

“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 recovery¹⁰ 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.

freezings¹¹ 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 movements¹² – 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-PDer's 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-dopa¹⁵ 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-PDer's 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

¹⁴Parkinsonism 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/psychotropic²³ 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.

²⁴An 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

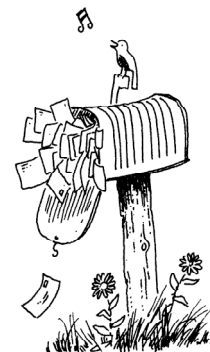
By the time Rose died, there had already been two others who had been lost to the drugs. I was wracked with the heart-breaking might-have-beens! And yet, as this book is coming together, I finally see that it might have been a blessing that I had not been allowed to prescribe medication. What I had considered 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 my article published in acupuncture journals every year since 1998. But no journal possibly accept an article of mine on medication. In the first place, the papers were due to people continuing meds after starting to recover – and no journal accept an article that presumes Parkinson's disease is curable. My advice was that it would be twenty years at least before my work became accepted by



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 reduction³⁴ – 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 *anticholinergic*. 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/antidepressant 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.