

CHAPTER THIRTY-TWO

IMAGINATION AND DISSOCIATION

After a year of partially-recovered PDers experiencing only short-term benefits from “connecting the heart and brain,” *if* they could even imagine the heart at all, I grudgingly admitted that this exercise and the dozens of variations that we tried might never give anything but temporary benefit. Partially-recovered PDers seemed to have extreme difficulty in *maintaining* a sustained visual relationship with their hearts.

And worse, as they failed to fully recover, they increasingly manifested, in both frequency and severity, mood-related symptoms of Parkinson’s, which also happened to be symptoms that resembled dissociation: rigidity; difficulty in initiating movement; tremor.

Their constant *physiological* symptoms of idiopathic Parkinson’s might be waning or gone, but in partially-recovered PDers, their susceptibility to symptoms that came and went according to thoughts, moods, or environment – symptoms that looked similar to automatic dissociation – were getting worse.

Symptoms that come and go

Often, the partially-recovered PDers reported, during the weekly session, that they had “forgotten” to have Parkinson’s several times during the previous week. During these times, they “accidentally” had moved perfectly normally. When I asked if they might try to “forget” more often, I was often surprised by the anger with which my suggestion was received. People who, in their own words, “accidentally forgot” to have Parkinson’s once in a while were adamant that these were just mental lapses into a false condition: their TRUE condition was Parkinson’s. It was *impossible* for them to see it from the opposite side: that their periods of dissociation symptoms might be temporary, “false” conditions brought on by some mental incongruity and that their underlying TRUE conditions might verge on good health.

Similarly, during the actual treatment sessions, PD symptoms might come and go. During our treatment sessions, patients could sometimes induce these periods of normalcy. For example, when they were able to *imagine* themselves moving, or in some cases, just imagining themselves as having a heart, they were sometimes able to move perfectly normally. When they could imagine a connected, working heart and imagine themselves moving, they were often stunned at the effortlessness with which they could move arms and legs.

But the imagination and the image of the heart never lasted very long. It was as if their hearts ceased to exist as soon as they stopped focusing on them. When I encouraged them to continue, outside of the session, to visualize themselves moving, they tended to be of the opinion that moving via imagination was a false, short-term construct and they were absolutely not interested in maintaining it. Even though many of them could move *perfectly normally* if they let their limbs move in time with their own imagined images of their limbs moving, they had no interest in practicing this “false” style of movement. And they were not interested whatsoever in the *fact* that normal, correct movement *is* mentally preceded by imagined movements.¹

¹ In healthy people, visualization of motor actions is a crucial part of healthy, dopamine-based, parasympathetic system movement. Although almost no clinical (working with patients) neurologists seem to be aware that the *real* problem behind the movement initiation problems of Parkinson’s is related to PDers’ poor ability to imagine movement, brain scans confirm this finding. In the article “A Dissociation Between Real and Simulated Movements in Parkinson’s Disease,” Frak V, et al, *Neuroreport*, 2004, June 28;15(9):1489-92, a research team

As they kept getting worse, and their symptoms kept turning our minds to what we knew about dissociation, we considered some non-motor aspects of automatic dissociation. In particular, we zoomed in on the heart changes that occur.

Dissociation rule #1: the heart shall be as if dead

A key rule for dissociation, a “prepare to die” condition, seems to be this: *all* aspects of heart function should shut down as much as possible. Heart rate *and* the heart’s role in feeling resonance with sensory experience are *both* minimized while a person is in this condition. When the heart function is minimized, the brain’s ability to freely imagine, including imagining feelings – including imagining the sensations and appearance of movement – is inhibited.¹

reported, “In individuals with idiopathic bilateral Parkinson’s disease motor imagery is impaired and execution of overt movements is spared.” What this means in basic English is that, in PDers, the ability to imagine motor function is impaired: has diminished activity. Activity in this area occurs when dopamine levels in the brain are high enough. Dopamine is a neurotransmitter that is released when a person feels safe or feels glad to be alive. The brain area that controls the ability to actually execute movement (actually move) is *not* impaired: it can work perfectly well *if* it gets electrical signals from the imagery area. The reason that easy, normal movement fails to occur in a PDer is that the PDer did not *imagine* it occurring.

In “Motor Imagery in Parkinson’s Disease: a PET Study,” Cunnington, R, *Movement Disorder*, 2001 Sept;16(5):849-57, the researchers used PET scans to discover the PDers do *not* have any damage to their Supplementary Motor Area (SMA) [the brain area that helps fine-tune motor function], but have inhibition of pre-SMA function [movement imagining function]. In this test, PDers were asked to imagine certain finger movements. The PET scans of the brains suggested that they had difficulty *imagining* repeated finger movements on the more affected side. When imagining the less affected side, the PET scans were able to show that the SMA *proper* had correct function. Again, this means that PDers have healthy function in the brain areas that execute movement. They have deficits in the brain’s imagination areas: the areas that precede the activation of movement.

In the above study, the researchers took PET scans of the SMA (Supplementary Motor Area) portion of PDers’ brains while the PDers tried to imagine moving a finger back and forth at the rate of 1 Hz. The finger used was on the side of their body that was more highly affected with Parkinson’s. In PDers, the SMA area of the brain showed *no* signs of damage. However, the pre-SMA function of PDers is inhibited. In other words, the PDers were not able to *access* the SMA area of the brain. The brain area regulating movement works fine, but PDers cannot conjure up the mental imagery that is necessary to activate the movement execution area.

Another article with the same gist is “Motor Imagery in Normal Subjects and in Asymmetrical Parkinson’s disease: a PET Study,” Thobois S, et al, *Neurology*, 2000 Oct 10;55(7): 996-1002. This article was published in the year 2000 and the introduction to the article states, “Previous work in PD has shown that bradykinesia (slowness of movement) is associated with slowness of motor imagery.” So, even though most clinical neurologists in 2008 have no idea that the imagination is the hang up in PDers’ inability to initiate movement, this is *not* a recent finding.

This article’s research used PET scans to measure activity in the portion of the brain that is activated via imagination of movement. The subjects imagined moving a joystick. The conclusion of article states, “in patients with PD...brain activation during motor imagery is abnormal.

Another study, published in 2007, shows that *extra* areas in the brain are employed when PDers are confronted with a motor imagery task. These extra areas seem to be attempting to increase intellectual processing of visual input, to compensate for the PDer’s inability to visually imagine movement. The study used fMRIs while subjects tried to mentally rotate their own hands in response to an image of a rotating hand. The study concluded, “we infer that, in strongly lateralized PD patients, motor imagery of the most-affected hand exploits additional resources in extrastriate visual areas. These findings characterize the cerebral bases of the increased dependence on visual information processing during the generation of motor plans in PD, pointing to its compensatory role.” Cerebral Compensation During Motor Imagery in Parkinson’s Disease,” Helmich RC et al, *Neurophchologia*, 2007, Jun 11;45(10):2201-15. The findings of this study, in basic English, are that PDers use analytical, problem-solving visual areas of the brain to try to compensate for their inability to use the visual imagination area.

¹ The non-PD reader will recognize that visualization is a *feeling*: it is an operation of the visual *sense*. The inability to imagine the tactile, proprioceptive sensations of movement and the inability to imagine visuals are both forms of the sensory feeling inhibition that is characteristic of partially-recovered PDers.

A person does not have unrestricted access to imagination when he is dissociated. His thoughts must necessarily be negative, and his imaginations are limited to *problems* and analytical problem *solving*. While in this condition, he *cannot* imagine hoped-for events with truly positive outcomes.

Therefore, if dissociation had, over the years, become the dominant pattern in a PDer's brain, any attempts at positive visualization, including imagining having a good looking, hooked up heart, might be difficult or short term.

Oppositely, during the "accidental" moments when the PDer was moving freely, he could evidently *imagine* freely. He was, therefore, not dissociated at these times.

But evidently, even if a PDer managed to temporarily rid himself of the dissociation, he usually soon slid back into dissociation. Very often, he slid back into rigidity or tremor in response mentally noticing that he was doing well – as if doing well was some sort of risk. As soon as he dissociated, he could once again only move in the PD manner: trying to *mentally overpower* his own fear-based compulsion to making him rigid.

Come-and-go movement ability suggested come-and-go dissociation ability. But how could that be?

Movin' easily when it's safe

Partially-recovered PDers tended to "accidentally" move easily or "forget" to have Parkinson's during one or several narrowly defined situations that, through some quirk, they had *subconsciously* deemed safe. Anything that wasn't "safe" had subconsciously been labeled "not safe." There was no neutral ground.

I've already mentioned examples of safe activities and safe times. Some more examples of these "safe times" show the range and seeming arbitrariness of individuals' "safe" conditions: watching an opera; dealing with public safety issues (as opposed to personal safety issues);

Telling research on this subject is included in *The Boy Who Was Raised As A Dog And Other Stories From A Child Psychiatrist's Notebook*, by Bruce Perry. He recounts how a young girl who taught herself to dissociate during her once-a-week rapes by her step-father found that she could go numb by dissociating and going "outside" of her body to an imaginary place. However, she was unable to create the visual mental fantasies that she *wanted* during her dissociation episodes.

During dissociation, her imagination's color choices were limited, as were her options for events that took place in her imagination. She could exercise some degree of control over the events that transpired in the imaginary land that she went to during the rapes, but she could not imagine *good* scenarios or *happy* outcomes. In her dissociation imagination, she could only imagine dark images consistent with being in sympathetic (danger) mode: dangerous, ugly situations that she could resolve using force and power. Her dissociative imagination was severely limited in other ways, as well. For example, during her dissociative events, she wanted to imagine herself as a bluebird but beautiful blue colors were not available to her mind. She could only be a black bird: a powerful, frightening black bird.

If asked, she might have said that she *was* able to imagine. I would argue that she had a limited ability to imagine, and that *full* reign over her imagination feelings was not possible while she was dissociating: in the dissociated condition, she was numb to positive outcome or beauty – just like most of the partially-recovered PDers would be numb to what it might feel like if they recovered.

Many PDers told me that they intellectually or even intuitively *believed* that they could recover. However, they could only *imagine* their PD symptoms worsening. They were *unable to imagine* that they might be recovering. And when they "accidentally forgot" about Parkinson's and moved normally, they could not imagine that this had been a good sign – after they re-dissociated. At the time of easy movement, they might feel certain that they were getting better. But as soon as the good phase passed, they then imagined, every time, that the brief period of ease of movement had certainly been the very last time that they would ever move easily – no matter how many times it happened.

washing dishes; performing in theatricals; making love; after eight forty-five p.m.; and so on. I have already sprinkled other examples throughout this book. And what was safe for one PDer was not safe for another. Some PDers were *more* anxious than usual during the evening hours or while attempting to make love, or while performing. The safety zone was a seemingly random, highly personalized choice.

Safe in the evening

Quite a few PDers' tremors ceased after some pre-determined hour in the evening. The actual time varied from one person to another. They tried to explain to me that this was perfectly normal because, after their specific time arrived, they were "done for the day," or they had "finished everything that was supposed to get done that day." The implication was that the rest of the evening was free time – so there was no need to tremor.

No amount of logic could convince them that, if they applied this "free time" attitude to the entire day, they wouldn't need to tremor at all. They dismissed my idea with explanations that were always pretty much the same: "I *do* have Parkinson's. Why should I pretend I don't? If I stop having symptoms when my day is over, that's because I'm not *really* doing anything any more. As long as I'm actually *doing* something that *needs* to be done, I do have Parkinson's. It would be futile and it would be a lie to pretend that I don't have PD.

It would be years before I realized that I could substitute the words "at risk" for their words "doing anything." So long as these people were "having to do something" that was "important" or "real," they behaved as if partially dissociated. My eventual translation of these words became "so long as they thought they were *at risk* of criticism or possible error, or at risk of *anything* that might lead to the slightest physical or emotional discomfort, they dissociated."

No heart: no movement; no heart: tremor

But I did see, based on our experiments, that partially-recovered PDers were not going to be able to sustain a healthy mental connection with their hearts so long as they were afraid of or not interested in having their hearts. And if they didn't have a heart, they couldn't *feel* their hearts. The heart performs the core processing of sensory perception, or feeling. The ability to feel safe is necessary for imagining the feeling of movement. If they couldn't imagine movement, they couldn't move. No heart connection = no movement

Also, when the heart doesn't perform its job of sending sensory information to the brain, the brain has to assume that the heart is struggling to come out of a dissociative state or a state of shock. The brain therefore institutes tremors, as it typically does when, following a shock, the body's autonomic nervous system doesn't receive the full complement of neural exchanges between the brain and the heart. In an otherwise healthy person recovering from shock, tremor can be helpful in restarting the heart: the involuntary tremor movement makes the person slightly more alert, causing him to take a deep breath, feel the inspiration of life pouring back into his chest, *feel* the "good to be alive" sensation, *feel* safe, and then, because of that feeling, *wham*: he snaps out of his heart numbness, the heart communicates a full message to the brain, the brain communicates with the heart, and voila! The autonomic (automatic) heart-lung-blood pressure regulatory system is back on line and running correctly. No more tremor. But as long as there is no heart connection, a person can be prone to tremor.

This means that tremor can have two sources: the brain shift that occurs in response to backwards flowing Qi from the foot injury, and from selective dissociation from the heart.

As noted earlier in the book, we came to realize that many PDers actually had two forms of Parkinson's: injury-induced idiopathic Parkinson's *and* psychogenic parkinsonism that derives from dissociation from the heart.

We'd seen people recover simply by having their foot treated. But many PDers also needed to re-establish a lasting connection with their hearts if they hoped to recover.

As these realizations broke over our heads, I started to understand the answer to my prayer: "She has to do it herself."

Basically, my heart had told me that, if a person has shut out God (heart resonance), God will not override that person's decision to be apart from God. God, or Love or Wisdom, if you prefer, is a perfect respecter of free will. If a person is making a decision to be apart from God, God will honor that.

In other words, if a person was selectively dissociating from his own heart, God wasn't going to step on his toes and force him to feel heart resonance. If a person wants to stop being dissociated from his own heart, he has to do it by changing his mind and deciding to have a heart again.¹

We were able to understand, finally, that there was no needle treatment or herb that could change the mind of a person who had decided to selectively dissociate from his heart. So long as he kept making that decision, we couldn't help him. But on the other hand, when we realized that our patients were selectively (mentally) dissociating, a pathology that was well known and well studied, we were relieved, in a way. At least our partially-recovered patients had a *recognized* mental problem: dissociation. They had a problem with a *name*.²

¹ I tried to research these two uses of the word dissociate to learn which came first. I was not able to discover the dates. But I did learn that another, more literary usage dates back to the 1700s. This meaning of dissociate also means to "be apart from," but it refers more to dissociation from one's customary social group or country.

I came across an essay from the 1930s in which a yogi used the word dissociate with this "social" meaning when he advises people to dissociate from their pain. The rest of the text makes it clear that he is not advocating numbness, but rather a lack of self-identification with the pain.

A person might be identified by the society (associations) he keeps. If he ceases to belong to those societies, he will no longer be identified by them. He will have "dissociated" from them. The yogi was using this word in this "social" manner. What he meant was, if you find yourself succumbing to pain and imagining that *you* are pained, stop and remember that you are the immortal soul. Your soul is not in pain – only your body. "Dissociate from the pain" means, don't identify with the body; do not imagine that your true identity is pained. When you remember what you really are, you will no longer identify with the pain. You will then be able to use your soul's energy to focus on the painful place, calm the erratic energy in the area of the pain, neutralize the nerves signals so that they convey mere sensation instead of fear and pain, and then mentally process the experience and instruct the body to institute any necessary healing mechanisms. This is what a great spiritual teacher means when he says "dissociate from the pain." He does *not* mean "make yourself numb" (automatic dissociation) or pretend it never happened" (selective dissociation).

² When I say "mental" (meaning "not structural or chemical") I have to note that the brain is extremely plastic and able to undergo physical change in response to thoughts. Psychological behaviors *can* cause brain change, which means that PDers' brains *can* be structurally affected by their pathological thinking patterns. For example, people with purely psychogenic parkinsonism (parkinsonism that starts in response to a terrible trauma and which goes away after the person processes the trauma) may still produce brain scans that show decreased dopamine-receptor activity – a temporary *physical* brain change – while they are in the depths of their parkinsonism.

The brain is extremely plastic: it changes in response to usage. Because many people were taught in school that the brain serves as an unchanging switchboard, and that brain cells don't change or grow, I will make this an extended footnote.

THE TREATABILITY OF SELECTIVE DISSOCIATION

I wrote earlier that people can recover from selective dissociation. I don't want to create false hopes that treating dissociative disorders is easy, or to imply that there is a standardized protocol for treating these disorders. In the most severe form of dissociation, multiple personality disorder, there is *no* consensus as to whether this condition is actually treatable. Some psychiatrists say yes, others claim that any improvements are due to coping mechanisms. As I searched for success rates for treatment of dissociation from specific body parts such as the foot or the heart, I found *nothing* in the readily available literature. There *is* an enormous amount of literature addressing the psychological pathologies that can arise from sexual abuse, which can include dissociation from the abuse *memories*.

As I studied hundreds of these cases and their treatments, I became increasingly certain that treatments used in these cases of sexual or other forms of abuse would *not* be helpful for my PDers. Based on thousands of hours of psychological work with PDers, I knew what most of them could and couldn't do in terms of mental exercises. I was digging into the materials about selective dissociation in 2008. By that time, I'd already spent nearly a decade doing psychological therapies with hundreds of PDers, and I had a very good sense of what they could and couldn't do. They could *not* do the treatments suggested for dissociation from events.

As an example of the brain's plasticity, studies show that the language portion of a bilingual's brain is much larger than the language area in the brain of a person who only knows one language. Not only is the grey matter (the portion that "knows" the words) larger, the white matter (which links words to other areas of the brain) is *also* larger in bilinguals and trilinguals. This is an example of a brain area increasing in size in response to use.

The brain also *declines* in response to *non-use*. An excellent study used juggling to demonstrate that both brain growth and brain decline occur in response to activity or non-activity, respectively. In this study, healthy volunteers had brain scans (MRIs) before being taught how to juggle. After much daily practice, they were able to keep three balls in the air for at least sixty seconds. A second round of MRIs at this point showed their brain's gray matter had increased in those areas related to this skill (in the temporal lobes of both hemispheres and the parietal lobes of the left hemisphere). Three months after of stopping the juggling, a third MRI showed that "the gray matter gains were reduced." (Both the bilingual study and the juggling study are written up in *The Wisdom Paradox*, by Elkhonon Goldberg, PhD, p. 255. The juggling study was cited as coming from "Neuroplasticity: changes in gray matter induced by training"; Draganski, B., Gaser, C., Bush, U., Schuier, G. Bogdahn, U., & May, A. *Nature* (2004), 427 (6972), 311-312.)

For some light, fascinating reading on the subject of brain plasticity, I recommend *The Brain That Changes Itself*, by Norman Doidge, MD, and *Mozart's Brain And The Fighter Pilot*, and *The New Brain*, both by Richard Restak, MD.

My point here is that the brain is plastic. So when I say that a problem is psychological instead of physiological, the reader has to understand that changes in thought patterns, mental behaviors, and physical behaviors will *create* physiological brain changes. In the case of PDers, psychological habits can affect dopamine production and dopamine receptor activity. However, these dopamine-decreasing changes do *not cause* a PDer's dopamine inhibition – these changes come about *because* of a PDer's constant dopamine inhibition. This inhibition can be both electrical *and* mental. I'll be redundant (again): *all* PDers with the "classic" look of idiopathic Parkinson's (asymmetric skin and muscle changes along the Stomach channel) have the electrical (physically caused) inhibition of certain brain functions that is a correct "shut down the body so healing can occur – as soon as the injury is actually recognized," result of a severe injury. This brain shift will cease when the injury heals. In addition to this electrical shift, *some* PDers and people with psychogenic parkinsonism have, to varying degrees, *mental* inhibitions (selective dissociations) that shut down the heart's ability to feel and thus inhibit dopamine release.

Based on modern understanding of the brain's plasticity, we hypothesize that both of these types of inhibition will, over time, cause measurable decrease in certain brain structures and functionality. Healing the injury *and* getting rid of any mental dopamine inhibitors *plus* experiencing the feelings that release dopamine will cause an *increase* in dopamine-related brain structures and functionality.

For example, treatments for dissociation from sexual abuse often involved creating a “safe, non-abuse” zone where they could have their usual, normal full range of feelings, and then teaching the person to expand that safe zone. This process would not work with a person who felt safest when numb or who didn’t know what the word feeling even meant. These treatments required people to modify the settings that triggered explosions of painful feelings.

With regard to painful experiences, my patients tended to not actually *have* feelings.

Dissociation from feeling as opposed to dissociation from an event

An abused person tends to dissociate from specific *experiences* so that he won’t have to feel his violent physical and emotional reactions to the event. Sexual abuse survivors were still very much able to feel, enjoyed feeling, and were constantly traumatized by their inability to stop their revisitations of painful feeling. They dissociated from painful *events* so that they could continue to have feeling for all other facets of life. Their problem was that the brain made linkages to other events – keeping them perpetually traumatized.

PDers dissociate from their ability to feel, period. The event may or may not be forgotten, and it may or may not be considered a problem. The problem, for many PDers, is *not* that a painful event occurred. The problem for many PDers was that they felt at risk of *feeling* a strong response.

PDers don’t necessarily dissociate from the memory of terrible *events*. Some do, but some don’t. However, all of our partially recovered PDers had, to some extent, dissociated from the ability to *feel* the experience. The negative *experience* wasn’t the problem; the problem was the ability to *feel* a physical or emotional response.

PDers were dissociating from feeling, period. When a person dissociates from his ability to feel his own physical or emotional pain, he does this by dissociating from the signals that are generated in his chest, in his own heart. Whether or not he consciously thought about not having a heart, a cessation in heart sensory function is the mechanism employed when a person decides that he is impervious to pain.

We now recognize that most, if not all, of our partially recovered PDers had dealt with unpleasant events by dissociating from their ability to feel. Many recalled doing this consciously, at some point in childhood, in response to an emotionally or physically painful situation. For most of them, they were now able to do it subconsciously, when any situation arose that might potentially result in criticism, error, or anything that could cause physical or emotional pain.

As I read books on treating selective dissociations, I knew that PDers would not be able to respond to the treatments that were being used. We’d actually tried some of the techniques already. We were going to have to come up with a treatment technique specific to the problem of fearing *feeling*.

Dissociating from the pain of diagnosis

When we figured out a way to treat this problem, we discovered that one of the most common emotional pains that actually needed to be specifically treated (as opposed to the pains that could be treated in batches, by focusing on generalities) was the pain of receiving the diagnosis of Parkinson’s. We saw an enormous beneficial shift in PDers when they were able to

correctly neutralize the pain of having been diagnosed with Parkinson's disease. This helped us answer a puzzling question.

You may recall reading in chapter xxx that the people who didn't have a diagnosis of PD tended to recover quickly. They didn't slide into partial recovery.¹

In our limited experience, the people who had not yet received a diagnosis of Parkinson's had only dissociated from the ability to feel their foot pain. But it became evident, in the last stages of our project, that people with a diagnosis of Parkinson's had dissociated from their ability to feel the emotional pain of bad news. This turned out to be a much harder dissociation to treat: it seemed as if it linked up to other emotional pains. When we starting working on the pain of Diagnosis of Parkinson's disease, it often brought forth other old, buried issues, as well. These other issues in and of themselves might not have been strong enough to create the symptoms of Parkinson's disease. But joined by the enormous pain of diagnosis, these various dissociated emotional pains seemed to assume the power of a small cabal, a gang that was, on top of everything else, highly susceptible to negative suggestions (the placebo effect).

After we learned how to teach people the necessary steps in re-associating with their hearts in general, the dissociation from the pain of diagnosis very often needed to be specifically addressed.²

We finally had a hypothesis that answered all our questions about partial recovery.

Next, we had two jobs. First, we had to *prove* that our hypothesis was valid. This proof would have two parts: we had to prove that PDers were, in fact, in a state of dissociation *and* we had to prove that they had been able to put themselves there by simply telling themselves not to feel.

¹ The fact that *none* of the twenty-five people who had PD symptoms but no official diagnosis were dissociated from their hearts is probably due to our small sample size. We have many PD patients who have told us, during or after recovery, that they had definitely shut down their hearts in childhood, and had never felt much physical or emotional pain ever since. Had these people been in the "no-diagnosis group," they may have experienced a brief respite from their symptoms when their feet healed and then, over time, continued to manifest symptoms of psychogenic parkinsonism. This would have altered our statistics, so that we would only be able to say *most* people who have not yet received a diagnosis recover very quickly.

A much larger sample size, involving thousands of PDers, might yield more variables, as opposed to the 100 percent finding of our small sampling.

² The PDer reading this section may well say to himself, "I don't remember feeling any pain when I was diagnosed." My reply would be, "My point exactly." Not all PDers are the same, of course. Some did recall feeling horribly betrayed by the body at that moment of diagnostic horror – but they didn't necessarily feel physical pain. Others were certain that they hadn't felt anything at all. An emotionally healthy person might respond to a terrible diagnosis of "incurable illness" by feeling as if he'd been kicked in the solar plexus, or feeling a knot in his throat, or feeling a knot forming in his stomach: some sort of physical pain.

When we developed a simple, safe, and pleasant method for processing these dissociated emotional pains and focused this method at the pain, gently reawakened, that *had* in fact occurred at the time of diagnosis, PDers have been able to feel the physical sensations of the emotional pain that actually occurred at the time. These pains were not anything special or rare. They were usually things like a lump in the throat, a gnashing or sinking feeling in the gut, a twist in the stomach, shortness of breath, etc: the usual responses to emotional pain. These are perfectly normal pains and can be processed in the normal way *but* only if a person knows that they exist. If a PDer had averted these sensations away from his conscious mind, then he had never felt them. But they were still sitting there in the body, unfelt, and they were also still sitting in some dark cubby-hole of the mind, sending fear signals that encouraged the PDer to keep making use of his skill in dissociating.

Second, we needed to come up with a treatment. We had to figure out a way to teach our PDers how to consciously induce *feeling* at will. After teaching them to feel, we had to teach them how to feel and heal from their buried pains without being overwhelmed or frightened by them.

And then we realized that we had already done the first job. One of our many experiments, “A Day at the Beach” had proved that partially recovered PDers were in a state of dissociation. Another experiment, the Heart-in-a-Box technique, had yielded proof that PDers had, in fact, induced this state intentionally, by deciding to be impervious to feeling.

