

# PART III

## RELATED RESEARCH

INCLUDING

THE DISCOVERIES AND A CHRONOLOGY OF THE RECOVERY PROJECT



“The mind can make a heaven out of hell or a hell out of heaven.”

- John Milton

## CHAPTER TWENTY-FIVE

# PLACEBOS AND PARKINSON'S DISEASE

### *The placebo effect*

A placebo is an inert or neutral substance or event that makes a person feel better. The western understanding of this phenomenon is that a placebo works via the power of suggestion.<sup>1</sup>

The placebo effect has been researched in many highly respected, rigorous, double-blind, scientifically conducted studies.

Placebos do seem to work for some types of illnesses, and do not work in others. The most recent research suggests that the determining factor in whether or not a placebo will work in a particular illness is this: whether or not dopamine plays a role in the illness.<sup>2</sup> If, due to a placebo treatment, a person *anticipates* that he will feel better, dopamine is released. The release of dopamine is the trigger that causes beneficial changes in the person's condition.

Placebos work very well in people with Parkinson's disease.

### *Negative-placebo effect*

Oppositely, fear or the expectation of trouble can behave like a “negative placebo.” In the case of a negative placebo, the expectation of *problems* can set in motion actual physiological changes, such as increasing the body's tilt towards the sympathetic system (and adrenaline) and the simultaneous tilt *away* from the parasympathetic (and dopamine). In other words, negative placebos can inhibit dopamine release. This sympathetic nervous response, if severe enough, can even set in motion instantaneous development of Parkinson's-like symptoms, even in people who do not have idiopathic Parkinson's disease.<sup>3</sup>

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<sup>1</sup> The word “placebo” comes from Latin and means “I shall please.” The word placebo these days usually refers to the medical use of dummy (sugar) pills or pretend treatments that make the person feel better even though the pills or treatments have no (known) healing mechanism. Placebos are sometimes used in medical trials when testing new drugs to determine if the benefit of the drug is coming from a physiological interaction or is merely coming from psychological suggestion.

<sup>2</sup> Illnesses such as insomnia, pain disorders, allergies, depression, digestive disorders, and even susceptibility to illness are immediately affected by dopamine levels. Illnesses such as broken bones and cancer are *not* immediately affected by dopamine levels. Then again, the *pain* associated with a broken bone or cancer is a dopamine-related problem.

<sup>3</sup> If the symptoms of fear or stress are severe enough to produce symptoms that actually resemble Parkinson's disease *and* the condition lasts for a significant period of time, the condition is referred to as “psychogenic parkinsonism.” But symptoms that resemble those of Parkinson's can also arise in a matter of minutes, and linger for a short period of time, such as an hour or so. I'll give a few familiar examples:

Consider a person with the beginnings of hypothermia (extreme chill) or the mind-altering stage of a severe flu during the alternating-fevers-and-chills phase. The shifts in body language brought on by these events can occur relatively quickly. What do they have in common with parkinsonism?

A person who is on the verge of severe hypothermia will be bent forward, his arms bent at the elbow and held close to the body, and his head pulled forward. His face may be nearly expressionless, his stride will be small, maybe even shuffling. His teeth may chatter and he may be trembling in his limbs. His speech will be very slow,

As for people who *do* have idiopathic Parkinson's disease, a negative placebo, an expectation of worsening problems, can cause a rapid acceleration of the symptoms of Parkinson's disease. This effect can even occur in a person who has physically recovered from Parkinson's *if* he imagines that he still has Parkinson's. His tremor and movement initiation symptoms may remain or might rapidly worsen even if his body structures are obviously healing.

In our experience, most unmedicated PDers *are* highly susceptible to negative placebos.

## Examples of PDer placebo studies

### *Moving with sugar water*

In a placebo study conducted in the early 2000s, placebo researchers injected PDers with sugar water. These were PDers who were accustomed to taking L-dopa based drugs and whose condition had advanced to the point where they were having On-Off behaviors in response to the medication.<sup>1</sup> Due to familiarity with drug effects, these people already knew how the drugs would make them feel: safe enough to move.<sup>2</sup> Thus, they had developed a specific expectation for drug-enhanced movement in response to their usual dose.

These PDers, while in an Off (rigid) phase, were told, falsely, that the sugar water was a dissolved form of L-dopa, and that the study merely wanted to see if the solution worked faster or at the same rate as the pill form of the drugs.

What happened after the PDers drank the sugar water? They uniformly responded to the placebo by experiencing ease of movement initiation in the exact same timing and behavior as if they had been given their usual oral dose of L-dopa. I repeat: they *thought* they had been given L-dopa, so they were able to move in the manner that their drugs usually allowed, even though

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and at low volume. It may be extremely difficult for him to perform any movements that require him to open out his body and stretch languorously. He may have trouble initiating *any* movement.

In the case of hypothermia, availability of dopamine appears to drop dramatically – even to the point where a person can be immobilized. This condition can come on very quickly.

Based on extremely rapid changes in condition in our PD patients following a severe chill or overheating, a change that can last for several days even if the chill is quickly remedied, we hypothesize that dopamine is heavily drawn on, rapidly depleted, in conditions requiring a severe temperature regulation effort.

Just as Parkinson's-like symptoms can be brought on very quickly in a person who is accidentally locked into an industrial freezer, these same symptoms can quickly be brought on by intense anticipation of pain or fear *if* there is not a concomitant increase in adrenaline release.

These examples are induced by physical factors. The same symptoms can occur rapidly, almost instantaneously, in response to devastating emotional news.

These examples are meant to show that manifestations of poverty of movement, rigidity, and tremor can come on quickly, if the physical or mental state calls for them.

<sup>1</sup> The On-Off behaviors that occur from addiction to antiparkinson's medications are discussed at great length in my book *Medications of Parkinson's or Once Upon a Pill*, available for free download at [www.pdrecovery.org](http://www.pdrecovery.org).

<sup>2</sup> I need to mention this: many PDers vigorously insist that dopamine-enhancing antiparkinson's drugs do *not* make them feel happy. Nevertheless, increasing the joy signals in the brain *is* the mechanism by which the drugs work. Dopamine is the neurotransmitter of pleasure and joy. When the motor area feels joyful enough, a person can execute uninhibited movement. Whether or not the conscious mind of the fear-oriented PDer is able or willing to cognize joy may determine if the drugged PDer can feel an overt mood lift from the dopamine. Typically, by the time the drug use and the Parkinson's is advanced, even the PDers who deny feeling "good" from their medications state that they feel "bad" when the medications wear off.

they had actually only been given sugar water. Based on what we now know from PET scan studies, this result occurred because the PDers' brains released dopamine in response to an expectation of feeling good.

The placebo effect in this study lasted just as long as each PDer's typical duration of benefit from a dose of his usual drug.<sup>1</sup>

The same type of study using sugar pills obtained the same type of results: PDers, when they think, mistakenly, that they have been given their medication, respond as if they had actually taken the medication.

Many variations on the above PD placebo study have been performed with similar results. Research abounds in this field. And nearly every study I have read that examines the placebo effect in a generalized way makes mention of the way in which PDers in particular respond to placebos.

Another different type of Parkinson's disease placebo study, described below, concluded that, possibly, the more dramatic the placebo action, the longer-lasting the placebo effect.

### ***Placebo holes in the head***

In the April, 2004, issue of *Archives of General Psychiatry*, an extreme placebo effect was described: 39 people with advanced Parkinson's had holes drilled in their heads. Half of the subjects had embryonic brain cells transplanted into their brains, the other half had "sham surgery:" holes, but no embryonic tissues. Neither the patients nor their regular doctors knew who had gotten the tissue transplants and who had the placebos (sham surgeries). Thirty of the patients agreed to participate in a long-term follow-up study. These people were asked whether or not they thought they had received the transplants.

"Those who thought they received the transplant at 12 months [after surgery] reported better quality of life than those who thought they received the sham surgery, regardless of which surgery they actually received," according to the write-up. It continued, "Some of the placebo patients made striking improvements. One patient said she had not been physically active for several years before surgery. After surgery, she was able to hike and ice skate. She eventually learned that she'd had sham surgery."<sup>2</sup>

As an aside, I have to wonder if the shock to the body caused by drilling holes in the head and brain was sufficient to amp up the sagging adrenaline levels a bit; after all, these people were able to sustain the placebo effects for such an extended period.

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<sup>1</sup> I have lost the citation for this study. A quick look at the Internet, trying to find it again, brought up a reference to a similar study done by the University of British Columbia in Vancouver. This study used placebo "injections of a harmless saline solution" (salt water). PDers were told that they were receiving L-dopa (a dopamine precursor). Using PET scans that measure dopamine receptor activity in the brain, researchers were able to see a boost in dopamine activity levels in the PDers' brains – a boost to the same levels as produced by "the [dopamine-enhancing] drug commonly used to treat the disease." From "Science File: Healing Body by Fakery," *Times* staff writer, Robert Lee Hotz, Feb 18, 2002.

<sup>2</sup> Danial DeNoon, reviewed by Michael Smith, MD, in "Strong Placebo, Strong Parkinson's Effect," *WebMD Medical News*, April 14, 2004.

Maybe the adrenaline response to having a hole drilled in the head, combined with the dopamine-releasing positive-expectation (placebo) effect, might account for a placebo benefit that was longer-lasting than expected.

Also, the psychological benefit from *imagining* oneself to be one of the lucky ones, one of the ones who got the actual transplant (even though, in fact, they might *not* have gotten the transplant) may actually work to open the heart of the PDer just a bit – just enough to turn down the sympathetic nervous system and thereby be able to trigger neurotransmitter release.

### ***A brain-implant placebo study***

One of my favorite PD placebo studies, done in 2005, involved PDers who had received deep-brain stimulating implants.<sup>1</sup> I like this one because, in addition to obvious (visible to the naked eye) changes in movement initiation and cessation of tremor, the brain scans showed clear proof of inner brain electrical changes: these changes corresponded, not to the treatments, but to expectations induced by spoken words.

The deep-brain stimulating implants work by distracting the little electrical anxiety signals in the brain that contribute to immobility and tremor. The implants perform this distraction via the method of sending a much larger, more focused electrical shock signal into the brain. The brain is thus able to shift from an attitude of “uh oh” to one of “*Omigosh!!!*”

The adrenaline-boosting shift from the implants enables the late-stage PDer, whose heart to brain communication has become so reduced that he can no longer raise his adrenaline levels up to Functional Level anymore, to suddenly rise to the occasion of this new, significant alarm: electrical jolts into the brain! The sympathetic nervous system is stimulated; adrenaline levels get an upward nudge. The lower-level anxiety static in the brain gets overridden by the wire-and-battery induced stimulation which provides a sense of electrical trauma in the brain. Thus, the PDer once again has the release of sufficient mental and motor neurotransmitter (adrenaline): he can move easily.<sup>2</sup>

Getting back to the implant/placebo study: PDers had the implants inserted, *but* the battery pack that activates the inserts was not yet turned on. Then, to study the effect of placebo on these patients, half of them were told, falsely, that their implants *had* been turned on and that they *should* experience rapid improvement in their symptoms. They were also told that their

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<sup>1</sup> L. Neergaard, “Expectations Can Help Healing,” AP, *Yahoo News*, Mon, Nov. 28, 2005.

<sup>2</sup> Although some uninformed clinical neurologists and other MDs who are out of the loop do imagine that the deep-brain stimulation (DBS) must somehow work by stimulating dopamine, the manufacturers of the product and researchers in the field know that dopamine increase has *never* resulted from DBS. The 1960 to 1995 preoccupation with dopamine in regard to Parkinson’s disease, and the subsequent assumption by the uninformed that “implants, if they work, must necessarily increase dopamine,” is outdated thinking.

The people who are actually doing current research in Parkinson’s know that the implants work by delivering a mild shock. The shock most likely encourages the release of adrenaline; it certainly does not cause the release of dopamine. The research that led to the approval of brain implants was done on lab animals in which parkinsonism had been induced. The stimulation from the implants allowed the lab animals to move, but their brains did not show an increase in dopamine.

In humans, unlike in the lab animals, the implants may have two mechanisms. They stimulate adrenaline, thus overriding the electrical disarray in the brain, but the surgery itself may also provide an *expectation* of feeling better. An expectation of feeling better makes a person feel safe, cared for. Thus, in some PDers, some amount of expectation-based dopamine release may be psychologically activated, especially immediately following the surgery.

tremors would stop almost immediately. These PDers rapidly began to experience normal movement initiation. Their tremoring instantly stopped. Brain scans of these PDers showed that the large number of tremor-inducing electrical firings in the “firestorm” area of the brain had calmed down.

Again, the PDers in this group were falsely told that their implants had been turned on. Their brain scans showed a calming effect. Their movement became easy and their tremors stopped *even though the implants had not been turned on*.

The other half of this group of PDers in the study was told, truthfully, that *their* implants had *not* yet been turned on. They were also told that they *should not* expect any immediate improvement until the implants were turned on later in the week. Subsequently, this latter group did not have any immediate improvement in tremor or movement, nor did their brain scans show any improvement; these people were waiting for the implants to be turned on so that they could begin to feel better. After the implants were turned on *and* the subjects were informed of the fact, this group could then also move easily. Only *after they were told* that they *should* feel better did their brains scans show a calming of the “firestorm” area.<sup>1</sup>

But getting back to the main point, this placebo study, like all the others, provides further support for the idea that PDers’ movement-initiation problems and their tremors are expectation dependant: movement becomes easy, and measurable amounts (measured with PET scans) of dopamine are released if the PDer *expects* that he will be able to move easily.

### **Using people with Parkinson’s disease in placebo research**

A person who follows the research on placebos might notice the frequency with which Parkinson’s disease is used in placebo research. There are two reasons for this. The first is, when looking for a placebo-induced change in behavior, the difference between “it works” and “it doesn’t work” is, in the case of Parkinson’s, visible from across the room. The PDers used in these studies nearly always are medicated. Very often, they are in the “on-off” stage of advanced Parkinson’s disease: they can move when their drugs kick in and they cannot move very well when the drugs wear off. This visible shift in movement when “On” or “Off” makes these people good subjects for placebo studies.

When doing scientific studies, the researcher finds it easier to work with subjects in whom the results are easily seen, glaringly obvious. If measuring the results of a study requires extracting molecules and measuring ever-changing brain waves, the results will be difficult

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<sup>1</sup> I suspect this study was originally inspired by the fact that many PDers, including three in my experience, could move more easily immediately following the surgery, *prior* to the implants being turned on. Since the implants aren’t “supposed” to work this way – and do not work this way in lab animals – I imagine that a curious researcher decided to do this quick, easy, and very telling experiment. After all, if patients received benefit from simply having the implants positioned, what need is there for the battery pack and the electric stimulation?

I suspect that the adrenaline surge brought on by having holes drilled in the head and wires placed inside the brain is the source of the immediate, “pre-turn on” improvement. This adrenaline increase from surgery may be accompanied, in PDers, by an increase in dopamine due to expectation of improvement.

In light of the fact that many PDers do have an instantaneous improvement, the study group that was told to *not* expect any benefit until the wires were turned on was also being given a placebo – a negative placebo: they felt no benefit because they had been specifically told that they would not have a benefit. As for whether one group or both groups were influenced by what they were told is clear; all subjects had brain scans and motor function that corresponded to their expectations based on what they had been told.

and/or expensive to read. People with advanced Parkinson's, on the other hand, are very easy to "read:" either they can move or they cannot. For this reason, PDers are frequently used in placebo studies.

The other reason that PDers are so often used in placebo research is that PDers are highly susceptible to suggestion. The daily, even hourly variability of PDer's movement ability is increasingly recognized (among placebo researchers and even by observant PDers and their loved ones) as being highly mood and/or expectation dependent. Placebos are a method for stimulating the positive mood and expectation.

### ***The physiological basis of the placebo response***

Researchers used to think that PDers responded to placebo through some purely psychological influence. New research shows that the reason for PDers' response to placebo is physiological: placebos trigger the release of ample dopamine – even in people with Parkinson's.

Also, since the late 1990s, researchers have used PET scans to measure dopamine-receptor activity in the brain.<sup>1</sup> In PET scan research studies that measure the activity of dopamine receptors in PDers, the PDers who have received a verbal or physical placebo show an increase in dopamine receptor activity – the result of an increase in one's ability to use the dopamine system.

Again, though I risk redundancy, this increase in dopamine receptor activity occurs when PDers think, mistakenly, that they have been given some treatment that increases dopamine levels.

When a PDer is given sugar pills or sugar water and told that the pills or water contain his usual dose of dopamine-enhancing medication, he can feel the onset of easy, dopamine-style movement in the time frame that he expects from his medication. When the time comes when he expects his medication to wear off, he starts to move slowly again.

*PDers who are given placebos can easily initiate movement. Under the influence of the placebo, they move and feel confident in exactly the same way that they move and feel when they take their antiparkinson's drugs. This effect lasts until the time arrives when they expect their placebo to wear off. The expectation of the placebo wearing off is usually based on the PDer's experience with his medications' wear-off timing.*

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<sup>1</sup> PET scans measure activity of dopamine receptors. In the PET scan process, radioactive dopamine-like chemicals are injected into the body. These radioactive molecules migrate into the brain and attach to dopamine receptors. If a significant number of dopamine receptors are become dormant through the decades (as they have in a person with idiopathic Parkinson's disease), the typical (healthy) number of radioactive molecules will not be able to attach. In PDers, the scan *may* show a dopamine receptor response that is lower than that of a healthy person.

In people who have rapid-onset symptoms of parkinsonism, as occurs in hypothermia or trauma, the dopamine receptors are still healthy. Therefore, the PET scan may show normal receptor activity with the dopamine-like radioactive tracers even though dopamine is not being released by the patient.

This is the theory, at any rate. However, as noted elsewhere, the medical jury is still out on whether or not these scans are an accurate diagnostic tool for Parkinson's disease.

SPECT scans are a new variation on PET scans. SPECT scans are currently used only for research, and are not available for the general public.

These movement-observation and brain-scan placebo studies make it clear that dopamine *levels* in people with idiopathic Parkinson's are sufficient. The problem in Parkinson's disease is insufficient dopamine *release*, and not dopamine insufficiency, per se.

The expectation-dependant symptoms of Parkinson's, movement initiation and, sometimes, tremor, are due to an *inability to create the mental state* necessary to trigger dopamine *release*. As placebo studies make clear, these symptoms of Parkinson's are *not* due to *insufficiency* of dopamine.

### ***The PD symptoms that do not respond to placebos***

In response to placebos, PDer's *channel*-related symptoms are not improved. The PD symptoms mentioned in chapter seven that are located on the Stomach channel, symptoms such as hardening of anteriolateral leg muscles and numbness in the toes, will not be altered in response to a placebo. These problems are due to injury and electrical distortion, not inhibition of dopamine release. A PDer who has lost his sense of taste and whose foot and "smile" muscles are physically distorted will still have no sense of taste and will still have his foot and facial distortion problems even if he has a good movement-initiation response to drugs or placebo.

Only the PDer's movement initiation, speed-of-movement problems, and in some cases, the relatively more severe, fear-based portion of his array of tremor problems – the problems that are dopamine related – will respond to the placebo.

### **Negative placebos**

In the brain implant study, the people who had no benefit from the deep-brain stimulating implant until they were told that they would be victims of a negative suggestion, a negative placebo. They were told that they should not feel better, and so they did not – even though many PDer's do have an immediate result from the implant process, even before the battery is turned on. Possibly this placebo benefit occurs in people whose doctors forget to tell them that the battery will not be turned on for a few days after the surgery.

In the placebo study, by *telling* the PDer's in advance that they would feel no benefit from the surgery until the batteries were turned on, the researchers were using a "negative placebo," a negative suggestion.

Almost all PDer's, even those who have never taken medication, respond strongly to negative placebos, to suggestions that the PDer should feel worse. The following two case studies will look at the role of negative expectation in the rapid worsening of Parkinson's disease symptoms in two PDer's in our recovery program.

### ***A mind game example: self-induced parkinsonism***

For the first case, I will quote directly from an email that we received. Extra information that I add to help the reader will be in brackets [ ].

"Dear Chris and or JJ,

"You may remember me. I've made three weeklong trips from Colorado to see you guys in the last 3 years. A few months after I saw you last I began manifesting...the half-healed state. The tremor intensified dramatically and I became profoundly fatigued and weak.

"This went on month after month and I became one of those people who panicked and went to a neurologist who put me on a very low dose of Sinemet [L-dopa]. I had a very violent

[excessive movement] reaction after the first dose and I vowed to never take it again. The fact that I would take it at all gives you some idea of how defeated I was.

“As luck or fate would have it, the new edition of your book became available for download the day after I took the Sinemet. I immersed myself in your writings (again) and gradually began to get some hope and energy back.

[The older edition did not have the material on the adrenaline-dopamine relationship, or information about fear and negative thinking, so there was much new material for this reader.]

“Eight months after the fatigue started, it began to lift until, by July 1 [2005], I was back to 95%. Particularly as the later chapters on “Fear,” “Negative Thinking,” and “Mind Games” became available and I integrated their ideas into my daily practice, my symptoms markedly improved. Progress was happening and the future was opening up again. Then your addendum of October 2005 was released.

[This addendum to our website was a warning: based on our findings, people who had taken dopamine-enhancing drugs (L-dopa, dopamine agonists, or MAO inhibitors) prior to entering our program had developed difficulties (symptoms that corresponded with those of drug-induced parkinsonism) that seemed to, in most cases, prevent full recovery. Worse yet, their recovery usually stalled in highly traumatic manifestations.

Because people who have even partly recovered are usually not able to tolerate the medications any longer, the people who were part-way recovered but who were suffering from symptoms of drug-induced parkinsonism were in a very difficult position. Therefore, with heavy heart, we made the decision that we would no longer include in our research project people whose experiences with these drugs had, on top of their idiopathic Parkinson’s, also set in motion drug-induced parkinsonism (semi-permanent brain damage). The writer is referring to this disheartening warning, a warning about these specific types of drugs, which was dated October 2005. The writer continues:]

“My intention is not to shoot the messenger, instead it’s to let you know how personally devastating [this addendum’s] effect was on me...Its effect has been to send me into a tailspin of negative thinking, hopelessness and depression with the *resultant worsening of symptoms*. [Italics are mine.] So the reason for this letter is to get clarity and guidance. I was on Artane for almost 2 years from 2000 to 2002 but never took a full dose...what are my options? Please let me know. – SP”

I will paraphrase our reply:

“Dear SP,

“Your tailspin is an example of the role of expectation on dopamine release and mood regulation. The problems with brain damage, as described in our October addendum, are set in motion – as we clearly noted – by the dopamine-enhancing drugs. The drug that you took, Artane, is not a dopamine-enhancing drug. Artane is an anticholinergic. Anticholinergics are a completely different family of drug. The book *Medications of Parkinson’s* explains very clearly that Artane is not a dopamine-enhancing drug.

The drug that you took is a mild muscle sedative. It is not a brain stimulant. It does not cause brain damage. Therefore, your symptomatic tailspin, which occurred because you thought you would not be able to recover – despite your own positive changes – was entirely the product of your imagination. We have seen *no* problems, no impediments to recovery, in those PDers who took Artane.

“It sounds like you’ve really experienced the influence of positive and negative thinking. Your improvement back up to 95% really happened. There is no need for you to listen to anyone or anything that tries to tell you that what you have done/are doing is impossible when you have clearly seen for yourself that you can do it. Yes, you can do it. You did do it.

“You might choose to view your recent tailspin objectively (having already fully experienced it subjectively) simply as the example of what happens when one does the opposite of what you had been doing throughout those several months of amelioration and improvement that you recently enjoyed. You might also choose to note that the difference between darkness or light is often as simple as the flick of a switch or a drug warning that, as it turns out, didn’t actually apply to your case.”

SP wrote back to us:

“Your words had the effect of loosening the choke-hold Devil Doubt (aka the fear-based mind) had on me. What had happened the last few months [SP’s gradual improvement] was undeniable and yet the frightened little guy in me was particularly vulnerable to [the addendum]...

“Thank you again. – SP”

### ***Another example of attitude-induced parkinsonism***

I received a detailed email from The Netherlands. It was several pages long, so I will paraphrase it:

“My young husband (early 40’s) was diagnosed in June, 2005, with early Parkinson’s disease. His symptoms were still very mild at that time. I am Chinese, an acupuncturist, and so we were looking for alternatives to the drugs offered by the doctor.

“However, by mid-September, four months after his diagnosis, he was no longer able to pick up our two young children, and had difficulty dressing himself or performing other activities of daily living. Although I gave him some herbs that – interestingly, in retrospect – support the Stomach channel, his Parkinson’s symptoms did not improve.<sup>1</sup> We were alarmed at his very fast rate of decline and were considering going back to the neurologist.

“Near the end of September, I discovered your website. I then contacted some people in Amsterdam who have been in your program. They were very helpful and positive about their experiences with your program. So I downloaded your book [the first 20 chapters] and gave it to my husband. He stayed up all night reading it; he finished it in just two days.

“Since he finished your book a few weeks ago, his condition has completely reverted to what it was back in June: very mild. We have not yet started doing any treatments, but he is now in approximately the same physical condition that he was in when he went to see the neurologist four months ago: he now has, once again, very mild symptoms of early Parkinson’s disease – symptoms that do *not* impede his ability to perform activities of daily living.”

In case the reader is wondering why the man improved without being treated, the most likely explanation is that his rapidly worsening symptoms were due to his *expectations* of rigidity and immobility. When he found that his illness was *not* incurable, the mood shift allowed him to revert to his previous condition, in which he was once again manifesting his actual, physical

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<sup>1</sup> Herbs that amplify Stomach channel Qi will not reverse Parkinson’s disease. The rules for treating Rebellious Qi are very straightforward, and include this warning: *never* tonify (strengthen) a condition of Rebellious Qi.

symptoms (which happened to be quite mild), without the extra onus of the psychological weight of his diagnosis.

### **The power of the diagnosis**

The above case study makes a very powerful point: the “curse” of being diagnosed with an incurable illness can accelerate the problems of the illness, especially when the illness has to do with mood and expectation-dependent neurotransmitters.<sup>1</sup>

As noted in previous chapters, I have seen many PDers who told me that their symptoms went into a tailspin when they received their diagnosis. On the other hand, I have treated many people who never suspected that their shuffling feet, slight tremor, cogwheeling wrists and ankles, expressionless face, non-swinging arms and postural stoop signified anything other than a passing “muscular thing.” These undiagnosed people all recovered easily and completely.

I have also had PDers (who had been told by a neurologist that they had PD) undeniably recover *but* then go into a terrible tailspin, complete with tremor and rigidity lasting for a few days, after tripping over an unexpected sleeping cat or some other darned thing. They’ve wailed at me something like: “I almost fell down! The Parkinson’s must have returned!” No amount of reassurance that *everyone* stumbles once in a while can shift their certainty.

It can then take a few days or a week before these “fallen” people accidentally perform some unexpectedly agile motor activities. These activities suggest to them that they have, once again, “recovered,” after which the recent PD-like symptoms evaporate.

This ends the section on placebo *research* in PDers. The next section in this chapter discusses psychogenic parkinsonism. In a sense, this form of parkinsonisms is similar to that generated by a negative placebo, inasmuch as it does not have a physiological basis, but is induced by the mind.

### **Psychogenic parkinsonism**

In the words of Michael S. Okun, MD, in [Askthedoctor@forum.parkinson.org](mailto:Askthedoctor@forum.parkinson.org), Dec 27, 2005: “These are folks who have slowness of movement and a lot of features that look like PD, but have another reason for the symptoms...this reason may be psychogenic – stress, anxiety, depression, affective disorder, rape, trauma, other event...or unknown... and if caught early they can be treated to complete resolution in many cases. They are not as rare as one may think!”

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<sup>1</sup> Back when I was still in medical school, we were told that, in China, it was against the law to tell a patient that he had cancer. A patient, if informed that he had cancer, might easily die within two weeks because of the powerful grip that the word “cancer” has on the Chinese consciousness.

On the other hand, if a person in China with incurable cancer was only told that he merely had a “deficiency” and that strong tonics might help, he would usually take the tonics (and pain-reducing medications, if necessary) and go on to comfortably live, in some cases, several more years (sometimes even ten years) before the cancer actually moved into a quick-moving lethal phase. This political issue, in which a patient’s “right to know” may conflict with the doctor’s knowledge that the patient will be better off if he doesn’t know, can be argued either way. I won’t even begin to go into it here, but you can see the ramifications that it has for the subject of Parkinson’s disease.

I know that these cases are not rare. I have seen some. Roma rapidly developed psychogenic parkinsonism. Roma's case was mentioned briefly in an earlier chapter: she was the patient who didn't tremor while eating desserts because "desserts don't matter."

### ***Roma, or "Desserts don't matter!"***

Roma came to see me after her neurologist diagnosed her with Parkinson's disease and her Ayurvedic doctor confirmed the diagnosis.

Roma walked gingerly, carefully, into my office and greeted me with a rapid vocal patter. Her posture was hunched and she held her arms tightly by her side. But after sitting down on the couch in my office, her spine relaxed, her left shoulder relaxed, and she leaned back easily into a comfortable position. She effortlessly crossed one leg easily over the other, and, while talking, gestured quickly with her hands to emphasize her points. Her very faint tremor came and went in her right hand. After observing her for several minutes, I said, "You don't have Parkinson's disease. What's going on?"

Roma was stunned and asked why I was so certain. I then had her perform a series of exercises including the cogwheeling tests, the balance tests, the "reach upwards and take a deep breath test," and so on. She had no Parkinson's-like responses to any of these tests. What she did have was a right arm that was stiff due to tightness at the shoulder and a right hand that tremored a little, now and then. Also, though she took small, slow, careful steps, carried her arms in a bent position and was slightly hunched over when *walking*, her body language always relaxed into a position of ease as soon as she sat down. Sitting, she gestured quickly with her arms and hands and moved her head, neck, and torso easily.

The Qi in her legs was working just fine, so I ended up working on her stiff arm, but my parting words to her were that I didn't think she had Parkinson's.

She called me a week later. She had revisited her Ayurvedic doctor and told him what I'd said. He checked her out thoroughly and then said he was chagrined that he had "missed" the diagnosis. He agreed that she certainly did not have Parkinson's.

So what did she have?

Roma came to see me again and I grilled her with lots of questions. Here were some of the answers.

Her tremor was the worst when she was eating. While this by itself is not unusual for a person with Parkinson's, note this: she never tremored when she was eating desserts.

It turns out, Roma had a PhD in nutrition. She was proud to have studied with one of the top nutritionists of the century. To her mind, food "mattered." Food mattered a lot. I came to suspect, later on, that she had put such faith in nutrition that she imagined "correct eating," whatever that is, might be able to prevent cancer, slow aging, keep the bones strong, etc.

So that filled in one part of the puzzle. She tremored when she ate nutritious food because she was anxious about nutrition. Since dessert "didn't matter," she didn't tremor during dessert.

But why had she started trembling to begin with, and why, when walking, did she take small steps and carry herself in that semi-rigid, PD-like hunched posture?

I asked her when the symptoms had first appeared. She said she wasn't sure, but she would ask her daughter.

### ***Roma's dread of osteoporosis***

The next time I saw Roma, she gave me the answer to the puzzle.

Her daughter had told her, “I know exactly when you starting walking in that weird way and trembling. It was the day the doctor told you that you had osteoporosis. You called me and said that you had osteoporosis, and when I came over to see you, you were moving all hunched over all of a sudden. You’ve moved that way ever since.”

I asked Roma what she had to say about that.

“Of course, since I have osteoporosis, I’m careful now when I walk. I have to worry about falling down. I need to be extra careful, take small slow steps, so that I don’t break any bones. I’m not trying to look like I have Parkinson’s, I just need to walk this way to be sure that I don’t lose my balance and break something.”

In fact, this was not true. Roma was incapable of walking with large steps or swinging her arms while walking. She wasn’t just “being careful.” When she walked, she was terrified of falling, and she moved as if she was nearly immobilized with fear. She took tiny, shuffling, PD-like steps.

I asked Roma how she had felt when the doctor told her that she had osteoporosis. She said that she remembered exactly. “I felt decrepit. That’s the word that flashed in my head. Decrepit. ‘You’re decrepit,’ I told myself.”

I asked Roma what it meant to her to be decrepit. She told me that it meant that she was disgusting. She was a failure. She hated being decrepit, it was the worst thing in the world.

I realized now why Roma was walking the way she did: she was deathly afraid of “decrepit.” I could also hypothesize about her horribly mixed feelings about eating. All of her nutritional studies had let her down. Her careful eating had not prevented her from becoming decrepit, the worst thing in the world. Possibly, subconsciously, she decided that she needed to be more vigilant about getting enough nutrition. Meals may have become, to some part of her mind, her only hope. Thus, the enormous emotional strain she felt when eating. Of course, desserts didn’t count; desserts, she assured me, were just for fun.

Roma’s symptoms all began on the day she was told that she had osteoporosis. One day she was fine, the next day she had full-blown rigidity and poverty of movement, and a small tremor – if she was walking. She had psychogenic parkinsonism.

Three years after she met me, Roma recalled the time she first consciously dissociated from her feelings; when she was nine years old she was sent off to summer camp because her mother was sick. When she returned from summer camp, her mother was dead and buried. She understood that she was not supposed to ask questions or “make a fuss.”

Her relatively recent diagnosis of osteoporosis was, for Roma, another horrible thing from which she needed to dissociate. Her body was decrepit: she dissociated from her body. She rapidly developed psychogenic parkinsonism. She did not have any of the classic physical changes of Parkinson’s disease, the symptoms that might show up in a photograph. She only had those symptoms that related to movement initiation and tremor: the mind/emotion related symptoms.

Over the next two years, she developed painful rigidities and movement initiation problems. Her movement problems became almost constant.

Not until I understood the role of the dissociation response in psychogenic parkinsonism was I able to help Roma in any way. I did not work on Roma’s feet. I taught her how to turn off the dissociation response. After she *temporarily* mastered it, she was once again, in her words, “flying high.” But she knew that when she left our program in Santa Cruz and went back to her home on the east coast, her symptoms would return and worsen. And they did.

The case of Roma is a powerful demonstration of how negative feelings and negative expectations can rapidly create symptoms that resemble Parkinson's disease, complete with tremor, rigidity and slowness.

Interestingly, after three years, Roma had a PET scan done which showed a decrease in dopamine receptor activity that was similar to the patterns seen in people with idiopathic Parkinson's disease. As mentioned earlier, doctors have seen that PET scans do not necessarily confirm Parkinson's disease. Many people who are diagnosed with PD have PET scans that are perfectly normal. Roma's case was clearly psychogenic: she who went into a rapid emotional decline after being told that she had Parkinson's; as mentioned in an earlier chapter, she developed highly specific PD symptoms in response to my specific suggestions; and recalled shutting off her heart when her mother died. These are all symptoms that suggest an utterly psychogenic cause for her symptoms. And Roma had a PET scan consistent that showed inhibition of dopamine receptors.

Wrapping up this chapter on placebos, I wish to emphasize one point. Placebos do not allow PDer's to move simply because PDer's imagine that they are able to move. The placebo studies that measure actual brain neurochemistry have found that PDer's have actual shifts in brain chemistry – an increase in dopamine release – in response to placebos. In response to positive placebos, PDer's release dopamine. The dopamine change is measurable.

In other words, PDer's do have sufficient dopamine to operate their mental and motor systems. What PDer's also have is an inability to *release* dopamine. Again, the decline, over decades, of the dopamine-producing cells in the midbrain, is due to the lack of *use* of the dopamine-producing cells. However, even with the decline in dopamine-cell numbers, the remaining cells are *absolutely able* to produce as much dopamine as is needed.

The brain, extremely plastic, increases development of those brain areas that are highly used. The brain can also render dormant (reverting even to an undifferentiated state) those cells that are not called on.

The next chapter will expand on this concept of inhibited dopamine *release*. Dopamine release is utterly, completely dependent on expectation and feeling safe. This concept is crucial.

A PDer must appreciate that his emotional posture and his ability to have positive feelings about his own body and mind are the determining factors in whether or not he can initiate movement or move easily. If the PDer does not understand this, then, following the healing of his foot injury, he may passively wait for his mobility to miraculously return. He may imagine that merely fixing his foot injury should allow him to move normally once again. But he will be wrong; fixing the foot merely allows the brain to be *capable* of releasing dopamine at whatever levels the heart calls for. The PDer is still responsible for instructing his heart to feel safe, and to interpret the sensory experience of having a body as positive instead of threatening.

If, however, his brain processes have become habituated to negative attitude or if he remains in the mental state induced by the conscious cultivation of the dissociation response, he will not be able to feel safe. He will not be able to activate the release of dopamine in the midbrain. Though he once again has the *capability* to release dopamine, he will remain locked in dopamine deficiency and dopamine deficient behaviors. Although his face may be more symmetrical, his circulation improved and his injury healed, he may still have difficulty with

movement initiation, slowness of movement, and tremor: he may look like a person who's been accidentally locked in an industrial freezer for ten minutes.

