

*“I keep six honest serving-men (They taught me all I knew); Their names are What and Why and When And How and Where and Who.”*

*The Elephant Child, by Rudyard Kipling*

## CHAPTER THIRTEEN

# DISSOCIATION QUESTIONS THAT ARISE

This chapter will consider some of the questions that often arise from a discussion of dissociation and the role of the mind in Parkinson’s disease.

### **Can a person actually cause physical changes by imagining his heart is gone?**

It was all well and good to *hypothesize* that a person can selectively dissociate from his own heart, and in so doing, create symptoms of automatic dissociation. But we wondered if this hypothesis could be confirmed. So, in experiments using PDers, recovered PDers, and people with no knowledge of Parkinson’s disease, we were able to prove that pretending to wall off the heart can inhibit sensory perception and reduce a person’s ability to engage the parasympathetic system. We learned that merely pretending to wall off the heart can cause a reduction in heart rate and breathing rate, and can inhibit the ability to feel the expansion in the chest that a person feels when he feels safe. In other words, pretending to utterly wall off the heart *does* activate modified symptoms of automatic dissociation.

Details of the various experiments that we ran are included in Part three of this book, “Related research xxx.”

### **Can one override the pain of physical injury with dopamine?**

In order to understand safe times and the placebo effect, it was necessary to hypothesize that a dopamine surge can override the movement inhibition that occurs following a severe injury.

It is easy to understand that adrenaline can override immobility. We can see examples of that in nature. But we also see examples of a dopamine override. Imagine a person with a very recent, very painful, badly sprained ankle, separated shoulder, and neck injury. Picture him limping about, feeling sorry for himself, and moving more slowly than normal. Now imagine how his movement ability changes when he is informed that he just won three millions dollars in the lottery. He can spin around, throw his arms in the air, talk a mile a minute and dance on his one good foot. This is a dopamine override. He is still injured, but the injury does not hurt, nor does it inhibit his ability to move at a normal pace.

Although a majority of PDers could relate to the idea of using adrenaline to override symptoms, a number of PDers had used dopamine-increasing methods. Some told us that, in the years before they were diagnosed, when they noticed that they were feeling stiff, clumsy, depressed or “just not right,” they would force themselves to sing songs or play a musical instrument. Even if they didn’t feel like making music, they had learned, over the years, that they could always banish the stiffening up or the feeling of wanting to curl up in a ball and stay there for a few years.

Some PDers used both: they forced themselves to enjoy music or other dopamine-releasing activities some of the time and created adrenaline-releasing “mental emergencies” at other times. Again, no two PDers are the same.

### *A chemical override*

A person who is immobilized with painful injury can move somewhat normally if he takes a wallop of dopamine-enhancing drugs such as methamphetamine or opiates. A person who is immobilized with Parkinson's can move somewhat normally if he takes dopamine-enhancing drugs such as methamphetamine, cocaine, or the pharmaceutical dopamine-enhancing drugs.

A huge surge of dopamine can override the immobility of pain, injury, fear, or automatic dissociation. But the surge of dopamine doesn't get rid of the underlying problem; when the surge wears off, the immobility resumes.

### *A safe activity override: how it works*

As noted in the beginning of this chapter, some PDers can initiate movement perfectly normally even if they have an unhealed foot injury, *if* they are engaged in an activity that they have subconsciously decided is safe. This phenomenon is well known and documented in many PD research reports. In these cases, the rare surge of dopamine that occurs from feeling safe is able to override the brain-induced immobility from injury and the physical damage and pain at the injury site. This proves 1) PDers do not have a dopamine deficiency, but in most circumstance they cannot *release* dopamine, and 2) feeling safe is what allows for the release of dopamine.

Of course, during these safe activities, the safe feeling automatically displaces the "not safe" feeling of heart inhibition that causes automatic dissociation. When a person feels safe, not only can he *chemically* override the brain-induced immobility from injury and the physical damage and pain at the injury site via dopamine release, he can also override his selective dissociation from his heart. When he overrides the selective dissociation from his heart, his automatic dissociation temporarily ceases, as well.

Of course, as soon as the safe activity ends or the mind is diverted back to the "reality" of some negative thought, the dopamine ceases to flow, the heart dissociation resumes, the symptoms of automatic dissociation resume, and injury-induced movement inhibition resumes.

*Maintaining* a "safe mindset" that allows for constant dopamine release is almost impossible if a person has a serious injury that needs to be addressed. As soon as a drug-induced or safe activity-induced dopamine surge is over, the dopamine inhibition held in place by the unhealed injury will once again come to the fore. Just because the mind had a temporary surge of dopamine is no reason to expect the injury to heal or the mindset to change. During a safe activity, the foot injury does not heal, nor does the mind re-associate with the foot injury. Again: as soon as the safe activity is over, the symptoms of Parkinson's resume.

But the larger point is this: when PDers are doing their "safe" activities, they never run out of dopamine: they have *plenty* of dopamine. They only stop being able to move easily only when their *minds* remind them that they aren't safe, after all.

### ***What about placebo research?***

In the same way, placebo research also proves that most PDers, even those with advanced Parkinson's disease, *do* actually have sufficient levels of dopamine to move perfectly normally. Research findings about the placebo effect on PDers make this glaringly clear. A collection of placebo studies conducted using people with Parkinson's is presented in chapter xxx. Following

administration of a placebo, PDers very often can move normally for a while. They feel safe because they think that something positive is going to happen.

PDers' hours, days, and even a *year* in the case of one placebo study, of "normal" movement, when under the influence of placebos or while doing safe activities, *prove* that a PDer's problem is not caused by dopamine insufficiency. The movement inhibition of Parkinson's is *not* a problem of "not enough dopamine." The real problem is the inhibition of dopamine *release*. When that inhibition is lifted, dopamine flows freely.

### **Can we prove that dopamine cells are merely dormant?**

We have hypothesized that dopamine-producing cells will emerge from their dormancy and resume their role as dopamine-producers if they are correctly stimulated. This assumption comes from the evidence of recovering PDers.

Yes, all PDers can release dopamine in sufficient quantities when they feel safe. However, some of our patients who recovered from Parkinson's did have, over time, a demonstrable *increase* in their amount of dopamine production and reserves.

Some patients, in the early stages of recovery, *if* they did a *significantly* greater amount of activity than usual, would actually *run out* of dopamine. The sensation was bizarre: their brains were calling for a release of dopamine, they were in parasympathetic mode, and yet, they had no ability to do anything, even think. They felt as if they were sleeping while awake, as if someone had turned off their power switch. The experience usually began with a feeling as if the body was becoming extremely heavy, weighing a thousand pounds. Within a few seconds, they would be immobilized.

We named this experience "a crash." These crashes were exactly like the OFF periods that PDers have when their antiparkinson's medications wear off *except* that the nearly-recovered PDers felt absolutely *relaxed* and calm when they became heavy and utterly motionless: as if their bodies were sleeping while the mind was awake. These crashes never lasted more than ten to twenty minutes – the time it takes for the brain to re-circulate dopamine back into dopamine-storage vesicles for re-use. After about ten to twenty minutes of sitting, motionless, on a bus stop bench or some convenient sitting place, feeling about the density of lead, or at least cement, the recovering PDer would suddenly feel a warmth and lightness pervade his body as if he was waking up. Instantly, he would be able to resume healthy, effortless movement.

Just like with the Parkinson's drugs, there was no "slow, medium, and fast" movement," as if dopamine supplies were running low or high. It was an all or nothing situation. Either there was enough dopamine to allow for perfectly normal, effortless movement, or there wasn't. If there wasn't, the PDer could not move, period (unless he ramped up the adrenaline).

The following example will explain what is meant in the above by *significantly* greater amount of activity: if, while recovering, the PDer customarily took a vigorous one-hour walk every day and then came home and rested for a bit, he would not crash. But if, one day, feeling extra good, he went on a vigorous *two*-hour walk, he might experience a crash partway through the second hour.

People who did have crashes usually had them in a predictable manner. For example, if the crash happened after an hour and a half of walking, it might happen after an hour and a half of walking every day for many days in a row. But after a few or many crashes at this exact level of work out, the crashes would cease: the recovering PDer could then walk vigorously for his

two full hours. If, after this, the recovering PDer added yet another hour onto his walk, he might have a crash during the third hour. This might happen once, or maybe twice, but he would even more quickly develop enough reserves to get through hour number three without crashing.

It seemed as if these PDers were developing greater dopamine-reserves. In the early stage of recovery, it almost seemed as if they developed larger stores of dopamine on an as-needed basis. But at some point, there were no more crashes, ever. At this point, it seemed as if dopamine-producing cells must have been continuing to develop *whether or not* they were compelled by crashes.

Within a matter of months after full recovery, recovered PDers stopped having crashes: their dopamine reserve levels had increased to provide *whatever* level of dopamine was needed.

These crashes only occurred in a few of the recovered PDers. *Most* of our recovered PDers never experienced *any* crashes. As soon as they were using dopamine – and the feeling of movement with dopamine was unmistakably different from their PD style of movement, which used adrenaline – their brains were able to provide as much dopamine as was called for.

But the PDers who had crashes when depleting their dopamine reserves were more exciting. They showed us that dopamine reserves *could* be increased. This increase suggested an increase in the number of dopamine-producing cells, or at least an increase in the amount of dopamine that was being produced.

### ***Use it or lose it***

Because we saw that increased use of dopamine caused increased dopamine levels, we are in a strong position to argue the reverse: failure to use dopamine will lead to decrease in dopamine production. No pathology needs to be presumed. No germ, gene, or environmental toxin needs to be found. In otherwise healthy people who develop idiopathic Parkinson's disease, the amount of dopamine production, like everything else in the body, is determined on a use-it-or-lose-it basis.

The increase in dopamine reserves, as evidenced by the decreasing number of crashes during recovery helped convince us that PDers did *have* dopamine production capability but they just didn't use it. The placebo studies and the evidence of safe activities just added that much more proof to our hypotheses. The dead dopamine-cell theory cannot make sense of any of the well-known dopamine-override events such as placebo-induced dopamine release and safe activity dopamine release.

But a hypothesis that dopamine release is being mentally *inhibited*, but can be overridden with a change in mental attitude, allows us to make sense out of the evidence.

### **Is dopamine inhibited because of injury or because of dissociation?**

*All* the PDers in our experience had an unhealed injury causing backwards Qi flow, so they *did have* the mid-brain dopamine inhibition that is supposed to occur with a severe injury. They could not make full use of their substantia-area dopamine producing cells, and very possibly had not have been using them for *decades*.

Also, *many* of the PDers, because they had dissociated from their ability to feel physical and/or emotional pain and were therefore manifesting symptoms of automatic dissociation, *did have* mid-brain dopamine inhibition. They could not make full use of their substantia-area dopamine-producing cells, and very possibly had not been using them for decades.

Whichever the cause, they had not been making regular use of their dopamine reserves.

The body is efficient and gets rid of things that aren't used or needed: in PDers, it seemed as if this lack of use resulted in mid-brain dopamine-producing cells becoming re-undifferentiated. Researchers report evidence of that in the autopsy studies.

When certain brain cells are not used, they revert back to a type of non-specialized cell that resembles an embryonic cell.<sup>1</sup> These non-specialized cells may contain some residue of their dismantled dopamine-building structures and other residues that are *also* sometimes seen in sick or non-functional cells, such as lewy bodies. But these midbrain re-undifferentiated cells are not sick or dead: they just aren't being used.<sup>2</sup>

## WRAPPING UP THE CHAPTERS ON DISSOCIATION

First, there is a time and a place for healthy inhibition of dopamine. Dopamine inhibition is a naturally occurring process. People with Parkinson's have gotten *stuck* in an electrical and/or neurological mode that inhibits dopamine. The electrical inhibition is set in motion by an injury. The neurological inhibition is set in motion by selective dissociation. In order to recover, PDers need to recover from their injury and they need to stop dissociating.

Next, and I will repeat this over and over and over in this book, not *everyone* with Parkinson's disease has selective dissociation from his heart or dissociation from feeling physical or emotional pain. Some PDers recovered from Parkinson's easily, as soon as their foot injuries healed. However, a majority of our PD patients manifested signs of mood- or situation-based parkinsonism after their foot injuries were gone, even after they had experienced many of the unpredicted, counterintuitive, and even bizarre symptoms of physical (as opposed to mental and emotional) recovery.

These *partially-recovered* PDers were unable to visualize light in their bodies, unable to visualize themselves having a functional heart, and unable to imagine themselves moving. They usually did not understand the meaning of the phrase "feel safe." They often thought that "feel

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<sup>1</sup> The substantia is supposed to be full of dark-colored cells. I read a research report back in 2002 that stated that the light-colored brain cells in the substantia of people with Parkinson's had characteristics more like embryonic cells and less like differentiated cells. The cells were not dead. They had just become more "immature," less specialized. At the time, it merely confirmed what we'd already started to suspect. I was heartened by it, but didn't think to save it so that I could cite it. At the time, I assumed that much more research would occur in this field, and I could snag as many citations as I needed when the time came. However, a cursory search today on the Internet did not bring anything up. I do wish now that I had put a copy of this particular bit of research into my files. But even without this research that described these cells as reverting to a more embryonic type of cell, evidence abounds as to the fact that the brain cells in the striatum of people with idiopathic Parkinson's disease are *not* dead. The few people who have drug- or toxin-induced parkinsonism have dead brain cells. But the vast majority of people with symptoms of parkinsonism – the people with idiopathic Parkinson's disease – *do not* have dead brain cells.

<sup>2</sup> Researchers of Alzheimer's disease were disappointed recently when their new drug, which successfully prevents plaque formation in the brain, failed to make any improvement in the mental degeneration of people with Alzheimer's. Because research had shown plaque on the brain cells of people with Alzheimer's, it was fallaciously assumed that the plaque was the *cause* of Alzheimer's. The researchers assumed that, if they got rid of the plaque, the Alzheimer's would go away. But it turned out that the plaque was just cellular debris. The plaque was a side-effect of Alzheimers; Alzheimer's is not a side effect of plaque formation.

Many PD researchers have made the same mistake. They find a reduction in dopamine producing cells, or they find cellular debris where cells have reverted back to immaturity and jettisoned their "mature" dopamine-producing structures, creating lewy bodies and bits of cellular jetsam. But this debris is a side effect, it is not a cause.

safe” meant “be wary:” the exact opposite meaning. Some thought that “safety” meant “silence” or even “lifelessness.” They were unable to feel sensations of vibration and expansion in the chest in response to emotional events or beautiful or glorious sensory events. They often recalled having decided, decades earlier, that they were not going to feel physical or emotional pain.

Some *partially-recovered* PDers *were* able to fully recover. But in order to recover, they needed to be willing to risk feelings of physical and emotional pain. They needed to recognize sympathetic (fear-based) mindsets, including excessively cynical, judgmental (of themselves and others), or overly self-conscious attitudes, and needed to learn how to turn them off.

Many partially-recovered PDers needed to learn what was *meant* by the phrase, “feeling safe.” They also needed to learn how to create the safe feeling in the chest so that they could re-associate with the pain sensations that they’d put on hold for decades. They needed to learn the correct, mature techniques for addressing physical and emotional pain so that their old pains, when they re-associated, could be transmuted into non-threatening sensations and processed by the *conscious* mind.

Strangely enough, very often, as soon as a PDer learned to feel safe, usually via gratitude and surrendering control of his life to a higher power, all the other learning, attitude changing, and pain processing happened *automatically*. Sometimes, it seemed as if their attitudes and dissociations were just temporary poses, and the real personality was just waiting for the opportunity to feel safe so that it could re-emerge.

Then again, some PDers just needed to heal their foot injuries. Or if the foot was technically unhealable, they just needed to restore healthy energy flow through the foot and heal their attitude towards their feet.

