

*“Why didst thou promise such a beauteous day,
And make me travel forth without my cloak,
To let base clouds o’ertake me in my way,
Hiding thy brav’ry in their rotten smoke?”*

Shakespeare’s Sonnet 34

21. GLIMMERS OF HOPE AND OTHER DANGER SIGNS

CONFUSING MINGLINGS: DRUGS AND NASCENT HEALTH

There are still a few medication complications that we have not discussed yet: the glimmer of returning dopamine, the trio of dopamine depleters, the deep sleep, the tenacity of the On-Off and other drug-induced patterns, the switching phenomenon, and the lingering siren song of the drugs. These phenomena are of interest because they enrich our thinking about dopamine-enhancing drugs as well as presenting challenges to any medicated person who is trying to recover from Parkinson’s.

GLIMMER

The glimmer is the name we’ve given to the first, shy blushes of native dopamine. The glimmer occurs when the body’s returning dopamine production is finally large enough to propel a few moments of natural, non-adrenaline movement. The sensation is one that has been long forgotten by PDers, and it seems somehow magical.

The return of dopamine

When patients begin recovering, they often notice glorious, if short-lived, new symptoms occurring now and then. These symptoms most often occur after a period of rest or sleep. The experience may feel like a moment, several minutes, an hour or a day of movement that was perfectly healthy, done to a normal tempo, that went smoothly and easily, and which required no thought.

Most PDers are stunned the first few times that they experience this movement. “I don’t know what happened: I thought about standing up from my chair, and the next second I realized I was standing up!” is the sort of description we hear.

There is absolute certainty on the part of the patient that this is a different form of movement than that felt under the influence of the drugs. Patients often struggle to explain how it felt to just move, but “effortless” is the most common description. They often say that they never moved that way even prior to their diagnosis of Parkinson’s.

Many people with Parkinson’s, hearing this description, protest that they never struggled with movement prior to their diagnosis. However, once they experience this form of dopamine-based movement, they realize that they had no basis for comparison. What they had thought was ease of movement was, in fact, adrenaline-driven, relatively conscious and intentional.

The people who have experienced these glimmers and who have also taken antiparkinson's medication have valuable experiences to relate: they invariably say that the glimmers of native dopamine are unlike the drug-induced movements. The movement that occurs has been described as more "simple" and more "sweet" than the drug-induced movements.

These words may not help the reader. Trying to explain to the reader the different feelings that accompany movement with different neurotransmitters is rather like trying to explain the taste of an orange. But I'm going to try.

How it feels to move

Under the influence of adrenaline, a PDer has been able to move stronger, harder and faster than most people during most of his life, prior to his diagnosis with PD. His movements were not effortless, however. He would create an inner command such as "Now I'm going to stand up!" and then he would make himself stand. Or else "This hill is high, but I'm just going to keep putting one foot in front of the other, and I'll make it to the top." And then, his adrenaline-rich brain would make it happen. A common attitude of the pre-PDer is, "It's just a question of mind over matter; I can tell my body what to do, and it does it."

Then, after the person is diagnosed with Parkinson's disease and starts taking DEDs (dopamine-enhancing drugs), he may notice a new type of movement. (These feelings may not be so obvious in the recently diagnosed PDer, but they become more familiar by the time a person starts having Ons and Offs.) As the drug washes through the brain, it appears that the areas that are altered the most are the limbic and mood areas, not the motor area. There is a feeling of confidence, of the disappearance of pain (somewhat, unless the medication causes painful dystonias or dyskinesias), lengthening of the limbs and then, just before the movement begins, a feeling of power and vigor may pervade the body.

Rigid limbs – like sitting in a cold car

This lengthening of the limbs can best be described this way: short limbs are the feeling that one gets while driving in a cold car on an icy morning. In that freezing car, you are stuck in a sitting position with no way to get your limbs moving, and the cold chill of the car sinks in and you slowly, ever so slowly, tighten up. Without realizing it, you are holding your hands a little too tightly on the steering wheel and leaning forward, hunching your shoulders in, making your body a bit smaller in order to conserve heat. And then, after five or ten minutes, the car heater starts to make a difference, and you feel your body lengthening. Suddenly you realize that you can relax, the shoulders can open up, the death grip on the steering wheel relaxes into a carefree pose, and you just feel more able to move. It is this sort of feeling that is imparted by the DEDs. It is as if the mood and the body temperature both warm up a bit.

Immobility from depression

Another way to understand this type of movement is to remember how you feel when you are deeply depressed. For example, maybe your true love was just reported missing at sea. You sink into despair. You don't want to move. You force yourself to

shuffle along. You have no bounce in your step. Your mood is making you drag. And then, suddenly, you get the phone call from your true love who had been thought lost at sea, and, not only has she been found, but she has discovered a sunken treasure of sixteenth century gold and rubies. At this point, you dance, you sing, you laugh for joy. This is the sort of movement that the antiparkinson drugs provide.¹

Dopamine-enhancing drugs impart joy, a mental and emotional warmth, and even for many people a physical warmth, which then allows movement as an expression of that joy.

“I don’t feel joy from my drugs”

At this point, there will be many readers who will say, “I never feel any joy from my medication. I do not like taking my drugs. You are wrong.”

Now, what I am saying is not that the drugs necessarily impart *obvious* joy. No. Although many people have awareness of being bright-eyed and more clever and happier when taking antiparkinson’s drugs, many do not. Sometimes, the person taking the drugs does not recognize any mood change at all, but loved ones can detect a brightening in the eyes and a mood shift, sometimes even a goofiness, that herald the beginning of an On.²

The drugs effect this change on a subconscious level; maybe this is why so many patients who are clearly undergoing a personality change when their dose kicks in will insist that they have no conscious awareness of any change. Although it *appears* that the dyskinesias and obvious adverse effects are coming from the motor area, most of the improvement in movement may actually come from the limbic center and frontal lobe use of the increased dopamine, both areas that play a role in subconscious movement.

Lightened body, lightened mood

The conscious mind may or may not be aware that it undergoes a change when under the influence of the drugs. But the type of movement, the quality of the movement, and what it feels like inside the body to generate that type of movement are consequences of an uplift in the mood and unthinking animal centers of the brain. When it is easier to move the arms after taking the drugs, it is because the arms feel lighter, less heavy and oppressive. This lightness of being is similar to the lightness one feels when one has a positive attitude and everything is going well.

Due to this lightness and sense of well-being, many people who are thrusting their arms or grimacing imagine that their dyskinesia is not as violent as it looks to their horrified friends. Often the dyskinetic PDer thinks his limbs are merely floating gracefully because they have become so light. *The drugs create emotionally enhanced movement.*³

¹ The agonist drugs do this to a lesser extent than the other drugs. The dopamine movement imparted by the agonists sometimes feels more natural as long as the doses are minimal. But when the agonists, by decreasing the dopamine need in the motor and mood area, free up some extra dopamine so that there is an excess of dopamine floating around, even though the agonists do not convert into dopamine per se, these symptoms may occur. This is why even an agonist like ergot can cause a person to dance and shout and hallucinate, even though it technically does not provide dopamine.

² This unnatural radiance can be quite obvious. I can usually spot an overmedicated PDer from a block away. There’s something uniquely strange about the eyes.

³ I have several patients who will refute this idea that L-dopa makes one feel good. They absolutely hate the way that L-dopa makes them feel. They have described the feeling of moving with L-

Takes one to know one

In response to all you drug-using readers who are going to suggest that you are unique, and that you don't feel this way, let me make this point: most PDers who take the meds are not able to accurately describe what it is the medications feel like until they are off the medication.

Only after they start to experience native dopamine for the first time in their adult lives are they able to realize, objectively, what it felt like to have artificial dopamine enhancers in their brain. It is only when they can compare the two side by side that they realize the difference.

Native dopamine

Native dopamine provides a type of movement that is completely different from the adrenaline or mood-created movements described above. By "native dopamine" I mean natural dopamine, produced in the brain (including that produced in the substantia nigra). This is the movement that most humans take for granted, and it is a type of motion that PDers typically have never felt since childhood. This movement is unplanned. It doesn't require that the mood be good or the body be heated up.

This movement occurs nearly simultaneously with subconscious thinking. It can also direct conscious movement. An example of unconscious movement is swallowing or speaking, or the movements that non-PDers make in their sleep. In sleep one is not supposed to use adrenaline to gently turn over. In our sleep we also don't really care if we are happy or dejected, we can still turn over, regardless of mood.

With PD, swallowing, blinking, and even speech increasingly become processes in which every step must be consciously executed. As many PDers will testify, speech requires a terrific amount of conscious thought if dopamine is absent. From word selection through to sound formation, every step of the way must be thought through and consciously initiated in order to produce speech. In healthy people, dopamine is the neurotransmitter that allows us to speak before we think – an activity we all regret at times!

Native dopamine provides the effortless movement that scratches at an itch and the perfect, harmonious working of the muscles so that one can sit still for a long time in complete comfort. These movements require no conscious initiation or uplifted mood.

Native dopamine coordinates the movement that enables a fast typist to produce a written word as fast as the word occurs in his mind. This movement is not a reflex, but it is not consciously initiated. Nor is such movement affected by mood – it is mood neutral.

This mood-neutral movement is not like the adrenaline drive that a PDer uses to declare, "I *can* get up from the sofa; I just need to make the effort." The native dopamine

dopa as being "kidnapped" or having a false energy that feels toxic. The people who have felt this way in my limited experience have all been people who, prior to being diagnosed with Parkinson's disease, spent many years of their life doing meditation or spent an unusually large amount of their daily time in prayer. These people say that the lightness of L-dopa brings with it a film of deceit that prevents them from being able to penetrate into inner peace or higher consciousness in the way that they used to. They recognize that the strange lightness of L-dopa is false, and that it is not coming from wisdom or joy, but from an illusion. On the other hand, many people who have gone through recovery and are able to get off their drugs continue to recall with wistful yearning how much easier, how much happier, how much more carefree they felt when they were on the drugs. These people continue to be at risk for returning to the drugs for the rest of their lives, even though the drugs may now have disastrous effects if they do so.

feeling is so smooth that as soon as the thought occurs “I would like to get up off the sofa,” the person finds that they have gotten up off the sofa. It is attitude-neutral, pure thought without the analysis.

“Brain waves taking form as movement”

Rose explained her experience of this phenomenon: “I got out of bed and for some reason, I didn’t even know I was getting out of bed. I looked at my clock, saw that it was time to get up, and as soon as my brain registered that it was time to get up, I was sitting on the side of the bed and then standing, and then walking into the bathroom using a perfectly normal stride! I brushed my teeth, and whatever I thought of doing, that’s what I did, and I was nearly dressed before the miracle ran out and then I was suddenly moving slowly and everything was slow and difficult again. It was a miracle. It didn’t feel like conscious work, it felt like brain waves taking form as movement.”

“I have never felt anything like it.”

The glimmer can occur even with very small, insignificant motor activities, as in the following description from pre-Mirapex Rudyard: “I was sitting in my chair watching TV last night and all of a sudden, I felt comfortable. I didn’t have to think where to put my arm so it wouldn’t be tight; I didn’t have to think about anything at all; I just sat there in my chair and I felt my body sink into the cushions. I wasn’t tired, I wasn’t happy or distracted from my problems, I wasn’t melting into sleep, I just felt this strange feeling as if things were just, well, sort of OK, and my whole body was relaxed. I wasn’t happy or sad, and it didn’t feel like I felt with my Eldepryl. I just ‘was.’ I have never felt anything like it.”

Heavier than the earth

Steve was never medicated. During late stage recovery, he had a nearly topped up supply of dopamine. He often went strolling for hours around the local neighborhoods to enjoy the feeling of unpremeditated movement. Now and then he overextended himself and would come to a complete halt. Sometimes he would have as little as half a minute’s warning before he was first halting, then shuffling, and then motionless.

During the times of non-moving, as he sat on a neighbor’s short retaining wall or at a bus stop, he would analyze how he felt. He described it as feeling as if his body was super-dense, heavier than lead, as if he was heavier than the earth, as if he might sink down into the ground from his intense weight, as if the earth could not support such heaviness. He felt like clay. He was not depressed nor moody, just purely heavy.¹ In fact,

¹ There is a common myth that if the earth were to stop spinning, we would all go flying off into space. Modern science says that the opposite would occur: if the earth stopped spinning, its terrific mass would suck our tiny bodies down into its core. It is the spinning of the earth that keeps us “floating” on the surface. Is it possible that the joy and dynamic of this spinning earth are related to the dynamic in a healthy brain that enables us to defy gravity and entropy and to turn our movement thoughts into physical reality? Is this “lightness” a role of dopamine in the motor-initiation area?

In contradistinction to the utter heaviness that one feels in a dopamine deficiency, as if one’s atoms are merging with the atoms of the earth, regard the great saints who cannot help but levitate when their thoughts of God transport them into bliss. Saint Joseph of Cupertino would literally float off the ground every time he walked past the image of the Mother in the Basilica of St. Francis in Assisi, Italy. Saint Theresa of Avila, Spain, was often chided for her inability to stay on the floor when she was

despite the alarm or despondency that usually accompanies immobility when one still has Parkinson's, the immobility that he felt was viewed as something mildly interesting. Certainly there was no panic associated with this experience, nor any particular joy. It was just that he couldn't move. Usually, in three minutes, or as long as ten, he would feel a return of uplift, and he would be able to stand up and walk completely normally again.

Understanding the On-Offs of undrugged recovery

Steve's episodes with immobility were not caused by fatigue or any emotional content. When he stopped moving it was because he had simply dropped just below the dopamine threshold for movement. Because his adrenaline had been turned off during recovery, he had no way to generate movement when the dopamine ran low. Apparently, his dopamine was still high enough to support equanimity of mood and thought at these times, but not high enough for movement. He was right at the threshold.

Most unrecovered PDers (except for those having On-Offs from drugs) do not go back and forth between effortless movement and stone cold motionlessness. Most PDers, including most of those who are taking medication, are able to use dwindling supplies of adrenaline, a neurotransmitter that enables one to keep going at least somewhat, even when the body is physiologically on empty. However, after recovery, the adrenal supply sets itself to near zero for months, if not years. Therefore, when a recovering person runs out of steam, he may be utterly incapable of moving. The adrenaline override is simply not there. And so, without emotional content or fatigue, when the new dopamine supply is temporarily used up, a person who was moving perfectly normally moments earlier may come to a complete halt, and cannot initiate movement again until the supply has been restocked. There will be no emotional override at these times, no sense of urgency to "keep on keeping on." The emotion may be one of bemused reflection.

Many former PDers have remarked on how events that would have been alarming or cause for agitation in the past become emotion neutral after their recovery. "I just don't get all worried about every little thing anymore," is a common, post-

overcome by Divine love. Is it possible that these witnessed events of lighter-than-earth movement are the opposite of the denser than earth feelings that are connected to dopamine deficiency?

Is it possible that the spinning of the earth that allows us to move, overcoming the powerful attractive force of gravity, might be a force that can be augmented or diminished by the forces generated by our thoughts as we are attracted towards or away from the earth, towards or away from Joy?

If this is so, then the feeling of density of PDers may be all too real and is not a mere figure of speech. This dopamine deficiency and its heaviness might be the opposites of the lightness and – maybe – a dopamine saturation – that are manifested by many of the great saints. If dopamine is in fact the neurotransmitter of spiritual ecstasy, one begins to see why the body so carefully guards and regulates dopamine levels: too much, and a person goes into ecstasy and cannot perform their earthly jobs; too little, and a person is plunged into the hideous world of pure matter, in which no vibrations of love or higher emotions can penetrate. A delicate balance indeed!

And while we are this far from the subject, I will share with you a favorite story about St. Teresa of Avila. She had an immediate and constant relationship with God – she was always sharing with Him in the language of her heart. Did you know, it was through her work that it became legal for Catholics to pray silently, using their own words, instead of the previous requirement that all prayers be repetitions of the approved catholic prayers? At any rate, one day while she was journeying, making a difficult crossing of a river in the wintertime, she fell into the river, soaking herself through. She stood up and spoke severely to God, "Why did you do that to me?" He chortled, "That's how I treat my friends." To which she snapped back, "That explains why you have so few!"

Parkinson's attitude. Therefore, the On-Off from *native* dopamine deficiency that may occur during recovery is not necessarily an emotion-inducing or an emotion-triggered event.

Slow increase in dopamine

As people recover, they increase from having mere glimpses or glimmers of movement, lasting a few seconds or minutes, to having hours at a time when they can move. But regardless of how much dopamine they have, when the native dopamine supply gets used up, dipping just below the threshold level, they can find themselves abruptly immobile, with a heaviness that feels denser than the earth. Unlike when they had Parkinson's, there is no longer adrenaline flooding the system, and so, when the dopamine runs out, they come to a complete halt. This is different from the Ons and Offs of medication. There is neither dyskinesia nor drug withdrawal trauma. There is just simply an abrupt transition during movement from effortlessness to impossibility.

This can be a challenging stage of recovery – it's impossible to know just how much juice is in your brain reservoir. There's no way to know how long you will be able to walk or drive.

Usually, once a person gets to the point where there is an hour or more worth of dopamine able to be stored up in the brain, they find that they can recharge just by resting or sleeping for a few minutes or a few hours, and then they can move again. However, this early-in-recovery supply is minimal – there are no hidden reserves.

Combining the glimmer with medication

This stage of recovery was particularly treacherous for our medicated patients. Back when we still were taking medicated patients, many of them reached the stage of recovery when they were having glimmers of native dopamine when there was no overt medication in their body, such as first thing in the morning or during the wee hours of the night.

Patients who experienced these glimmers of movement, only to fall back into motionlessness, simply could not refrain from taking their next pill of the day. No matter how often I suggested that these increasing moments of dopamine meant that their body had switched over to making its own dopamine, they were loath to sacrifice any movement time by making decreases in their medication. The typical refrain was, "When the new dopamine can go all day long, then I'll reduce my drugs."

That day never came, of course. If the brain is capable of making dopamine again, but a person insists on shoving dopamine-enhancing drugs into their body, the body appears to learn reliance on the drugs. It appears as if the body will only make as much dopamine as is necessary. In fact, it may be necessary to challenge the brain via dopamine insufficiency for up to ten frustrating, slow-moving weeks before the brain will increase its dopamine producing potential.

We simply don't know what the mechanisms are for regulating dopamine quantities in a person who is recovering from Parkinson's and also taking the medication. In our small sampling, most people who started having these glimmers who were taking the medication were never able to make further reductions in their medication. Those who did, like Becky, went through agonies.

Rose

Although Rose had reduced her medication considerably prior to the appearance of native dopamine, she was never able to make another reduction in her medication after she started to have moments (which slowly extended up to forty-five minutes before she left our program) in the morning of perfectly normal, pre-pill movement.

Sonny

Sonny started having glimmerings of native dopamine. For years he had been unable to move at night. However, less than a year into his treatments, he noticed that at three in the morning, a time when he had previously been the most rigid, he was able to move. He could not move much; at first he could only move his least affected limb, his right arm. Over the course of the next year, while we worked to remove more blockages and restore feeling in his long-numbered left side and right leg, he slowly regained use of these limbs during the night as well. The period during which he could move grew longer. He would wake at two o'clock in the morning and move his legs and arms, slowly and weakly, but with ever-increasing accuracy. Sometimes he was awake for hours, just practicing moving. Sometimes he used his new ability to move at night to readjust his blankets by himself. For years he had needed his wife to move his blankets for him and turn him when he cramped up. Now he was able to lift one leg over the other and back again – but only in the wee hours of the night, when his drug crash was over and his native dopamine was working.

He had slowly eased his medication from over 1200 mg/day of levodopa down to around 300 mg/day. At this level he no longer had the dyskinesia or cramping that he had at higher levels; and his Off times were less painful, but he did still have Ons and Offs. Once he started having these glimmers of nighttime movement, he became more reluctant to further reduce his medication because he did not want to have more Off time than he was having, despite the fact that, following every one of his past reductions, he had recovered all the movement that he had prior to the reduction. When he broke his hip, he had been vacillating on the verge of a drug reduction for nearly two months. He was grateful that the broken hip gave him a reason to stop the reductions. Prior to the glimmers of nighttime dopamine, he did not suffer so much with each drug reduction. After his nighttime movement began, each reduction was more miserable. Strangely, he never had the full-blown drug withdrawal symptoms, but he did appear to be increasingly attached to the drugs as his nighttime movement increased.¹

The glimmers at night never got any longer than four or five hours, which, coincidentally, was just long enough to enable him to move in the night, but all ability to move ceased about twenty minutes after he took his first pill in the morning. He couldn't move after that until his first pill of the day "kicked in" forty minutes later. It was as if his body was darned if it was going to make any more dopamine than it absolutely needed. If it needed to make enough dopamine to make it through the long, nighttime hours with no drugs, it would do so, but only exactly enough and no more.

¹ Sonny's changes in recovery and drug addiction moved along at a slow pace. Some patients made lightning fast transitions from undermedicated to addicted. There will be more about these differences in chapter 24.

After Sonny broke his hip he made no more decreases, but instead, increased his medication. He started having worse Offs and less predictable On times (see Sonny's charts in chapter 18). As indicted by his charts, this increase caused him to become grossly overmedicated (severe adverse effects of On-Off, freezing, and dyskinesia). His case was interesting especially because he had made slow, steady decreases for such a long time, and assumed that he would be able to continue making these decreases until he was off the medication. He was not able to. We now assume that his window of opportunity for getting off the medication was probably the first few days when he started having this strange new ability to move all by himself at night, using this new type of slow, languorous movement during which his arms appeared to "move all by themselves."

Conclusion on glimmers

We have learned that if a person starts having these glimmers of native dopamine and is still taking the medication, it is too late to avoid addiction. Not one person has been able to get off the medication without going through the sort of hell that Becky described in her journal if they are still taking ANY antiparkinson's medications at the time that the first glimmers of native dopamine appear. This creates a dilemma for any medicated PDer who wants to be in a recovery program but is thinking of delaying drug reduction until starting to feel some positive signals of actually recovering; if you can tell that you are recovering, it's already too late. On the other hand, if there is a chance you are not going to recover, why should you go through the bothers of drug reduction?

Therefore, because it is unreasonable to stop taking medication before knowing if a recovery is forthcoming, and because, once recovery becomes apparent, the brain damage due to addiction will have already started, we do not recommend a recovery program for PDers who are taking the medication.



DOPAMINE DEPLETERS

Another challenge to people going through recovery, especially if they have been accustomed to taking drugs, is the dopamine depleters: cold, illness, and social stress. During the time when glimmers of movement are just beginning to appear, and dopamine levels are trembling on the threshold, almost any sort of physiological challenge will cause the dopamine levels to drop just below the threshold level. When this happens, it appears as if there is no dopamine in the system whatsoever. This can be emotionally devastating for a person who has finally started to experience the long-awaited recovery from this “incurable” illness.

A person who has ever taken the medication who starts recovering but who then has any moments of immobility will typically assume that all of his seeming progress was false, merely psychological, and that he actually still has Parkinson’s disease. Even if the drugs don’t call to him during these times of brain stress, his friends and his doctor may insist that, when he took his drugs like an obedient fellow, he never had any problems. They may use powerful emotional leverage in their attempts to either coerce or cajole him into resuming his medications.

The trio: Cold, illness, and emotional stress

There are three primary factors that can deplete dopamine so that the brain drops below the effective level: getting chilled (or any exposure to extremes of temperature), illness, or emotional stress. If this happens, it will seem exactly as if a person has profound Parkinson’s disease. In this stage of recovering from Parkinson’s – when there is no adrenaline and just barely enough dopamine – when the movement stops, there is no way of knowing, just from looking, whether or not a person is within a few hairs of normal movement or a thousand miles away. A person in this condition is just like Steve, sitting by the side of the road, as heavy as lead, with this exception – if the immobility is due to one of these three dopamine depleters, it may take days instead of minutes for the dopamine levels to be restored.

Cold

We have seen this repeatedly, and yet it seems very difficult for patients to understand – the effects of these three dopamine depleters can last for several days after the event is over. A full six months after Steve had experienced what he’d imagined was the last of his sudden droppings of dopamine below the threshold, six months of consistently reliable movement, apparently free from any semblance of Parkinson’s, he was out on the prow of a boat on a crisp, windy February day. His socks were wet, but everyone was having a good time and he stayed on the deck for six hours. He was cold for the rest of the day and didn’t really get warm until he got home and had a hot shower. Three days later, going for an amble, he recognized the warning signs that he was rapidly approaching zero dopamine again. He made it as far as a bus stop and then sat motionless for ten minutes. He was able to start moving normally again after his ten-minute rest, but he was stunned that he should have had a depletion event when he had been doing so well for six months with no signs of dopamine deficiency.

When he asked me about it later, I asked if he had gotten chilled or had a mild flu in the week preceding the event. He recalled the chill on the boat deck but wanted to

dismiss it: “That was three days earlier, and I felt fine afterwards.” But looking back, he had not gone walking or in any way challenged himself in between the chill on the boat deck and the subsequent shutting down. He had probably been just barely at the threshold, and when he went for his walk, he dropped below and experienced, once again, the absolute nature of the dopamine threshold.

Here is the point I am working up to: had he ever taken the medication, he would have been tempted at this point to say, “I had an unexpected episode of immobility after six months of feeling perfectly normal. I guess this is proof that I still have Parkinson’s after all. I will never be completely better; I might as well be taking the drugs.”

We have seen this scenario over and over.

Life sustaining measures have priority over movement

It may seem baffling, at first glance, that a chill or flu can have such a lingering effect on the dopamine levels. But it appears that the crucial life-sustaining functions of temperature regulation and supporting the immune system are high-priority items for the body. It appears that, in the case of these high priority events, the limbic system can sacrifice its dopamine content rapidly, just as it does in the case of an emergency. However, as we have already seen, the restoration of limbic dopamine after the crisis can take two or three months. If a person has recovered from Parkinson’s disease to the point where he has just barely adequate dopamine, he may appear (if his muscle damage has been repaired), for all intents and purposes, completely normal. However, if his dopamine level drops just below the threshold, he will be utterly unable to move.

Any event that decreases his limbic dopamine might create an illusion of profound Parkinson’s that can last for weeks or a few months, until he body restores its dopamine reserves, after which he might appear perfectly normal again.

These events are probably a good thing in the long run: following each of these events, it appears as if the brain cautiously decides to up the dopamine levels just a tiny bit more. The brain, ever efficient and exacting, will not create more dopamine than it needs. However, these crashes, similar to the stunning, complete collapses of a tired infant, seem to be indicators to the brain that the dopamine-making processes need to be increased by just a little bit more.

During the entire time a person hovers at the very threshold of movement, a person is at risk for seemingly inexplicable crashes. If there is a rapid depletion of dopamine during times of either obvious or subtle body emergencies due to the stresses listed above, interludes of immobility, worse than their PD ever used to be, may come and go, or even appear permanent for a period of several months. Due to the slow restoration of dopamine, these seemingly brief, insignificant events may explain the terrific, long-lasting crashes and immobility that we have seen in people who had thought they had recovered but who have appeared to have a Parkinson’s setback following an infectious disease, a chill, or an emotionally stressful event.

Taylor Paul

If you will forgive the inclusion of another case study, I would like to give an example of this. Taylor Paul, age 69, diagnosed five years earlier, had been taking 400 mg/day of Sinemet when he started our program (in summer of 2001). He began reducing his medication and steadily reduced down to 25 mg/day (one fourth of a 25/100 tab). He

then stayed at 25 mg/day for six weeks before making the final reduction – down to no medication – in September of 2002.

When he was at the 25 mg/day level, he fared pretty well; he lived alone, and he was able to perform all the activities of daily living, plus putter around in his workroom and do his beloved projects. Shortly after his reduction to no Sinemet, he went through a period of drug withdrawal that lasted fourteen weeks, during which he felt shaky, had festinating gait, and often woke in the night with horrible, even violent shaking. But after fourteen weeks, he felt he had turned the corner: he was drug free and feeling better every day.

Two months later he started feeling worse. He felt weak and shaky. He just didn't feel right. He had urgent urination throughout the day, and slight burning pain when urinating, but assumed that it was a recovery symptom. His doctor told him that it was the Parkinson's and suggested taking the drugs again. Taylor Paul refused and went home, but he kept feeling worse and worse.

After a month of increasing weakness and shakiness, his son became alarmed and demanded that I do something. I suggested that there might be many causes for the decline and to find out if Taylor Paul had been exposed to colder than usual weather, any illness that was going around, or any unusual stress. The son asked Taylor Paul, but Taylor Paul couldn't think of anything that was unusual.

By the end of the next month, Taylor Paul was profoundly weak, dizzy, confused, and feverish. He was unable to care for himself, and he wasn't eating. Finally, at his son's insistence, he started taking Sinemet again. The MD suggested starting at 500 mg/day (five 25/100 pills), more than Taylor Paul had ever used in his life. After three weeks at this drug level, Taylor Paul was still worsening. Finally, after his daughter came to his house on New Year's eve and found him paralyzed, utterly unable to move, she called the ambulance.

At the hospital, they chided him for having stopped taking his Parkinson's drugs, even though he had been now taking 500 mg/day for three weeks and his condition had continued to worsen. They did a round of blood work, and the blood work confirmed that he was seriously ill – but not with Parkinson's. His white cell count was extremely high. He had a body-wide infection of unknown origin. They put him on intravenous antibiotics. After several days of this, they switched him to pill antibiotics, but they kept giving him the 500 mg/day of Sinemet. He was still in bed, unable to get himself in or out of bed or to walk more than a few steps, when he started having facial grimacing from the medication and feeling stoned and unfocused.

When he told his doctor that he was starting to have spasms from the medication and maybe the med level was too high, the doctor retorted that he obviously needed the drugs – after all, he could barely move.

Taylor Paul tried to point out that he was having symptoms of overmedication, that he had been moving well on only 25 mg/day of his meds a while before, and that possibly the reason that he was still too weak to move was the infection. His face was still gaunt and ashen from his long-term bacterial infection. He had some kidney damage, probably from the months of untreated illness. Though his body had been ravaged by an infectious disease, still, the doctor told him that his main problem had to be his incurable Parkinson's disease, and that the best treatment was Sinemet.

After Taylor Paul's white cell count dropped back down to a healthy level and he was able to walk a few steps, he was discharged to his daughter's house. At this point, because he was no longer so sick, his dopamine levels were starting to climb back up. The extreme levels of Sinemet were starting to accumulate (it had now been six weeks), and he suddenly, for the first time, had full limb dyskinesias. They were frighteningly severe – not only his face and arms but his throat went into spasms: he could not swallow, and for one terrifyingly long night, he feared he was going to choke to death. His doctor should have been curious about the fact that he was having dyskinesia even though he was still too weak to perform normal movements. He could not walk or even get up from a chair without support, and he could barely move his arms. His muscles had deteriorated during his illness. However, despite this immobility of conscious movement, his brain was employing dyskinetic movements both huge and small in response to the drugs. Paradoxically, he couldn't yet move, but his drugs were causing him to thrash about uncontrollably.

He decreased his medication the next day down to 400 mg (four 25/100 pills). He felt less confused by evening. After several days, he decreased further, down to 300/day. He felt better still, though he still felt overmedicated, his arm was still twitching, and his eyes had that characteristic Sinemet glow.

At this point, he was still extremely weak. The doctors had concluded that his infection had probably been a bladder infection that had gone into the kidneys. He had probably been fighting it for months before he collapsed. The pallor in his face was just beginning to ebb, and he was starting to feel as if he had been seriously ill, which he had, but that he was going to recover. He also felt deeply discouraged by the fact that he was going to have to go through drug reduction all over again. He feared that the five weeks at 500 mg/day had probably done him real damage, creating an addiction. He was correct.

His children, watching the whole thing closely, decided that the doctor had been wrong about the cause of immobility during the recent hospitalization being Parkinson's disease. They felt that his immobility had been caused by his infectious illness. They recalled that prior to the illness, Taylor Paul was moving fine on 25 mg of Sinemet. Also, more damning, the 500 mg/day of Sinemet, more than he had ever taken in his life, had not helped his ability to make conscious movement whatsoever, even after five weeks. Certainly, after he started the Sinemet, prior to going to hospital, he had continued going rapidly downhill. The thing that had reversed that downhill trend was the antibiotics.

However, his doctor had completely ignored the possibility of an infectious illness because of Taylor Paul's pre-existing diagnosis of Parkinson's disease. When Taylor Paul was dismissed from the hospital, the doctor insisted that he never wanted to see Taylor Paul playing around with his Parkinson's medications again, as if the medication reduction had been the cause of the illness.

We have seen this over and over. When a person who used to have Parkinson's disease has a new condition, whether it is stroke, infectious illness, grief from a profound loss, or anxiety from social stress, the doctor will tell them that the subsequent heaviness of body and spirit is simply a symptom of Parkinson's disease. The new illness will not be treated. The answer will be, every time, "Increase your antiparkinson's medications."

In Taylor Paul's case, I learned that there had in fact been the entire trio of depleting forces at work. He had started feeling lousy in November – he lived in Canada, where the late autumn nights grow very long and the weather can be abruptly cold as it

shifts into winter. Next, he had just decided to sell the family farm. His wife had died five years earlier, and his adult children were not interested in continuing the family farm. He himself had continued to play an active role in the farm up until they decided to sell. The farm had gone on the market in October, just before he got sick.

When I discussed it with him the following February, he insisted that he wasn't emotional over selling the place, that it was too much work, and that everything changes over time, but there was a set to his chin and a look in his eye that said more than his words.

There you have it: it was growing colder and darker, he was under emotional stress, and then illness struck. With his brain just barely able to make enough dopamine to keep him at the threshold, these three dopamine depleters used up so much dopamine that by New Year's Eve he was literally paralyzed.

Dangerous illness may be ignored

Again, the risk is not so much that a person may, for the rest of his life, be somewhat susceptible to these factors, and that his brain may never be restored to the dopamine excesses of his youth.

The risk is this: anyone who has ever taken the medication who subsequently succumbs to any of these other factors may never be taken seriously by his physician.

If he is no longer taking his medication, even if he has not used it in years, even if he was doing perfectly well without it, at the first sign of any trouble, his doctor may well blame the absence of PD drugs and refuse to look more closely into the matter.

A person who has never even taken medication is much safer. Even though he has the PD diagnosis on his chart, it is highly unlikely that a person who never needed so much as a sniff of PD medication would suddenly become paralyzed. Such a person might point out that he is early stage and doesn't yet need meds. The symptoms of a person with a history of medication who has stopped taking his drugs are not only going to be ascribed to the cessation of meds, but the person will be considered to be mentally incompetent – only a nut case would imagine that he no longer needs his PD drugs.

If Taylor Paul had not had blood work done when he entered the hospital, he might have died of kidney failure due to staph infection. His doctor would have erroneously signed the death certificate, "Cause of death – Parkinson's disease."

Therefore, due to the very real possibility of death, due to misdiagnosis of a life-threatening condition by a doctor who assumes the only problem is a need for PD drugs, we do not recommend a recovery program for PDers who are taking the medication.



THE DEEP SLEEP

The deep sleep phase of recovery is included in this section on dopamine depleters because the effects of this phase on friends, loved ones, and doctors can be the same as in the case with Taylor Paul.

As you may know from reading the *Patient's Handbook*, there comes a time in recovery when a person is absolutely unable to move. This profound inertia usually occurs between 7 and 9 in the morning, although it can occur at other times instead or as well.¹ A person who is undergoing this bizarre daily zombification may decide that he is getting much, much worse, even if he is able to get up and start moving normally every day at 9:15.

This deep stillness in the mornings occurs in nearly all recovering patients, unmedicated or medicated. This makes it hard for a medicated patient who has recently reduced his meds to know if he is having Off time in the morning because of the deep sleep or because the med levels are too low.

It was this stage of recovery that started Zoe on her rapid drug increases. The terror induced by this weird, two hour immobility every morning like clockwork caused her to triple her morning dose, even though I assured her that the deep sleep thing was normal, and even though the increased drugs did nothing to ease the paralysis-like condition. She balked at the prospect of surrendering to the sleep and turned to the drugs instead.

Other patients have also succumbed to the lure of the drugs when they began to have the hours of immobility.

Anyone who has taken the drugs will be terribly drawn to resume taking the medication if the weakness phase stretches into weeks, then months, and possibly even years. This phase is an unpredictable span that may partly depend on how advanced the PD was – a quality that is impossible to measure in a medicated patient.

Of course, when such a person does resume the drugs, “just for a few days,” he is most likely lost for good. Becky had been feeling just slightly less vigorous than she liked when she decided to start taking the drugs again, “just for the weekend.”

Therefore, because of the heightened risk of returning to drugs during the Deep Sleep or the Profound Weakness stage – the latter has been, for some patients, more painful and debilitating than Parkinson's – we do not recommend a recovery program for PDers who are taking the medication.



¹ The Asian understanding of this is that most repair work to the tissues related to the Stomach channel usually occurs between 7 and 9 in the morning. Each of the channels has a certain two-hour time zone during which the energy in this channel surges. If there is massive repair work to be done in this channel, the body may fall into a motionless paralysis during this time. Surgeons have long known that certain surgeries have better outcomes at specific times of the day. They have ignored this powerful information, for the most part, because it doesn't fit into their theory of anatomy. However, this awareness has been in the records of Asian medicine for over two thousand years.

TREMOR

Tremor is a symptom that merits a special paragraph of its own. Just as long-hidden pain can become exposed during recovery, so can long-suppressed fear, which the body manifests as trembling. When a person with tremor recovers from Parkinson's, his tremor may continue for months or years.

If a person took antiparkinson's drugs for very long, even if he did not have tremor to start with, he may very likely develop tardive dyskinesia, which has a similar appearance to tremor.

As long as there is trembling or tardive dyskinesia, even if all other Parkinson's symptoms are gone, a person who has once used drugs might be tempted to resume the drugs to "control the tremor." Considering that the tremor may actually be tardive dyskinesia, the drugs may worsen the condition. By the time it becomes apparent that the drug is not helping the ticcing but rather worsening it, the addiction will have begun, and with it, long term damage.

LINGERING ONs AND OFFs

Another difficulty for the person who is trying to decrease his medication is the appearance of On's and Off's. While some people find that their On's and Off's decrease when their medication gets down to a non-threatening level, other people find that they have On's and Off's right up until the time when they take their final pill. This may be a brain habit, but it may also have to do with altered thresholds and baselines.

As you have seen above in the section on dopamine depleters, even a person who never took the medication may have moments of "off" time during recovery, as they dance back and forth across the threshold. While these immobility events are very different from the drug-induced Off's and freezings, it can all become very confusing for a person who anticipated a predictable cycle of drug decreases.

Since unmedicated PDers do not have On-Off cycles, and the only PDers I have seen who have these distinctive, brief periods of off time following an exertion that depletes dopamine are those who are drug-free and have in every other way recovered, it should seem obvious that *dose-related* On-Offs are caused by the medication and are not a symptom of worsening Parkinson's.

If a person does begin to recover and is still taking the drugs, the addiction response and the increased severity of adverse effects may cause drug-induced symptoms such as On-Offs to increase, rather than decrease.

And yet, despite these arguments, those people who are still taking medication after exhibiting signs consistent with recovery who still have On-Offs usually use the On's and Off's to justify an increase in their medication or else refuse to make any further decreases until such time as the On-Offs have lessened. This maintenance of medication at levels that cause On-Offs or any other adverse effects may lead to worsening of those adverse effects and heightened addiction symptoms.

Therefore, we do not recommend a recovery program for PDers who are taking the medication.



SWITCHING

Many people have noticed that shortly after they take a pill, before the pill begins to work, they go through a brief period during which they feel much, much worse than they did before taking the pill. They may freeze or have dyskinesia or dystonia of such ferocity that they will scream out in agony. This phase is short-lived, and as the medication begins to make its presence felt more, these movements and the unpleasantness may come to an end, and the good On may begin. Then, when the drug begins to wear off, there may be another period during which severe freezing or terrible dystonia or dyskinesia once again occurs. When the benefits of the pill come to a complete halt and the basic Off begins, these symptoms of enhanced freezing, muscle tension, or dyskinesia ebb.

It can feel, in the period just before the pill really takes hold, and again just as the pill begins to wear off, as if the body is more traumatized, less able to move correctly, than it is during a full Off, a full On, or if there is no pill in effect at all.

We named this period the Switching phase because it occurred when the brain was switching from non-pill status to pill-affected status and vice versa. We have heard this phase also referred to as diphasic dyskinesia.

We have no explanation for this very difficult period which usually only lasts for two to twenty minutes. It seemed to many patients as if their brain was struggling with the medication, resisting it; every time the meds went On, they had to endure a struggle for a short while, until their brain's resistance was overcome by the drugs; once the drugged condition was established, there was no more fighting. Then, when the drugs were wearing off, the brain was inspired to take up the fight again, with the resultant difficult movement, either excessive or inadequate, and then, when the drugs were gone and the body was back to its more natural state, the fighting ended – until the next pill was taken.

Sometimes, as with the mouth burning switching that Rose experienced (as described in Chapter One), the switching would be very painful; when the switching ended and the full strength of the medication kicked in, you could almost see her eyes glaze over in relief. A smile spread across her face, and it was obvious that she had no awareness of pain anywhere. As the medication wore off, the mouth pain would resume, and then, when the medication was completely gone, such as when she woke up in the morning and was moving about on her own dopamine for half an hour to an hour, there would be no mouth pain or only a mild version. After she took a pill, as her own dopamine appeared to be turned off and the medication dopamine was just starting to take over, the searing pain would come again. Depending on the particular drug side effects that a person has, they may see that these side effects are at their very worst while the brain is making what appears to be a transition from native dopamine to the medication-induced dopamine.

Incompatible systems?

Observing people making the transition from their own dopamine, however inadequate, to the drug-induced form of dopamine, it really did seem as if the brain was having to make a switch, as if it was moving from one system to another. There appeared to be an incompatibility of the two systems, as if they could not work smoothly side by

side. Most people only noticed this switching phase when their medications levels got high enough to have distinct Ons and Offs.

People who are in the early years of taking these drugs, who are still getting continuous coverage from their medication, do not experience the switching. However, the people who have switching problems during the transitions from their natural dopamine, however inadequate, to the drug form of dopamine, might feel as if the brain is being buffeted between the two systems, creating the extremes of pain and erratic movement.

Unnatural dopamine?

The switching problem suggested that there are two different forms of dopamine or two dopamine systems. It appeared as if the dopamine or the dopamine distribution system that derives from the drugs is somehow different from the dopamine that is produced and distributed in the brain. We have no chemical or radiological evidence to support this, but it did seem almost as if the two dopamine types, native and pharmaceutical, did not work in an additive fashion, but rather in a combative fashion.

Glimmers provide an answer

Our theory of two different types of dopamine, or at least two different dopamine systems, was finally proved to be true: when recovering patients began having noticeable glimmers of native dopamine at levels high enough to breach the threshold to good motor function, after long, Parkinsonian years without it, they invariably noticed that the sensation of the natural dopamine was very different from the dopamine feeling that came from the medications.

This noticeable difference suggests that either the location, receptivity, chemical structure or some other aspect of native dopamine is different from the pill-provided dopamine.

We can only guess at why this is. It may well be that the switching occurs when the brain, flooded by the outside dopamine, shuts down the native dopamine factories, so as to protect against dopamine excess. During this time when the native dopamine is shut down and the pharmaceutical dopamine is not yet positioned, there may be the worsening of symptoms that is noticed during the switch.

Switching rigidity

This idea might explain why the Offs just before a pill starts to work can be much worse than the pre-pill condition. Even if a person with this pattern can move slowly before taking his first pill of the day, or in between pills, he may find that he has utter rigidity after taking each pill, before the pill begins to work. This shut down may be a learned, protective mechanism.

Switching dyskinesia

Other people have just the opposite response: while the switching from one system to the other is happening, they have very powerful and uncontrolled movements during the switching – their arms flail, their legs spasm, and their whole body tenses with painful cramping. We are guessing that those people who move too much are showing a brain pattern where both dopamine systems are functioning at the same time, creating an

excess, and that the brain is trying to rid itself of the excess through the frenzied movements.

In those people who become absolutely motionless or some degree thereof during a switching phase, it may be that the brain is choosing the “Shut Everything Down” method of dealing with the dopamine excess. In those who have violent spasms, their brain may be in a “Get Rid of It” program.

Switching with rigidity and dyskinesia

Some people will have both patterns, unpredictably. The first pill in the morning may be closely followed by a complete cessation of movement, lasting twenty minutes, after which the medication begins, and the slow, unmedicated movement of the early morning is replaced by the drug-induced movements. Then, when the pill begins to wear off, there may be a brief period of frantic movement, or there might be a period of freezing that was even worse than at the beginning of the pill’s onset. As the day wears on and the drugs build up, the switching may become more unpredictable with each dose.

People who have painful symptoms, such as the burning tongue and mouth, light-headedness, or hallucinations, may find that all of these side effects are at their very worst while the brain is switching from one system to another. During recovery these switching-related problems may become much, much worse. As the brain makes dopamine in greater amounts, if the person is still taking some amount of dopamine-enhancing drugs, all of the side effects might worsen.

However, if and when there is an end to the switching phase, bringing with it the blissful deliverance from switching symptoms, Parkinson’s symptoms, and the nasty reality of life itself, a person may feel just fine – until the drugs begin to wear off. Once a person has started having switching symptoms before and/or after his dose’s effective period, these On times might be the only moments of the day when a person feels half human.

One cannot assume that, as drug levels are decreased, the switching will also decrease. In our experience, just the opposite occurs.

Hua To and Laurel both have made steady progress with decreasing their medication, but, probably due to starting recovery symptoms before getting off the drugs, they both have much worse switching now than they did in the past at higher drug levels, but prior to the onset of recovery symptoms.

Hua To

Hua To is now down to .75 Permax, 75 mg levodopa, and Amantadine from a high several years ago of 4 mg Permax, 600 mg levodopa, and Amantadine. His switching worsens with every dose of the day. By evening, his last switching and crash from his 4 p.m. dose can be so severe that he cannot move for hours afterwards. Very often, his wife has to spoon his dinner into his mouth. Finally, by bedtime, the switch and crash are over, and he is able to slowly get into bed by himself. If he needs to use the bathroom at night, he can do it, very slowly, by himself. In the morning, he can usually get up and get dressed and have breakfast – all very slowly – before the morning pill heralds its onset with an attack of freezing, during which he cannot move. Because he must work to help support his family, aging in-laws, and a young child, he cannot stop

taking these seemingly small, “sub-therapeutic” bits of medication which both allow him to keep working and may be, inexorably, destroying his dopamine-producing cells.

Laurel

I recently received an email from Laurel. She has probably the worst pill-induced dystonia I have ever witnessed. (Her consultant – the British word for “medical specialist” – presented a research paper on drug-induced dystonias in 1999: see appendix 10.) When her drugs work, she may be perfectly pain free. While she is switching, a searing pain burns and twists her hip and leg. If she is in a Build Up phase so that her afternoon or evening pill does not work due to excess, the pill that fails sets in motion a ferocious version of this pain – pain that is not touched by even the most powerful anti-pain medications, including apomorphine. As you will read in this email, her consultant, though acknowledging that the pain is due to the drugs, is not suggesting that she decrease the drugs but that she begin taking a powerful anticonvulsant.

Laurel experiences that particularly cruel joke of nature, the phenomenon noted earlier of symptoms of excess and withdrawal being the same. When the brain wants to protest against a drug decrease, it can employ the same vicious spasms and pains that it learned when trying to use up excess dopamine. Both events, a drug deficiency and a drug excess, are seen by the brain as a horrible problem. When either deficiency or excess occurs, the brain may select dyskinesia or dystonia from its learned repertoire of Horrible Problem Solvers. It will then institute these excruciating – but from the brain’s perspective, effective – attention-getting methods.

In Laurel’s case, her most painful dystonias occur with the afternoon or evening dose, as the drugs build up over the course of the day. If her threshold has risen too quickly, she will not get any On time from her mid-afternoon pill but will spend the entire three hours of maximum pill coverage in a state of scream-level pain. This condition will begin with a feeling as if the pill is just about to come on, but it never makes it into the good On. It suspends in the pre-On condition for a while, and then goes straight into the Excess Zone agony and holds there for several hours. Usually, if this intense pain condition lasts for hours, her evening pill will work better than usual. This may be due to the dispersal of dopamine from the limbic area during times of extreme pain, which is serving to alter the baseline or threshold. We have no idea what is actually happening in this case, but it is not uncommon for one of her agonizing “pill failures” to be followed by a fairly successful pill.

When Laurel is Off, she cannot move well without assistance. When her pills work well, she has a few hours of time during which she is animated and active. She, like Becky, seems to have that rare overview which allows her to observe herself objectively during her Off times and times of extreme pain. She usually gets reasonable (several hours) coverage from her first and second pills of the day, a small amount or none from the third, and, depending on what happens with the third pill, some or no On time from the last pill. However, there is a great deal of instability, and her daily Ons and Offs are affected by all of the other factors of daily life. She has two teenage children and a wonderful husband who have been supportive of her in all of her trials and her decisions.

You will notice in the emails that she has very brief Vacations or none at all. She is addicted; she started having symptoms of recovery over a year ago, when she was still taking 900 mg/day levodopa. Her brain now responds very quickly to any decrease in the

meds. Also note, she uses the words “switch on” or “switch off” to mean the onset or ending of a pill’s effect. She does not use the word “switch” in the meaning that I have defined for this book. She uses the word “tablet” and “carer” where we in the USA would use the words “pill” and “caregiver.” I have added comments and explanations in brackets.

Here are Laurel’s most recent emails:

January 2003

Janice,

Re: my email before Christmas asking about SSRI drugs - in the end I decided against taking them but instead gritted my teeth and decided to try reducing again. To remind you, I have had several attempts to reduce from 350 mg to 325 mg [levodopa in a Madopar format]. (During September, October and November, I tried reducing but always ended up returning to 350 mg.) I wasn't getting any better on 350 mg so on Christmas Eve, I decided I must grit my teeth and reduce again. I just want to explain what happened to see if you can shed any light on it.

Pattern of medication: 7:00 – 100 mg; 11:00 – 75 mg; 15:00 – 75 mg; 19:00 – 75 mg

*For first 4 days reduced by 25 mg/day every other day [and after that every day], - painful and poor 'Ons.'

*5th day good switch Ons with some dyskinesia at 11:00 but shorter length of switch on at 15:00 and 19:00. Slept well - didn't wake up until 5:45 in morning and then fell asleep until 8:00. Unusual.

*6th day less pain and quick switch Ons at 8:00 and 12:00 (doses an hour later because I'd slept in). 16:00 dose didn't switch on until 19:00 and stayed on for one hour.

*Next 6 days less pain - switched on well apart from 15:00. Didn't switch on at 5:00 but felt 'almost on' - during these sessions I had dexterity in my hands but my feet felt icy cold and 'cut off' - my right foot felt worse than my left even though usually my left side is my most affected.

*Next day noticed some dyskinesia at beginning of dose plus loss of balance. Treatment from T. [acupuncturist] She held my right foot first and then left. 5:00 switched On but more pain again when Off. Good night's sleep.

* Next 3 days switched on each session and noticed feet weren't as cold when off but gradual increase in pain again.

* Another session from T. who felt Gall Bladder channel was clear and Stomach channel more responsive again but not clear.

Now a week after reduction have started having bad days again with extreme pain in my left hip and very poor mobility again. Unbearable.

Can you tell anything from this pattern? Why do my good days turn into such awful ones? Where do I go from here? Stay at 325 mg for awhile and hope things improve or reduce to 300 mg? I know it's impossible for you to advise but does the pattern described give you any clues or do you need more information?

Love, L

I wrote to her that the poor mobility and extreme pain were probably due to drug reduction and might be unavoidable. I reminded her to keep in mind that she had been having intermittent mobility and extreme pain now and then prior to the decrease as well as after. As for the “good days turning into bad,” I thought that might be because of medication building up, even though she had decreased her medication. The first four days, when she had poor Ons every time, were her vacation – she was sleeping well and actually feeling good, mixed in with symptoms of withdrawal – pain and poor Ons. After a person has become addicted, the theoretical separation of drug reduction phases can become a blur, with mingled symptoms of vacations and pills not working well. Also, keep in mind that sometimes people can go through the entire “ten-week” cycle of drug reduction in a matter of days.

Here is her next month’s report:

Feb 2003

Dear JJ

Reduced again on 17th January - from 325 mg to 300 mg. For the first 6 days every other day, then every day.

*Day 1: took longer to switch on but only 3:00 pm dose failed. Bad night 10:00 ‘til 2:00 then slept well until 8:15 - unusual - had also turned myself in the night. [This is new – she had not able to turn herself over in bed while Off.]

*Day 2-7: On for first two sessions of the day, Off 3:00 and variable at 7:00. (Pain when Off almost unbearable: severe cramping in left buttock and the same twisting I was experiencing when you came over. Impossible to move myself at all when Off.)

*Day 8: Reduced every day. Switched on for 1 hour after first dose. Hour and half after second dose and Off rest of day.

*Day 9: 1 hour On after first dose but only On 10 minutes from second dose and Off for rest of day.

*Day 10: 10 mins of On after first dose but then On after 11:00 dose, off 3:00 and On briefly at 7:00. The pain was getting worse as the day went on.

*Day 11: Didn't switch On all day. Horrible!

*Day 12: Not On first dose but then switched On briefly each session.

*Day 13: Pattern changed again: On after pills #1 and 2 but Off rest of day

*Day 14 & 15: On mid morning only.

*Day 16: Off all day.

*Today (day 17) Slightly better day hence I can email you. Life is very difficult at the moment with the pain being almost unbearable. One thing I have noticed is that although the pain is centred in my left buttock and T. has been holding my left foot, my skin on my *right* foot has become very dry and is peeling. Is this significant at all? Over the last few days, by the end of the day, my right knee becomes swollen and has a lot of heat in it. I can't imagine being able to cope if the pain persists at this level for much longer. Do people whose main symptom is rigidity suffer more pain and have you had patients that have come through it? The trouble is that the time On is getting shorter and shorter and when I am Off the pain is excruciating (sometimes it feels as if I've been stabbed in my buttock and I'm sitting on the knife!) and I can't move my lower body at all. The

pain also becomes so bad that I can't move my hands or hold myself upright when sitting. Have you seen people in as much pain as this? Can you reassure me that I can come out the other end? Does the pattern above give you any clues as to what is happening and can you give any guidance as to what to do next?

Possible explanation

In this email she describes many patterns that appear in the drug reduction cycle. The first pattern is her slide, which ended on day eleven. During the slide, her pills became more unpredictable, but she didn't actually become immobile until the eleventh day. During the slide, her first pills of the day sometimes worked, but not the later ones. This is her most common pattern – she was having it prior to her reduction – and it corresponds to a Build Up. She was still having this Build Up pattern even after making a reduction and feeling as if her drugs were not working as well.

In other words, even after making a small reduction she was still overmedicated: by afternoon her pills were “failing” (she was freezing, or Off). However, her addiction threshold had grown so high (due to taking drugs while recovering) that this very small reduction made her feel worse, in general – typical for drug withdrawal. To sum up, she was experiencing a small degree of withdrawal suffering, and still manifesting Build Ups by afternoon. Despite her raised movement threshold, the Safety Zone, the level of drugs that sets in motion adverse effects (including freezing, stabbing, painful dystonia, and pill failure) is a somewhat fixed amount, and does not go up proportionately when the threshold goes up. Her threshold was high; to surmount it, her pills invariably ended up in the Excess Zone, even though she was reducing and experiencing drug withdrawal. She was deficient by habit, and Excess by the numbers – both deficient and excess at the same time. This is not unusual in the early stage of drug reduction, while the brain is still slowly coming to equilibrium with the new, lowered dose. After the slide is done, none of the pills might work, but at least there may be no symptoms of excess – unless this person's body is one of those that exhibits the same sort of symptoms in times of severe dopamine insufficiency as it has learned to do during dopamine excess.

Also contributing to the Build Up, in Laurel's case, is this problem: she has started recovering, and she does have glimmers of native dopamine, especially in the earliest hours of the morning. This is why she is sleeping better, especially in the dawn hours. This extra dopamine may be contributing to her morning Ons, which, in the early days of this reduction cycle, are still good. However, once she has expended this bit of native dopamine – which accrued very slowly during the night – the other doses of the day do not work as well. Possibly, due to the glimmers of morning dopamine, her morning doses are excessive even though she has reduced her medication. If so, that excess dopamine could contribute to a rising threshold, which would prevent the subsequent doses, the afternoon and evening doses, from working. (This is the Build Up.)

This pattern continued, with ever-decreasing amounts of On time, for nearly ten days. By day eleven, the effects of the first reduction and the second reduction were clearly visible. At this point, she became immobile, and the drugs appear to not be working whatsoever. She was now in the darkest part of the drug decrease cycle.

On days 12 through 17, she is having only faint On times. Day 12 had a Deficit in the morning, but by afternoon she had built up her dopamine level so that she was On in the afternoon and evening. This meant that she had, overall, a good day, one that was

hovering on the edge of just right. This nicely balanced day allowed her to build up some native dopamine overnight, so that the next day she went On first thing in the morning; but the glimmer of dopamine combined with the pills pushed her threshold over the top, and her other pills that day did not work.

Bear in mind, the threshold may be going up and down every day, but the baseline is still dropping, due to the decrease in pills set in motion over the preceding weeks. (As the baseline drops, her susceptibility to pain will soar. The stripped limbic area cannot protect against incoming pain signals.)

As her baseline continued to drop (due to dose decrease), her pain increased. She was no longer having On times during the day; these Ons had afforded her mental protection against the pain. Now that she was having no recesses from the pain, it was becoming much more fierce.

She was in a downward spiral, where pain causes a panic or sense of emergency, which may lead in turn to dopamine decrease, and because a dopamine decrease allows more sensation to get through, the decrease causes more pain. This is the classic dilemma of the person in drug withdrawal: damned if you do take drugs, damned if you don't. The animal mind reminds you that if you do keep taking the drugs, you will have intermittent respites from the pain. If you don't take them, you will have the spiraling. Your logical mind, if accessible, may point out that if you do keep taking the drugs, the pain will increase on its own, over time, as the body uses spasms to get rid of the excess dopamine.

I wrote to her most of the above, and also a note that I had no idea what was going on with her right knee, but possibly there was an old injury there, and as her mind was less drug addled, and now that she had recovered to the point where she could feel and heal injuries, her body might be presenting her with yet another case of long-forgotten damage.

Mid-February 2003

I got the following from her in mid February, together with a copy of her consultant's research paper:

I don't know whether (his paper) has been published but you were correct – the paper was given at the Parkinson's Disease Society's research conference in Coventry in 1998. It is interesting because yesterday I went to see a new consultant in Liverpool (because I can no longer travel down to London) and she openly acknowledged that the long-term use of Levodopa caused the pain. She suggested that I have implants. Needless to say I am ignoring her. I read in a magazine that an herb *Macuna Puriens* can be used to treat Parkinson's and have found out that it contains natural dopamine. Do you know anything about it? I know I still need to reduce but would this lessen side effects in the mean time? Pain is still my biggest problem. Will be in touch later about my progress - going off!

March 2003

I thought about quitting (!), but decided I was too 'chicken' to stop taking my L-dopa completely so instead I cut out the first dose of the day to see how I would feel with no drugs in the morning.

*The first day went quite well - it was a day when T. was treating me at 9:15 in the morning. I was obviously off but she said my feet felt much better than when I was medicated. I switched on with my 11:00 dose and was On for one and a half hours. Next dose failed and I was in a lot of pain. 7 p.m. dose worked briefly then to pain again. I slept reasonably and was relatively comfortable when I woke up.

*Day 2 - I noticed that I could move more easily than when I had taken my tablet and it hadn't worked. My carer commented that I felt much lighter to move around and in fact I could just about walk to my chair when I got downstairs. At about 10:00 though I had increasing pain at the base of my spine and down into my feet. It felt different than the pulling/cramping/twisting pain that I get with the meds but it was equally unbearable. The 11:00 dose worked, 3:00 and 7:00 failed and by this time pain was excruciating and instead of easing overnight as it usually does it increased. I felt forced to take my morning dose because I couldn't bear the pain.

*Day 3 - Excruciating pain all day which only eased slightly when my 11:00 dose worked. Strangely though I slept much better last night and felt more comfortable in the morning.

*Day 4 (today) trying again to do without my morning dose but already by 1:45 my pain is at screaming level again.

Any ideas or comments? Is there any other information you need? My GP prescribed clonazepam for the cramping - I know you can't advise about drugs but do you know anything about this?

Look forward to hearing from you

xx L

Pain relief medication

In my response to this email I commiserated with regard to the horrible pain, and asked if she could possibly ask her doctor for a mild pain reliever. She had tried pain relievers in the past for her spasms and they hadn't worked, but this pain seemed to be of a different nature. I also pointed out that T., her carer, and her own observations showed that her body felt lighter and able to move better when she didn't take the morning pill. That should be of significance. I had to wonder if the horrible pain that she was now able to feel – a pain distinctly different from the spasming, twisting pain that she usually got when her drugs built up or failed – was one of the underlying pains that had first started the Parkinson's disease. It might also be sciatic pain, brought about from the years of twisting at the hip – her body's most common form of drug-induced dystonia.

I suggested that she work very aggressively at dealing with this new pain. I suggested that she try counseling, hypnotherapy, massage, Tui Na, acupuncture, craniosacral work, or anything and everything that felt right in her heart, that might make her confrontation with this pain move along most quickly. I felt that a small amount of pain-masking medication might be helpful if she could keep the pain at a level where it was present but not breathtaking.

I made the further point that taking Parkinson's medication to treat hip and leg pain was not appropriate. Parkinson's medications should be used to treat Parkinson's

and pain medication should be used to treat pain. In general, most pain medications seem to disturb the frontal lobe dopamine levels much less than do the antiparkinson's medications.

Meanwhile, pain aside, it was clear from her new experience of a "lightness of movement" that the rest of her body greatly preferred having less levodopa. She seemed to move better than she had moved in a long time when she had *less* drugs in her system – she was now taking less than she had ever taken since she had first been prescribed levodopa. I therefore included in my response that, although I could not make a recommendation, I could suggest that she listen to her body on that count, at any rate.

As for the antispasmodic recommended by her GP, I reminded her that this new pain did not seem to be a spasm.

Her remark that "I could move more easily than when I had taken my tablet and it hadn't worked" refers to the painful Roller Coaster failures and violent freezing which usually included painful switching symptoms before and after the mighty spasms that constituted "failures." As you can see, once there is addiction, along with the painful, drug-induced dystonias, glimmers, and switching, it can become very difficult to negotiate a drug reduction. Even when the body feels better and everyone notices that the body is lighter and more relaxed, the raw pain that the brain experiences when its shroud of dopamine is wrenched away can turn a person away from thoughts of drug decrease. During the hours, days, and weeks of immobility and pain that might occur during drug decrease, the tortured limbic brain, recalling radiant memories of angel-voiced pills, may call on the mind with fervent longing to please, please, just take one more pill.

It is unreasonable to expect a person who is only having a few pain-free hours every day to decrease or stop taking the medication that provides those fleeting interludes. Therefore, we do not recommend a recovery program for PDers who are taking the medication.

