

“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.

¹J. 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 *undermedication* 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 become 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 insistent, 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 PWP's [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 pattering 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 PDer's 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.”

