the world like sagging, lifeless muscles, were attributed now to excess muscle tension! In textbooks of this period, the feeble tremor of Parkinson's was attributed to excess motor strength. The heavy weariness of Parkinson's was described as a struggle against excess muscle tone. Although people with Parkinson's insisted that this was not the case, that their problem was difficulty in figuring out how to initiate movement, they were assured by the more modern doctors of this period that, in fact, their Parkinson's was due to excess motor function and too much muscle tone.[2]

---

[1] Technically, DOPA should be all capital letters, as it is an acronym for di hydroxy phenyl alanine. In much of the older, technical writing, it is all caps. However, it is common now to see it as "dopa," or "L-dopa."

[2] Michael J Fox, a popular actor who has Parkinson's disease, wrote in his autobiography that it felt to him as if his Parkinson's disease was coming from his foot – which it was. His doctor assured him that his hunch was wrong. This modern problem of doctors denying a person's intuition about the source of a malady is particularly repugnant in light of the constant stream of errors and retractions coming from the medical community. Insult is added to a patient's injury or illness when the medical community, so rife with unproven hypothesis, squelches these insights from patients. It seems to me that if more doctors listened to their patients, they might find the cause of illness much more quickly. In the naturopathic school of medicine, it is said, "If you listen to your patient long enough and let him speak freely from his heart, he will eventually tell you, whether he knows it or not, exactly what the nature of the problem is, and what is needed to cure it, even though he may not consciously know the root of the problem nor its solution." Such listening requires not only extreme patience, but also humility.

Questioning the cause of Parkinson's

The older doctors were still uncertain. At this point no one had proved that dopamine deficiency was the cause of Parkinson's. Some still held to the turn of the century idea that Parkinson's was fear-based. Others were still interested in Charcot's 19th century idea that Parkinson's was an electrical disorder. (Both of these hunches, as it turns out, were partially correct.)

Proof: acetylcholine excess is the cause of Parkinson's disease

In the early 1960's a belladonna-related chemical was shown to reduce Parkinson's tremor. This chemical was found to be an anti-acetylcholine molecule, also called an anticholinergic. Because this drug reduced tremor by reducing acetylcholine, it offered proof of the cause of Parkinson's disease: Parkinson's was caused by excess acetylcholine. Again, the proof was this: both tremor and also the high anxiety level of some PDers were reduced following the application of anticholinergic medication. Therefore, Parkinson's was caused by too much acetylcholine.

This school of thought had many adherents despite the curious observation that the other symptoms of Parkinson's disease (poor balance, slowness of movement, and rigidity) were all unaffected by anticholinergic medications. In fact, it did appear as if just the opposite was the case – anticholinergics *increased* slowness of movement. However, since the only drug that appeared to have any benefit whatsoever for PD patients was an anticholinergic, Parkinson's disease was, beyond a shadow of a doubt, caused by excess acetylcholine. This became one of the mid-twentieth century facts about Parkinson's.

Proof and corollaries

1) Medication that decreases acetylcholine (anticholinergics) reduces the cause of Parkinson's disease tremor and anxiety.
2) Anticholinergics, drugs that reduce muscle tension, are an effective treatment for Parkinson's disease.
3) People with Parkinson's disease have excess muscle tension. This muscle tension is the cause of their slowness of movement, rigidity, tremor, and balance problems. This tension is obviously caused by an excess of acetylcholine. (This was obvious because acetylcholine was at that time accepted as the neurotransmitter that caused muscle contraction.)

Please do not memorize the above. It has since been proved false.

Gallons of levodopa – a new approach

Meanwhile, experiments were ongoing with levodopa, trying to figure out why people who supposedly had acetylcholine excess didn't respond to levodopa. Because levodopa was a muscle relaxant, a supposed opposite to acetylcholine, it was assumed that Parkinson's could be managed by either using anticholinergics or by using dopamine. To this way of thinking, dopamine was, in effect, a form of anticholinergic.

Rat brains were able to convert levodopa into brain dopamine. People with Parkinson's, however, did not respond whatsoever to oral doses of levodopa. The failure of levodopa to help people with Parkinson's nearly rang a death knell to the theory that dopamine deficiency was a factor in Parkinson's disease.

Then, in 1967, a bold researcher, Dr. George Cotzias, tried using doses of L-dopa that were *thousands* of times higher than had been used previously. At these shocking, stunning levels of levodopa, an amazing result was obtained: people with Parkinson's appeared quickly to regain their lost motor function! Dr. Cotzias was hailed as a hero, and L-dopa was rapidly pronounced, by many, to be the cure for Parkinson's disease.

Proof: Parkinson's is caused by insufficient dopamine

In the late 1960's, it was clear that people with Parkinson's were able to move more easily, albeit only somewhat normally, when their brains were flooded with levodopa. They also developed bizarre side effects from the drug. These side effects were attributed to various causes, and whole new theories sprung up. (One of the earliest theories, blood dopamine versus brain dopamine, still has adherents in the first decade of the twenty-first century, even though research has repeatedly shown that levodopa's adverse effects are due to brain levels of dopamine.) This finding led to some new (and since proven wrong) facts about the function of dopamine.

Corollaries

(Note that these corollaries are added onto the corollaries about acetylcholine excess. Unless absolutely necessary, outmoded medical theories are NOT abandoned. New theories are expected to build on existing theories.)

1) Dopamine allows people with Parkinson's disease to move, somehow neutralizing the tension from excess. Therefore, dopamine is *obviously* a relaxant.

2) Correct muscle movement requires a balanced blend of acetylcholine, a muscle tightener, and dopamine, a muscle relaxant.

3) Parkinson's disease is caused by an imbalance between acetylcholine and dopamine.

4) Since dopamine is a relaxant, it is undoubtedly released in large quantities during the night and especially during sleep. Adversely, during waking hours, dopamine levels decrease.

The above is wrong

Please do not memorize the above. It has all been proved false. High school kids today can tell you that the above is incorrect.[1] Your doctor, however, may not know that

[1] I was sharing the material from this appendix at a weekend seminar. When I listed these above four points, I was interrupted by a high school student who chided me, "That's not the way dopamine works. We learned about dopamine in health class, in the unit on drug and alcohol abuse. It's a disinhibitor." This was in the year 1999. And yet the old theory of dopamine as relaxant was still being taught in medical schools up into the 1990's. To write an example here, I just grabbed off my shelf *Principles of Anatomy and Physiology*, G. Tortora, N. Anagnostakos, fifth edition, 1987, Harper & Row, NY, p 332. (Note: "DA" is biologist code for "dopamine.") This medical school text states that PD is caused by "severe reduction in DA in the basal ganglia…(and) diminished levels of DA cause unnecessary skeletal movement." There you are – not enough DA causes too much movement. Dopamine is a relaxant. In the same book, on p. 325 in the section on dopamine, the book states "DA leads to inhibition."

An example of how new data that conflicts with a cornerstone theory must be twisted to fit the existing theory is on this same page. It had been observed for nearly two decades that L-dopa caused excess movement in PDers, not increased relaxation. To make this conform to the existing "facts," this movement was explained away thusly on p. 325: "DA is involved in gross, subconscious movements of skeletal muscle." (Footnote continued on next page.)

this is no longer current. In retrospect, it is easy to see how the acetylcholine/dopamine imbalance conclusion came about. When I was in college back in the 1970's, busily engaged in growing germs in a petri dish, the above is what I was taught. I was taught it, by the way, not as hypothesis, but as stone cold fact.

Problems with the dopamine deficiency theory

PDers and their doctors soon noted that, while PDers could move more quickly after administration of levodopa, many of their other PD symptoms, such as tremor, cogwheeling of wrist and ankle joints, and balance problems very often did not respond to levodopa. Also, despite regular daily dosings of levodopa, PDers found that their condition continued to decline. This led the deeper thinkers of the medical world to refute the suggestion that levodopa was a solution and that Parkinson's was due to a dopamine deficiency. These doubters, as it turned out, were correct – but since the only treatment available that gave short-term relief was L-dopa, most doctors bought into the theory that dopamine deficiency is the cause of Parkinson's disease.

False assumptions

The observation that dopamine could impart faster movement to PDers turns out to be as irrelevant to the true underlying cause (original trigger) and correct treatment of Parkinson's disease as the observation that rum or whiskey imparts joy to a person with depression. No reasonable researcher would conclude, based on the temporary uplift from alcohol, that the underlying cause of clinical depression is alcohol deficiency in the brain. And yet a parallel to this supreme bit of illogic was applied to Parkinson's disease: since the bradykinesia (slow movement) of people with Parkinson's was assuaged for a few hours after partaking of levodopa, many doctors signed up to the school of thought that held dopamine deficiency to be the root cause of Parkinson's.

An aside about levodopa research

A famous L-dopa experiment took place in the late 1960's, two years after Cotzias gave massive doses of levodopa to PDers. This later experiment was so dramatic that it was made into a major motion picture (*Awakenings*, based somewhat loosely on the work described by Oliver Sacks in the book of the same name). This well-known experiment involved a group of eighty people with encephalitis caused by a viral plague in the 1920's, who had been in a condition of relative stupor for decades. Under the influence of massive amounts of L-dopa,[1] they were suddenly able to move, talk, and behave somewhat normally after having been more or less in suspended animation for three decades. The experiment developed unexpected side effects, however, as the patients soon began having hallucinations, violent spasming, and personality aberrations.

(Footnote continued from previous page.) This was a way of keeping the old facts intact: DA was defined as a skeletal muscle *relaxor*, a movement *inhibitor* for conscious movement. Therefore, the excess movement that occurred as a result of L-dopa was due to DA having the *opposite* role for *subconscious* movement. There was NEVER any research that supported this idea of DA behaving oppositely in conscious and unconscious movement – the theory simply had to have these two contradictory parts so that the existing DA-as-muscle-relaxor theory could continue to stand in light of the facts that people exhibited excess movement when taking L-dopa.

[1] These dosages were as high as four *grams* (4000 mg) a day.

No matter how the L-dopa was titrated (carefully measured, with incremental increasing or decreasing of dosages), it appeared impossible to find the correct dose for these people, a dose level at which they could move and be awake and yet not suffer the monstrous side effects of the drug. Most of the patients died or had severe setbacks, some within a very short time, others within a few years of the experiments. Although there were a few exceptions where the L-dopa seemed somewhat beneficial if combined with other countering drugs, L-dopa, for purposes of this experiment, was ultimately decreed too dangerous and unpredictable for further use.

1970's

Levodopa use for Parkinson's grew in popularity

Despite the uncertainly as to whether or not Parkinson's was actually caused by a dopamine deficiency, people with Parkinson's were finding L-dopa to be helpful in treating their slowness of movement, even though, within a few years on levodopa therapy, they too were beginning to show side effects similar to those of Sacks' patients. Curiously, people with Parkinson's who were using L-dopa had side effects that were less violent, less unpredictable, in the short term than people with other movement disorders who also experimented with this new drug. This observation, that people with Parkinson's had fewer side effects (at first) than other people with movement disorders, lent support to the faction that considered Parkinson's to be caused by a dopamine deficiency. But even people with Parkinson's fell victim to the unpredictable side effects of L-dopa within five years.

Within five years of Cotzias' experiments, it became apparent that L-dopa was not, in fact, a cure for Parkinson's. People with Parkinson's who had L-dopa therapy developed, within two to five years, traumatic side effects from the L-dopa. Increased doses of L-dopa appeared to accelerate the decline of the underlying PD symptoms and exacerbated the side effects.

L-dopa was no longer considered a cure. Worse, those doctors who had worked closely with Parkinson's patients during the years prior to L-dopa use and were familiar with the normal, undrugged pace of advancing Parkinson's noticed that the PD symptoms in people using levodopa therapy advanced more quickly than should have been expected based on the rate of Parkinson's decline in their undrugged patients. It appeared as if L-dopa accelerated the worsening of Parkinson's disease. Doubts were expressed as to whether or not dopamine deficiency was the cause of Parkinson's after all. Meanwhile, the acetylcholine excess theory was still holding firm in some quarters, and the dopamine theory was also strong. Many doctors did, and still do, continue to embrace both.

Proof: Parkinson's is caused by substantia nigra cell death

Research conducted on brain autopsies of people with Parkinson's presented a puzzle. It did appear that an area in the dark, almost blackish, center of the brain, the substantia nigra, was different in PDers. The cellular change was curious. Some of the cells in this blackish area were no longer black, but had reverted back to a more neutral color. In some, though not all, of the brains there was even evidence of cell death. Because few people suspected at this time that levodopa itself could cause brain cell

death, it was assumed that any and all changes seen in the substantia nigra of PDers, including cell death, must be due to Parkinson's.

This did not prove that changes in the substantia nigra caused Parkinson's, but it did suggest an association. Some people held that Parkinson's disease caused the change in brain cells, others proposed that the change in brain cells caused Parkinson's. Researchers discovered that this darkly pigmented area was rich in dopamine. Jumping to an unfounded conclusion, they decided (erroneously, it turns out), that all brain dopamine was produced in the substantia nigra. This suggested a connection between the facts that 1) PDers had changes in their substantia nigra and 2) they responded to levodopa.[1]

Still, even with all this information, the fact remained: levodopa only addressed a few of the many symptoms of Parkinson's disease. It had not been conclusively proven that dopamine deficiency, and certainly not cell death in the substantia nigra, were the underlying cause of Parkinson's.

1980's

By the 1980's dozens of neurotransmitters had been discovered. Dopamine was no longer considered to be the opposite of acetylcholine. Dopamine had been reassigned: it was now the opposite for serotonin, a frontal lobe neurotransmitter. The "opposites" theory was dying; too many neurotransmitters had been found and their functions appeared to overlap more often than oppose. However, some turn of the millenium pharmaceutical texts still refer to dopamine as the opposite of serotonin.

Frozen Addicts

In 1982 a new piece of information appeared which was vigorously massaged to "prove" the cell death theory of Parkinson's. A group of drug addicts (made famous in the book *The Frozen Addicts* by Dr. Joseph Langston) overdosed on MPTP, a designer drug that is supposed to resemble heroin, after which they were unable to move. Their paralysis suggested, somewhat, the immobility of Parkinson's. These addicts were able to move again after they were administered high doses of L-dopa.

Using lab animals, it was shown that this drug (MPTP) was lethal to certain brain cells, substantia nigra cells in particular. Dr. Langston jumped to the conclusion that, because these brain-damaged addicts could not initiate movement, and their movement

---

[1] It was discovered much, much later, in the late 1990's, that a decrease in the darkened areas of the substantia nigra is a normal consequence of aging. This normal decrease occurs in the back part of the substantia nigra, while in Parkinson's, the decrease is usually in the front part.

(I should reference this study, of course, and all of the studies that I quote. But I have been very bad at organizing and saving all of the thousands of references that I have read. But then, I never intended to write a book, so why should I have saved references to everything I was reading about? At some point, when I realized that my writings on Parkinson's medications should be assembled into book form, I had to decide between putting in a few extra years to find all the footnotes, or else getting this book out as soon as possible to help the greatest number of people. Possibly future editions will pay more attention to scholarly attribution.)

Another study (one was done in Germany, another similar one was done by either the Mayo or Johns-Hopkins clinic, in the late 1990's – forgive me) found, based on post-mortem brain exams, that a *significant percentage of people who had been diagnosed with Parkinson's disease did not have any abnormal decrease in the dark cells of the substantia nigra.* I have never seen any follow up on either of these studies, a common fate for research that does not conform to expectations. I seem to remember that a conclusion to the study was that doctors had evidently been incorrect in their diagnoses.

initiation was restored with levodopa, he had proved the cause of Parkinson's disease: Parkinson's was caused by brain cell death, and more than likely, the source of the cell death was some as yet unknown external agent.

In retrospect, Dr. Langston probably should have said that the drug-induced brain damage caused Parkinson's-like symptoms. Instead, he boldly declared that he had confirmed the cause of idiopathic Parkinson's disease: Parkinson's disease was caused by the death of substantia nigra cells. This hypothesis and his extra hypothesis, that damage to the dopamine-producing area was probably caused by environmental agents was touted *regardless of the facts that the symptoms of the addicts were significantly different in many ways from the symptoms of Parkinson's disease, and the brain cell changes were also significantly different.*

Langston's questionable logic was hailed by most neurologists.[1] The previous theory about acetylcholine/dopamine imbalance was integrated into a new theory that worked with the substantia nigra cell death/environmental toxin theory.

1990's

By the 1990's, more than 50 neurotransmitters had been identified. The opposites theory was long dead (although still used by some neurologists in their explanation of Parkinson's disease, and the manufacturers of anticholinergic medications still refer to their drugs as adjusting the dopamine/acetylcholine imabalance). The hormone vs. neurotransmitter debate was not only dead, it was long forgotten; neurotransmitters were obviously made in nearly every cell of the body and traveled freely from stem to stern. Even though the blood-brain barrier prevented some neurotransmitters from moving between the extracerebellar (body) areas and the brain, neurotransmitters manufactured anywhere in the body were clearly influencing neurotransmitters in all other body parts.

Proof positive

These 1980's hypotheses about dopamine were so widely embraced that the dopamine – substantia nigra cell death – Parkinson's connection was accepted as unassailable fact by the 1990's. During this decade various official numbers were even assigned describing just how much substantia nigra cell death had to occur before Parkinson's appeared: PD symptoms appeared when "60% of the nigral nerve cells disappeared."[2]

---

[1] This theory has been a goldmine for researchers. Millions of dollars in grant money are currently being poured into research on how to stop this inexplicable cell death from mystery environmental toxins, even though other research has proved that the substantia nigra cells in idiopathic Parkinson's are not, in fact, dead.

At a lecture by a prominent Parkinson's researcher, he bragged about the twelve million dollars that his company had received to study drugs that would protect cells against "Parkinson-causing cell death." A member of the audience raised his hand and queried, "Why are you researching how to prevent cell death when the new research indicates that the cells are merely dormant and not dead?" The researcher didn't miss a beat, and countered, "It doesn't matter whether the cells are alive or dead, there is still not enough dopamine so we want to stop these cells from dying." Was he listening to himself? He didn't make much sense, but I suppose twelve million is hard to argue with.

[2]A. Lieberman, MD, Curing Parkinson's Disease in our Lifetime, *Parkinson's Report*, National Parkinson's Foundation, Fall 2000, p. 10. This nice, round, theoretical number has no basis. This number was determined by guessing backwards. (Footnote continued on next page.)

Also in the 1990's, research proved that L-dopa accelerates the worsening of Parkinson's disease. Dopamine agonists were shown to also cause acceleration of Parkinson's, but not to the same extent as L-dopa.

Researchers in the field of Parkinson's rely on rodent "models" of Parkinson's disease for their experiments. Even though it was suspected (and has now been proven) that idiopathic Parkinson's disease is not caused by toxins, rodents are purposely brain damaged via toxins, and then studied as if they represent a valid model for Parkinson's. While I am on this subject, please be wary: whenever you read about a new breakthrough for Parkinson's disease, read carefully in the fine print. If the research was done on a "model" of Parkinson's disease, or done on animals with "parkinsonism," the results have little or nothing to do with idiopathic Parkinson's disease, and the researchers are well aware of the fact. However, since blinding amounts of research money is available for Parkinson's, and there is no way to actually create idiopathic Parkinson's disease in a lab, these "models" of Parkinson's are used in research.

Again, drug- and toxin-induced cell death parkinsonism has almost nothing in common with cell-dormant Parkinson's disease. Studying the former in hopes of learning about the latter is pointless, especially since the direction of research is usually oriented towards finding a way to prevent cell death – a cell death that does not occur in Parkinson's disease. Your tax dollars at work…

How science grows

Right from the start there were inconsistencies with the Frozen Addict = Parkinson's disease conclusion. There were notable physical differences between people with Parkinson's disease and those who, like the frozen addicts, had drug-induced or toxin-induced movement inhibition.

For example, the addicts had developed a rapid onset, complete body paralysis, and PDers usually develop rigidity very slowly. The addicts had immobility due to inability to initiate movement. PDers, in addition to difficulty in initiating movement, have a host of symptoms that are not found in "frozen addicts," symptoms such as a

---

(Footnote continued from previous page.) At the time this number was proposed, there were no methods of measuring brain change except for autopsy. This beautifully exact number, 60%, was pure guesswork based on autopsies of PDers. The number was attained by extrapolating the amount of cell change visible after death and comparing it to what it might have been at the time of diagnosis. The now-known fact that *the drugs that had been given to the PDers who were autopsied actually cause cell death* was not considered at the time these numbers were created. Very possibly, the brains that were used to create this 60% statistic were actually damaged by the drugs, more than by the idiopathic PD.

Moving beyond the numbers game, it is fun to read further in *this same paragraph* by A. Lieberman that, in Parkinson's disease, "although dopamine is depleted, *the cells in the striatum are preserved*. This is unlike the PD-like disorders (drug- and toxin-induced parkinsonisms) where, in the striatum, the dopamine content is decreased and *the cells are lost*" (emphasis added). In case that got by you too quickly, what the MD director of the National Parkinson's Foundation states, all in one paragraph, is that, 1) by the time PD appears, "60% of the cells have disappeared" (or "are lost," the old established fact), and 2) the exact opposite position (the new evidence), namely, that in Parkinson's disease, the cells are preserved! The cells are "disappeared" and "preserved" both, all in one paragraph! Hats off!

This just in! In a new book that just crossed my desk, I read that Parkinson's disease "results when 80 to 90 percent of the dopamine neurons are lost." This tidbit is from the 2001 edition of *A Primer of Drug Action,* R. Julien, MD, PhD. Henry Holt and Company, p. 358. Don't memorize these numbers – they will doubtless be changing again soon.

characteristic rigidity in specific joints that causes a cogwheeling motion in the wrists and ankles, and they have inflexibility of specific muscles of the torso, unresponsiveness to brain command in the anteriolateral muscles of the legs and neck muscles, use of certain facial muscles but collapse of others, degeneration of the bicep, atrophy in the muscle alongside the second metacarpal, and dozens of other PD-specific symptoms. Most significantly, Parkinson's disease usually develops on one side of the body and only after some time does it become two-sided. Even after it spreads to the second side there is usually a distinct difference between the sides.[1]

Also, the addicts did not have the same type of tremoring along the Large Intestine and Stomach channel lines, balance problems, personality profile, circulatory problems, constipation, hardening of anteriolateral skeletal muscle, or seborrheal skin problems that were seen in PD, and other details that should have suggested a difference between drug- and/or toxin-induced parkinsonism (as it is now called) and idiopathic Parkinson's disease.[2]

However, the L-dopa as cure theory had already been embraced even though L-dopa was suspected to increase the advancement of Parkinson's symptoms and only some, not all PD symptoms responded to L-dopa. The brain cell death theory was added on. As there was no reason to discard the excess acetylcholine theory, that theory was also maintained.

The way of all science

Scientists like to build on previous information. The PD-is-caused-by-dopamine-cell-death theory, together with the dopamine-as-relaxant theory were established, by the mid 1980's, as the cornerstone theory upon which all future PD/dopamine research would have to stand.

Once a theory becomes a cornerstone, it requires a paradigm shift – ideological dynamite – to change the theory. The problem with changing a cornerstone theory is that all of the theories that have been built on top of the cornerstone will topple. The political

---

[1] This may not seem significant to you, but an all-over disease can be best attributed to a systemic failure in the system or ingested toxins, whereas an asymmetrical disorder can usually be traced to an injury, insult, or localized illness on only one part of the body, such as is seen in stroke or polio. The one-sidedness of Parkinson's onset is actually a major clue as to the source of the physiological changes that eventually trigger the PD symptomS.

[2] Dr. Langston's work was done back in 1982. Now, in 2003, the idea that drugs, including legal, pharmaceutical drugs, do cause parkinsonism is old news. The latest edition of *Parkinson's Disease, Questions and Answers*, Merit Publishing, with a forward by the medical director for the National Parkinson's Foundation, has an entire section on "Which medications cause parkinsonism?" In that section the author notes that "Many pharmacologic agents can produce features of parkinsonism, including tremor, bradykinesia, rigidity, speech disturbances, and akathisia. These include dopamine-blockers such as the neuroleptics" (My note – these drugs are often prescribed to PDers who are having dyskinesia rather than telling the PDers to reduce their dyskinesia-inducing drugs.) "and antiemetics, as well as dopamine depleters, the gastrointestinal motility drug metochlopramide, lithium, alpha-methyl-dopa, and some of the tricyclic antidepressants." The author goes on to list other drugs that can cause parkinsonism, and ends with stating that "anyone who has recently used these medications should be observed for at least six months off the medication before a diagnosis of Parkinson's disease is made."

Despite these published warnings, I frequently meet patients who were taking antidepressants from the above group who developed signs of parkinsonism and whose MDs then started them on PD drugs without a moment's hesitation.

force behind a cornerstone theory is strong: trying to disprove these theories can destroy a scientist's career.[1] Evidence that contradicts the cornerstone is assumed to be faulty. In the case of Parkinson's disease, those patients whose responses to L-dopa contradicted the new theories were accused of having the wrong response!

An example: patients are wrong if their response doesn't match the theory

In the first decades of L-dopa use, many, if not most, PDers complained to their doctors that L-dopa was causing them, among other adverse effects, a severe type of insomnia. As an example of the inertia of science, and what happens when the cornerstone is contradicted, here is what happened to these complainers: *they were told that they were wrong*.

Here's why they had to be wrong:
1) Parkinson's is a disease of excess tension (ACh excess theory).
2) L-dopa cures Parkinson's.
Therefore:
3) Dopamine is a relaxant.
4) Humans are more relaxed when sleeping.
Therefore:
5) Dopamine is at highest levels at night and when sleeping.
6) L-dopa is therefore a sleeping aid.
7) Patients who take L-dopa should not attribute their insomnia to the drug, because the drug helps them sleep.

Please do not memorize the above. The above has since been proven wrong. Native dopamine levels naturally drop to very low levels during sleep. Also, as it turns out, L-dopa is not a muscle relaxant, it is an anxiety suppressor and motor *stimulant*.

The above seven-step proof was so logical that a drug company-sponsored study was conducted to disprove the contention made by PDers that their L-dopa medication was causing severe insomnia. Experiments were run comparing the sleep patterns of PDers who took the L-dopa at night or late in the evening compared to those who did not. The results of the study *proved that the complaining patients were correct*: *taking the medication late in the evening or at night was associated with increased insomnia*.

Despite the finding that L-dopa was, in fact, linked to insomnia, the official (if illogical) conclusion of the study was that (and I paraphrase), even though the medication *appeared* to be a major cause of insomnia in users of L-dopa, the *obvious* (their words, my italics) benefits of having consistent levels of L-dopa in the body throughout the day and night should override the insomnia concern.[2] Also, it was a fact (see step 5 in the

[1] As an example of the dangers inherent in challenging a current scientific paradigm, Galileo Galilei, who insisted that the movement of heavenly bodies was more orderly if the sun, not the earth, was the center of the planetary orbits, lived out his days under house arrest. His fellow astronomers, who, for the most part, snapped up his writings and recognized him as a genius, were afraid to publicly support him.

[2] A citation for this article is in order, but I don't have it. The abstract on this article was one of the ones that came up in the 100 abstracts mentioned later in the next appendix. Go to med base and do a search for dopamine/sleep. I did the search in August of 1998.

above proof) that dopamine levels should be higher at night. Therefore (concluded the study), the insomnia problem, though seemingly caused by the L-dopa, was probably being generated by some unknown.

This trend, in which patients who have problems with their drugs are told that they are wrong, and that the drugs are not causing problems, is increasingly the norm. In medical schools today, would-be doctors are often taught that patients' adverse responses to medications are usually psychosomatic, and that patients should not even be warned of the side effects for fear that they will develop them. But the above study was even more alarming: the study indicated that insomnia was being caused by the drugs, and yet the stated conclusion of the study was that the drugs should still be used during the night. The implication was that the drugs should be used even if they caused harm.

Other patient complaints about the drugs were usually met with the same sort of nonchalant shrug on the part of doctors. The accepted medical position was, by the late 1990's, that Parkinson's disease was caused by dopamine loss due to substantia nigra cell death, and people with Parkinson's should take whatever medications they needed to increase brain levels of dopamine.

2000

By the year 2000, over sixty neurotransmitters had been identified. They appear to influence nearly all cellular functions. The complexity of neurotransmitter interactions, the effect of neurotransmitters on mental activity and the effect of volitional mental activity on neurotransmitters has begun to make the old idea of brain-as-switchboard look positively quaint. Increasingly, some molecular biologists now suspect that thought initiates release or creation of neurotransmitters, rather than the reverse. However, some neurologists, especially those who went to school prior to 1980, still consider that there are only a few crucial neurotransmitters, and that Parkinson's disease is caused by dopamine deficiency and possibly excess acetylcholine.

As for Parkinson's research, the largely ignored research at the turn of the millennium proved that a substantial difference existed between people with idiopathic Parkinson's and those with drug- or toxin-induced parkinsonism. In those with idiopathic Parkinson's, the cells of the striatum are not dead.

This should-be shocking news has been, for the most part, ignored by most MDs and PD researchers. The problem with this new finding is that it suggests that there might be some obscure, unknown reason that the cells are not making dopamine. The cell dormancy seen in PD might mean that what is needed for PDers is not necessarily dopamine, but a way to make these cells start producing dopamine again. Since western researchers don't know what causes cells to differentiate and create their various cell components, they have no way to use this new information about cell change. Since there is no way to pharmaceutically benefit from these new findings, the new research is largely ignored in the applied medical (clinical) realm.

Further research has shown that, in idiopathic Parkinson's, the dopamine receptor cells do not necessarily accept dopamine even when it is foisted onto the brain via dopamine-enhancing drugs. In other words, it begins to appear as if the brain of idiopathic PDers is *resisting* dopamine, not suffering from a dopamine shortage.

This conclusion, that PDer brains are trying to resist dopamine, is not helpful to MDs because western medicine has no method by which to reverse this shunning of

dopamine. Therefore, because the only tools available to ameliorate a few of the symptoms of PD are drugs that force dopamine onto a brain that is trying to resist dopamine, those tools are the ones that are used. Rather like the man whose only tool is a hammer and for whom all problems therefore look like nails, MDs have no tools other than dopamine-enhancing drugs, and so all PD problems continue to look like dopamine deficiency to this group of doctors.

Acetylcholine, again

Recognizing that PDers have difficulty initiating movement, combined with the fact that acetylcholine is a muscle stimulant, some bold young researchers and doctors have started using drugs that enhance acetylcholine in the treatment of Parkinson's. While these drugs exacerbate tremor, they can sometimes help, briefly, with the Parkinson's slowness of movement. This is a 180º reversal of the treatments offered from 1960 to the present. I have even heard from patients simultaneously being prescribe both anticholinergics and cholinergics! This type of error is not shocking; I have learned that many doctors prescribe pills based simply on whether or not a drug is on the list of "antiparkinson's medications," while having no idea about the mechanism by which the drugs perform their job.

Dopamine and addiction

In 2000, dopamine was recognized as the chemical of addiction, the neurotransmitter of pleasure. It has now been proven that the various addictive drugs and substances, including cocaine, opiates, methamphetamine, alcohol, and cigarettes are all addictive because of their ability to elevate, however briefly, brain dopamine levels.

Dopamine's many hats

Dopamine is recognized as a powerful mind and motor stimulant. Dopamine decreases pain awareness. Excess dopamine combined with insufficient frontal lobe activity causes schizophrenia. Excess dopamine alternating with insufficient dopamine is the neurotransmitter imbalance that causes bipolar (manic-depressive) behavior. Dopamine helps regulate body temperature. Dopamine levels affect the immune system. Dopamine is used in sedating stress from social interactions. Dopamine levels are highest during waking hours, and decrease drastically during sleep. Dopamine is used not to stimulate motor function nerves, but to bridge the gap between thought and motor function.

The brain is exquisitely calibrated to maintain dopamine at exactly correct levels: too little causes severe depression and too much causes illogical ecstasy. It is impossible to administer dopamine-enhancing drugs in a manner that can replicate the sublime balancing act performed by the brain in regulating dopamine. Any application of a dopamine-enhancing drug that attains the desired effect must necessarily broach the line above which drug addiction and brain cell damage occurs.

As for the relationship between thought and neurotransmitter release, it has been shown that dopamine is the most expectation-dependent of the neurotransmitters: if a person imagines that he will feel better, he does in fact feel better. This improvement in his mood and energy is directly related to an increase in dopamine that occurs in response

to the expectation of improvement. Conversely, a conviction that one is going to feel worse is accompanied by a decrease in dopamine.

The placebo effect, a benefit that can occur in treatment of some illnesses and not in others, appears to primarily play a part in those illnesses that have a dopamine-related component. Dopamine imbalance plays a part in a variety of illnesses, from asthma to insomnia, from schizophrenia to Parkinson's disease.

Summary

The theories about Parkinson's disease changed frequently from 1950 to 2000. Doctors are currently practicing medicine who subscribe to the excess acetylcholine theory, or the acetylcholine/dopamine imbalance theory, the dopamine deficiency theory, the substantia nigra cell death theory, or most recently, the dopamine resistance theory. The theory to which a doctor subscribes very possibly is determined by the year he attended college.

Drug prescriptions

The drug prescribing patterns of a doctor may very well be determined by the prescribing that was popular during the doctor's intern years. Some doctors still use anticholinergic (anti-acetylcholine) medications. Some young doctors are using the opposite: acetylcholine-enhancing medication. Older doctors usually tout L-dopa. Younger doctors often shun L-dopa or tell patients that it is a drug of last resort, while exhorting the virtues of the newer dopamine enhancers.

Most doctors, believe it or not, do not realize that the various antiparkinson's drugs have different effects. Few doctors in my experience have understood that anticholinergics should only be used in patients whose dominant problem is tremor or anxiety, patients who do not have serious motor initiation difficulty. Other doctors do not know that L-dopa, a drug that causes increase in motor initiation and an increase in tremor should *not* be used for patients whose main complaint is tremor.

In my limited experience, I have seen that most doctors, even Parkinson's specialists, simply throw at their patients whichever antiparkinson's drugs they are most accustomed to using, despite the tremendous variety in PD symptoms presented by their patients. If one drug doesn't seem to work, or becomes ineffective, the useless drug is almost never removed from the medication roster, but is retained, while more drugs are piled on top.

What a neurologist learned about dopamine in medical school may or may not be the view of dopamine in your Sunday newspaper column on Ask The Doctor. The view of dopamine that your spouse or loved ones might hear on TV talk shows about dopamine can vary dramatically, depending on which year's medical dictionary is being used as a reference. Your fourteen-year old niece probably knows that dopamine is the neurotransmitter of addiction. If your health care provider went to school some time between 1960 and 1995, he probably does not know this. Your neighborhood biochemist may know about the exciting new world of neurotransmitters. Your neurologist may not know about any of the exciting new findings in the field of neurotransmitters, and may instead subscribe to some or all of the outdated dopamine/Parkinson's theories included in this appendix.

The various theories explained in this appendix were the cornerstone facts for Parkinson's disease during the second half of the twentieth century. They were the paradigms of Parkinson's, and most neurologists that are currently practicing medicine will not let go of them without a fight. These various outdated theories are still, for most neurologists, the alpha and the omega of Parkinson's disease.

This appendix was written to hopefully make the point that medical facts are inconstant, and that there is no such thing as a standard of medical knowledge. For those who are struggling with family members who demand allegiance to the all-knowing doctor, this appendix may not help, but on the long-shot chance that it might, I offer it to you. I also enjoyed writing it because it was a History of Science class, a college course that traced the creation and destruction of scientific myths, from the days of Plato to the Einsteinian twentieth century, that first opened my eyes to the realization that the "facts" that I was memorizing in my biology classes were simply the current theories, and no more related to Truth than the now laughably false dictums of Aristotle. This class was the catalyst that turned me away from my pedantic study of biology "facts," fanning my interest in both history and modern physics, and giving me the strength to pursue my medical research even when it flew in the face of the established theories. As an example of my own run-in with hard-to-change establishment thinking, I have included in the next appendix the true tale of my first confrontation with dopamine fallacies.

Appendix 6

DOPAMINE FALLACIES:

THE STODGINESS OF SCIENCE HITTING HOME

The human tendency to evaluate current observations through the looking glass of previous theory often leads to perpetuation of disproven ideas even in the face of conflicting evidence. This appendix is a collection of ongoing dopamine myths and falsehoods, some of which affect ongoing research, some of which directly affect patients' daily lives.

For example, as noted previously, the scientific gospel of my youth asserted that dopamine was at its highest levels at night. This mis-fact continued to influence research even into the late 1990's despite voluminous evidence to the contrary. The resistance to change in the field of science was driven home to me personally when I published my first article on Parkinson's disease in 1998.

In it I had hypothesized that the pathological electrical patterns present in PD triggered a constant, if only partial, go-to-sleep signal in the brain. This partial (only present on one side of the brain) go-to-sleep signal had been present in the brain for decades, long before the PD became apparent. This errant "bedtime" signal flashed all day long. I proposed that the decrease in dopamine, as seen in PD, was consistent with this go-to-sleep signal. It was likely, therefore, that the sleep-time electrical pattern is a pattern that causes a shut down in dopamine. To put it more simply, dopamine levels decrease during sleep.

I further proposed that it was the decades-long presence in PDers of a pathological, asymmetrical (so that it was only half a signal rather than a full one), 24 hours a day sleep-inducing electrical pattern (a non-dopamine pattern) that explained why the dopamine-producing cells eventually became dormant: the cells never received a full (symmetrical), daytime, dopamine-producing signal.[1] A waking signal, electrically different from the sleeping signal, is necessary to trigger the production of new dopamine-producing structures in the cells. Because the dopamine-producing cells never received a full electrical signal to produce dopamine, the DNA of the cells, over time, ceased or slowed the building rate of the dark-colored cellular structures in these cells.

All cellular components are always being broken down and replaced with new ones. In Parkinson's disease, the cells of the substantia nigra continually dismantle yesterday's dark-colored, and presumably dopamine-making structures, as per the normal, healthy process. But because, over previous untold decades, there was never an adequate waking signal, no building of replacement structures was ever initiated since the time the aberrant signals began. I proposed that after decades of slow dismantling and no replacement, the dopamine producing cells would slowly revert into cells that were no longer dark. They would be sort of neutral cells, closer in structure to embryonic cells, cells that had not yet been assigned tasks. Biologists say an embryonic cell that doesn't

[1] The waking hours of PDers are dominated by adrenaline instead of dopamine. Adrenaline is a neurotransmitter that provides ample alertness even when dopamine, a stimulant, is absent or diminished.

yet have a specific task is "undifferentiated." In Parkinson's disease, the cells of the substantia nigra were no longer dark but had become, instead, a pale pinkish. They had very nearly reverted to an immature, or non-specific condition[1] in which they were re-undifferentiated.[2]

The basic electrical theory portion of this hypothesis was not an original idea. It was ancient Asian medicine. The substantia nigra cell change portion was a logical guess as to what the Asian science might imply in the case of Parkinson's disease. The shocking part of this proposal, the part that was the hardest for me to swallow, was the radically new idea that dopamine was a day-time neurotransmitter, that dopamine was more predominant in an awake person than in a resting person. This paradigm shift was necessary because the electrical alteration that I had seen in Parkinson's disease involved an increase in electrical current in the Gall Bladder channel, a channel that runs at highest levels from 11 p.m. to 1 a.m. – the time when a person is traditionally supposed to be falling asleep, or at least experiencing specific, often sleep-related changes in physiology. This electrical alteration in PDers, combined with the recognized dopamine deficiency in PD, suggested that dopamine in healthy people must be at higher levels in the daytime!

Dopamine by day – the challenge

Though it was in conflict with all my previous training, I had come to this difficult conclusion via two bits of evidence. First, the electrical evidence – my patients all exhibited, *even during the day*, a detectable (by hand) electrical pattern that was identical with a nighttime sleeping pattern.

– A bit of Asian medical background is necessary here: a significant portion of this particular current, known as the Gall Bladder channel, runs alongside the head, directly across from the midbrain. From 11:00 p.m. to 1:00 a.m there should be an amperage increase in the energy flowing through the Gall Bladder channel.[3] A change in any body current will necessarily change the electrical influence on its nearby cells. These faint changes in amperage, their effects on adjacent currents and cell-current iterations, as well as their effects on cells within their sphere of influence are considered, in Asian medicine, to be drivers of the cellular changes that occur during the circadian cycle, as well as the drivers of selective DNA expression. The Gall Bladder channel runs posterior to the temple, passes above the ears, and runs over to the nape of the neck before flowing down to the feet. The head portion of this channel very nearly follows the curve of the brain stem and the midbrain, and changes in this channel exert a large influence over the inner brain. As seen in chapter 24, the Du channel is the main driver of the midbrain. The Gall Bladder channel, running somewhat parallel to but in the *opposite direction* of the Du channel, suppresses the power of the Du channel considerably. When

---

[1] It has been known for years that the substantia nigra cells in people with *idiopathic* Parkinson's are not dead. The substantia nigra cells in *toxin- and drug-induced* parkinsonism are dead or severely damaged. The cells in idiopathic PDers are merely altered.

[2] This has recently been proven. With regard to the article that proved reversion to brain cells back to an undifferentiated condition (I believe the word they used was "embryonic"), I have a memo to myself on a scrap of paper, "see Jan 1, *Neuroscience*, University of Houston." (No year, but possibly 2002.) I am still trying to relocate this reference.

[3] Though this subject is far beyond the scope of this book, it should be noted that every channel in the body has a characteristic period during the day when it experiences a surge in power. The circadian cycles are driven by these surges.

the Du channel is suppressed, brain function, especially frontal lobe function and consciousness, is also suppressed. When the midbrain is suppressed, dopamine production is put on hold. –

I suspected, based on my patient sample, that this electrical aberration of excessive energy in the Gall Bladder channel during the daytime was present in all PDers.

I could also assume, based on their diagnoses of PD and the dopamine connection with PD, that these patients all had decreased dopamine and changes in their dopamine-producing cells. This evidence suggested a relationship between the decreased dopamine of PD and the Gall Bladder channel's go-to-sleep electrical signal. The conclusion drawn from this evidence, that dopamine was an awake-time neurotransmitter, flew in the face of the long established fact that dopamine was a relaxant, present primarily at night.

Secondly, dopamine being a daytime neurotransmitter fit the behavioral evidence: if dopamine were a stimulant, rather than a relaxant, it would explain the strange behaviors of my medicated patients. These patients behaved as if their dopamine-enhancing drugs gave them mental alertness and physical spontaneity, not relaxation or drowsiness. I had been observing the behaviors of my patients who were trying to reduce their L-dopa levels: after they made drug reductions, they behaved just as if they were having withdrawal effects from some of the better-known stimulants (cocaine and methamphetamine). This behavior made no sense according to the dopamine-as-relaxant theory but it suggested, instead, that dopamine was a stimulant.

The logical conclusion, and one that I wrote up in my article, was that dopamine was a waking-time neurotransmitter. I stated that in a healthy person, dopamine levels dropped at night, and guessed that the daily electrical signal change was a contributing trigger. It appeared obvious, based on the evidence. I felt very uneasy about this evidence, and I kept looking for the flaw in my thinking; I had been training for over thirty years in Biology – I knew, like I knew the earth orbited the sun, that dopamine was a relaxant. It was painful for me to propose this radically opposite idea. It is never easy to be a traitor to one's training.

The editor of the journal that was considering publishing my research said that the rest of my hypothesis was well formed and supported, but she had a problem with the dopamine idea: it was an accepted fact that dopamine is produced at higher levels at night. Dopamine is a relaxant. She was willing to give me a chance, however. She said that if I could find even one recent research article stating that dopamine levels were higher in the daytime, refuting all the current books on the subject, she would run the article. She was most dubious.[1]

Dopamine by day – the proof

Here is where it got exciting. I did a search at the local university's Medline computer station. I printed off the first one hundred abstracts that came up under the search topic Dopamine/Sleep. Those hundred abstracts gloriously demonstrated how we scientists transform a guess into a theory and then try to force subsequent information into the theory, whether it fits or not.[2]

---

[1] B.G. Grace, chief editor of the long-running and highly respected *American Journal of Acupuncture*, was the most thorough and exacting editor I have ever worked with. Thank you, B.G.

[2] Some famous examples of forcing the foot of evidence into the wrong size shoe of established facts are the increasingly convoluted charts of the heavenly bodies that were drawn up by pre-Galileo

Of the one hundred articles that I pulled up in my search, there were ninety-eight articles in which the subjects had, strangely enough, higher dopamine levels in the daytime than at night! Shocking!

In every one of these studies, the main subject of the research was not dopamine or Parkinson's disease, but was some other illness being studied. The night and day blood dopamine levels had merely been checked as a part of the general blood work. In every case the researcher had noticed that, contrary to the "normal" pattern, all of his subjects had higher blood levels of dopamine in the daytime, lower levels at night. In each article, the researcher concluded that the unanticipated and obviously pathological dopamine levels might be contributing to the illness that was being researched. The various illnesses being researched included PMS, narcolepsy, epilepsy, mood swings, muscle cramping, mental retardation – the list went on and on. In every case, the researcher for each article had suggested that maybe the cause of the illness at hand was the pathologically elevated daytime levels of dopamine and decreased nighttime levels.

Since it was a recognized fact that dopamine is a relaxant, and is therefore present in higher quantities at night (based on the acetylcholine/dopamine imbalance = excess rigidity theory of Parkinson's disease), every one of these studies concluded that the cause of the illness in question might be this reversal of the "correct" dopamine pattern.

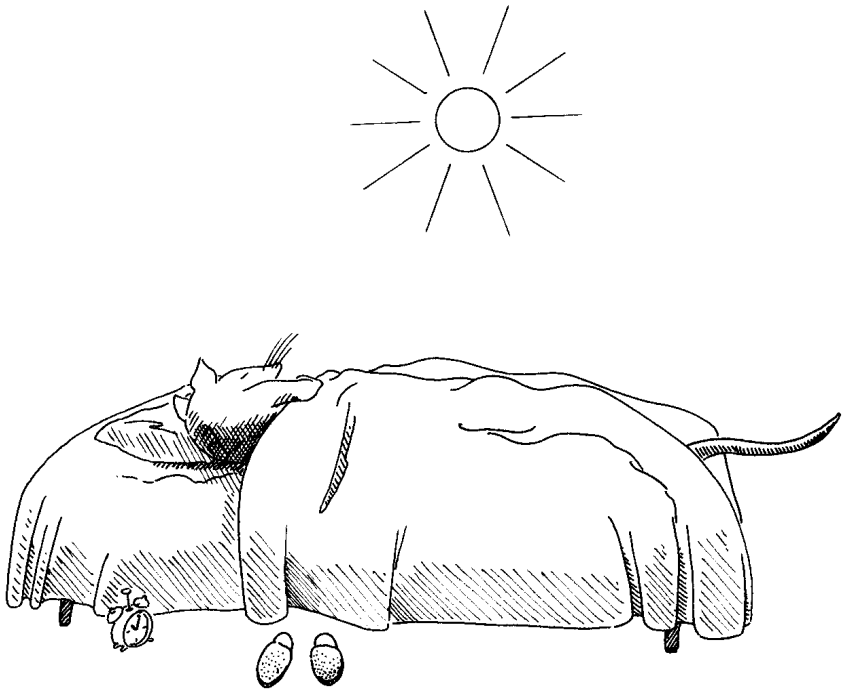
In other words, if a patient was in the headache study, it was probable that this abnormal reversal of the normal dopamine pattern was causing his headaches. Ninety-eight of the one hundred studies followed this pattern. It didn't matter what they were researching; the conclusion in each case was that the illness at hand might be related to the abnormal, reversed situation of blood dopamine levels being higher during the daytime and lower at night.

But, to be fair, there were two studies that had the opposite result. In these two studies they measured not blood dopamine levels, but the actual brain levels of dopamine. This was done by chopping off the heads of the subjects, tossing the heads in a blender, and quickly assaying the results. This gave the most accurate possible reading of brain dopamine levels. In these studies, the dopamine levels were higher at night, thus confirming the facts of dopamine as a nighttime relaxant. There was only one detail that had missed everyone's attention: the subjects in these experiments were rats. Rats are nocturnal – they are active at night, they sleep in the day.

Armed with these research abstracts, abstracts that suggested that 100% of the time human dopamine levels were higher during awake, active time and lower during sleep, and the clinching evidence that nocturnal animals had higher dopamine levels at night during their waking hours, my editor agreed to run my article. She also said, only half in jest, "This contradicts the current facts; they're going to kill you." This was in 1998, just five years ago. I haven't been killed yet, but hopefully, this vignette illustrates just how hard it is to defy the established "facts."

---

scientists. The invention of the telescope allowed increased accuracy in tracing the paths of the planets. In order that the charts of these bodies might be proven to have an earth-centric orbit, thus supporting the status quo, the descriptions of their travels developed into fantastic shapes and patterns, complete with full stops and mid-air reversals. On the other hand, if, as Galileo proposed, the sun was placed at the center of the solar system, their paths became ovals of simple elegance. The long-standing paradigms of the day made it impossible for such simplicity to be acceptable.



Rats are nocturnal.

The role of dopamine in hallucinations and psychoses: a subordinate fallacy

Research published in 1999 confirmed that the drug-induced hallucinations or psychoses experienced by many PDers occur most often in those patients who take dopamine-enhancing medications at night.[1] This corresponds with the well-researched finding that the hallucinations of schizophrenia appear to be caused by a combination of excess dopamine in the lower brain centers coupled with decreased activity in the frontal lobe.

And yet, even today, when my PD patients[2] report terrible problems with insomnia, restlessness, excess movement, and even hallucinations to their doctors, they are nearly always assured by their MDs that what they need is more L-dopa, not less. This thinking is a continuation of the dopamine-equals-relaxation fallacy, combined with the inability of most MDs to keep up with the latest research. Fortunately, most MDs do recognize that hallucinations are a sign of excess dopamine. However, even in my limited experience I have run into a few doctors who needed to have the drug warning's words mailed to them, with the bit about hallucinations highlighted with yellow marking pen, before they accepted that these drugs might be responsible for their patients' hallucinations.

More dopamine fallacies

In their attempts to explain away the twitching and spasming being caused by excessive L-dopa, a supposed muscle relaxant, I heard the following explanation from several of my patients: dyskinesia is caused by excess dopamine in the blood and insufficient dopamine in the brain. It was when L-dopa converted into dopamine in the blood before it had a chance to pass through the blood-brain barrier that it triggered all the adverse effects of L-dopa, especially the dyskinesias. Whether they learned this from their doctors, speakers at their support groups, the literature, or they simply misunderstood, I cannot guess. But I heard enough of this theory from my patients, and even from a few doctors, that I include it here.

I have never, to this day, found any actual research that supports the theory that blood dopamine is a stimulant and brain dopamine is a relaxor. In fact, it does appear, based on all the current research in drug addiction, that nearly all the adverse effects of L-dopa, including the dyskinesias, are coming from the brain dopamine excess. (There are a few adrenergic dopamine receptors located outside the brain, especially effecting the bladder and stomach, but these do not relate to the great majority of levodopa-related adverse effects.)

So why is this excess-dopamine-in-the-blood-and-deficient-dopamine-in-the-brain-causes-dyskinesia theory so widespread? Maybe this theory is acceptable because it permits dopamine to remain a relaxant, as previously "proven." In science, any new theory that allows a previous theory to remain standing is going to be a winner.

---

[1] "Significant risk factor for developing psychosis in PD is nighttime use of long-acting dopaminergic medications." R. Hauser, T. Zesiewicz, *Parkinson's Disease, Questions and Answers*, Merit International Publishing, 2000, p. 29. (in text reference to Juncos JL, Management of psychotic aspects of Parkinson's disease. J Clin Psychiatry 1999; 60 (suppl. 8): 42-53.

[2] These include email correspondents from across the USA and around the world.

L-dopa additives: how they work

The idea that blood dopamine causes dyskinesia and brain dopamine does not has played a part in the ongoing reliance on the levodopa additives, also called buffers, and the new levodopa-helper (anti-digestive) drugs, such as Comptan and Tasmar. In the United States, the most popular L-dopa additive is carbidopa (Sinemet). In Great Britain, the most popular is benserazide (Madopar). There are two theories as to why adding carbidopa (or similar molecules patented under different names) to levodopa may be beneficial.

One guess presented by the manufacturer[1] of Sinemet is that carbidopa slows the conversion of levodopa into dopamine in the blood (the blood being an extra-cerebral tissue; in other words, anything outside the brain). The other reason, well-proven, is that carbidopa isn't digested very quickly: by attaching a molecule of carbidopa to the molecule of levodopa, the pair of them can pass through the gut without being mistaken for a bit of dinner. [2]

Without the attached carbidopa, the protein of levodopa can be quickly broken down into small, protein building blocks that the body then uses in the same way as protein from bacon or eggs. In other words, in its pass through the stomach and liver, most levodopa is rendered useless as a drug, and becomes, instead, a snack.[3]

The quick digestion of levodopa was probably the reason that the earliest experiments with levodopa showed no results in Parkinson's patients – the minuscule doses were being digested like hors d'oeuvres. Only when the doses were increased a thousand fold did enough dopamine slip past the digestion process that the tiny amount needed for brain upheaval could get into the cranium, where it then manifested its stupendous effects.[4]

I don't know which of these two carbidopa processes is actually the more significant, the extra-cerebral conversion prevention or the digestion prevention. The latter benefit has been proved. However, many physicians use the former theory to explain dyskinesia despite there being no test/proof basis for the idea that extracerebral dopamine causes dyskinesia.

Therefore, if your doctor is trying to get more dopamine into your brain in an attempt to stop your dyskinesia, he is probably laboring under a false premise. Your problem is that you have too much dopamine in your brain.

---

[1] *Physicians' Drug Handbook*, Springhouse, 2001, p. 596.

[2] Ibid.

[3]There is another advantage to carbidopa/levodopa that is rarely mentioned: it can be patented. L-dopa exists in nature. In its natural form, it cannot be patented. If levodopa is combined with other compounds in a form that does not exist in nature, this combination can be patented. Such a patent can be worth billions for the drug company that is the first to file for the patent rights.

Conveniently, congress members were persuaded that levodopa, a naturally occurring compound found in fava beans and certain herbs, should be considered a dangerous drug, like opium, and no longer be available for sale over the counter to the general public. At the same time that L-dopa was being legislated out of the health food stores, the patented and expensive version of carbidopa/levodopa was being heralded with great fanfare as the new, improved cure for Parkinson's disease.

[4] Drug addicts have known for decades about the importance of bypassing the voracious digestive system. Bypassing the digestive system via smoking, snorting up the nostril, or injecting drugs directly into the bloodstream enables one to get a drug effect with much, much less drug, up to a thousand times less drug, than is needed to get past the stomach.

More levodopa adjuncts

Many of the new drugs, such as Comptan and Tasmar, are attempts to further prevent metabolism of L-dopa in the gut by shutting down critical digestive processes. This tummy turn-off can, in theory, get more of the L-dopa to the brain. These drugs are supposed to be taken when the levodopa drugs show decreasing effectiveness over time, or adverse effects appear. The implication is that it is L-dopa's failure to make it into the brain that is causing these difficulties. The helper drugs increase the payload to the brain.

The possibility that it is L-dopa's presence in the brain that is causing these problems of dyskinesia or decreased effectiveness is never, ever, considered.[1]

This idea that brain dopamine is always the Good dopamine and all other forms of dopamine, including digested dopamine or blood dopamine, are Bad dopamine, goes hand in hand with the idea that brain dopamine is a relaxant and blood dopamine is a stimulant and a causer of problems.

Why am I even bringing this up? When a patient's ever-increasing L-dopa dosage becomes so high that it causes muscle spasms and twitching, eye rolling and tongue thrusting, jerking and shaking, most MDs assure their patients that what they need is still more L-dopa. Since brain dopamine is supposed to be a relaxant, this excess movement, in the eyes of some doctors, indicates a worsening of Parkinson's. (Whatever happened to poverty of movement? Don't forget: many doctors hold that the rigidity is due to excess vigor, and excess movement could conceivably be considered an extension of this excess of strength. The 1960's transformation from viewing Parkinson's disease as a condition of "poverty of movement" to one of "excess strength" is responsible for this honest mistake.) Hence we have the ongoing effort in drug design to get ever-increasing amounts of dopamine into the brains of PDers who are moving *too much*.

Most MDs have embraced the "ever-increasing dopamine" program wholeheartedly – to the extent that many of them have completely abandoned common sense. Now, when a PDer who previously suffered from immobility and poverty of movement suddenly reverses his symptoms and is moving far too much, ticcing and grimacing, his MD may prescribe still more medication.

Under the weight of its own self-belief and political/financial clout, medical science is ponderous and slow to change. Even in the face of obvious problems with the late twentieth century theories of PD, the western medicine search for a cure is still focused, for the most part, on how to get more L-dopa into what is often a dopamine-saturated, dopamine-resisting brain.

Now, in 2003, this may finally be changing. It is not changing because of an admission that the drugs are causing many of the movement problems in PD; it is changing because many American neurologists are beginning to recommend brain surgery for patients whose drugs are propelling them into violent dyskinesias, which are falsely considered to indicate a worsening of Parkinson's disease.

But that is getting ahead of our story. Our story is about how difficult it is to get rid of an incorrect theorem or fact.

---

[1] These new drugs appear to cause a rapid worsening of symptoms within a few months. This is not surprising: they serve to increase dopamine levels in the brain – and it is brain dopamine that is causing the adverse effects, including the Ons and Offs that these drugs are trying to prevent. Meanwhile, some doctors continue to prescribe the new drugs, convinced by the ever-worsening adverse effects that what is needed is yet more dopamine!

Food fallacy: the protein myth

While we're here separating the wheat from the chaff and the myths from the science, let's look at the popular old wives' tales about protein. The grapevine has it that people with Parkinson's disease should not eat protein. That is NOT the advice given by the pharmaceutical industry.

This myth is an outgrowth of the fact that levodopa is a protein. The advice given by the industry is this: a person should not take levodopa-containing pills immediately before or after a protein-heavy meal. This is a very, very different statement than "a person with PD can't eat protein anymore."

When a person begins to chew a meal that has considerable protein in it, the mouth and tongue detect the protein and signal the stomach to pump some protein digesters into the stomach. Different foods require different digesters that are specific for that type of food. If the stomach is told to produce protein digesters and a person throws back a pill of levodopa, the stomach will digest the pill along with the beef and cheese. Therefore, the wise levodopa user will take his pill an hour before or three hours after he eats a protein-heavy meal. With no obvious, large protein molecules present in the mouth to stimulate a release of protein-digesting enzymes, and with carbidopa somewhat disguising the proteinaceous nature of the levodopa, the small levodopa/carbidopa pill can slip through the stomach pathway without being digested. It is that simple.

This myth, that people with Parkinson's cannot eat protein, has grown to terrible proportions. Easily a fourth of my patients are courting protein deficiency because they have not eaten any significant protein in the years since they were diagnosed with Parkinson's. This is terrible, this is ridiculous, and this is dangerous. I had one patient in my office in tears of self-recrimination because, in a moment of weakness, she'd had eggs for breakfast. She was certain that her Parkinson's would be worsening quickly due to this unforgivable lapse.

And making it all the more strange, if a person refuses protein for a long time, this very absence of protein makes that person's body become so desperate for protein that the little pills of levodopa, which ordinarily would be sneered at by a body that has adequate beans or brisket, now begin to look to a protein-starved body as something worth bothering about. If this pill might be the only protein it is going to get all day, the stomach will produce protein-digesting enzymes just for the pill! Consequently, the starved body focuses its protein-grabbing function on the pill. Ironic, eh?

When my patients started eating protein again, and carefully having their pills an hour before or three hours after a protein-heavy meal, they felt much healthier – *and their pills worked much better*. A person who is getting enough protein and not taking his pills at the same time as protein-heavy meals will find that his pills are not nearly as affected by meal times as they are if he plays games with his protein intake.

By the way, if one is eating a meal that consists primarily of fruit or vegetables, the pill can be taken much closer to meal times. As long as the body is getting adequate protein, a little pill taken about the same time as a fruit salad will slip right past the tongue sensors and the protein-digesting enzymes won't even be alerted.

I was wondering where to fit this in to this book. I suppose this appendix on dopamine myths and false facts is the perfect place. Life with Parkinson's is challenging enough; there's no need to make it a time of protein deprivation as well.

The fallacy of treating immobility with sedatives

In accordance with the old theory of acetylcholine/dopamine imbalance, people were given anticholinergic drugs to reduce acetylcholine levels. Some patients noticed a subsequent decrease in their tremor and the sedation of their nervous, shuffling pacing. It was assumed, on the basis of these symptom changes, that the acetylcholine/dopamine balance had been improved.

Tremor and restless pacing are the two PD symptoms that are stress-sensitive; an increase in stress can cause increased tremoring. A decrease in stress sometimes allows tremor to decrease. Falling asleep usually stops the tremor completely.

The reason that the anticholinergics appeared to work is that they are essentially knockout drops: they made a person weak, groggy, and sleepy. Anticholinergics decreased tremor because they made a patient tired. This tiredness, meanwhile, from inhibition of acetylcholine, caused most of the other PD symptoms to worsen. Symptoms such as poverty of movement, cogwheeling, micrographia, slow voice, slow thinking, and all the other symptoms that require muscle power or alertness were made worse by the administration of anticholinergics.

To the eye of the doctor, the decrease in tremor seemed to indicate that the patient was doing better. The fact that these sedative drugs sometimes robbed the patient of the strength to get out of a chair or even to think straight had to be balanced against the fact the tremor was repressed. What these doctors didn't realize is that any relaxant, from classical music to antihistamines, would have decreased the tremor. But not knowing this, it did seem as if the calmed tremor was proof of the theory that Parkinson's disease was caused by excess acetylcholine, or, more recently, an imbalance between acetylcholine and dopamine. There are doctors today that still subscribe to this theory and point to the anticholinergic drugs as proof. Certainly, the language of the drug manufacturers supports this thinking: in 2002, in keeping with the decades old acetylcholine/dopamine imbalance theory, the published purpose of these drugs was to "balance cholinergic activity in the basal ganglia."[1]

Today, in a complete reversal, some of the new drugs for Parkinson's disease *elevate* ACh levels. The new thinking here is that, since ACh is necessary for motor function, and PDers move slowly, maybe they need more ACh, not less. As noted earlier, some doctors, unaware of the processes involved and only aware that both drugs are listed as "antiparkinson's," may well prescribe both these medications to a trusting patient.

"Facing" the facts

For another glaring example of the way in which medical evidence doesn't necessarily fit the facts, look carefully at the expressionless face of a PDer in advanced, unmedicated (if you can find one) PD. The cheeks sag down heavily. Sometimes, the weight of the lifeless cheeks pressing on the facial bones makes the sinuses feel as if they are collapsing down into the mouth, causing the extreme snoring and sleep apnea sometimes seen in PD.

---

[1] *Physician's Drug Handbook*, p. 1064.

And yet, as late as 1989, the physiology text at the school where I teach stated that in Parkinson's, "the *rigidity* of the facial muscles gives the face a mask-like appearance"[1] (emphasis added). This is just plain wrong. Rigid muscles are hard, not sagging. Overly tight muscles can cause burning pain, like the pain of a muscle cramp. Rigid muscles in the face would produce the risus sardonicus, the hideous grin that is seen in the case of too much medication. The mask-like, expressionless, *limp* muscles of the Parkinson's face are cold, weak, and numb.[2] The facial muscles in Parkinson's are not rigid with excess tension. They are limp, or even deathly limp, with an utter lack of tension.

When patients instead of doctors describe PD, they say that what they feel is heavy, slow, and dead inside. Though they often have tremendous inner drive, intensity of purpose, and strength of personality, the rigidity that they feel in their bodies is the opposite feeling from strength – it is a feeling of absence, a feeling of leaden lifelessness. However, the claim by the researchers that the loss of muscle tone in the face and the rigor mortis-like woodenness in the muscles along the anteriolateral side of the leg (especially on the side of the body where the PD started) come from an *excess of strength* still remains today in some literature on PD.

Slanted conclusions

Going far beyond the problem of mere stodgy adherence to faulty conclusions and historical precedent, another problem arises when trying to sort the facts from the fallacies: drug companies have a vested interest in the conclusions of their studies.

For example, the makers of Mirapex, an antiparkinson's dopamine agonist, sponsored a four year study with over 80 participants. The results, written up in January 2002[3] in the *Journal of the American Medical Association* showed, using SPECT scans, that over the 48 months of the study the Parkinson's patients taking L-dopa had a decrease in dopamine transport of 25.5%, while Parkinson's patients using an agonist had a decrease of only 16% in dopamine transport.

Completely ignoring studies that prove that L-dopa causes a decrease in dopamine transport, the Mirapex-sponsored study ingenuously stated that L-dopa did no harm. After all, L-dopa is only dopamine, and people with Parkinson's are dopamine deficient, so giving dopamine cannot hurt them. They then leapt to a wild conclusion that the damage seen in L-dopa users during the study represented the normal damage that occurs from untreated Parkinson's. Because people with Mirapex had measurably less damage than did the people using L-dopa, the manufacturers of Mirapex brazenly were able to make the false conclusion that Mirapex *slowed the progression* of Parkinson's disease!

---

[1]G, Tortora, N. Anagnostakos, *Principles of Anatomy and Physiology*, Harper & Row, New York, 1987, p. 332.

[2]The painful cramping and dystonia that sometimes manifest in PD occur when the leaden and unresponsive muscles of damaged tissues cannot maintain a balance with their oppositional, and still-healthy muscles. The healthy muscles pull, but their counter pullers are too ineffectual, and so torsions of the limbs ensue. These torsions are relieved by medication in part because the medications are powerful pain-relievers and mood relaxors – aspects of dopamine that were not recognized until recently. In the absence of pain, the muscles can do splinting, in which working muscles compensate for missing muscles, taking on jobs for which they were not designed. This splinting leads to further pain, but it provides function. As long as the pain relieving function of the drugs is effective, this allows for a joyful, if temporary, resumption of somewhat normal movement in tissues that were dystonic.

[3] *JAMA* 2002; 287: 1653-1661.

A report footnoted earlier in this book (see page 42, the Elldopa study) presented measured evidence that people taking L-dopa have a decrease in dopamine transport more than seven times greater than those taking a placebo. Given this information, it does seem that the Mirapex logic, and especially their contention that L-dopa does no harm, is suspiciously self-serving. A less prejudiced conclusion, derived by combining the dopamine transport loss rate seen in the Mirapex study with the result of studies done on placebo (dummy pill) patients, might have been that Mirapex, though less damaging than L-dopa, still nearly doubles the rate at which untreated Parkinson's progresses.

The Mirapex study was conveniently done without any placebo (control) patients, but the conclusion that Mirapex might slow the progression of Parkinson's was widely advertised.

While we are on the subject, and so you will know that I am not personally taking sides in the Mirapex versus Requip contest, it is most likely that all dopamine agonists, not just Mirapex, cause acceleration of Parkinson's disease.

A nice change of pace

As I dig into the slimy pit of drug company research manipulations, I realize that this chapter is rapidly developing a negative slant. The original aim of this chapter was not to show that profit-seeking pharmaceutical companies do distort the results of their research and publish their false claims in doctors' journals where they are often accepted at face value. My goals in this appendix are to show that old research is hard to uproot; the most well-meaning of doctors have a difficult time in overcoming the paradigms that they learned at their professors' knee. The false root assumptions can not only branch upward and outward into long-lasting fallacies but can also send spreader roots so that, even when the first false root is killed, the errors and fallacies live on.

Unhappily, pointing out the all too human tendency towards adherence to our first teachings can come across, in this medical tome, as anti-doctor. To correct this tone and to bring us back onto a more nourishing perspective, I would like to share the famous prayer written by Maimonides.

The Doctor's Prayer

Thy eternal providence has appointed me to watch over the life and health of Thy creatures. May the love for my art actuate me at all times; may neither avarice nor miserliness, nor thirst for glory, or for a great reputation engage my mind; for the enemies of truth and philanthropy could easily deceive me and make me forgetful of my lofty aim of doing good to Thy children.

May I never see in the patient anything but a fellow creature in pain.

Grant me strength, time, and opportunity always to correct what I have acquired, always to extend its domain, for knowledge is immense and the spirit of man can extend indefinitely to enrich itself daily with new requirements.

Today he can discover his errors of yesterday and tomorrow he can obtain a new light on what he thinks himself sure of today. Oh, God, Thou hast appointed me to watch over the life and death of thy creatures; here am I ready for my vocation and now I turn unto my calling.

This humble prayer by Rabbi Moses Maimonides (1135 –1204 AD), a Spanish philosopher and physician who spent most of his adult life in Egypt, is read at some medical school graduations. This prayer makes reference to the ever-changing nature of medicine and, rather than asking for perfect wisdom, asks for the wisdom to be always learning. Most people take it for granted that these noble sentiments motivate their doctors. I know that many doctors work hard to uphold these ideals.

Continuing education: a doctor's prerogative

Contrary to common belief, there is no requirement that a clinical neurologist must keep up with the all the rapidly changing "facts" of brain science. In fact, it would be impossible for him to do so. The new information is coming in so fast that only the researchers can keep up with the changes, and even then they can only stay current within the very narrow field of their specialty. While MDs are required to take a certain number of course hours per year to maintain their license, they are not even required to take these continuing education courses in their own field of specialty, if they have one, nor are they required to take regular exams to test their command of the current findings.

One of the most common email queries I receive is: "How is it possible that my MD has incorrect/outmoded information?" The answer is this: your doctor is human.

Keeping up with the latest: fact or fallacy?

Summary

The science most often being used today to explain Parkinson's disease is based on logic dating back to more than fifty years ago: logic proven to have been false. The medications being prescribed today for Parkinson's often work in the opposite way from the way they are intended. Because of the multiplied errors in Parkinson's disease theory and misunderstandings about the PD drugs, many treatments are currently being prescribed which actually accelerate the illness or cause side effects that are then treated as if they were an advanced form of the illness.

This premise was stated in chapter one. This appendix was written to show you how and why some of the "scientific" fallacies about Parkinson's have been perpetuated. I have included some of the revelations that I had while doing this research, the first being dopamine's role as a daytime stimulant – despite all the rhubarb about its being a relaxor. A disappointing corollary to this new tidbit was the realization that massive amounts of data already existed that indicated that dopamine was a stimulant but that, with their noses buried in the notebooks of assumptions, no researcher had bothered to stand back and take a gander at the amassed data from a larger perspective.

A bemused moment was enjoyed when I read, in a single paragraph in a respected Parkinson's journal, both that dopamine cells disappeared and that dopamine cells were not lost (see footnote, page 569).

As I pored over thousands of the latest research reports in this field, and was swamped with nearly as many reports from patients about their doctors' advice, theories, and prescribing patterns, I awoke to the fact that modern medicine simply does not reflect modern research; many MDs are practicing medicine based on illogically "proven" or even long-since disproven fallacies.[1] This was perhaps the most disheartening insight of all.

Ultimately, these painful revelations of doctor fallacies were all to the good. When, eventually, we had to advise our medicated patients, "The advice that your highly qualified doctor gives you may be wrong, and even harmful," I was ready, thanks to my experiences with outdated or outright wrong medical pronouncements, to stand by those strong and painful words.

Your inclination may be to disbelieve that your doctor is giving you dangerous, even deadly, drugs. Your spouse, friends, and loved ones may vehemently disagree with you if you say that the drugs are dangerous. They will probably side with your MD. Most of all, they will want to believe in Science. Unfortunately, there are wise MDs and there are ignorant MDs. There are wise practitioners of Asian medicine and there are ignorant practitioners of Asian medicine. A well-meaning heart and a diploma, even prizes and acclaim, do not necessarily translate into accurate or up-to-date knowledge.

Because of the tremendous amount of rapidly changing information in the realm of medicine, and because, ultimately, who we are and why we have the particular illness we do may be more related to deep mysteries of spirit than to the results of lab tests, the greatest qualities a doctor can have, whether he is eastern or western, are intuition,

[1] See Appendix 8, p. 560, which quotes the article "Clinical Practice Lags Behind Medical Research" from the *Journal of the American Medical Association* (*JAMA* 2002; 287:1653-1661).

willingness to learn, and enough humility to say “I don’t know,” or even, “Forgive me, I was wrong.” These qualities are not yet being taught even in the best of medical schools.

Hopefully, this appendix demonstrated that science is not actually about facts. Science is about politics, money, and faith. New evidence is usually convoluted to make it conform to the status quo. Proofs, like statistics, can always be arranged to make the desired case. As long as you regard science as a sacred cow and your MD as its priest, you may not be able to make intelligent decisions about your antiparkinson’s medications.

Appendix 7

THE ALPHA PRIMATE/COCAINE STUDY

In the first chapter of this book a reference was made to a published research project that studied cocaine addiction in primates.[1] This appendix gives more details on that study. This 2002 study helps support our findings about addiction in PDers before and after recovery. There are two parts of the alpha primate cocaine study that are particularly significant: first, the subjects who became alphas still used cocaine but no longer needed an ever-increasing dosage of cocaine to maintain their response, and secondly, SPECT scans showed a difference in dopamine receptor activity after a primate changed from isolation status to alpha status. This appendix will explore the results of this study in two parts.

PART 1

The study and its cocaine related findings

In the study, male primates were first kept in isolation cages for months and had free access to cocaine. They were allowed to self-dose. It was observed that *all* the animals were equally addictable: they all used ever-increasing amounts of cocaine. The increase in need was assumed to be due to addiction. Following this study, the animals spent several months with no cocaine to get the cocaine out of their system.

A subsequent study determined that when the same animals were caged together in four-member, all-male social groups, the primates that became the alpha male of their group (the dominant male at the top of the pecking order) still used cocaine but they never needed to increase their daily doses beyond their starting levels. The social groups' lowest ranking members, the omega males, were *more* subject to addiction than before: their cocaine dosing increased more rapidly and soared to a higher level than when all the subjects had lived in isolation.

A key feature of addiction as it is currently defined is the need for ever-increasing amounts of the addictive substance to attain an effect. Because all primates needed ever-increasing amounts of cocaine in the first part of the study, the conclusion was formed that all the participants were subject to addiction. After the housing change that led to social formations, the now alpha males did continue to use cocaine, but did not need to increase their doses over time. It appeared as if these primates, when alphas, were not susceptible to addiction.

[1] *Nature Neuroscience* 5(2): 169-174, 2002, Mike Nader.

More details

This research was exploring a possible link between social standing and addiction. The brilliant and very helpful researcher, Mike Nader, implied that there is such a link: elevation of social standing might have caused a decrease in susceptibility to addiction.

Happy, carefree alphas

The author of this study hypothesized that the dominant primate had more of the good things in life, and therefore had less need for cocaine. He proposed that the alphas had a higher dopamine level (possibly due to all the fun they were having as alphas) and therefore did not need as much cocaine. Since less cocaine was being ingested, the addiction rate, both pace and quantity, was lower. The chimps at the bottom of the totem pole were assumed to be less happy, and therefore craved cocaine to help make themselves happy despite their dire circumstances.

The assumption being made is that alpha males are happy, or at least contented, males. This assumption is, I propose, in error.

Wary alphas

I hypothesize the opposite: alpha males are more alert, less able to let their guard down. Upon attaining alpha status, the dominant subject must rely more on adrenaline than on dopamine. The dopamine/adrenaline relationship must shift towards the adrenaline end, away from the dopamine end. This adrenaline shift would be necessary to maintain safety, as well as maintaining alpha status: a dominant primate must be continually alert. He is the subject of relentless scrutiny by all other males. The other males behave with subservience, but they are also always watching him to see if he shows signs of weakening. The alpha is the one for whom any show of weakness would be dangerous, even fatal.

The top monkey is always looking over his shoulder. He needs constant vigilance against predators and the constant threats of bodily harm from challengers. He needs to show constant readiness to defend females, infants, and children from any and all threats. He needs the ability to, from a lonely distance, oversee the harmony in the group of females and children. He must behave fearlessly, even pretending to be unhurt when injured.

The pack of challengers will quickly mount a potentially deadly group challenge if any sign of weakness is shown. Although his superior fighting tactics and focus helped him to attain the honored alpha seat from which he can contribute his superior DNA to the harem's gene pool, he must suffer his "honor" in loneliness and constant wariness.

Research review

One wag from *The Economist*, reviewing Mike Nader's conclusion, even went so far as say that it seemed unjust: alphas had all the sex, all the fun, and everyone kowtowing to them, plus they didn't get addicted. He noted that the losers, the ones with no sex and therefore no fun, also had the greatest propensity for addiction. The *Economist* chappy felt that this just wasn't fair – the animals having the least fun also were most prone to be drug addicts!

I wonder if it ever occurred to this reviewer that the life of an alpha male in a cage with three jealous challengers, no privacy, and no females or children would be about as

far away from “fun” as any lifestyle I can imagine. The constant edginess of maintaining his dominance in a close setting with three smirking subordinates, all of whom were just waiting, waiting for the alpha to make a slip so that they could kill him in a group attack, does not seem especially jolly.

A PD parallel with alphas

Many people with Parkinson's disease, whose dopamine levels coincidently are very low, can relate to this feeling of constant alertness, a high level of self-control, and refusal to show any sign of weakness. They are often very successful in life, very intelligent, and may, in work and play, maintain a more self-controlled persona than their colleagues.

Famous PDers who have exhibited this personality include Adolph Hitler, Chairman Mao Tse Tung, General Douglas MacArthur, Yasir Arafat, Pope John Paul II, and many others. It has been proposed that Napolean Bonaparte kept his hand in his shirt front to control a tremor – certainly his famous sleep pattern was consistent with that of many unmedicated PDers. As for the Great Emperor who unified China in 221 BC, it seems likely, based on his “premature aging” and early death from what sounds like aspiration pneumonia, that he too was an alpha male with Parkinson’s disease.

The intensity of activity and purpose and high level of emotional self-control of PDers is well recognized. The underlying fear that drives them is described as “emotional harm avoidance,” and is a recognized part of the Parkinson’s personality.

Many PDers are not obviously “dominant:” some PDers choose to use their adrenaline to maintain an unshakeable equipoise. This stability is not based on inner serenity, however. For the most part, those PDers who behave with undisturbable balance are doing so because they dread any sort of tension. A pathological fear of emotional, and in some cases physical, harm or conflict of any sort may lead them to use their terrific powers of intelligence and alertness to behave as if subordinate, even though they are actually running the show to their own specifications. This form of manipulative behavior is much more subtle than passive-aggressive; this behavior is actively harmonizing, and, in the case of PDers, is based on a pathological level of fear.

“Harm avoidance” may well be a dominant factor in the behavior of an alpha male. It may well be that alpha males, like PDers, are adrenaline-dominant, dopamine-subordinate. If this is the case, this primate research may help support our own observation, made during our four years of research, that PDers appear to be fairly resistant to addiction. It may also help explain why, upon beginning to recover, PDers become normally, or even abnormally, susceptible to addiction.

Addictability is variable

Regardless of whether or not the changes occurred due to more fun or heightened vigilance, the most exciting thing about this study was its conclusive proof that addictability was variable. External (non-genetic) change in social standing could alter addictability. This is a very new concept.

The abrupt changes seen in our patients who began recovering from Parkinson’s disease while still taking antiparkinson’s medications defied anything we had read in our medical texts. If, in fact, susceptibility to addiction could change quickly, and in response to something so “superficial” as a change in emotional condition or social status, as was

suggested by this primate study, then possibly our findings were no longer quite so much of a bizarre aberration. As one colleague said to me with regard to this timely primate study, "You never would have thought of doing this study; they did your research for you!"

PART II

The study and its findings with regard to SPECT scan changes in alpha males

SPECT scans employed in the study measured the level of activity at the subjects' brain dopamine receptors. These scans use a radioactive "tag," or "tracer," a molecule that is attracted specifically to dopamine receptors, and to which a radioactive bit has been added. The subject is given the tags intravenously, and the scanning machine then measures the amounts of radioactivity that moves into the various brain areas.[1] The researchers in this study were wondering if monkeys with different social ranking would have different levels of activity in their brains' dopamine centers.

As an aside, the relationship between social standing and addictive drug and alcohol use has been pondered for millennia. Because addicts sometimes lose their social skills, to say nothing of jobs and relationships, when their brains become preoccupied with drugs, the apparent relationship between drugs and social standing is this: low social standing is sometimes related to drug addiction. On the other hand, one might say that it is low social standing that causes one to reach for a mind-altering drug in the first place. Sociologists argue strongly back and forth as to whether or not the poor social standing preceded the drug addiction, or if the drug addiction led to the poor social standing. The study with primates was looking for brain differences between individuals prior to drug use and again after the socialization changes in hopes of finding a clue about these ancient questions.

Alpha males have more dopamine receptor activity

The before-socialization and after-socialization SPECT scans of the animals' brains indicated that primates living alone in separate housing all had similar responses to the tags; their dopamine receptors' activity levels were all comparable.

Subsequent to the group housing, the levels of dopamine receptor activity changed. This change corresponded to the social ranking of the primates; the radioactive dopamine-like tracer showed up as greater receptor-area activity in the alpha male and decreased receptor-area activity in the omega male.

Conclusion of the researcher: increased activity means increased dopamine levels

The researcher who assessed the results of this study guessed that alpha males' increased tracer activity at dopamine receptor sites probably equaled higher levels of native dopamine activity. He further concluded that this elevated dopamine level was the reason the alphas did not need ever-increasing doses of cocaine. Because cocaine's

[1] For a fun aside, one of the first researchers to use this technology became infamous. In the USA the laws prevented him using humans for his research, so he went to China in 1999 and used the machines there. He used himself as a subject and used a radioactive cocaine analog, measuring his own brain's response. He published his work and was hailed as both a brave hero (by his own university) and a villain (by competing universities, who insisted that he should somehow be punished).

pleasure enhancement comes from its ability to elevate dopamine, the researcher assumed that the underlying dopamine increase, as suggested by increased receptor activity, eliminated the need for more dopamine enhancement.

This logic may be completely wrong. To show how this assumption may be 180º backwards, let's look at the findings of SPECT scans on the brains of PDers and consider the assumptions. Hopefully, as you read this, you are recalling the preceding appendix on fallacies in science, and how wrong assumptions lead to convoluted proofs further downstream.

What SPECT scans show and what they do not show: still anybody's guess

I want to consider the possibility that the radioactive tags that *purportedly* measure dopamine receptor activity and thus imply a certain level of dopamine activity, may not actually measure these things. They may do only this one, much smaller thing: they may show how many empty dopamine receptors are available.

Let us assume that a radioactive tag that is designed to fit in a dopamine receptor succeeds in getting into the brain. The extent to which the brain absorbs these tracers and lodges them into the receptors might depend to a large degree on how many receptors are sitting by idly. It may be that, in a situation where the receptors are already saturated with dopamine, the injected tags will not be able to find a resting spot – it may even be that they will have increased difficulty in crossing the blood-brain barrier.

On the other hand, if a person has a dopamine deficiency, the tracers may find dopamine receptors open armed and waiting for a hookup. In this latter case, an increase in SPECT scan radioactivity in the area of the dopamine receptors will indicate that the subject has an excess of *unused* receptors; these receptors may be unused due to an underlying *shortage* of dopamine – just the opposite of the conclusions given in this study.

SPECT scans in PDers: an aside

SPECT scans in PDers usually show reduced receptor activity. This finding, coupled with the long-held (fifty years) assumption that PDers sole problem was a lack of dopamine, inexplicably was used to "prove" that low dopamine levels were connected with low receptor activity as seen in SPECT scans.

Logically speaking, one would have thought that low receptor activity would indicate low receptor activity, and nothing else. If anything, a deep thinker should have proposed that PDers, having insufficient dopamine, would also have lots of empty dopamine receptors. These empty receptors would have been sitting ducks for the radioactive tags. This would then mean that decreased dopamine availability, as seen in PD, should lead, theoretically, to *increased* receptor activity in the presence of the radioactive tags, increased compared to a normal brain that supposedly has a full complement of dopamine. This would have been logical.

Since the opposite result was found in PDers, namely that PDers have both lowered receptor activity compared to normal and are also traditionally purported to have lowered dopamine levels, one might hope that some researcher might have considered the possibility that our overly simplistic view of Parkinson's might be wrong. This hope, of course, is far-fetched. Logic scarcely entered into the thing, and the idea that the preexisting ideas about Parkinson's were wrong was never entertained. It was announced,

shortly after SPECT scans were first employed in PDers, that low receptor activity must naturally be an indicator of low dopamine levels! The reverse was then dutifully noted: high receptor response to the radioactive tags meant that the subject must have high levels of dopamine.

No one has ever actually tested this theory by performing SPECT scans on rat brains and then assaying the results to see whether or not a high tracer level at the receptors corresponds to a high level of dopamine. Instead, a sort of backwards thinking, based on assumptions about Parkinson's disease, has led to the current mode of interpreting these new scans – a mode that may be completely wrong.

While one might still use the tests to decide whether or not a PDer's dopamine receptor activity level is consistent with the scans of others who have also received a diagnosis of Parkinson's, the expanded conclusion that high receptor activity indicates high dopamine levels may be completely wrong.

Logic: the road not taken

It might be that not only dopamine itself, but all parts of the dopamine system, including dopamine transporters, dopamine reuptake enzymes, dopamine-producing cells and dopamine receptors are all slowly, over the years, decreased in a person with Parkinson's disease. This would explain the low dopamine receptor response in PDers' SPECT scans.

This more logical assumption might lead to a better understanding of Parkinson's disease, a disease in which the body has turned off the dopamine *system* altogether, or at least turned it down very, very far for a very long time. This would explain why PDers have both a decrease in dopamine and, eventually, a decrease in dopamine receptors. Let's look at the thing from this angle. After that, we can go back to the primate study and consider a conclusion quite different from the one made by the paradigm-weighted researcher who imagined that his happy, contented alphas were refraining from cocaine because increased dopamine receptor activity – and therefore high levels of dopamine – were keeping them content.

PDer SPECT scans: let's suppose

SPECT scans of people with Parkinson's disease show decreased receptor activity: the radioactive tracer drugs simply find fewer resting spots in the brain of people whose various dopamine structures have decreased or been dismantled over decades of unuse. During their decades of subclinical PD, their dopamine-producing structures, dopamine receptors, dopamine transporters, and other dopamine-related enzymes have been decreasing.

Were this not the case, and if all the dopamine receptors were still in place along with all the rest of the dopamine-related chemistry, and the only thing missing from a PDer was the dopamine itself, the infusion of tagged, dopamine receptor-seeking drugs should swarm onto the receptors, the tagged drugs filling in the otherwise vacant receptors.

This is NOT what we see in a SPECT scan of a person with Parkinson's. Even though a PDer is given an adequate quantity of tagged drugs, the scan still shows a lower receptor activity than a normal brain. This may mean that not only dopamine levels, but *receptor* levels (the number of receptors) are eventually decreased in Parkinson's disease.

Or possibly receptor responsiveness is decreased. Or for that matter, maybe the amount of tracer pulled inside the blood-brain barrier is the commodity that decreases in a person with Parkinson's disease. Or the correct answer may be "D," all of the above. No one knows.

While these low-receptor SPECT scan tests are beginning to be used to confirm a diagnosis of Parkinson's disease, they might be better used to confirm that Parkinson's disease is not caused by mere dopamine deficiency, but by some alteration in the entire dopamine processing system over time, an alteration that prevents radioactive tracers from collecting on PDers' receptors in quantities as large as they can on healthy people's receptors.

Primates vs. PDers: where we are going with all this

Following their change in social status, primates in the cocaine study underwent rapid changes in their response to cocaine and their SPECT scans also showed a quick change from the previous scans. The alpha males were *no longer addictable* to cocaine, and their SPECT scans showed an *increase* in dopamine receptor activity.

People with Parkinson's also have a *low level of susceptibility to addiction*, **but** their SPECT scans show a *low* level of dopamine receptor activity.

If there is any relationship between the lack of addictability and the level of dopamine receptor activity, why do these two groups have such opposite SPECT scans?

Short-term neurotransmitter change as compared with long-term neurotransmitter change

The primates in the study were only observed over a period of months. During this brief period, the alphas may well have changed their adrenaline/dopamine ratio so that their adrenaline was higher and their dopamine was lower. This would make them less subject to addiction. Their healthy brains would still have a full complement of dopamine-related chemistry and structures, but they would be temporarily not in use. Radioactive tags, if inserted into the bloodstream, might easily find their way into the system and cluster onto the unused dopamine receptors, thus making a large showing at the receptor sites, a showing that would indicate much activity in these areas on the scans.

In sharp contrast to this temporary drop in dopamine manifesting in the alpha males, a PDer has brain changes that have been taking place over decades. Is it possible that the brain change of Parkinson's that stifles the entire dopamine system might lead to a gradual decrease in dopamine, dopamine-producing cells, dopamine-related enzymes, and dopamine receptors? By the time the PD is diagnosable, decades later, there might be a substantial decrease in all structures or functionality of structures that relate to the dopamine system. If this is possible, then it only makes sense that a SPECT scan of a PDer will show less activity in receptors: he may have fewer receptors, period!

If so, then we have the answer to the puzzling difference between PDers whose dopamine systems have been shut down for decades and primates whose dopamine systems have been recently put on hold while they perform as alphas. A SPECT scan of the PDer's brain will show a decrease in receptor activity, particularly on the damaged side of the brain, the side that has not been receiving a healthy electrical signal to its dopamine system. A SPECT scan of a recently promoted primate may show a significant

number of vacancies in the dopamine receptors, as manifested in larger than usual numbers of the tag molecules being picked up.

Hypothesis

The long-term changes in PDers may not be analogous to the quick alteration in primate brains that occurs in response to a change in social status. Comparing these situations and trying to make a connection between diminished SPECT activity in the former and increased SPECT activity in the latter may not be as worthwhile as previously imagined. It may be that the fast time frame of neurotransmitter release, and the slow, concomitant structural changes that occur over time make it inappropriate to form conclusions about a primate's *short*-term change based on previous data gathered from observing PDers' *long*-term change; the historical basis for concluding that low receptor activity is related to low dopamine stems in part from SPECT scans of PDers, plus lots of conjecture. The researchers, trying to impose interpretations of PDer SPECT scans onto the SPECT scans of primates, may have been comparing dried apples and fresh oranges.

Further proof: uncooperative brain cells

Recent research suggests that even in PD brains that are supplied with adequate dopamine levels via levodopa, some dopamine receptors, especially those that have had long term exposure to dopamine-enhancing drugs, increasingly refuse to use the dopamine that is thrust upon them.

This reluctance on the part of PDer receptors to accept dopamine may also contribute to the lower tracer levels seen in PDer SPECT scans. PDers' SPECT scans may register low receptor activity levels even when dopamine is present! In other words, the results of a SPECT scan do not actually reflect dopamine levels or the quantity of receptors. They merely indicate how many tracers are being used by receptors – nothing more, nothing less.

What SPECT scans really show

The deeper meaning of these SPECT scans is still cloaked in mystery. Like Plato's cave dwelling fellows making conjectures about the real sizes, shapes, and colors of objects which the fellows – remaining in their caves – only perceive as inconstant shadows[1], the people who employ SPECT scans are only guessing as to the meaning of the black and white images generated by the radioactive tracers deep in the brains of their subjects.

Looking ahead

What we really need here are SPECT scans of people who have just made the transition from normal to pre-parkinson's. When their foot injury occurs and they switch into ferocious denial of the injury, that would be the time to get a SPECT scan of these latent PDers. It may well be that they too would show increased activity of tracers in that first flush of powerful denial and self-control, when adrenaline surges through the body and dopamine receptors are lying around empty.

[1] See Plato's *The Republic*.

This is all very much fun to ponder, and it doesn't need to be further addressed in this book about medication. But this little foray into the meaning of SPECT scans might, if nothing else, help the reader to remember that science, with all its reproducible data, does not provide Truth. The real value of this appendix's musing on SPECT scans is pointing out that it is naively assumed that increased tracer activity means increased dopamine when, in fact, increased receptor activity in response to tracers may mean less native dopamine.

It used to be assumed, because the sun rose every morning, that the sun went around the earth. It turns out the best assumption to be made from the fact that the sun rises every morning is simply this: the sun *appears* to rise every morning. Every other possible theory that derives from that observation is only a guess. Science is not about Truth. Science is about converting observations into guesses. The guesses necessarily reflect our desires and prejudices.

The extreme value of this study

The most valuable aspect of the primate study, from the perspective of Parkinson's research, is that it suggests that a model for Parkinson's can actually be found in an animal setting. Until now, the only way to get an animal cell model that even remotely resembled the dormant dopamine-producing cells of PD was to destroy cells via toxins. There was no known way to induce a long-standing adrenaline/dopamine imbalance in an animal, that I knew of.[1]

Because animals in nature do not have the emotional constraints and personality/ego perversions that are the necessary basis for denial of injury, I had presumed that there would never be an opportunity to observe an animal model of Parkinson's disease. However, I had never considered the sad situation of the alpha male.

The alpha male, friendless, extremely responsible, even selfless, always on alert, looking over his shoulder for potential attacks or betrayal from his boyhood playmates, living *for* society but not *of* it, is a beautiful, natural example of several aspects of the Parkinson's personality. That these noble, selfless animals – after the heavy burden of alpha-hood is won – should also be, like PDers, unaddictable to dopamine-enhancing drugs, may be another indication of their brotherhood with the unaddictable PDer.

Most medicated PDers have pooh-poohed the idea that their vaunted will power will be affected when their Parkinson's-inducing fear pattern is altered; they often refuse to believe that such a change is possible – often with disastrous results. The rapid change in alpha primates provides proof that such a change is possible: the measured, rapid change in primate addictability seen in this study in response to a change in social setting presents a new way to think about addiction.

If high or low resistance to addiction can be understood primarily to have a physiological basis rather than the will power basis with which most PDers credit themselves, possibly they will better comprehend the dangers that confront them if they begin to recover from Parkinson's disease while taking supremely addictive medication.

[1] While I believe much of the animal testing in labs today constitutes unnecessary abuse, animals in the wild do provide a valuable window into natural phenomena, a window uncluttered with the "civilized" emotional twistings and sociologically induced illness that humans are heir to. Animals in the wild make a valuable contribution to our observances of natural history.

Summary

The abrupt reduction of addictability in alpha males when they are under stress may be related to the decreased susceptibility to addiction that is seen in people with Parkinson's. Also, SPECT scans, while they may not offer the proof of high or low *dopamine* levels per se, may be valuable in this regard: alpha primates show a change in their dopamine receptor areas when their social status changes. This indicates an objective basis for noting that brain neurotransmitter levels may be influenced powerfully by sociological (external, also known as "environmental") factors.

Also, there may or may not be a relationship between the decreased addictability of alpha males – a presumably dopamine-related phenomenon – and the objective, concomitant finding of altered SPECT scans in the dopamine receptor area of these animals.

Appendix 8

WORKING WITH YOUR DOCTOR:

BUILDING A GOOD RELATIONSHIP

Your doctor may be an all-knowing saint or avatar. On the other hand, he may be a mere human. If the latter, he will tend, like the rest of us humans, to be just a tad defensive, proud, and worried about his professional liabilities. Therefore, if you really want to have a good, working relationship with him, you will start by acknowledging him as a human. You will not, at some time in future, attack him with diatribes based on something you've read in a book. Your doctor is a professional, but he is not necessarily a psychic or able to keep up with all the information available about Parkinson's. There is a very good chance that he is being the best doctor he knows how to be.

When it comes to Parkinson's medications, while you need to be aware that he is probably un- or misinformed, it is not your place to educate your doctor. He must work within the Standard of Care for his profession, no matter how many clippings you bring him showing him that he is wrong.

That having been said, the single most common problem that my patients have had with their doctors comes from the MDs' resentment towards patients' "insubordination," patients' refusal to follow orders unquestioningly, right or wrong.

Be aware of your rights.

Doctors do not have the right to tell you what you must do. Their prescriptions are only suggestions – so long as you are competent and not a danger to yourself or others, you have the right to use medications or not. However, many doctors, for noble reasons or lowly, decide that their duty is to command and yours is to follow. Therefore, if your doctor is of this type, you may wish to placate him. He is, after all, only human. Then again, you may need to change doctors.

If you, on your own, decide that you wish to reduce your medication, you are implying that the doctor has made a mistake. No one likes to be told that they made a mistake. My patients tried various methods of vocabulary to avoid hurting their doctors' feelings while informing the doc that they were reducing medications, but we have found that only one method was universally successful.

Doctors respect hallucinations

As noted in the text, the advent of hallucinations was the one sure method of receiving doctor support for reducing medications. During the three years while Hjalmar had been getting treated by me and reducing his medication, he had seen his neurologist every six months. Dr. Rafferty had praised him for how well he was moving, and asked him what he was doing differently. The first time, Hjalmar said "Acupuncture." Dr. Rafferty laughed and said, "No, really, what are you doing?" Six months later, when Hjalmar again answered the question with "acupuncture," Rafferty laughed again, and said, "Your wife sure is taking good care of you." This happened again six months later.

When, six months later still, Dr. Rafferty asked him for the fourth time, "What's your secret? You're doing so well! Do you have any advice for my practice?" Hjalmar thought for a moment, then suggested, "Keep practicing." (Hjalmar was very proud of this crack, and retold it to me many times. Hjalmar was just starting to feel bitterness towards his good doctor of more than twenty years – he was giving the doctor honest, truthful input, and the doctor every time simply assumed that Hjalmar was joking.)[1]

Every time that Hjalmar reduced his medication, the doctor asked why. Sophia, Hjalmar's wife, always said that it was because Hjalmar was having hallucinations. It was Sophia, by the way, who first discovered the power of the hallucinations argument. We were to find later that no matter how well a patient was doing on lowered drugs, even if he was in better shape than he was at a higher dosage, most doctors, especially the ones educated in the 1970's and 1980's, would get a bit huffy if you told them the medications had been reduced. This was an implication that the doctor had prescribed the wrong amount, I suppose. Sophia first, and then others, found out that if you said the meds had to be reduced because the patient was having hallucinations, the doctor would always agree to the reduction.

It was as if the doctors didn't care about the dyskinesia, and they were inured to the stories of insomnia and agitation, altered personality, and the rest. They considered that to be a part of the worsening Parkinson's. The solution to all of those symptoms was to increase the medication. But hallucinations? Those were Bad. For some reason, all the neuros that we have heard about in this program in five years seem to agree: it is reasonable to reduce the medications in cases of hallucinations. No other reason is particularly valid. Hallucinations were the magic line of side effects over which the doctors would not cross. And so as Hjalmar continued reducing his medication, glimpses of his old self became more frequent, his agitation decreased, and his doctor continued to support him.

I must note, one MD in our experience, Zoe's doctor, didn't care about hallucinations. He felt that, in some cases, any decrease in L-dopa could precipitate fatal seizures, and that "you can still be alive with hallucinations, but not with seizures."

A word you maybe shouldn't use with your doctor

Iatrogenesis (ee a truh JE nuh sis): "the creation of illness and injury in patients as a result of medical treatment. From the Greek: iatro, *doctor* + genisis, *produced or generated by.*

[1] I sent Dr. Rafferty a form asking with regard to a list of specific symptoms whether Hjalmar was worse, the same, or improved, since 1998 (when Hjalmar started the project). The list included symptoms such as falling down (which he was no longer doing, and which he had previously done several times a day), volume of speaking voice (he had had no voice when we started, and the voice had returned), ability to straighten the fingers (his fingers had been unusable when we started), and so on. The MD had returned the form with "same" checked off in every box. Considering that Hjalmar had improved in every area (of course, we had purposely written up the form to play up his improvements), and had reduced his meds (at that time) to less than one third of what they had been, this response was very upsetting to both Hjalmar and Sophia. They felt betrayed. They had told the doctor that Hjalmar was doing well, and for two years the doctor had expressed amazement at his improvements. But this man refused to put it in writing. At any rate, the doctor has written that everything was the same. Considering that the disease is supposedly incurable, and that Hjalmar had reduced his medication by two thirds, I decided that even the statement that "everything was the same" was something of a victory. I wrote the doctor a thank-you note for his statement of support for our project. He did not answer.

> "There is a long list of iatrogenic diseases – those induced by physicians and by the drugs, vaccines, and other new therapies they use. Certain anemias have resulted as a complication of the surgical insertion of artificial heart valves, for example. Such diseases are essentially diseases of medical progress. They would not have occurred in the days when doctors could do little beyond prescribing worthless medications and holding the hands of patients as they died. Today many therapies actually work, but their undesired effects make them double-edged swords."[1]

Doctors in the dark

As promised in an earlier appendix, here is information from an article from the *Journal of the American Medical Association* (JAMA) that arrived recently (February 2003) from my neurology webline:

"Clinical Practice Lags Behind Medical Research"

The title says it all. But if you want details, the gist of this article from the Clinical Research Roundtable is that research findings don't always make the transition into practical application. Here are some excerpts:

"Despite the proliferation of medical research, much of this new information is not affecting clinical practice…(and) findings of clinical studies are slow to influence medical practice and healthcare policy. Factors contributing to these translational blocks include high costs, slow results, lack of funding, regulatory burdens, fragmented infrastructure, incompatible databases, and not enough qualified investigators or willing subjects… "

"In an accompanying editorial, Roger N. Rosenburg, MD, from the University of Texas Southwestern Medical Center in Dallas, suggests that one solution might be to establish a Department of Biomedical Research analogous to the Department of Homeland Security. 'A unified voice from the biomedical community must speak clearly and resolutely to emphasize that the CRR (Clinical Research Roundtable) report [on clinical practice not matching research] amounts to a national crisis,' he writes. "The American people need to know that the current system for bringing …research to the bedside is operating at an obsolete level of efficiency, causing great delay, and consequently resulting in the loss of many lives."

Later in the article, Dr. Sung, PhD, program officer of the Burroughs Wellcome Fund in Research Triangle Park, N. Carolina, responded to the questions posed by Medscape.

"Clinical studies…in many cases, are only sporadically applied to the patients who need them. Where strong evidence-based practice guidelines exist, they are frequently not followed by physicians and other practitioners. Comparative data on the effectiveness of old versus new technologies is often lacking…consequently, significant holes exist in the body of evidence from which clinicians [doctors and nurses who actually treat patients, as opposed to researchers] and consumers base health promotion and treatment decisions, and on which health plans and employers make coverage decisions...

---

[1] Greenman, Robert. *Words That Make a Difference*, Levenger Press, Delray Beach, Florida, 2002, p 181.

"Insurance benefits may limit coverage inadvertently by placing some new and potentially better…therapeutic interventions out of reach of ordinary consumers. Conversely, benefits may include coverage for therapies that are of questionable value or have even been proven ineffective. These problems will become more acute as the pace of medical and health sciences innovation quickens…"

"The clinical research enterprise presently contains inefficiencies at almost every step. We lack data on the numbers of adverse events or the denominator of how many are actually enrolled in clinical studies…Most medical records are kept manually. Clinical research staff are often overtaxed. Replicated entry of data on charts, insurance claim forms, clinical trial forms, and adverse event forms is the rule…

"Standardization (an electronic record keeping) in other industries has facilitated vast savings through process improvement, and information technology has been an integral part of this transformation. This same revolution has yet to happen in healthcare – indeed, the entire healthcare industry continues to invest significantly less in information technology than any other information-intensive industry."[1]

Although the person interviewed in the above article blames the problem on funding and lack of standardization in record keeping, a large part of the problem may actually be the human component: the psychological resistance of people – even doctors – to a change in their status quo.

There is no legal requirement that doctors be up-to-date, or that they modify their treatments from what they learned in school thirty years earlier. The continuing education requirements only require that doctors take classes, but not necessarily classes in clinical application in their own field. Current medical certification does not require that a doctor be regularly tested on his rapidly-changing area of expertise. But as demonstrated in the International Parkinson's conference, even doctors who do try to keep up often decide to ignore the new findings. This human tendency for inertia, more than any amount of funding, may be the reason that so few doctors, including "top authorities," are actually up to date in their field.

Finally, remember that a person gets to be a "top authority" by producing a smidgeon of new data early in his career. In hopes of being someday honored by his finding, his job is to defend his data and its conclusion against all comers during the following decades. If his data becomes outmoded or proved wrong, his findings will be sneered at and his status reduced. The current system only allows a person to become a "top authority" *if* his previous work remains unchallenged. If you are working with a top authority, you are most likely working with a person who is desperately trying to maintain the status quo and force all subsequent research to conform to his own previous contribution.

A little something for your doctor.

Here is an example of the articles that cross my desk regularly. The gist of this one is that mice were brain damaged via a chemical that kills dopamine-producing cells. They were then given levodopa. Over time, some of them developed dyskinesias. The ones with dyskinesias were seen to have damage to certain nerve synapses (nerve

[1] *JAMA*. 2003; 289:1278-1287. 1305-1306.

junctions). The drug that caused the cell death didn't cause the nerve synapse damage – the levodopa did. The conclusion is that levodopa, although helping to give movement to dopamine-deficient mice, also causes damage, and there is an association with the damage and dyskinesia. The damage was associated with dyskinesia. Dear reader, do not jump to a cause and effect conclusion! That would probably result in bad science. Whether damage or the dyskinesia came first is not known. Nor is it known if the mice had equal amounts of original brain damage, so that the levodopa was more or less excessive in some mice than in others.

However, a relationship has been seen between levodopa, brain nerve damage, and dyskinesia. This is merely an example of the sort of research that is available and which shows that doctors who attribute dyskinesia to advancing Parkinson's may be wrong. Your doctor may appreciate, or may not, knowing that there is new information on this subject. Here is an excerpt from the article.

> "L-DOPA Dyskinesia Linked to Reduced Plasticity of Corticostriatal Synapses"
> source: Reuters Health Information, April 4 (2003).

"Levodopa (L-DOPA)-induced dyskinesia is associated with reduced synaptic plasticity in the corticostriatal pathway in a rat model of Parkinson's disease. According to a report in *Nature Neuroscience*, published online March 31st, maintenance or restoration of synaptic plasticity represents a potential therapeutic target for these patients.

"Even though L-DOPA is considered the most effective therapy for Parkinson's disease, long-term treatment often results in the gradual development of dyskinesia. According to Dr. Paolo Calabresi, of the University of Rome, and associates, this complication may be caused by overactivity of the striatal output pathways [due to the levodopa].

"A lack of depotentiation of corticostriatal inputs may have profound pathophysiological consequences in Parkinson's disease patients treated with L-DOPA," the investigators conclude.

In basic English, L-dopa causes damage in the substantia nigra.

Educating your doctor

Whether or not you want to learn to read articles like the above and foist them off on your doctor, who, if he is normal, will not alter his prescribing patterns regardless of the new findings, is up to you. But I included the above just as an example of the research that is available that makes the point that levodopa causes brain damage. Enjoy.

Educating your patient

For the benefit of all the non-MD health care practitioners who might be reading this book, I will tell you what I did for my patients. I read out loud. I read to them from the *Physician's Drug Handbook*. I read them the lists of adverse effects from their drug inserts. I read to them out loud the correct dosages of the drugs, which often conflicted with their prescribed doses. I read to them the warnings about the drugs.

Sometimes I would read the list of side effects to patients several weeks in a row because each week they would fixate on the symptoms that matched their problems that particular week, and then the next week, they couldn't understand the new symptoms that

appeared. Sometimes my patients were so well informed that they told me how they were going to determine which of their drugs to decrease next, based on their changed side effects.

You cannot tell a patient what to do with his meds unless you are an MD. I am not an MD. But I'm pleased to say that my patients now know the published, legally available information about their medications better than their MDs. They needed to know this in order to take the correct amount of medications. I also shared with patients what we were finding with other cases. Every week, nearly every medicated patient would be told about what the other patients (unnamed) were noticing with their medication changes. And there was a lot to talk about.

Do remember to observe patient confidentiality. If you only have two Parkinson's patients, you may not be able to make suggestions based on your experience with your other patient, as the patients may know each other. You may wish to use the case studies in this book, instead, for your examples.

Never say that you will work with a patient on the condition that he first stops taking medication. To do so is to make prescriptive advice about the drugs; you are saying, in essence, that he should stop taking drugs – you have no right to say that. If you are willing to take my advice, you will simply not work with medicated patients, period. Do not enter into negotiations and do not make suggestions. You do not have the right to do so. If you are not an MD and you make suggestions about the drugs, you will be violating the law, and certainly, if you lose your license, you may not be working in the patient's best interest. It is not in the best interest of the world for compassionate health care professionals to lose their licenses.

While the refusal to treat a medicated PD patient may seem rather harsh on those people who are currently taking antiparkinson's medications, working with them may prove to be a false kindness.

Appendix 9

ANOTHER SINEMET CASE STUDY

These emails from Taylor Paul have been appended in this book because the most frequent request I get from patients is, "Give us more case studies! More individual examples!" Hopefully, with every new edition of this book, I will find time to type up a few more case studies. Maybe someday, an entire book of case studies relating to antiparkinson's drugs will be published.

Do bear in mind that every PDer is different, but that the general principles demonstrated by this case may be helpful.

Taylor Paul

These are some emails that I got from Taylor Paul when he was nearly off the medication the first time. (Taylor Paul is introduced in chapter 21 of the text.)

These notes describe his experience with the vacation – the fleeting feeling of peace that can occur immediately following a drug reduction. The first email states that he had just quit taking his meds altogether, as of a few days ago. He had been slowly reducing his medication (from a high of 450 mg/day) down to 150 mg/day of L-dopa. He had just decided that he was going to quit altogether, since 150 mg a day was practically nothing, and he had taken no medication for three days and was feeling great. I sent him some frightening case studies of people who had just stopped cold from 150 mg, and even 100 mg/day. He evidently decided to reduce his drugs more slowly after that. He went back up to 150 mg/day.

In the second email he had just reduced from 150 mg/day of L-dopa down to a reduction in which he took 100 mg/day on two days of the week, and took 150 mg/day on the other five days of the week. He was spacing the low-dose days a few days apart.

Here's his missive:

Hello:

I'm back on 150 mg per day. 50 mg at 7:30 AM; 50 mg at 11:30 AM and 50 mg at 5:00 PM. I was planning on cutting off the 7:30 AM morning dosage for a couple of days next week because I am definitely having meds dyskinesia after taking these doses (most of the time). I am not feeling great but OK or as normal as I can feel with this PD. I haven't felt "really good" since going back on the Sinemet.

Yesterday I went to see my doctor. I gave him an update on what I am doing. He is a great guy. Very attentive and compassionate but he loves drugs. He loves Sinemet as he has used it for years with his PD patients. I asked him...what about when Sinemet quits working and dyskinesia sets in? He said then it is "awful." I told him about my experiment of going off Sinemet for a couple of days. He asked me why I didn't just stay off the drug (cold turkey). He has never taken anyone off Sinemet because none of his patients have ever requested it. They have all been so "happy" with the drug. Sound familiar?

The only big change I have observed (other than my PD symptoms gradually worsening) is that I have a sleep interruption at 2:00 AM mostly every morning. I am usually awakened by shaking and neck pain and general weakness. When I first started FSR, I was able to sleep thru the night for a long time. Quite a blessed change. But a month after I dropped the dosage of Sinemet to 150 mg was when the sleep interruption started again. You mention in your book to try a sleeping pill or Benadryl to get by. (Author's note: I no longer make this recommendation. Too many patients became addicted to their sleeping pills.) *My Dr. suggested that I go on Amitriptyline. No thank you. I have used Tylenol. But I don't like using it often.*

Two weeks later

Hi:

I have for the last two weeks dropped two doses (total of 100 mg) of Sinemet per week (two days). The first week...no problem. The second week after the second reduction day (spaced two days away from the first day of withdrawal), I experienced a sleeping deprived night and uncontrollable shakes day and night. After resuming 150 mg per day (for the past two days) I am OK now. Wow!

Isn't it strange, I can go off the drug cold turkey for three days and feel wonderful...but two weeks later…

What do you attribute my right arm pain – shoulder, arm and hand too? I remember my dad complained about pain in his shoulders too. Anything I can do about it? [Now that the meds are so low] I am FEELING the numbness in my feet and legs now.

Love,

Taylor Paul

In review, two and a half weeks earlier he had gone from 150 mg/day to none, and stayed at that level for three days. He had felt great during that time. He then started taking 150 mg/day again. For the next two weeks, he took 150 mg of L-dopa on five days of the week, and only took 100 mg on two of the days of the week. After one of the days when he took only 100 mg, he had two days of powerful drug withdrawal symptoms. After that, he felt "normal" again.

And here was my answer to him:

"Dear Taylor Paul,

"It actually takes nearly ten days for the limbic part of your brain to realize what is going on at the head office. That's why there was a delay in noticing the three days when you went cold turkey. What you were probably feeling when the shaking set in that night was the result of taking no pills, for three days, almost two weeks earlier!

"Repeat: the uncontrolled shaking was probably coming from the cumulative effect of not taking any meds at all for three days, back when you quit cold turkey ten days ago. You are lucky; a night or two of shaking is not very bad, in terms of what drug withdrawal can potentially do. Sounds as if you got off easy. And you might not be done with the withdrawal. It will be some days before you really know if you are finished responding to your three day hiatus, and if you have adjusted to your new, lower weekly level already or not. The shaking response tells you that you were overmedicated, or at

least mildly addicted, at your previous dose level. If you had been mildly undermedicated at the time you made the further reduction, you would merely be even more undermedicated and moving slowly, looking more PDish. But if your brain is slightly addicted, so that when your reduce, your limbic area was exposed, showing a drug-induced deficit, that's when the brain will do the withdrawal tantrum – the shaking that you had – as an attempt to get its goodies back (the L-dopa pill) without having to grow the dopamine itself. Those three great days when you stopped your pills and had no problems at all, that was just a little vacation. Most people love the clear-headed feeling that they get when they stop the pills briefly, even though they don't move too well. But clear-headedness doesn't compensate for the hellish drug withdrawal symptoms, once they begin. That's why most people choose to go slowly instead of going cold turkey."

So that demonstrates the vacation. He felt just fine for nearly ten days. And then, boom!

Taylor Paul, the second time around

The following brief description of a drug reducer's dilemma is taken from Taylor Paul's second round of getting off Sinemet. As you may recall from chapter 21, his doctor had mistakenly put him on Sinemet during a hospital stay for what it turned out was not Parkinson's but a life-threatening bacterial infection.

Taylor Paul had come to a difficult point in the reduction and was uncertain whether he was getting better or worse, or needing to decrease his drugs or increase them again; he wanted advice. His adult children were supportive of his plan to reduce his medication, but his wife had passed away a few years earlier, so he lived alone and was very much alone in his drug decisions. He wanted an answer from me but, as you know, I cannot advise. I asked him to describe his situation in hopes that, while describing it, it might become obvious what he should do next.

He described himself thus: he was at the point where he could function slightly better every day. He was down to 100 mg levodopa/day from a brief, post-hospital high of 500 mg. He was driving his car again and had recently moved back into his own home after staying with his daughter during his weakest phase. However, every night at around 4:00 a.m., he would startle awake and proceed to shake violently until 6:00, when he got out of bed. He asked me if this meant that he had decreased his drugs too abruptly.

I asked the following questions: Was he once again able to initiate movement? Yes. How was his balance? It was improving daily. Was his stride lengthening? Yes. Was he able to resume his daily walks? Yes. Was he having dyskinesia? No, and the facial grimacing had ebbed. He had normal facial expression. Had he fallen recently? No. Were his arms swinging? Yes. Was there anything that he could do, short of taking medication, to reduce the shaking from 4 to 6 a.m.? Yes: he had tried taking Tylenol and that would put him right back to sleep, but, he asked, wasn't it bad to take Tylenol every day?

My answer was, "Yes, it can be hard on the liver to take Tylenol every day."

He mused for a bit. He then proposed that he try having some toast at 4:00 in the morning. He also suggested that a bit of wine would probably knock him right out, and he would sleep like a baby if he had a bit of wine at 4:00 in the morning.

I told him that if he could deal with his shakes by eating some toast or having a drink of wine, then those might be healthier options in the long run than using L-dopa to deal with his 4 to 6 a.m. shaking. Considering that in every other way, he was doing better and showing marked improvements in his symptoms, he might want to think about the possibility of overmedication rather than undermedication. (To me, though I was not able to give advice, his overly bright eyes and overquick smile seemed more of a concern than his shaking in the night. He seemed slightly stoned to me, but I couldn't mention it.)

He tried the toast; it worked. The old Ayurvedic saying, "Never use medicinal herbs when food will suffice," applies neatly in this case.

A month later he made a further decrease, taking only 50 mg two days a week and 100 mg on the other five days. The stoned look in his eyes faded away. He may always need a small amount of dopamine-enhancing medication due to the damage he has done to himself. In order that the needed pharmaceutical dopamine never exceed his Safety Limit, he may always need to be careful that he takes just enough that he never feels really good, but enough so that he doesn't fall into a Slough of Despond. He is determined to use spiritual inspiration to make up the dopamine difference between the sub-feeling-good drug amount and the way that he really wants to feel. I supportively include him, and all my patients, in my prayers.

Appendix 10

DRUG PAIN IN PARKINSON'S DISEASE

Because many doctors feel that all problems in a PDer, including pain, must be signs of advancing Parkinson's, I am including these excerpts from a paper read at a Parkinson's research conference in Coventry, England, June 19, 1998. In my own experience, I have seen that most pain in Parkinson's comes from dystonias. These dystonias can be the result of old injuries causing muscle flaccidity or tension, and they can also be the result of overmedication. In the latter case, it appears as if the brain employs muscles with decreased muscle-motor command communication to burn off some of the excess dopamine via frantic activity. The muscles may shake or they may spasm instead. If they go into spasm mode, the resultant pain can be excruciating and does not usually ebb until the medication wears off. If pain first appears an hour or so after taking a pill, whether or not the pill ever seems to be effective, it is a good guess that the pain is the result of overmedication. The apparent "failure" of the pill may be, in fact, a symptom of freezing from overmedication.

The following is from a paper by Kylash Bhatia, MD (Department of Clinical Neurology, Institute of Neurology, University College London).

"…Pain can be the presenting feature of PD but more commonly pain in PD patients may be associated with motor fluctuations induced by treatment….

"…Quinn and colleagues in 1986 classified PD pain into four main categories. The first category was patients in whom pain preceded the diagnosis of PD. The other 3 categories were related to motor fluctuations with regard levodopa treatment. The first category was *'off period pain without dystonia'* and included morning pain, beginning of dose pain and end of dose pain. The second category was *'pain associated with dystonic spasms'* which could again occur early morning, during the off-period, at the beginning of a dose or at the end of a dose. The third category was *'Peak-dose pain',* occurring when the patient was turned on."

"…However, others believe that pain symptoms which accompany PD may not be directly related to motor phenomena and cite the following reasons.

1. pain can often occur on the opposite side of the body to the main motor signs
2. sensory symptoms and pain can exist for many years before the usual motor symptoms of PD develop
3. there is often no relationship between pain and tremor or rigidity
4. while antiparkinson medication sometimes relieves motor and pain symptoms, this therapy does not always alleviate pain and <u>in some cases can increase sensory symptoms</u>.
5. "…in some individuals, levodopa dosage reduction may be required (rare cases where only levodopa withdrawal led to cessation of pain have been recorded)."

Appendix 11

GUESS WORK ABOUT BUILD UPS:

HOW RESEARCH STUMBLES ALONG

So that you can follow along with our thinking, and maybe do some of your own that will help you see your way clearly through all this, let me share what was going through our minds as we pondered the Build Up.

What exactly goes on with the Build Up? Just what mechanism(s?) does the brain use to initiate short-term (single dose-related) Offs when dopamine levels rise? Does it shut down vesicle doors? Does it inhibit transport molecules?

It doesn't really matter, but we remain curious. I think curiosity is an inherent human condition. So here is our thinking about the build up.

We figured there were two likely possibilities: an enzymatic response that affected dopamine metabolism, transport, or uptake, or else a short-term receptor shutdown. In lay terms, to use a deliveryman analogy, either the deliveryman wasn't making the delivery, or else the deliveryman was there with the goods but no one was answering the door. You don't really have to read the following Fun Ideas, but if you want to see the kinds of thinking we research types tumble around in our heads, here it is. If you'd rather walk the dog or wash the car, go right ahead.

Fun Idea #1

The first idea, in which the deliveryman is not able to deliver the package, requires a more complex mechanism than the second. To explain why the Build Up occurred in a 24-hour cycle, it was first necessary to propose a dopamine half-life in the brain of, let's say, twelve hours. This "educated guess" is based on the observation that some patterns repeated every twenty-four hours. In this case, the first dose would work fine, but as overall dopamine levels increased during the day, the dopamine-resistance mechanisms (decreasing the transport and other enzymes) or an emergency rigidity mechanism (dopamine override) would be initiated. But in which part of the brain would this be registered? The limbic area might be too slow to change; it might have to be a mid brain, motor area, or frontal lobe response. To explain what we saw, it would need to be a graduated response: the quantity of shut down mechanisms was tied to the quantity of the (relatively) excess dopamine. That would be the only way to explain that Build Ups could be either mild or severe, and that each subsequent dose might work slightly less well. The brain response would have to be graduated and not a threshold event,

Of course, maybe the symptom of freezing from overmedication could be caused by a threshold sort of action, an upper threshold above which movement is not allowed, just as there is a lower threshold below which movement cannot occur.[1] The upper

[1] The upper threshold idea is not without a corollary in everyday (non-drug related) life. We all recognize that with deep concentration (an act which increases energy flow through the middle of the brain as opposed to the sides, and which therefore might increase dopamine levels), the body does grow still and breathing becomes slow. Unlike the fretful monkey who is in constant motion and breathing fast, a person

threshold idea might apply in this case, as the build up of dopamine might be increasingly poking its nose above the upper threshold. Even though the brain might be trying to dismantle the incoming dopamine, each new dose would shove the total that much higher above the threshold, and the Offs from excess would increase in length. By the next morning, the dopamine levels would be low enough so that the first dose of the day wouldn't violate the upper threshold.

This thinking would require the dopamine break-up mechanism to be fairly quick, completing its job within 24 hours at the most. Considering that doses were very often taken in the evening, and yet a person was back down to the daily starting place again by morning, we might need to propose a 12-hour break-up mechanism.

The above is just musing, to show you the kind of thinking we were doing.

Fun Idea #2

The second idea, in which no one is home to receive the package, is based on the finding that the dopamine receptors in the brain do have a shut-off mechanism, as demonstrated in nicotine research: once a dopamine receptor has been used by a nicotine molecule, it can't be used again for a period of twelve hours. A short-term (twelve-hour) shut off would explain why the end-of-the-day drugs don't work as well as the beginning-of-the-day drugs.

Again, this is just a guess. We don't know exactly what is happening. These are just some thoughts that we've had. Let's leave something for the biochemists to detail out. But it's important for us to recognize that, whatever mechanism is being used, the brain does *not* go back down to zero after each dose wears off, but is in fact still responding, somehow, to the dopamine that has already come in that day. Either the receptors are shut down, a threshold has been crossed, or some unknown mechanism is

---

intent on a specific task can be very still for a long time. An extreme example of this is the whole-body rigidity that occurs during deep meditation when the breathing stops or very nearly stops. This rigidity is not at all like that of a soldier who is trying to stand still during roll call. The soldier is making an effort. The yogi, sage, or saint is utterly relaxed. His is a rigidity that occurs when the mind literally withdraws from the sensations of the body.

As the sensory nerves are turned off and the mind is completely centered (literally centered, in many cases, on focal points (chakras) in the medulla, midbrain, or forehead), the body becomes as if frozen. It is a rare sensation. It is not so much that the body cannot initiate movement, but that the body has no desire whatsoever to initiate movement.

It does feel, during such a time, as if the brain chemistry has been altered. The concomitant state of deep peace or bliss that accompanies such stillness is a noble relative of the spritz of bliss that is experienced by a drug user. The fleeting, false joy of the drug user is evidently seen as dangerous by the brain: it triggers an addiction response. The peace that wells up within a person of prayer or meditation appears to be a response to patient brain restructuring. This restructuring may make a permanent increase in dopamine levels. This lasting redirection of the brain towards peace, wisdom, and ever-new bliss by increasing divine attunement in the soul-radio might be attained via the vibrating molecule of dopamine. Unlike the deluge of dopamine that accompanies drug use, the dopamine increase in a person of prayer is gradual and exact. Obviously, in the latter case, the proposed Safety Limit is never violated, and may, in fact, through training, be raised to allow for beneficial, ever-higher dopamine levels.

The possible relationship between dopamine and spirit is fascinating and, as you have guessed already, beyond the scope of this book. But even the possibility of a relationship between dopamine and divine joy helps to explain both the allure of dopamine and the brain's cut-and-slash response to any dopamine excess. While the man of spirit may want to "die daily," into the breathless stillness of God communion, the animal limbic system wants no part of death!

responding to the accumulation of dopamine. This is why the later pills of the day can be so often unreliable or their side effects so violent.

Just by looking at the Build Up, we can't really tell which mechanism, if either, is more likely. But when we consider the evidence of the Daily Deficit, we lean more towards the idea that dopamine is accumulating over the course of the day. However, given the proven (for what that's worth) 12-hour reset button that has been seen in DA receptors in the nicotine experiment, and given the complexity of the brain, it's possible that both effects are occurring, if not three or even fifteen systems that we haven't even imagined. There may be both a build-up of dopamine over the course of the day, triggering reduction of dopamine-processing enzymes and closing of vesicles, and *also* a temporary shut down of receptors, plus dozens of other subtle brain-balancing acts. But what is important for us is this: neither one of these ideas has any relationship to the purported 1 to 3 hour half-life of the various medications in the bloodstream that are preached by the drug companies.

Of course, we have no idea what the actual mechanism is. Possibly the mechanism has to do with shutting the doors to all dopamine vesicles so that no more dopamine can be released. Or maybe transport enzymes are involved. We have no idea of the process, but we named this Off process that was occurring at highest dopamine altitudes the "Shut Off switch."

Our mental picture of the shut off switch is a circuit breaker that can be flipped in response to an upper threshold breach. This upper threshold is far above the Safety Limit. The Safety Limit merely registers that dopamine is present in any quantity above that which is needed. A violation of the Safety Limit begins an addiction process to moderate future dopamine levels. The addiction process occurs when the Safety Limit merely notices an unhealthy level of dopamine.

The Shut-Off switch is an abrupt, immediate, curtain-dropping shut down that freezes the body on the spot while it rallies all of its dopamine-reducing forces. Once the demolition crew has broken up and carted away enough dopamine to bring the levels back down to a merely addictive level, rather than a near-death-inducing one, the circuit breaker flips on again, and the On might resume. Sometimes the second On of a Roller Coaster might be followed by a loud Crash, but often, after enduring a Roller Coaster, the next pill of the day might work more predictably. This might be because the demolition crew overplayed its part and brought dopamine levels so drastically low that the next pill would play out its part completely within the On zone, rather than going up into the excessive, dyskinesia range.

What are your ideas on the subject? Isn't science fun?

Appendix 12

ALCOHOL

The following is a case story about a person who feared alcohol more than Mirapex. Although I have used levodopa examples for much of this book (because of the preponderance of levodopa in treating Parkinson's), please be aware that any dopamine-enhancing drug will eventually have the same sort of symptoms I'm describing throughout this book, including addictiveness and semipermanent brain changes. So, in this discussion of alcohol, permit me to stray from the levodopa theme and share with you a story about Woody.

Woody

Many patients have chosen to use alcohol to ease symptoms of drug reduction, especially the insomnia. None of them have had any difficulty quitting the alcohol when they found the pain and rigidity easing up. Woody refused to drink alcohol because he had been a heavy drinker decades earlier, long before he had ever heard of Parkinson's.

He was taking Mirapex when he started our program and had gotten off it with ease. He had not been addicted. He had been completely off Mirapex for over half a year when he started having the deep fatigue in the mornings. He panicked and started taking Mirapex again at an extremely low level of .125 mg per day, a mere 1/24 of the suggested therapeutic dose. He was certain that he would be able to quit Mirapex a second time as easily as he had quit the first time; he pointed with pride to his years of sobriety and leadership in the local branch of Alcoholics Anonymous. We suggested that people who have started having recovery symptoms are extremely prone to addiction. He laughed us off. The Mirapex did not help with the fatigue in the mornings, but the rest of the day he felt wonderful, better than he had in a long time.

He continued to point to the fact that he had quit Mirapex easily prior to his Tui Na treatments. To prove to his wife that he could quit anytime, he stopped taking Mirapex. After a week with no medication, he started to feel vaguely uneasy and resumed the Mirapex. After this episode, he told his wife he had quit Mirapex twice, which, he felt, proved that this drug could have no power over him.

He increased his Mirapex again a month later, up to 1/16 of the therapeutic dose. This increase occurred just after his wife told him he was starting to act "funny, snotty and know-it-all, just like when you used to drink." When we asked why he had increased the Mirapex, he said he felt like it, and that he could do whatever he wanted. He retorted that he was fine, had never felt better in his life, and was not subject to addiction. He insisted that he had easily stopped using alcohol because of his twelve step program, and that, when he chose to, he could just as easily stop taking Parkinson's drugs. He refused to believe that his recovery symptoms were a warning that he would soon be highly addictable.

After a month during which he told his wife he could quit the Mirapex anytime, he quit taking the Mirapex to prove to her that he could take the drugs or leave

them. He stayed off the drugs for nearly a week, but when the withdrawal symptoms started, he resumed his medication at a still higher level than before, .75 mg/day. Within several weeks at this very low dose, a mere one third of that required for therapeutic effect, he began having dyskinesia for the first time in his life. He was visibly overmedicated. He announced at that point that the dyskinesia wasn't a problem since he obviously could quit Mirapex anytime. He was stronger than the drugs. He insisted that he had, in fact, succeeded in quitting three times already. No amount of logic could show him that, in fact, he had never quit – he had only been able to stay off the drugs until the withdrawal symptoms started, and then he had quickly caved in. He told us that we didn't understand.

His wife even suggested that, despite his previous use of alcohol, he might consider alcohol rather than anti-PD drugs if he felt he needed something. She felt the effects of the drugs, especially his dyskinesia and the strange look in his eye, were bizarre and much worse than anything she'd seen even when he was drinking heavily. He refused, dropped out of our program, and no longer goes to AA, where he had been a highly respected counselor for over a decade. He has now increased his drugs again, is constantly euphoric, is wracked with dyskinesia, and will tell anyone who will listen that the drugs are mild, and not to be feared: "Alcohol is dangerous, but not the PD drugs." His wife is planning to leave him soon, and he appears unaffected by this. He is a licensed family counselor.

I have run into the fear of alcohol many times, and I find it puzzling. People who have been taking mind-altering drugs that cause permanent damage and accelerate their symptoms by adding drug-induced parkinsonism to their idiopathic Parkinson's can get cold feet about having a withdrawal-easing glass of beer or a hot rum at bedtime, for fear of becoming accustomed to the alcohol. Medicinal alcohol does have a place in the roster of mild treatments that can alleviate pain in the short term. As long as care is taken when alcohol, a very mild drug, is used in the withdrawal from the heavy artillery of anti-PD drugs, it may be safe and appropriate to use alcohol in moderation.

It is also noted in the antiparkinson's drug inserts that these drugs should never be combined with alcohol. If you read carefully in the insert, what it says is that alcohol contributes to the effect of the drug, enhancing the properties of the drug and making it stronger. This is because these drugs work very much like a form of super-alcohol. When you add more alcohol to this mix, you are merely adding a bit more fuel to a fire.

However, when a person is reducing medication and hovering near pain or the insanity of drug withdrawal, it might be well to ease the suffering a bit and modify the withdrawal. If adding a spot of alcohol to the mix once or twice can make slight, quick modifications to the otherwise dangerously deficient drug level, possibly avoiding the downward spiral into withdrawal, this might be a very good thing.

Some patients have even figured that a glass of beer feels comparable to a dose of about 5 mg of levodopa, but more heavily loaded towards the relaxation area than the motor area. At any rate, none of the patients who used alcohol to help with sleep during drug reduction became addicted to alcohol.

Woody made it a point of pride to refuse alcohol, and he has been lost in a haze of drug-induced confusion and euphoria for over a year now.

Addendum October 2005

Notice: the Parkinson's Recovery Project's email support and practitioner training programs will *not* provide support or training for people who have ever used antiparkinson's drugs for more than three weeks. The Parkinson's Recovery Project's research arm, the PD Team of Santa Cruz, will not accept into its recovery program people who have ever used antiparkison's drugs for more than three weeks.

We no longer recommend a recovery program for any person who has ever used antiparkinson's drugs for more than three weeks. Even if such a person has stopped taking the medications, he is not a good candidate for our recovery program. This announcement, being added to our website and amended to our writing about Parkinson's medications, is an update in our ongoing research with Parkinson's disease.

History of our drug findings

By the year 2002, we had realized that no person who was using antiparkinson's medications should enter into a recovery program. Since 2002, we no longer accept medicated patients into our program. To explain our position, many case studies were included in a book, *Medications of Parkinson's, or Once Upon A Pill: Patient experiences with dopamine enhancing drugs and supplements.* This book, released in late 2003, is available for free at our website. The case studies in this book detail the disastrous results that occurred when medicated patients began to recover.

Ongoing research

By the fall of 2004, we were beginning to recognize a different problem: drug-induced parkinsonism in people who had ever taken any antiparkinson's medications, even in small amounts. Those PDers who had ever taken any antiparkinson's medication for longer than three weeks ultimately did not follow the same recovery path as people who had never taken dopamine-enhancing drugs – *even if they stopped taking the drugs prior to entering the program.*

The difficulties of these people were not nearly as dramatic or as deadly as the problems encountered by people who recovered while still taking medication. However, these people have not, in our experience, been able to permanently recover from their symptoms of tremor and difficulty initiating movement, even if, through the treatments we recommend, they temporarily recover from body-wide symptoms and permanently recover from all of their localized Parkinson's symptoms. (For a description of localized, Stomach channel symptoms, as opposed to body-wide symptoms, please see chapter 8 in *Recovery from Parkinson's Disease: A Practitioner's Handbook*, available for free download at <www.pdrecovery.org>.)

It now is becoming apparent that people who have ever taken dopamine-enhancing drugs evidently have semipermanent brain damage that corresponds to the adverse-effects warnings provided for these drugs by the drug manufacturers: tardive tremors, tardive dyskinesias, tardive body-wide slowed movement, tardive body-wide difficulty in initiating motor function – all symptoms of drug-induced parkinsonism.

Our program very effectively treats idiopathic Parkinson's disease, a disorder in which brain cells fail to produce or release dopamine, and in which dopamine-producing

brain cells revert to a dormant (non-dopamine producing) format. We have *no* effective treatment for the drug-induced cell death that causes parkinsonism, a disorder that outwardly resembles some of the symptoms of idiopathic Parkinson's disease.

The problem

People who have used dopamine-enhancing drugs for more than three weeks have the following problem if they enter our program: in the beginning, they undergo some distinct, pleasant or sometimes unpleasant symptoms of recovery, and many of their Parkinson's symptoms disappear. This is consistent with recovery from Parkinson's. However, at some point in the process, they find that, once their adrenaline is turned off, they are unable to focus their minds on either movement initiation or the mental processes required for full recovery. Their behaviors suggest that the brain centers that regulate mental focus and will power from the dopamine mode (as opposed to regulation via the adrenaline mode that has been in use during most of the PDer's life) have been damaged by the drugs.

Therefore, even after many Parkinson's symptoms are gone, the PDers who did use antiparkinson's drugs, *even for a short while*, find themselves, within a few months or a few years, in a new type of very unsatisfactory condition. To ameliorate this problem, they or their loved ones often hope that the medication, at very low dose, might solve the remaining difficulties.

However, because during recovery, even partial recovery, their bodies have switched from a state of pure adrenaline dependency to a healthier state of dopamine reliance, drugs that were previously appropriate or even seemed to "do nothing" become highly dangerous. Those PDers who have tried to resume even small amounts of medication to address the lingering, somewhat milder problems of drug-induced parkinsonism have had horrible problems: death, insanity, and, in two cases, violent thrashing that requires the drug user to be permanently strapped to a bed. This issue, the problem of using antiparkinson's drugs after the adrenaline system has been turned off and recovery has started, was addressed in the medications book. What was not addressed in the first edition of that book was the problem of drug-induced parkinsonism in people who stopped taking the medication or used the medication only briefly (more than three weeks). We have learned that this problem is a significant one.

Questions that arise

Some issues that were not addressed in that book are these: what happens if these partially-recovered people, whose recovery is stalled because of brain damage from drugs, refuse to take any antiparkinson's medications? What will their condition be like if, after their idiopathic symptoms are gone, they just "suffer" along with those symptoms that have come about from drug use? Will they be better off than if they'd never been in the program? Is a person better off staying on the medication or getting off the drugs, recovering, and then dealing with the milder symptoms of drug-induced parkinsonism?

These are very good questions. Because every case in our experience has been unique, we cannot answer these questions with specifics. A medical statistician who visited our program with the hope of creating a model for prediction left us saying "Each person's situation is so different. Until you have treated at least a thousand patients, it will be impossible to generate any meaningful data."

Also, it is important for the reader to understand that the motor, mental, and emotional problems of drug-induced parkinsonism are not static. A person with these

problems will continue to decline over time. Many of the parkinson-like symptoms that can be caused by dopamine-enhancing drugs are tardive. ("Tardive" means that the adverse effects set in motion by these drugs may not appear immediately.) The list of dopamine-enhancing drugs includes antianxiety and antidepressant drugs (SSRIs and tricyclics), most anti- ADD/ADHD drugs, as well as antiparkinson's drugs. Tardive adverse effects from these drugs may not appear until years after a person has *stopped* taking the drugs. It appears as if the brain changes set in motion by these drugs continue to build upon themselves; as a consequence of drug use (of dopamine-enhancing drugs), the brain moves inexorably, relentlessly, towards parkinsonism even after the drugs have been stopped. That march towards ever-increasing immobility and ever-increasing tremor may very likely continue for the rest of one's life. The drug warnings for nearly all dopamine-enhancing drugs note the risk of developing tardive tremors, tardive dyskinesias, tardive slowness of movement and the other tardive symptoms that constitute drug-induced parkinsonism.

Historical precedent

The earliest research on this subject, written up by Oliver Sacks in his book *Awakenings*, shared the unexpected results of administering, for just a "short" period of several months, dopamine-enhancing drugs to people with sleeping sickness, people whose physical immobility had not changed significantly in decades. After it was determined that the drugs were causing these people more harm than good, the drugs were stopped. The truly unexpected part of the experiment was the after-effects: those people who had used the powerful dopamine-enhancing drugs for a relatively short time were no longer in a static condition of immobility. Their post-drug condition, for most of them, was much worse than their pre-drug condition. Also, their condition *continued* worsening, even after all the withdrawal effects from the drugs were finished. In other words, short-term use of dopamine-enhancing drugs had set in motion a new brain pattern than caused dynamic, steady worsening of their condition even after the drugs were no longer in use. This is a pattern that we are beginning to recognize in people who used antiparkinson's medications, stopped using them, and got into our recovery program.

A person who has ever used dopamine-enhancing antiparkinson's drugs may well have created a drug-induced condition in his brain that will, at some point, render him depressed, immobile, and tremoring. If he has recovered, meanwhile, from idiopathic Parkinson's, he will not be able to use antiparkinson's medications to treat these symptoms of drug-induced parkinsonism. I repeat: it will not be safe for him to use antiparkinson's medications ever again, if his idiopathic Parkinson's is gone. As our book on medication demonstrates through hellish case studies, a person with the above symptoms who no longer has the adrenaline-dominance of idiopathic Parkinson's cannot tolerate the antiparkinson's medications at any level.

Therefore, we now recommend the following*:*

People who have ever used any dopamine-enhancing antiparkinson's medications for longer than three weeks should ***not*** *enter into a recovery program.*

Proceeding with caution

We have been cautious about jumping the gun with this gloomy recommendation, However, our ongoing research has slowly convinced us this is the correct stance to take. In fall of 2004, we started sharing the above recommendation with new PD correspondents

who wrote to us about their plans to stop taking medication so that they could start being treated for idiopathic Parkinson's. We were still hoping that possibly our findings were wrong, but we were seeing a definite negative trend in those previously medicated patients who had quit the medication and thought they were on the road to recovery, only to find themselves stymied. Those who tried to revert back to medication could no longer tolerate the medication.

By early 2005, we were more certain than ever that the problematic symptoms we were seeing in previously medicated PDers who had gotten off their medication prior to starting the recovery program were not coincidence. We decided that we should no longer offer hope for recovery to those who had ever used antiparkinson's medications for more than a few weeks. We decided that we could no longer, in good conscience, provide recovery-oriented treatments for these people.

We started communicating this new recommendation by emailing long, detailed warnings about our change in policy to any new drug-taking correspondents who contacted us to say that they hoped to seek treatment as soon as they were drug-free. We found that they, almost universally, disregarded our warnings and started reducing their drugs in anticipation of starting a treatment program. When they wrote to us again, saying that they were completely off their drugs and now wanted advice or support because of unexpected difficulties, or because they wanted to come to Santa Cruz for treatment, we repeated our statement that we could not support a recovery program for people who had ever taken antiparkinson's medications for more than three weeks: we would not treat them. They were often shocked, or even outraged by our regretful but increasingly adamant adherence to our new position about the unsuitability of trying to recover after having ever used antiparkinson's drugs.

A very common response has been, "But I assumed that your statement about not recommending this program for people who ever had taken the drugs for more than three weeks must have just been a legally protective stance. Surely you did not actually mean what you said!"

Now, in late 2005, our finding that people who have ever taken antiparkinson's drugs for more than three weeks are not good candidates for recovery is being posted on our website and appended to the medication book using firm, unambiguous language and an illustrative case study.

Responses to our new findings

As soon as the PD Team of Santa Cruz (www.pdtreatment.com) started enforcing our new position, refusing to advise or provide therapeutic treatments for new inquirers who have ever taken antiparkinson's drugs for more than three weeks, we began receiving emails, ranging from pleading to furious, telling us that we must change our "drug policy." The typical email says, "My father's medications no longer work / have horrible side effects; his condition is destroying the quality of my mother's life. Your program is our only hope. Therefore, please tell us that my father can recover, at least partially, if he gets off his medications and enters your program."

Others say, more or less, "Who do you think you are, to condemn me to Parkinson's and a lifetime of drug hell just because I took medication for a few months! How dare you tell me not to try and get off the drugs! You must make an exception for my case, and tell me that there is hope. I have the right to be treated by you. The very least you

should do for me is train my health practitioner so that he can treat me even if you will not."

These painful emails are heart wrenching, and yet they do not alter the fact that, in our limited experience, people who have used antiparkinson's drugs for more than three weeks have ultimately not fared well in our program. Even if these people did attain some benefits from our therapies, the long-term result could not be considered "recovery from Parkinson's."

Our job

I must make it perfectly clear that it is not our place to "give permission" for someone to take *or* stop taking medication. A person who feels strongly that he wants to make a change in his medication must consult his doctor and his own "small, still voice within." If such a one feels certain that a particular course is right for him, though it flies in the face of western convention or the limited experience of the Parkinson's Recovery Project, then that person must follow his convictions and the callings of his heart. If a person who, on his own, stops taking medication and subsequently recovers from Parkinson's, we keenly desire to learn about it. But at this point in time, we will no longer participate in such an experiment.

We are working in a research project: our role in this project is to try new approaches and report what we are finding. Our job is not, and has never been, to destroy hope. However, we certainly will not personally offer training or emotional support for a medical situation that, in our experience, has thus far had worse-than-unsuccessful results.

What do I mean by "worse-than unsuccessful?" I will try to explain. Certainly, a person who stops taking his medication and goes through the recovery-oriented treatments will no longer be in the same condition as a person who never takes these steps. But even if this person is able to reverse the processes that set the idiopathic Parkinson's in motion, his end result may not resemble "recovery from Parkinson's disease." A person who stops taking medications and goes through the process to recover from the idiopathic Parkinson's *may* end up with a slightly higher quality of life, despite his slowly burgeoning problems stemming from his drug-induced parkinsonism, than a person who does not go through the program. *But he possibly may not end this way*. The following case study may help clarify my point.

An illustrative case study

I will give an example by using the case study of Hjalmar. The first twenty years of Hjalmar's experience with Parkinson's, his subsequent recovery from idiopathic Parkinson's, and his nightmares in drug withdrawal are described in the book, *Medications of Parkinson's Disease or Once Upon A Pill*. In the three years that have passed since late 2002, when I wrote up that case study, I have been in infrequent touch with Hjalmar. He has been in a nursing home since his wife broke her hip in 2004.

Hjalmar cannot move. He cannot chew food or talk. He is taking no medication: he can no longer tolerate even an "ineffective," subminimal drug dose of 50 mg/day of L-dopa. He becomes completely dyskinetic and crazed if he takes even this tiny amount.

Here is the good news: his tremor, one of his first symptoms of Parkinson's, is now extremely mild and only slightly effects one hand. When I most recently saw him, he was sitting, motionless, in his wheelchair at the nursing home. His legs were stretched out in front of him in the wheelchair's leg support. His feet flopped gently out towards the outer

side of the body. Note: a person with unmedicated, advanced idiopathic Parkinson's would have rigidity in the legs and a tendency for the legs to be pulled inward, with the feet hitting or crossing over each other. Also, the severe tensions in certain leg muscles in unmedicated Parkinson's tend to cause flexing of the legs at the hips and knees, bringing the legs into a more fetal position.

When I first met Hjalmar, his legs could not relax. Sitting in the wheelchair at the nursing home, his legs were much straighter than I'd ever seen them, and they were no longer steely hard – they were no longer characteristic of idiopathic Parkinson's disease.

Sitting in his chair in the nursing home, Hjalmar's arms were somewhat relaxed; they were hanging easily from the shoulder and bent gently at the elbow so that his hands could rest in his lap. Note: a person with advancing idiopathic Parkinson's disease will typically hold his arms rigidly bent at the elbows. The pain and tension in the arms can be excruciating. When I first met Hjalmar, his arms were perpetually bent at the elbow and the arm muscles were like lifeless steel, unable to relax. Now, they are more relaxed and comfortable – uncharacteristic for idiopathic Parkinson's disease.

Hjalmar's hands, like the hands of many people with advanced Parkinson's, had been knotted and painfully dystonic. At the time of our very first meeting, his useless, rigid hands had been bent so hideously to the side that his smallest (fifth) finger was nearly touching the side of his wrist. When I recently saw him in the nursing home, his hands were resting peacefully in his lap, with only a small distortion at the wrist as a reminder of his previous deformity.

Sitting in the wheel chair at the nursing home, his head was reclined, resting on a pillow. When I had first met Hjalmar, in 1998, the rigor mortis-like rigidity of his neck muscles kept his head pulled forward so far that his lower jaw and chin rested on his sternum. His neck was always in pain, as were the muscles behind his shoulder blades.

During my recent 2005 visit at the nursing home, I saw that Hjalmar's neck was somewhat relaxed, and his chin was up in the air where it should be. His head was tipping to one side so I straightened it for him. As I did so, I could tell that his neck was still somewhat stiff and hard to move, but it was nowhere near as rigid as it had been during his years with Parkinson's disease, and it no longer pulls forward and down.

After I'd adjusted his head, he tried to communicate with me; he made some guttural noises without moving his lips. Hjalmar and I had always had a fun-filled relationship. So I joked, "You realize, of course, that I have no idea what you just said, but since you have always been so courteous, I shall assume that you just said, 'Thank you, my angel.' If you were actually trying to curse me, too bad for you, for I shall never acknowledge it!"

In response to my tease, the corners of his eyes crinkled up and his gaping mouth moved open a bit wider and turned up at the corners to form a soundless smile. When I had met Hjalmar, he had been unable to smile, even with his antiparkinson's medications, for nearly twenty years. The return of his smile had been one of his first recovery symptoms.

In the nursing home, he clearly could not initiate any movement, even though his body was now relaxed and did not hold the agonizing extreme tightnesses and tensions that are characteristic of advancing idiopathic Parkinson's disease – tensions that Hjalmar had had in full measure, despite the benefits of his medications, when I first started working with him.

He appeared to have no localized symptoms of Parkinson's disease (symptoms that occur in Parkinson's-specific muscle groups) and the adrenaline-induced intensity of his

personality had been replaced by a peaceful acceptance: I chatted with/at him for half an hour. Though the conversation was mostly one-sided, at one point, when I was monologue-ing my sympathy for him being assigned a shared (two-person) room, I was able to understand enough of his gargling to detect the phrase, "It's OK; I'm used to it."

His attempts at whispered, throat-compressed speech had been almost impossible to understand when I first met him. During his recovery, he was able, for a while, to speak clearly and loudly. Now, with his previous adrenaline-induced tensions and intensity gone, but with damage from his past years of medication-use creating a classic drug-induced parkinsonism situation, his throat was somewhat more relaxed but he could barely initiate enough movement to activate his tongue.

Hjalmar no longer had many of his previous characteristic symptoms of advanced idiopathic Parkinson's disease: he was no longer restless, shaking or tense, and his limbs and head were no longer painfully retracted into the characteristic shape of a person with advanced Parkinson's. Unlike most PDers who are taking the anti-PD meds, his mind is clear. (His wife can understand him better than I can, and she feels that he understands everything she says and takes a compassionate interest in news from home.)

However, aside from some feeble eyelid movement, some weak tongue squirming and a tiny bit of movement in his lips, he cannot move. As for giving him antiparkinson's medications to assist with his movement, that is out of the question. We have already seen, dramatically, that even minute doses of the medications which, in the past, had imparted mobility, now cause him to writhe helplessly and hallucinate (see: *Medications of Parkinson's or Once Upon A Pill* / Hjalmar).

But I must be honest; Hjalmar did make one movement that, I think, surprised both of us. As I was taking my leave, I brought my palms together and bowed my head to Hjalmar as I had always done, touching my joined hands to my forehand. Hjalmar brought his hands together in reply and, with unexpected control, lifted his joined hands halfway to his forehead and held them there for a few seconds before his arms dropped limply again to his side. As I gaped in astonishment, I saw that his eyes were twinkling especially brightly as they gazed deep into my own.

In retrospect

Whether Hjalmar might have been better off had he stayed on drugs and not entered our program, we cannot say.

When he entered our program, he was in pain from the rigidities of Parkinson's when his meds were "Off." He had not been truly communicative in a deeply thoughtful way in over a decade. His On/Offs from his drugs had become increasingly unpredictable. When he was Off, he was utterly immobile – "stiff as a board," as he used to say. When he was On, he had been squirmy with dyskinesia and sometimes had difficulty feeding or dressing himself. Then again, sometimes, while On, he *could* walk or use the bathroom by himself. At all times, whether On or Off, he had been agitated and anxious. He had no time during the day when he could be considered "healthy" or capable of sustained normal function in activities of daily living.

When we first met him, the slope along which he had been sliding was increasingly slippery. He was heading for utter immobility and painful tension. But is his current condition significantly better? It may very well be that he is more relaxed and comfortable now than he would have been had he not been in our program. However, it is also clear that the phrase "recovery from Parkinson's" cannot be used in Hjalmar's case.

Of course, many of the other medicated PDers in our project had not had Parkinson's for as long as Hjalmar, nor had they taken the drugs for as many years. Even so, the long-term picture that is emerging for people who ever took antiparkinson's medication is grim. Even those who had stopped taking medication prior to starting our program do not appear to be capable of recovering in the same way that an unmedicated person is able to recover.

Therefore, to speak of "recovery" in these cases is misleading.

Another "long-term" case study – a case of a PDer who entered our program in 2000 – features a relatively younger person who had "only" taken Eldepryl (Selegeline) for "only" one year before stopping the drugs ***prior to*** beginning our program. His preliminary improvements and unfortunate conclusion are written up in an extensive footnote in chapter 21 of the book *Recovery from Parkinson's Disease*.

Therefore, our new statement for people who have ever taken antiparkinson's medication for more than three weeks is this: they are not good candidates for our program. We will not treat or advise people who ever took drugs for more than three weeks.

Exceptions to the rule

People who have used the medications invariably assume that their own case will be exceptional: they will fully recovery even if they have used medications. Our own findings are that, in our very limited experience, these assumptions have been proven wrong. No one yet, in our very limited experience, has been an exception.

We will not nurse false hopes, and we certainly must conform to the ideal of "do no harm." We cannot know for which people the damages of drug-induced parkinsonism will be more dire than the problems of idiopathic parkinson's, or for whom the reverse might apply. Certainly, it has become apparent to us that anyone who has used the medication for more than a few weeks will have sustained enough brain damage to set in motion, sooner or later, the symptoms of drug-induced parkinsonism. Therefore, we will not work with people who have ever used antiparkinson's medications for more than three weeks.

Hope

We highly suggest that any person who is taking antiparkinson's medications read the book, *Medications of Parkinson's Disease, or Once Upon A Pill*. This book includes much information on how the drugs work, how patients have learned to dose themselves in such a manner that drug damage is minimized, and the doses used by patients who had gotten the greatest number of years of good movement while taking the drugs. This book does *not* make prescriptive suggestions for readers. This book provides information that a PDer might use in figuring out for himself the medication course that he wishes to travel.

Historically, these meds retained their effectiveness for up to ten years before the drug-induced problems started and the effectiveness of the drugs waned. Now, as doctors have become more accustomed to these mind-altering drugs, they have become more cavalier about prescribing them. Uninformed doctors, including some neurologists and Parkinson's specialists, usually, right from the start of a person's diagnosis, now prescribe the meds at much higher dosages than were used in the 1970s and 1980s. Therefore, most people now have only two to five years, not ten, before the meds cease to be effective and become more of a problem than the Parkinson's itself.

Our findings suggest that in the rare cases when medications are used judiciously, the effective period for the medications might be decades – a period long enough to

provide for a full and meaningful life for most PDers. Most MDs do NOT prescribe the drugs according to the manufacturers warnings. Also, current use of the meds often includes a drug increase when adverse effects appear, when what may be needed is a drug decrease. *Once Upon A Pill* includes information about what constitutes correct and/or safe dosages.

Our suggestions

What suggestions do we make about the drugs? We, the Parkinson's Recovery Project, do not give advice. We will not say that any given individual should start or stop taking medications. We are not prescribing physicians and we cannot, will not, give prescriptive advice. Nor do we think that anyone, even an MD, should give prescriptive advise to a person with whom he has not worked closely.

However, the new understandings about how these drugs actually work in the brain, the time frames of effectiveness and the case studies of people who have successfully reduced or stopped taking their medication – all provided in the book *Medications of Parkinsons or Once Upon A Pill* – have been very helpful for people who are struggling to make changes in their over-amped prescriptions so that they can get the best long-term use of their medications.

We feel strongly that people need to find their own way through such important life choices. We also wish to note that people taking antiparkinson's medications are not very good judges when it comes to making choices. The medications work by altering brain function to create a false sense of well-being, wisdom, and even omniscience. It is almost impossible for a medicated person to clearly assess his own situation. Therefore, although people do need to find their own way through the various options regarding Parkinson's disease, people who are taking medication might be well advised to ask trusted friends or family members to help them make their decisions.

However, if a PDer who has ever used antiparkinson's medications for longer than three weeks decides to enter into a Parkinson's recovery program that is based on our findings, we cannot support this person's decision. We will *not* accept such a person into our own treatment program. If he chooses to attempt recovery via working with someone else, we will *not* offer any advice for such a person or his practitioner. To accept someone into our program or offer advice to a person who is pursuing such a path – recovery from Parkinson's disease after having used antiparkinson's medications for more than three weeks – would be to imply that we are hopeful about the results. We are not, and therefore, we will not be a party to these heartbreaking cases.

As for whether or not it is reasonable for a PDer using meds to go through the hell of cautious drug withdrawal and the physical and emotional rigors of recovery symptoms in the hopes that he will somehow, miraculously, not have drug-induced parkinsonism when he is done, we will not say. We will say that, if such a one does go through this process, experiences even a few symptoms of recovery, and then finds that his drug-induced parkinsonism symptoms are intolerable, he will NOT have the option of returning to antiparkinson's medications; if he has begun to recover, the medications will have become far too strong for him. (The case study of Rudyard (in this book) is an example of this type of event.)

The meds book is full of case studies showing what happens to people who use the medications in even the smallest amounts after having recovered from idiopathic PD. And as for some of the cases in the meds book that looked as if they might have happy outcomes, I must sadly report that, as the last three years have gone by, these outcomes have slowly turned sour or been abruptly disastrous. I will also say that our position is not completely unexpected; on pages 382 and 383 in the meds book, I wrote that, due to the inevitability of brain damage from the drugs or the risk of life-threatening misunderstandings with MDs or hospital personnel, we recommended, even at that time, that a person already using medication should not consider entering a recovery program.

At the time that the book was released, we realized that most people who had ever used the meds could not have a satisfactory recovery, but we were nurturing the hope that it was still possible that some, a few, might: a few people did, at that time, seem to be bucking the odds. We did not wish to dash hopes if there were any hopes to be had. Therefore, although the risks for a person who had *ever* taken medication were presented in that book, I did not dwell on them. With heavy hearts, we are now changing our position from one of ambiguity to one of regretful conviction.

Recovery from Parkinson's

On the other hand, those people in our program who were never medicated and who have recovered from idiopathic Parkinson's disease have not only retained their positive changes, but are continuing to note subtle increases in vigor and flexibility through the years. Our earliest cases of recovery for PDers who never took any medication whatsoever date back to 1998, and those people are still thriving.

Conclusion

We cannot, at this time, point with deep, long-term satisfaction at any case in which a person had used antiparkinson's medications for more than three weeks prior to entering our program, whether or not that person stopped using drugs prior to starting the program. We are deeply sorry to come to this conclusion.

We know that millions of people are currently taking antiparkinson's medications. We hope, over the next few decades, to learn more about the opportunities that exist for people who have already started down the medication road. We know that the problems of drug-induced parkinsonism will soon be a major problem in this country: antianxiety and antidepression drugs, anti-ADD/ADHD drugs, as well as the antiparkinson's drugs, all can cause the cell changes and cell death associated with drug-induced parkinsonism. With the increasingly rampant use of these legal drugs, we anticipate that the problem of drug-induced parkinsonism will, within a few decades, be in the spotlight. (Of course, since most MDs cannot or do not bother to differentiate between idiopathic Parkinson's disease and drug-induced parkinsonism, it is likely that the increase in parkinsonism from legal drug use will be wrongly ascribed to "unknown environmental changes that are causing more Parkinson's disease.")

We hope that recognition of and a solution to the problem of drug-induced parkinsonism will appear prior to that time. If it does, that solution will also provide hope for people with idiopathic Parkinson's disease who have taken antiparkinson's medications for more than three weeks.

But until such a time, those people are not good candidates for our program; we will not treat them, provide individualized advice, or offer false hope.

INDEX

A

A Primer of Drug Action 491
abdominal pain
  Comtan.....518
  Eldepryl 522
  Nat w/ Requip/case study.....306
  Permax 497, 498
  Sinemet 527
abdominal spasm 206
abnormal thinking
  Mirtazapine.....524
accommodation
  Amantadine.....511
  definition.....126
accumulation 33, 334
acetaminophen.....104
acetylcholine
  imbalance theory 564
addiction5, 18, 34, 83, 124, 139, 149, 334, 346, 377
  agonist drugs, long-term 400
  and dyskinesia 203
  Asian channel theory.....447
  cocaine 196
  cycles of.....163
  dormant process.....341
  during withdrawal 285
  emotional 49
  fear of alcohol addiction 619
  from dopamine 36
  from herbal medicine 201
  Hermann case study 419
  higher power.....175
  how, why, and when.....157
  hypothesis summary.....338
  if recovering 219
  indications of 171
  meditation 176
  National Institute on Drug Abuse.....220
  nicotine 301
  parkinsonism 186
  PDers resistant to.....335
  permanent 170, 238, 260
  renewed susceptability 52
  resistance 171
  Safety Limit.....617
  subset of dopamine processes.....339
  twelve-step programs 296
  vs. habituation 172
  vs. will power 297
  Xanax.....154
adrenaline14, 50, 57, 85, 93, 121, 128, 136, 166, 172, 198, 214, 248, 307, 339, 348, 369, 458, 510, 594
  earthquake example 123
  Super-dopamine 94
adrenaline tremor 93
adverse effects*See* specific drugs, specific effects in the index
agitation
  Amantadine 512
  Comtan 518
  Sinemet..... 527
agonist ..... 138, 497. *See* individual drugs
  Birdie/case study..... 221
  blood pressure 404
  Coach/case study 355
  definition 192
  delayed refresh..... 116
  dopamine receptors..... 161
  general description..... 191
  half-life..... 41
  hard to quit 193
  Hermann/case study..... 415
  Hjalmar/case study..... 264
  neuroleptic malignant syndrome 124
  nicotine.....44, 158
  orthostatic hypotension..... 404
  short-term withdrawal symptoms..... 49
alcohol 18, 161
  anti-depressant 83
  avoidance 619
alpha male 343
Alprazolam..... 110
Amantadine 198, 305, 400, 510
  adverse effects..... 512
  Hua To/case study..... 387
  recommended dosages..... 511
  small amount..... 301
Amantadine hydrochloride 485
Ambien..... 552
American Academy of Neurology
  Elldopa study 43
amphetamines 161
anemia
  Permax..... 499
angina
  Coach/case study 350
Angus 185
ankle rotation 206
anorexia..... 179
  Amantadine 512
  Eldepryl..... 522
  Mirapex 506
  Permax..... 499

Sinemet 527
Tasmar 520
anti-anxiety drugs 160
addiction 128
Birdie/case study 222
Buzz/case study 413
industry cover-up 112
overview 552
parkinsonism 260
tardive dyskinesia 83
antiarrhythmics 203
anticholinergic 195
Artane 514
stress reduction 586
switcheroo 586
tremor reduction 586
antidepressant ..... 160. *See* tricyclic
antihistamine 197, 524
anxiety
acupuncture for 552
Amantadine 512
anticholinergics 195
Artane 514, 515
Atenolol 516
Becky/case study 155, 156
Buzz/case study 410
caused by sleeping pills 552
causing tremor 93
Comtan 518
dopamine deficiency 166
during drug reduction 257, 552
Eldepryl 522
Ivy/case study 499
Permax 499
Sinemet 527
tension
Hermann/case study 421
Apo-Trihex 485
arm tremor 87
arrhythmia 401
Permax 499
Sinemet 527
Artane 195, 514
recommended dosages 514
side effects 514
Artane Sequels 485
arthritis
Tasmar 520
asthma drugs 518
Atapryl 193, 485
Atenolol 516
atrophy 88
audio pain, oversensitivity to sound 537

B

B6, vitamin 526, 532
back pain
Becky/case study 56
Comtan 518
Fred/case study 24
Hermann/case study 418, 428
Permax 497
balance 19, 25
Becky/case study 69
loss of 538
parkinsonism 113
baseline 35, 46
changing 35
diminishing (graph) 48
Becky 55
background 55
Benedryl 429
Breath Monster 59
El Twitcho 58
first resumption of Sinemet 58
first Sinemet decrease 56
hallucinations 67
hallucinations from Mirapex 465
Mirapex 463
sleepwalking 59
starting Xanax 110
Tylenol PM 70
bee sting 543
behavioral changes
Eldepryl 522
belladonna 196
Benedryl 106, 197
Becky/drowsiness 109
to stop anaphylactic reaction 544
benzodiazapines (footnote) 139
beta blocker 516
Bhagavad Gita (footnote) 448
Bhatia, K. MD 613
Birdie 221
bitter taste in the mouth
Sinemet 527
blood cell decrease (red or white)
Sinemet 527
blood in the urine
Permax 499
blood pressure, high or low
Eldepryl 522
blood pressure, low
Sinemet 527
blood sugar, high or low
Atenolol 516
blood-brain barrier 37, 525, 560
bloodstream, dopamine levels 37
blurred vision
Bromocriptine 502
brain messenger 560
breath monster 60

drawing.....64
breathing problems.....*See* dyspnea
Becky/case study.....59, 144
Mirapex.....506
Mirtazapine.....524
Permax.....499
Zoe/case study.....29
breathing unevenly.....206
breathing, rapid
Artane.....515
bromocriptine.....191, 478, 502
adverse effects.....502
recommended dosages.....502
Bromocriptine mesylate.....485
bronchitis
Tasmar.....520
bruising
Comtan.....518
buffer.....201, 532, 583
Build up.....234, 254
burning mouth
Rose.....*See* Sinemet/adverse effects
BuSpar.....410
buttocks
twitching or spasm.....206
Buzz.....409, 533

C

Cabergoline.....191, 485, 509
Carbex.....193, 485
carbidopa.....525, 583
ratio to levodopa.....410
carotid sinus.....403, 497
cell loss (footnote).....569
chaos theory.....557
Charcot.....19
charts.....*See* graphs
chest pain
Eldepryl.....522
Mirapex.....506
Tasmar.....520
chin tremor.....*See* tremor
description.....87
choreiform
Sinemet.....527
cigarettes.....44, 50, 124, 158, 400
clinical practice lagging.....591, 605
clonazepam.....*See* Klonopin
Clozaril.....11
CNS depressants.....518
Coach.....228, 347
cocaine.....18, 160, 196
crack.....161
cocaine analog.....596
cocaine research
smaller doses vs. fewer doses.....395
coffee (footnote).....335
cold
dopamine depleting.....378
oversensitivity to cold.....537
Columbia University.....558
Comptan.....198
COMT inhibitor.....517
Comtan.....517
adverse effects.....518
recommended dosages.....518
Conference Highlights.....558
confusion
Mirapex.....506
Mirtazapine.....524
Permax.....499
Sinemet.....527
Congress of Parkinson's Disease.....558
constipation.....475
Artane.....515
Becky/case study.....108
Bromocriptine.....502
Coach/case study.....349
Comtan.....518
Eldepryl.....522
Mirapex.....506
Mirtazapine.....524
not dopamine-related.....162
Permax.....499
Sinemet.....527
Contraindication
definition.....495
Requip.....507
Sinemet.....528
Controlled Release Sinemet.....396
coordination, loss of
Eldepryl.....522
Cotzias, G. Dr.....564
cramping.....206, 538
abdominal/Nat/case study.....306
Birdie/case study.....221
Birdie/leg cramps.....222
Bromocriptine.....502
chest, face, neck/Rudyard.....362
clonazepam for.....393
from medication.....401
hamstring, calf/Rudyard/case study.....358
Hermann/case study.....418
Mark/case study.....530
Mirapex/Rudyard.....362
Tasmar.....520
violent freezing.....210
cramps.....401
cranking.....71
Crash.....244, 254
crushing pills.....526, 531
Cycles.....255

D

D1 receptors 497
D2 receptors 497
D3 receptors 497
daily patterns 233
Datura 196
DBS
  deep brain stim implants 478
deceit 289
DED ....*See* dopamine-enhancing drugs
deep brain stimulation implants 478
Deep Sleep 282, 369, 383
deficit 233, 235, 254
  summary 243
delusions
  Bromocriptine 502
  Mirapex 506
dementia
  Sinemet 527
Deprenyl 193, 486, 523
  joint pain 523
  Lance/case study 200
depression
  after stopping drugs 260
  alcohol 83
  Amantadine 512
  Ambien 552
  antidepressants/tardive dyskinesia 209
  Becky/case study 155
  Bromocriptine 502
  causing immobility 370
  Coach/case study 353
  Deprenyl 523
  dopamine deficiency 131, 166
  dopamine imbalance 138
  drug treatment for 189
  Earle/case study 303
  frontal lobe neurotransmitters 166
  hangover example 245
  in winter 303
  long-term, from agonist drugs 400
  Mirapex 505
  overcome w/music, singing 426
  Permax 499
  placebo 177
  Rudyard/case study 364
  Sinemet 527
  symptom of Parkinson's 406
  when drugs wear off 219
  Xanax 148
diaphragm spasm 60, 70, 206, 527
diarrhea
  Atenolol 516
  Bromocriptine 502
  Comtan 517, 518
  Eldepryl 522
  from milk 493
  Mark/case study 533
  Permax 499
  Sinemet 527
different function in various brain areas 115
dilated pupils 30
  Artane 515
diphenhydramine ....197. *See* Benedryl
dishonesty 291
dizziness 538
  Amantadine 512
  Artane 515
  Atenolol 516
  Comtan 518
  from drug reduction 538
  Mirapex 506
  Mirtazapine 524
  orthostatic hypotension 404
  Requip/Mitch/case study 507
  Sinemet 526
dizzy
  Taylor Paul/case study 380
DNA damage
  from cocaine, ecstasy. (footnote) 159
doctor's prayer 589
dopamine
  "cells are preserved in idiopathic PD" (footnote) 569
  "rewiring the brain" 178
  1950's 559
  abrupt cessation of 375
  accumulation/build ups 233
  accumulation/half-life 41
  addiction from excess 202
  after recovery 50
  allocation 116
  as relaxant (theory) 564
  at night 582
  baseline 38, 46
  below threshold 45
  build up/freezing 41
  catecholamine 521
  cigarettes 191
  Cocaine 196
  cocaine research 179
  comparing natural and artificial 372
  DA producing cells 339
  deficiency 209
  deficiency (symptoms of) 166
  deficiency in parkinsonism 113
  density from deficiency 176
  depleters (footnote) 570
  depletion due to pain 360
  dislodged during emergencies 248
  dormant cells 171

during emergency....123
during sleep....332
dyskinesia....208
excess....33, 125, 223
excess (on graphs)....38
excess, danger of....125
excess/addiction....36
excess/danger of....160
fatigue....348
fear suppressant....122
glimmer of....369
graphing....35
immune system....116
increasing using acupuncture....473
inner peace....125, 616
levels after addiction....47
levitation....374
life force....176
limbic area....120
low levels = addiction resistance....336
Macuna....200
meditation....176
motor area....131
motor initiation NT....331
muscle relaxant....120, 188
National Institute on Drug Abuse....178, 220
native (definition)....12
native amount....46
nicotine....191
night time levels....571, 579
oversaturated but not addicted....338
Parade Magazine....178
parasympathetic system....341, 346
perception of pain....452
perception of time....452
permanently lowered....47
placebo....177
pleasure/bliss....616
prayer....176, 616
precursor....120
prioritization....116, 126
Proceedings of the National Academy of Sciences....179
Prozac Backlash....112
rapid rate in motor area....131
reduction mechanism....43
release via MAO inhibitors....195
researching....178
restoration of....51
resumption of dopamine system....345
return of native DA....370
reuptake blocking via cocaine....178
role in addiction (NIDA)....18
role in various brain regions....120
saints....374
serotonin opposite theory....130
sharing in the brain....332
slow rate of change....124
SPECT scan....333
SSRI's and tricyclic antidepressants....112
stimulant....120, 131, 579
street (illegal) drugs....191
subconscious movement....565
suppressor....120
system....34
tardive dyskinesia....211
temperature regulation....116, 136
ten-week readjustment....124
three systems....330
threshold....38, 134
Time magazine....178
time perception....116
two systems....330
vs. adrenaline....339, 342
Xanax....112
dopamine depleters....378
Dopamine Doorman....120
dopamine hydrochloride....561
dopamine precursor....525
dopamine transporter
decrease....43
dormant brain cells
(footnote)....31
double vision
Mirapex....506
Permax....499
Dr. Gelfand....24
Dr. Leslie
Becky....56
Becky's visit....68
Rose....10, 13
Dr. Pender
Hua To....292
Nat....305
refusing business card (footnote)....10
Zoe....29
Dr. Rafferty....17, 205, 264, 325, 478
Dr. Rafferty, (footnote)....268
dragging foot....80
Nat/case study....304
dragging leg....81
Rudyard/case study....357
dream, abnormal
Permax....499
dreaming, excessive
Tasmar....520
dreams, vivid
Amantadine....511
drug withdrawal....536
Eldepryl....522
drooling....205, 223, 264, 404, 540
Buzz/case study....410

Hjalmar/case study 264, 404
Sinemet 527
drowsiness
Artane 514, 515
Atenolol 516
Benedryl 67
Bromocriptine 502
Eldepryl 522
Mirapex 503
Mirtazapine 198, 524
not a PD symptom 107
Xanax 150
drug failure 53
dry mouth
Amantadine 512
Artane 515
Becky's journal 108
Comtan 518
Eldepryl 522
Mirapex 506
Mirtazapine 524
Permax 499
Sinemet 527
Du 15
acupoint for confusion 552
Du channel 447
addiction theory 447
in utero 97
regulator of dopamine system 453
relationship with Stomach channel 454
dyskinesia 85, 62–64
"only sign of excess" 50
Birdie/case study 221
brief description 85
chapter 203
chart 319
chest muscle 438
Comtan 518
corticostriatal synapses 607
daily pattern 233
definition 82, 85
diphasic 385
Eldepryl 522
excess line (graph) 35
extracerebral dopamine 583
Fred/case study 24
freezing, form of dyskinesia 53
from drugs, not PD 83
heart pain 228
in the overmedication/reduction cycle 256
increasing (after FSR) 402
indication of overmedication 252
Laurel/case study 238
literal definition 84
Mirapex 506
National Parkinson's Foundation (footnote) 214
on a roller coaset 241
Permax 499
preferred by patients 205
quote, "dyskinesia caused by meds" Natl Prknsn's Foundation 83
Rose/case study 14
Rufino/case study 16
sign of toxicity 499
Sinemet 526, 527
switching 388
tardive 31, 217, 260
Zoe/case study 29
dyspnea ..... *See* breathing problems
Atenolol 516
Comtan 518
Mirapex 506
Permax 499
dystonia 538
counter muscles (footnote) 587
definition 81
drug-induced 82
masking pain via drugs 587
Mirapex 506
painful, Laurel 388
Permax 499
Sinemet 527

E

ear acupuncture 551
ear ringing
Tasmar 520
Economist 594
edema
Mirapex 506
Eileen/Buzz's acupuncturist 414
El Twitcho 58
Elaine 302
Eldepryl 24, 193, 218, 302, 357, 521
adverse effects 522
Ivy (case study) 499
recommended dosages 522
small amount 301
tremulous voice 25
Eli553
Elldopa study
American Academy of Neurology 43
embryonic cells
reundifferentiation 577
emergency
mobility during 340
emotional stress 378
Entacapone 486
epinephrine ..... *See* adrenaline
ergot 371

ergot (footnote).....193
Euclid.....183
euphoria.....5, 28, 219, 416
Hermann/case study.....416
Sinemet.....527
excess
line on graphs.....35
excess line.....35, 39, 209
eye pain
photosensitivity.....537
eye, pressure inside
Artane.....515
eyeball rolling.....206
eyelid.....539
Eldepryl.....522
Sinemet.....527
eyelid twitching.....203, 206
eyes, unnaturally radiant.....371

F

fainting
Tasmar.....520
falling
Tasmar.....520
fatigue
Atenolol.....516
Comtan.....518
Eldepryl.....522
Sinemet.....527
fava beans.....201
FDA.....16
Tasmar.....520
FDA approval.....493
FDA approved drug
Baycor lawsuit.....399
festinating gait
description.....80
during drug reduction.....539
Taylor Paul/case study.....380
fibrillations
Amantadine.....512
finger movement.....206
finger tremor.....87
flu-like symptoms
Permax.....499
foot pain.....359
foot spasm.....145, 206
foot sticking.....80
description.....80
during drug reduction.....539
Hermann/case study.....428
foot tremor
description.....87
fourth hypothesis.....314
Fred
case study.....23
freezing25, 35, 38, 41, 53, 75, 80, 211, 233, 539, 615
Comtan.....518
description.....80
drug reduction cycle.....256
drug-induced.....211
Eldepryl.....522
freezing zone.....41
graph.....40
Sinemet.....527
tips for dealing with.....550
violent.....210
Freud quote regarding cocaine.....78
frontal lobe.....128
DA change rate.....129
definition.....118
dopamine deficiency.....130
dopamine excess.....131
dopamine prioritizing.....129
review.....133
Frozen Addicts by Dr. Langston.....567

G

gait.....*See* festinating gait
gait abnormalities
Mirapex.....506
gait, abnormal
Permax.....499
gait, shuffling,
from schizoprenia drugs.....117
gait, uneven or loping.....206
gait, unsteady
Xanax.....148
Galileo Galilei.....571
gambling.....79
gas pains.....206
gasping.....29
gastric lavage.....203
GDNF (footnote).....159
Gleick, J. quote on chaos theory.....557
glial cell neurotrophic factor (footnote).....159
glimmer.....369, 375
Laurel/case study.....391
glutathione.....199
graph
build up.....40, 234
changing threshold.....45
Crash.....244
deficit.....236
diminishing baseline.....48
reduction cycle.....258
Roller Coaster.....241
tracking Sonny's On/Offs.....319
graphs.....*See* charts
gravity (footnote).....373, 374
grimacing.....13, 86, 206

dyskinesia 85
Eldepryl 522
Fred 25
Mark 528
quote from Dr. Sacks 68
Sinemet 527
Stephanie 402
Taylor Paul 380
Zoe 28

H

hair loss
Eldepryl 522
half-life 326
blood drug levels 37
levodopa 41
hallucination 337, 536
Amantadine 512
Artane 515
Becky 66
Bromocriptine 502
Comtan 518
during drug reduction 536
early L-dopa research 565
Eldepryl 522
Hjalmar 265
medication at night 396
Mirapex 505
Permax 499
quote from Dr. Sacks 67
Sinemet 527
hallucinations 223, 293
lasting five months 105
Mirapex 506
nighttime dopamine 582
schizophrenia 117
telling your doctor 603
hand clenching 206
hand pain 359
hand tremor ..... 87. *See* tremor
hangover 245
headache
Amantadine 512
Artane 515
Eldepryl 522
Tasmar 520
heart arrhythmia 206, 401
Eldepryl 522
heart attack
Bromocriptine 502
heart beat
irregular ..... 206. *See* arrhythmia
heart beat, rapid or racing
Artane 515
heart failure
Atenolol 516
heart fibrillations
Amantadine 512
heart pain 350
Eldepryl 522
heart palpitation
Tasmar 520
heart rate, slowed
Atenolol 516
heart valve problems 498
heartbeat irregularities
Sinemet 527
heartburn
Eldepryl 522
heat, avoidance 547
hemorrhage 10
herbal dopamine 201
Hermann 414
hiccups
Sinemet 527
hip, broken 226
hip, pain 206
Hjalmar 264
holiday, drug 167
honeymoon 212
hot, oversensitivity to heat 537
Hua To 261
Switching 387
humility 175
hyperventilation
Sinemet 527
hypothermia 544
hypothesis
first 37
fourth 314
second 334
third 50
hysteria 539

I

iatrogenesis 604
illness, depletor of dopamine 378
illogic 295, 538
immature cells 578
implants, deep brain stimulation 478
impotence
Tasmar 520
incontinence
Sinemet 527
indigestion
Comtan 518
individual drugs
adverse effects *See* find the specific drug in the index
details on each drug 485
insert, drug information 494
insert, terminology 495

insomnia....536
Amantadine....510, 512
Artane....514
Becky....66, 111, 151
Bromocriptine....502
case study....553
during drug reduction....536
Eldepryl....522
Eli553
helpful tips for....547
insomnia drugs cause insomnia....554
Lila....183
Mark....530
Mirapex....506
Mirapex, Requip....503
Permax....499
Xanax....553
insubordination....603
insurance (footnote)....551
intensity of purpose....587
internet....179
intestinal spasm....206
irrationality....293
Ivy, case study....499

J

JAMA....181, 335, 587, 605
jerking movements....206
Jonesing....71
Journal of the American Medical Association*See* JAMA

K

Kaasinen, MD, Valtteri (footnote)....8
kicking....206
Kidney channel....452
Kidney Yang Excess....125
Klonopin....290
Buzz....410

L

Langston, J. Dr.....567
Large Intestine channel
(drawing)....89
Laurel....237
dystonia....388
L-deprenyl hydrochloride....486
leg cramps....222
leg spasms....206
leg tremor....*See* tremor
Lemual....480
lethargy
Atenolol....516
Eldepryl....522
levitating saints, (footnote)....374
levodopa....202, 238
400 mg maximum....162
addiction research....179
Angus....185
Becky reducing last little bit....310
below 400 mg/day....259
Birdie/case study....221
Buzz....410
Coach....347
Coach, sensitized....350
current half-life theory....38
dangers at 100 mg/day....286
decreasing if using an agonist drug....399
definition....561
dyskinesia after 2 to 5 years....83
Earle/case study....303
effectiveness compared to carbidopa/levodopa....201
Euclid....184
for life....259
Fred....24
from fava beans....201
from Macuna....201
Hermann....417
Hjalmar....264
Hua To....387
Laurel....388
Lila....182
love-hate relationship....400
manufacturer's warning w/re reduction....273
neuroleptic malignant syndrome....124
problems with controlled release....396
Sinemet website....180
sleep aid following brain implant....477
small amount....260, 301
Sonny/case study....290
Stephanie....307
use with MAO inhibitors....195
Yves....445
Zoe....28
Levodopa-carbidopa....486
Lieberman, MD
on dyskinesia (footnote)....213
Parkinson's Report....31
percent cell death....568
lightheadedness
Bromocriptine....502
Lila
background....182
limbic area
dopamine deficiency....128
dopamine excess....128
rate of change....122
review....133
limbic system
definition....118

slow rate of change....122
Linnaeus, Carl (quote)....203
lip spasm....206
liver failure....520
liver problems
Sinemet....527
lizard brain....118
losing weight....*See* anorexia
loss of appetite....*See* anorexia
loss of balance
Eldepryl....522
Tasmar....520
low blood pressure....404
agonists....497
Amantadine....513
Atenolol....516
Bromocriptine....502
Eldepryl....522
Mirapex....503, 506
Permax....499
Tasmar....520
lying
during drug reduction....538
to patients....7, 181

M

Macuna....200
Madopar....486. *See* Sinemet
Mahatma Gandhi....95, 294
Maimonides....589
Mandate of Heaven....168
Mandell, Arnold, modern dynamicist (quote)..557
mania
Bromocriptine....502
MAO inhibitors....193, 521
combined w/ levodopa....522
type B....521
Mark....528
10% plan....529
death....533
mask....587
massage....550
Maurice....79
meditation....125, 176, 372, 616
nun research....176
Medscape....558
megalomania....79
melanoma....528
melatonin....144
memory loss....296
methamphetamine....18, 160, 195
Mic....310
Mirapex....503
adverse effects....506
agonist function....191
apparent effective life....41
Becky's hallucinations....466
Birdie....221
Coach....355
D3 receptors....120
Earle....303
Elaine....302
half life....41
happy hallucinations....505
Hjalmar....264, 265
Hjalmar's journal....265
Lila....182
low blood pressure....404, 503
low levels....170
manufacturer's warning....203
Mark....528
neck tension....504
neuroleptic malignant syndrome....273
official half-life....327
pro-Mirapex study (footnote)....44
recommended dosages....506
Rose....11
Rudyard....357
six months to see benefits....124
stroke....504
study suggesting slowing of PD....588
subtext of depression from quitting....400
with brain implant....479
Woody....619
Mirtazapine....197, 524
adverse effects....524
Nat....306
recommended dosages....524
missing paperwork....29
mood....47, 126, 326, 550
after addiction....47
-altering drugs....128
frontal lobe....119
rate of change....129
role of frontal lobe in....118
Mother Teresa....175
motor area
DA prioritization....132
definition....131
dopamine deficiency....132
dopamine excess....132
rate of DA change....131
Movement Disorder
conference....558
MPTP....568
multiple injuries....446
muscle heaviness
Mirapex....506
muscle spasm
Mirapex....506
muscle tension
Permax....499

music
helpful tips for drug reduction 547
imparting movement 80

N

NADA protocol 551
Nader, Mike 593
primate research (footnote) 18
NADH 193, 199
Becky 145
narcolepsy 465, 503
Becky 465
Mirapex 506
Nat 304
National Institute on Drug Abuse 18, 220
article on addiction 178
website address 179
National Parkinson's Foundation
percent cell death (footnote) 568
National Parkinson's Foundation
dormant brain cells (footnote) 31
dyskinesia 213
native dopamine12, 35, 46, 112, 130, 237, 375, 385, 402
switching 141
Nature Neuroscience 593
nausea 115, 122, 418
Amantadine 512
Artane 515
Atenolol 516
Becky 62
Bromocriptine 502
Comtan 518
dopamine deficiency 166
Eldepryl 522
from heightened sensitivity 537
Mirapex 506
Permax 499
reduction cycle 257
Sinemet 527
symptom of drug reduction 536
neck pain
burning pain 360
Mirapex 497
Tasmar 520
neck, stiff
Eldepryl 522
nervousness
Artane 515
Bromocriptine 502
neuroleptic drugs (footnote) 570
neuroleptic malignant syndrome 124, 273, 519
Neurology (Journal of)
L-dopa addiction 34
Newsweek, article on brain sites 161
nicotine 18, 44, 160, 191, 192, 193, 199
12-hour delay 44, 616
preventing Parkinson's 335
nicotine patches 199
NIDA 18
nightmares .....536. *See* vivid dreams
nighttime use of meds 396
nigral cell disappearance 569
Nobel prize 559
non-addictive products 199
nonsympathetic state
(footnote) 457
norepinephrine 211
definition 128
norepinephrine re-uptake blocker 198
nuns
meditation brain change 176
Nutt, MD, Elldopa study (footnote) 44

O

Off 233
definition 75
partly Off 77
Oliver Sacks 17
Awakenings 565
Awakenings (patient quote) 67
comparing his patient and Birdie 223
patient seeing atoms 79
ticcing description 207
Omar Khayamm 448
On
definition 75
On!
definition 78
onset time 323
opiates 18
overmedication, phase in drug reduction cycle 258

P

pacing 537
pain
during recovery 541
from medication (research) 613
pain in chest, neck, or back
Permax 499
palpitation
Tasmar 520
palpitations
Eldepryl 522
Permax 499
paradigm shift 570
paralysis 80
paranoia
acupoints for 552
Deprenyl 523
drug reduction 223

from dopamine deficiency 128
Mirapex....506
symptom of drug reduction....537
parasympathetic system 50, 51, 339, 344, 454
Du channel relationship 452
Parkinson, James....19, 93
parkinsonism 113, 405
definition(footnote) 6
drug-induced.... 31, 167, 186, 189
Parkinson's
personality (footnote)....8
Parkinson's Disease, Questions and Answers, by
Merit Publishing....570
Parkinson's Report....569
Parlodel.... 486. *See* Bromocriptine
particles....79
passing out
Permax....499
patient rights....603
Paxil....18
Pergolide.... 191, 486. *See* Permax
Pergolide mesylate 486
permanent ticcing (footnote)....208
Permax 191, 497
adverse effects 499
Ivy 499
recommended dosage 498
sex-enhancement 500
small amount 301
personality disorder
Permax 499
Pert, Candace....42
PET scan....333
Physician's Drug Handbook....491
pill-rolling tremor....87
placebo....177
pleasure, addiction....116
posture
fetal position 543
hunched....539
pouting....206
Pramipexole hydrochloride.... 486. *See* Mirapex
prayer 176, 372, 616
increasing Kidney Yang....125
St. Teresa, role in....374
prescribing drugs
scope of practice (footnote) 9
priapism
Sinemet 527
primate addiction....18
primate study 593
prioritization of dopamine....137
Proceedings
National Academy Sciences (footnote)....8
protein 583, 585
Prozac 18, 123
with Mirtazapine 524
Prozac Backlash 112, 161, 396
SSRIs, tricyclics....128, 151, 161, 396
SSRIs, tricyclics (footnote) 18
psychoactive, definition (footnote) 8
psychosis 582
Permax.... 499
psychotherapy 415
psychotropic, definition (footnote) 8

R

Rafferty, MD.... 604
rage
limbic response 118
random brain respose.... 557
Rebellious Qi 22
(footnote).... 21
Remeron 197
reptile brain 118
Requip 191, 304, 507
adverse effects.... 507
Mitch, case study 507
recommended dosages.... 507
small amount.... 301
respiratory distress....7, 24, 29, 60
Becky.... 111
Euclid 184
restless leg syndrome.... 5
restlessness
Eldepryl.... 522
rhubarb 591
rigidity, in meditation 616
rigor mortis.... 587
risus sardonicus.... 587
Roller Coaster 240, 254, 319
Ropinirole....486. *See* Requip
Ropinirole hydrochloride 486
Rose.... 10
medication levels 11
mouth pain 12
Rubaiyat 448
Rudyard.... 357
Rufino.... 73
violent freezing 210

S

Sacks, Oliver, MD ....*See* Oliver Sacks
Safety Limit 158, 213
saint .... *See* St.
Samantha.... 218
Sammy.... 302
scalp acupuncture.... 21
schizophrenia 117
scope of practice, (footnote).... 9
scopolamine 196
sebhhorhea 162

seizures
Amantadine....512
Bromocriptine....502
Selegeline....486
selegeline hydrochloride....193, 486
self-assessment....175
Selpak....193, 486
serotonin....112, 128, 160, 198, 211, 410. *See* SSRI
"is the opposite of dopamine" theory....153
effect on dopamine levels....128
Xanax....150
serotonin reuptake inhibition
effect on dopamine....128
sexual dysfunction
Eldepryl....522
Shakespeare
quote from Macbeth....289
shaking....223, 535
Shaking Palsy....19, 86
shock....544
shoulder twitching....206
Shut down....211
side effects, broader meaning....553
Sinemet....525. *See* levodopa
"is my only comfort," Becky....151
adverse effects....527
arrhythmia....401
Becky resumed....58
Becky's journal....145
Becky's journal, urinary frequency....149
Becky's visit to Dr. Leslie....69
Buzz....410
case study....528
Coach....347
comparing the 25% and 10% config....532
crushing pills....532
drooling....404
Earle....303
Eli, insomnia....553
Elldopa study....43
Fred....24
Hermann....420
Hjalmar....264
Hjalmar's journal....264
is no longer reliable for Becky....151
Lila....182
manufacturer's contraindications....528
manufacturer's website....525
Mark....482, 528
not working during withdrawal from Xanax....150
overmedication (Angus)....185
overmedication from (Becky's journal)....154
recommended dosages....525
Rose's dose....11
Rose's mouth pain....13
Rudyard....407
Rufino....74
sleep aid following brain implant....477
snakes crawling under the skin....155
Sonny....290
Stephanie....307
Taylor Paul....379
Taylor Paul, emails....609
with Eldepryl....521
with vitamin B6....532
with Xanax (Becky's journal)....153
Yves....445
Zoe....28
Sinemet CR....396, 486. *See* Sinemet
sinus collapse....586
sinus problems
Mirapex....506
Permax....499
skin cancer....531
skin rash
Eldepryl....522
sleep
7a.m. to 9a.m....541
excessive/deep....541
sleep aids....548, 552
sleep disorders
Mirapex....506
sleepiness
Comtan....518
Eldepryl....522
Mirapex....506
Mirtazapine....524
Slide....247, 254
slowness
Amantadine....512
Eldepryl....522
snakes, crawling sensations under the skin....26
social crisis....283
Sonny....289, 376
spasm in the airpipe (bronchospasm)
Atenolol....516
specific drugs, details on each drug....485
SPECT scan....333, 598
speech
difficult to understand....206
loud or rapid....206
slowness....539
speech disorder
Permax....499
repeating words....537
Tasmar....520
speech, slurred
Amantadine....512
spiritual change....176
SSRI....128, 160, 524
effect on dopamine....112

Mirtazapine....198
St. Francis' basilica....373
St. John's Wort....108
St. Joseph of Cupertino....373
St. Teresa of Avila....373
talks with God....374
St. Vitus' dance....193
stabilization....30
statistics....473
Stephanie....307
stiffness
Eldepryl....522
Tasmar....520
Stomach channel....383, 454
(drawing)....91
stress
adrenaline....95
adrenaline tremor....95
causing tremor....586
regulated by DA....136
stroke
Bromocriptine....502
substantia nigra
changes due to aging....567
substantia nigra cell death
Parkinson's Report (footnote)....31
suicidal tendancies
Sinemet....527
suicide....353
Sunday supplement....178
superdosing....243, 254
supplements....199
suppressor
dopamine....120
surgery....584
surrender....175
swallowing, difficult
Eldepryl....522
Mirapex....506
sweating
Comtan....518
Eldepryl....522
Switching....246, 254, 385
Symmetrel....198, 486, 510
sympathetic system....50, 52, 172, 338, 458
Asian perspective....458
quick transition to/from....342
sliding scale....446
turned off during recovery....344

T

tardive dyskinesia....31, 112, 168, 198, 211, 217, 260
definition....211
resembles tremor....209
side effect of anti-parkinson's drugs....384
Tasmar....198, 517, 520
adverse effects....520
taste perversion
Comtan....518
Taylor Paul....609
bladder infection....379
teeth clenching....206
ten percent....220, 273
ten weeks....42, 47, 103, 218, 247
cycles....255
limbic adjustment....124
Tennyson, Alfred, Lord....33
Tenormin....516
tension....416
during switching....385
Hermann/case study....416
in drug reduction cycle....256
Nat/case study....306
tension, neck (Rudyard)....362
termperature regulation....537
theta wave....87
Thorazine....117
thought abnormalities
Mirapex....506
threshold....134, 313
changing....35
rising (drawing)....164
rising (graph)....45
throat, swollen
Tasmar....520
ticcing....206
violent....209
ticcing, definition....207
Time magazine....178
toe curling....206
toe spasms....206
toe tapping....206
toes, rising up on....206
Tolcapone....486
tolerance, addiction (footnote)....6
tongue thrusting....206
tremor....535
adrenaline....93
Amantadine....512
Artane....514
Bromocriptine....502
definition....84
description....82
during drug reduction....535
during meals....98
during recovery....384
Eldepryl....522
from hypothermia....544
Mirtazapine....524
Permax....499
self-awareness....96

Sinemet 527
stress-sensitive 586
Tasmar 520
theta wave 87
violent 209
waking up 97
weakest part of the body 543
Xanax 553
tricyclic drugs 128
tricyclics 526, 570
effect on dopamine 112
Trihex 195
Trihexane 486
Trihexy-2 or 5 486
turkey dinner 503
Turnaround 237, 254
twitching
acceptable level 204, 213
DED overdose 132
drug warning 204
dyskinesia 206
Eldepryl 522
Mirapex 506
Permax 499
Sinemet 527
Sinemet overdose symptom 527
twitching muscles 203
Tylenol
Taylor Paul/case study 611
Tylenol PM 103
liver damage 104
tyramine 522

U

urinary frequency
Eldepryl 522
Permax 499
urinary frequency or retention
Sinemet 527
urinary hesitancy
Artane 515
urinary incontinence
Tasmar 520
urinary retention
Amantadine 512
Artane 515
Bromocriptine 502
Eldepryl 522
urinary tract infection
Permax 499
Tasmar 520
urination
frequent 536
painful 536
urine, dark
Sinemet 527

V

vacation 256
vaginal pain 499
Vicia fava 201
violence 289
violent behavior 538
violent ticcing
Rufino 74
viral protection 510
vision abnormalities
Mirapex 506
vision, blurred
Artane 515
vision, blurry
Amantadine 512
vitamin B6 ..... 526, 532
Voltaire, quote 217
vomiting 536
Amantadine 512
Artane 515
Comtan 518
Eldepryl 522
Sinemet 527

W

Waldo 275
walking, overly fast 206
Walt Whitman 352
water retention
Mirtazapine 524
water swelling
Mirapex 506
water wheel
(drawing) 251
weakness
Artane 515
Comtan 518
Mirtazapine 524
weakness, during recovery 541
Website warning 9
weight loss ..... 537. *See* anorexia
Eldepryl 522
Sinemet 527
which drug to stop first 398
will power 174
window 135
window, decreasing (drawing) 165
Woody, case study 619
World War II 351
writhing 206

X

Xanax 18, 112, 123, 139, 168, 553
effect on dopamine 112

Y

Yin Tang.....448
Yin Tui Na.....12
Yves.....445

Z

Zoe.....28, 475, 477
Zoloft..... 18

Janice Walton-Hadlock

Janice Walton-Hadlock, Licensed Acupuncturist, teaches at Five Branches Institute of Traditional Chinese Medicine in Santa Cruz, California. She has a BA in Biology from University of California and a Master's degree in Traditional Chinese Medicine. Her research articles on Parkinson's disease have been published in peer-reviewed journals of Asian medicine. Her books, including *Recovery from Parkinson's Disease: A Practitioner's Handbook*, and *Medications of Parkinson's Disease: Once Upon A Pill*, are available on the internet for free at www.pdrecovery.org.

photo by Peggy Patterson

John Bateson

John Bateson, the illustrator, lives in Pembroke, Ontario with his wife Peggy and their two children. He graduated from Ontario College of Art and has worked as a commercial artist, as well as doing landscapes and humorous illustrations. His favorite job around the house is cooking. He loves to spend time with the children, in the garden, reading, and exploring the wild woods. He generously donated the art in both this book and in *Recovery from Parkinson's Disease*. He is recovering from Parkinson's disease.

Paul Saxon

Paul Saxon, of Vancouver, BC, performed the graph magic, took Janice's photo, helped tremendously with proofreading, and kept us laughing. He is recovering from Parkinson's disease.