

“ ‘Playing possum’ is not a strategem but a swoon; ‘the animal can no more stop the involuntary action than a sensitive plant can withhold the folding of its leaves.’ ”

The World of the Opossum, by James F. Keefe

CHAPTER ELEVEN

AUTOMATIC DISSOCIATION

What I refer to as “automatic” dissociation is a recognized set of physiological shifts that can occur in response to severe physical trauma, puncturing of the skin, or a high level of blood loss. When automatic dissociation sets in, the injured person or animal experiences an extreme decrease in heart and breathing rate. His blood moves towards the deep interior of the body, away from the arm and leg muscles and the digestive system. The extremities and skin may even become cold. Midbrain dopamine release is inhibited, preventing easy movement, hunger, or “seeking” behaviors such as curiosity. Adrenaline release is *minimized*: adrenaline levels will be just sufficient to maintain *minimal* breathing and heartbeat. At the same time, endorphins (opiate-like chemicals) are released into the injury area(s). The endorphins block the perception of pain. If a very high level of endorphins is released, the endorphins can get into the general blood supply, whence they can trickle into the brain, causing sedation or even loss of consciousness. If a human experiences some degree of automatic dissociation but does *not* lose consciousness, he may undergo an *alteration* in consciousness: he may feel as if he is outside his body, *observing* his body rather than *feeling* as if he is inside of it.

This altered consciousness is called “depersonalization.” Depersonalization can cause “a sense of detachment from the self. Patients sometimes describe depersonalization as feeling like a robot or watching themselves from the outside. Depersonalization disorder may also involve feelings of numbness or loss of emotional ‘aliveness.’”¹

Depersonalization can, in some cases, make a person feel apart, or different, from others, just as he feels apart from himself. If a person is unable to resonate with his own sensations and his own physiology, he will also be unable to resonate with the underlying “human resonance” of himself or others. Lacking awareness of this resonance, he will tend to *evaluate* others critically, based on their variations in actions and words – their differences. If he could feel his heart, he would be able to wordlessly *resonate* with mutually similar, vibratory heart patterns and characteristically human electromagnetic fields – and thus appreciate others as being basically similar to himself.²

¹ *Health A to Z*; from website www.healthatoz.com, date accessed 1-14-8, derived from *Gale Encyclopedia of Medicine*; essay author Frey RJ PhD; Dec, 2002.

² Curiously, most of our PD patients have been insistent that they are different from all the cases I have written up. To our eyes, and in the eyes of their spouses, they often conform quite nicely with many of the case studies and with the generalities that I make about people with Parkinson’s disease. However, many PDers grasp at small differences, or aggrandize their bits of uniqueness to such an extent that we were, at first, baffled. It was only when we learned that depersonalization *makes* a person view his relationship with others analytically, even critically, by examining *differences* instead of being able to enjoy and resonate with *commonalities*, that we began to understand. What had seemed like compulsive nit-picking was actually part of the depersonalization that occurs with dissociation.

During an automatic dissociation response, the heart's role in sensory interpretation is temporarily shut down: the heart nerves that tell the brain *which* neurotransmitters to release and *how much of each* become temporarily inhibited. In other words, the nerve communication between the heart and brain that ordinarily determines the heart rate and the breath volume, and all other sensory-resonance heart-brain communications, are temporarily suspended. In this respect as well, the short-term physiological shifts of the automatic dissociation response resemble the long-term heart-nerve changes that are *only* seen in people with Parkinson's disease.

In this neurological mode, the chest moves only as much as is needed for breathing: the chest area is unable to expand with pleasure or contract with displeasure or fear in response to sensations in the body or in the environment.

Like Parkinson's disease, automatic dissociation symptoms can range from mild to severe. The symptoms of *mild* automatic dissociation resemble the symptoms of *early- to mid-stage* Parkinson's disease. The symptoms of *severe* automatic dissociation create a condition in which a living animal appears to be dead because he's encased in a rigid, seemingly dead body – very like some people with *late-stage* Parkinson's disease.

Is automatic dissociation a variation of the sympathetic nervous system response?

A sympathetic response (as opposed to a parasympathetic response) swings the body over to an adrenaline-dominant, dopamine-reduced condition. Because automatic dissociation also causes an adrenaline-dominant, dopamine-inhibited state, it might be classified as a variation on the sympathetic nervous system response. On the other hand, enough differences exist that the automatic dissociation response very possibly should be in its own category. Even so, it can be helpful to compare the automatic dissociation response with the sympathetic response.

When most people hear “sympathetic” or “adrenaline-type” response, they think “fight or flight.” This is gross oversimplification. The adrenaline-dominant response actually can take many forms, including the relatively rare “fight,” or “flight,” the more common “freeze,” and, the most common of all, “wariness.” These responses to danger or challenge feature varying amounts of increase in adrenaline, a corresponding *decrease* in dopamine, and a corresponding *increase* in heart rate, bronchial dilation, alertness, and blood supply to the muscles of the arms and legs.

As noted earlier, the automatic dissociation response – even though it is a type of adrenaline-dominant response inasmuch as adrenaline levels are higher than dopamine levels – causes a *decrease* in heart rate, breathing, alertness, and blood supply to the muscles of the arms and legs. This is because, in automatic dissociation, adrenaline is released at very *low* levels, not high levels.¹

¹ Actually, the sympathetic/parasympathetic classifications need an overhaul. These two modes only define the body's behaviors during times of alertness. When sleeping, a different mode kicks in – a mode similar to the automatic dissociation mode. More than a century ago, doctors decided that anytime a person wasn't in sympathetic mode, he must automatically be in parasympathetic mode. How simple. However, parasympathetic mode is usually understood to be the mode in which a person is relaxed and his heart and brain are oriented towards the vagus nerve – the nerve that activates the stomach, intestines, and all the organs that run in tandem with the gastro-intestinal tract. In the 1960s, when I was in high school, we were taught that we could think of parasympathetic mode as the “cud-chewing mode.”

During sleep, as during automatic dissociation, the vagus nerve is somewhat inhibited (we don't have bowel movements in our sleep, nor do we digest food well when we are sleeping), consciousness is altered, heart

Dissociation in prey animals

In nature, dissociation usually occurs when a predator's teeth or claws have punctured or severely cut the skin of his prey. The dissociation response is beneficial to the prey animal in the following ways: because heart rate is slowed and blood is shunted to the interior of the body, bleeding from the torn flesh is minimized. More importantly, the endorphin-induced sedation, the severely slowed heart rate, and the slow breathing of the prey animal can cause it to become cold and rigid. To a casual observer, the animal will look as if he is dead.

The supreme benefit of the dissociation response is this: if the predator was catching the prey for sport instead of for food, he may very likely lose interest in his seemingly dead prey. He may drop his prey and go in search of livelier fun elsewhere. Several minutes to an hour later, the seemingly dead animal will return to an alert state and make his getaway.

Many of us have seen a cat drop a mouse when the mouse lapses into a deathlike rigidity. The cat may make a few swats at the mouse to see if he can stimulate the mouse to movement. However, if the mouse remains cold and motionless, the cat often loses interest and moves on.¹

rate and breathing slow down, and blood flow is shifted away from the muscles of the arms and legs – a combination of conditions that does not match the parasympathetic *or* sympathetic modes. The outdated classification system leaves us no way to easily refer to the physiological shifts that take place during sleep – or during automatic dissociation. If we use the existing system, we are reduced to describing all physiological responses in terms of “happy or hungry” *or* “fight or flight.” Simple, but incorrect.

¹ Children who are regularly abused sometimes teach themselves to consciously activate an automatic-type dissociation response when the abuse is about to begin, for the same reason: an enraged or sadistic attacker sometimes redirects his energy or his energy becomes diminished when his “prey” is unresponsive.

When abused children intentionally dissociate from their ability to feel *anything*, opiate-like chemicals (endorphins) are released and numbness pervades the body. Then, if the child bothers to observe the abuse from his depersonalized perspective *outside* of his body, it seems as if the abuse is happening to his numbed physical form – but not to *him*.

As noted earlier, in humans, dissociation from the heart can allow a person to feel no pain and to perceive himself as apart from the body that is being hurt, as if he is watching the event from outside his actual body. While not *all* PDerS have taught themselves, via dissociation, to maintain this type of outsider's perspective, many of our PD patients did maintain this outside-the-body perspective almost continuously.

As an illustrative aside, some people who have learned, often during abuse, the pain-numbing benefits of the dissociation response may later perform self-mutilations such as cutting or burning to trigger the pain-deadening rush of opioid-like chemicals, endorphins, that naturally occur when the skin is perforated. Children who frequently engage in this type of dissociative response can develop significant brain changes – presumably from regularly flooding their brains with endorphins (or, more exactly, the *dopamine* that is released in *response* to endorphins). In brain SPECT scans, the brain changes – patchy areas of decreased levels of cellular activity in the *frontal* lobe (not the midbrain) – that occur in children who frequently dissociate resemble the brain changes seen in people with frequent use of powerful dopamine-enhancing drugs such as methamphetamine. It is presumed that these children's brain changes are due to frequent periods of high levels of endorphin-triggered dopamine that is released during their dissociation responses.

Sometimes, during an extreme dissociative response, even if the dissociative response was self-induced via superficial slashing that does *not* cause much blood loss, *if* the endorphins are released at high enough levels, a person can have a response similar to a heroin *overdose*: coma and even stopping of the heart. To rapidly reverse the life-threatening heart stoppage or coma, the hospital's emergency room can treat a person in this condition by administering the anti-opiate drug noxalone. For more information on this subject, see: *The Boy Who Was Raised As A Dog And Other Stories From A Child Psychiatrist's Notebook*, by Bruce Perry, MD, PhD, Basic Books, New York, 2006, p. 189.

How does this relate to Parkinson's disease? Understanding that mental control over dopamine release *and* dopamine inhibition is an innate ability and not a reflection of pathological genetics or illness can help the PDer who had been emotionally sandbagged by the idea that Parkinson's occurs when an unknown “something” kills off dopamine cells for “no reason,” or as many PDerS have bitterly described it: “My body is betraying me.” PDerS

Dopamine inhibition and turning dopamine back on

Following automatic dissociation, the “safe feeling” must occur in order for dopamine release in the substantia area of the midbrain to resume.¹ We’ve been able to determine that, in humans, the “safe feeling” can best be described as the physical sensation of expansion that occurs in the chest when the sensory cues for danger have ceased. Perception of the safe feeling leads directly to the release of substantia (midbrain) dopamine. Release of substantia dopamine initiates “seeking behaviors:” curiosity, interest in food, playfulness, uninhibited self-expression. Indulging in these behaviors leads to the release of yet more dopamine in the ventral striatum, which induces still more positive, safe feelings, thus activating a positive feedback loop.²

Seeking behaviors need to be inhibited during times of 1) potential danger, 2) trauma-induced dissociation, and 3) during times of initial healing from severe injury. Therefore, because *dopamine* is the neurotransmitter that initiates seeking behaviors, dopamine release is *inhibited* during 1) a strong sympathetic (danger) system response, 2) an automatic dissociation response, and 3) the initial phase of shock and healing that follows a severe injury.

may be uplifted by learning that altered dopamine-cell function can be set in motion by something curable: either an unhealed foot injury or a long-term, fear-based mindset; a *dopamine-inhibiting*, automatic-dissociation mindset.

¹ *In Search of Memory: The Emergence of a New Science*, by Erik R. Kandel, Nobel prize winner; p. 350. Research linking the safe feeling and the release of midbrain dopamine used mice that had received Pavlovian training so that they “felt safe” at the sound of a specific tone. When they heard this tone, a surge of activity occurred in the substantia: they subsequently moved more freely, and over a larger area. Prior to this research, no one knew exactly what the stimulus was for the initial surge of activity in the substantia that then led to seeking behaviors. Chapter xxx goes into great details on this highly significant research. The safe feeling has nothing to do with logic or thoughts: mice do not consider whether or not they might reasonably deem themselves to be in a safe situation. In order to help PDerS who were locked into mindsets that couldn’t imagine feeling safe, we had to figure out the *biological* events that occur in a mouse when he is “safe”: the events that, through Pavlovian training, are activated automatically when the mouse hears the “safe” sound. Because most of our PD patients had no idea what was even meant by the phrase “feel safe,” part of our research was directed at figuring out how to *describe* the physical processes and sensations that occur when a healthy person feels safe, so that the PDer could learn to produce them.

² Research and general news articles supporting the finding that dopamine is the “reward” for indulging in seeking behaviors – behaviors that can be performed after the initial release of dopamine – are easy to find. While writing this up, I looked for an additional citation to support this relatively new finding, and within minutes I had grabbed one off the Internet, titled “Brain Scientists Discover Why Adventure Feels Good.” This article referenced a study by Wittman and Daw, of the Wellcome Trust Centre for Neuroimaging at University College in London, that had been published in the online journal *Neuron*. The study drew the conclusion that “Seeking new and unfamiliar experiences is a fundamental behavioral tendency in humans and animals [because of the reward of additional dopamine that is released when doing something new or unfamiliar].” (Continued on next page.)

For our purposes, we might add a correcting modifier: “*If a person feels safe*, then seeking new and unfamiliar experiences is a fundamental tendency, etc.” Oppositely, one of the manifestations of the harm avoidance that is a prime component of the Parkinson’s personality is the increasing reluctance to engage in new or unfamiliar experiences.

The next article I pulled up, “What’s in a Smile? Maternal Brain Responses to Infant Facial Cues”; Strathearn, Li, Fonagy, Montague; *Pediatrics*; Vol. 122. No. 1, July 2008, pp. 40-51, stated that new research shows that parents’ midbrains release dopamine when their babies smile at them. The reward of dopamine release makes new parents become “addicted” to making the child smile. This article suggested that parental bonding and wanting to keep the child happy is driven by the dopamine reward. In terms of animal behavior, trying to stimulate a baby’s ever-new smile is a type of “seeking behavior.”

These examples of dopamine-inhibiting conditions demonstrate how *inhibition* of movement and seeking behaviors via inhibition of dopamine *can* be a correct, naturally occurring event.

Relating this information back to Parkinson's disease, PDers sometimes exhibit all three of the above dopamine-inhibiting conditions: 1) a perpetual sense of wariness or an inability to feel safe, 2) varying degrees of automatic dissociation brought on by selective dissociation from the heart's ability to resonate with physical or emotional pain, and 3) an unhealed injury that, because of its location at the terminus of the Stomach channel, causes the type of electrical disarray that is usually activated by *severe* injury.¹

Turning dopamine back on: feeling safe

Most of the PD patients that we worked with almost never felt safe. They had taught themselves to be perpetually wary. Many PDers were proud of their constant wariness, considering it to be a sign of high intellect. Some of our PD patients felt negatively towards the very idea of "feeling safe." Some insisted that only an idiot could ever feel safe.

To help our patients who got stuck in partial recovery who were, for the most part, deeply confused by the concept of "feeling safe," we also tried to define "feeling safe" more technically: we told them that the feeling was "a subtle sensation of physical expansion in the chest, like the feeling of chest expansion that occurs when you perceive something of great beauty or grace." Many patients claimed to have no memory of ever having experienced a sensation of expansion in the chest in response to relaxation, or to something beautiful or stirring, or to feeling safe, or in response to anything. Others thought that they *might* have felt something along those lines, years or decades earlier, but they had no idea how to recapture the feeling. Many had no interest in trying to feel safe, even if it meant a release of dopamine. One PDer expressed it this way: "I'd be lying if I made myself *feel* safe when the truth is, no one is actually safe."

When we discussed the importance of feeling safe with PDers, many of them protested, saying something along the lines of: "The whole purpose of life is to protect yourself from strong emotions. If a person feels safe, he might let his guard down."

Then again, a few insisted that they always felt perfectly safe. They admitted that their tremor and other symptoms were worse in certain situations, but the worsening of symptoms had nothing to do with being *unsafe*. I asked them *why* their symptoms worsened in certain situations if fear and/or not being safe weren't involved. Their reasons ranged from "Social stress" to "I have no idea." They often added statements such as "Worry isn't related to fear or safety," and "Social stress has nothing to do with fear." Some of these people refused to admit that worry or stress had *any* relationship with fear or not feeling safe. Many felt very strongly on this point, explaining to me that they *did* feel safe; that they had built their entire life around the idea of keeping themselves safe. A few went so far as to say that *everything* they did was an intentional effort to stay safe. I tried to suggest that a person who actually felt safe wouldn't have to make a constant effort to feel safe, and that a person's constant efforts to make everything safe might be

¹ An aside: I do realize that this chapter is about automatic dissociation, and not about injury. I included the fact that injury can also cause dopamine inhibition because the same benefits of dopamine inhibition apply in the case of injury. I am redundantly including the fact of dopamine inhibition during injury in this section on automatic dissociation because it helps make the larger point: dopamine inhibition can be a perfectly natural, beneficial response to certain situations. For the same reason, I have introduced the idea that dopamine may be inhibited when danger looms. An editor was concerned that, by introducing the subjects of injury and danger into this already meandering chapter, I might confuse the reader. This footnote is an attempt to clarify.

considered *proof* that he didn't yet feel safe. These patients were usually unable to comprehend the point I was making.

But a large majority of PDers, after reading the earlier editions of this book, volunteered that they did tend towards constant wariness, overthinking, and/or fear of letting their guard down.

It wasn't until 2008 that we realized that many PDers did not understand the literal meanings of words and phrases such as "feeling" or "physical sensation of expansion in the chest," let alone "feeling safe." They had spent so much of their lives in a state of altered consciousness, in which they perceived themselves as being outside their own body, observing their body but not feeling it, or feeling only those strong signals of pain that made it past their mental blockages, that they only accepted metaphoric meanings for literal phrases such as "feel your heart," "open your heart," and for other sensation-related words and phrases. Many of them truly thought that "feeling safe" meant "thinking about how to protect oneself from risk or criticism." They had no idea that feeling safe could include an actual feeling, a physical sensation, a relaxation or expansion of the chest.

Actually, a few PDers' symptoms instantly worsened for up to two minutes when I slipped any form of the word "feeling" into a sentence, even in an innocuous usage such as "I *feel* like the weather should be nice by the afternoon."

Many of our patients had selectively dissociated from their ability to feel, period, and thus had dissociated from their ability to *feel safe*. Because they could not feel safe, they could not activate the surge of midbrain dopamine that should kick in when trauma, injury or immanent danger is *over*. They remained locked into wariness – an attitude that triggers the sympathetic nervous system and inhibits the parasympathetic system: a neurological posture that *also* increases adrenaline and inhibits dopamine! Yet another dopamine-inhibiting behavior!

But I am getting ahead of myself. This section was merely to make the point that dopamine inhibition can be a perfectly normal function *and* the inhibition ends when a person feels safe. In a physically and emotionally healthy person, mentally induced dopamine inhibition can be an appropriate, healthy *short-term* event in response to a fleeting fear, danger, or trauma. We saw that, in many people with Parkinson's disease, it had become a lifestyle.

What dopamine actually does, and how this relates to automatic dissociation

Because many of my patients with Parkinson's are fascinated with the subject of their so-called dopamine insufficiency, are somewhat well read on the subject of dopamine, and drill me with all sorts of questions about dopamine, this section will explain a little more about other roles of this major neurotransmitter. To fully appreciate the automatic dissociation response, during which dopamine in the substantia is inhibited, it can be helpful to know what dopamine does when it's not being inhibited.

Dopamine is a neurotransmitter that doctors have, at one time or another, defined as being the neurotransmitter of sleep, waking, relaxation, alertness, pleasure, addiction, pain relief, muscle movement, joy, parasympathetic system-based imagining of movement that *leads* to motor initiation, and now, most recently, "seeking behaviors." Some of these ideas were just plain wrong. Some of them were only partly wrong. Also, many doctors are ignorant of the fact that dopamine behaves differently in different parts of the body, being a stimulator of nerve transmission in some areas and an inhibitor of nerve transmission in other areas. Many are also

ignorant of the proven fact that thoughts, as well as chemistry, can release dopamine and turn on or off the different types of dopamine *receptors*.

Today, most school children correctly learn in health class that excess dopamine is related to the addiction process. From what I can tell, many newspaper science writers have learned that dopamine is the neurotransmitter of happiness: a *mood* chemical. But they also repeat the cant that the death of dopamine-building cells causes idiopathic Parkinson's disease, which was found to be *not* the case at the turn of the twenty-first century, nearly a decade ago. And from what I've learned from patients and various other sources, most MDs who were educated prior to 1995 still think that midbrain dopamine initiates sleep and muscle relaxation – two of the proposed dopamine roles that have turned out to be completely wrong. So, purely for those who want more information about dopamine, and its *necessary* inhibition during times of danger, automatic dissociation, or severe injury, here is the latest.

Dopamine in different places

In and around the spine, dopamine *inhibits* the transmission of nerve signals. In the midbrain, dopamine *stimulates* neural signals. (A nerve inside the brain is called a “neuron.”)

Dopamine in the substantia, in the deep inner core of the brain, known as the midbrain, activates seeking behaviors *if* the animal is awake and *if* the animal is in the parasympathetic (joyful and/or content) mode. Seeking behaviors include the uninhibited, self-expressive *movement* that PDer's cannot perform (except during safe activities). The neurotransmitter that actually activates muscles is acetylcholine; dopamine allows activity in the *imaging* area of the brain, which, if approved (because one is safe to self-express), then *leads* to the initiation of motor function.

Dopamine's role in feeling good

Dopamine in the midbrain also contributes to a feeling of well-being. In this capacity, dopamine is exquisitely regulated so that a person never has too much. If a person felt too good, he might not bother to inhale or he might jump off a cliff just for fun. (People have been known to do both these things while under the influence of dopamine-enhancing drugs.) Dopamine-enhancing drugs include methamphetamine, cocaine, opiates, alcohol, and nicotine, as well as the dopamine-enhancing products produced by the pharmaceutical industry. Because of the real risk of sudden, carefree, *willing* death if dopamine levels are ever too high in the consciousness and the fear-inhibiting parts of the brain, the body will instantly institute a decrease in the brain's dopamine receptivity if a person, even briefly, experiences an excessive level of dopamine in these areas. After an event in which dopamine levels are deemed excessive, the brain may wait for up to ten weeks before it will once again release or be receptive to enough dopamine to provide for a minimal level of the feeling of “well-being.” The process by which the brain reduces dopamine receptor activity or dopamine release in response to even a momentary excess of dopamine is called “addiction.”¹

During an automatic dissociation response, the body inhibits the release of the *midbrain* dopamine that is used to initiate seeking behaviors. *Imagination* and/or *visualization* of motor

¹ More details on the addiction process are available in *Medications of Parkinson's or Once Upon A Pill*, available for free download at www.pdrecovery.org.

initiation are among the midbrain-initiated processes that are inhibited during automatic dissociation.

An aside: inhibition of visualization during automatic dissociation

Inhibition of the *thoughts* of movement and the mental *images* of movement that precede parasympathetic motor function is important during automatic dissociation. For example, when a mother bear is contemplating taking another swat at your seemingly lifeless body, or when the enemy soldiers are giving “just in case” bayonet jabs to any almost dead soldiers on the field who are still moving in the slightest, you don’t want to be able to even *think* about moving, or even *visualize* movement.

As an aside, we were to discover that most of our patients who got stuck in partial recovery from Parkinson’s disease were either *unable* to visualize themselves moving, or they felt uneasy when doing so. Only while they were consciously forcing themselves to work at it could they imagine themselves moving. If they were in my office, and if I was standing right there, encouraging them to practice the hated imagining exercise, and if they managed to visualize themselves moving, then they could move effortlessly. They *loved* the feeling of “weightless” limbs and effortless movement. However, this pleasure and ease of movement was never enough of an inducement for them to work at learning to *maintain* the highly unpleasant process of forcing themselves to visualize movement. They didn’t like to visualize themselves moving: it didn’t feel *safe*.

Dopamine’s role in pain relief and altered consciousness

Getting back to the subject of what dopamine actually does, consider its role in preventing pain. During automatic dissociation due to severe injury, *location-specific* opioid-like endorphins (molecules similar to morphine or the other opiates) are released in the vicinity of the injury. Endorphins then cause *localized* (point of injury) dopamine release. (Opiates also work by causing dopamine release.) The endorphins cause the release of pain-relieving dopamine in very specific, pain-numbing, parts of the body (usually at pre- and post-synaptic spinal nerve junctions). At very high levels of endorphin release, endorphins and dopamine can both overflow the localized pain management areas and get into the bloodstream.

Endorphins in the bloodstream can get into the brain. But dopamine in the bloodstream cannot pass through the blood brain barrier: dopamine cannot pass into the brain from the bloodstream. This is a safety mechanism that serves to prevent excess dopamine appearing in the midbrain.

When endorphins can get into the brain, they flow to the areas where endorphins have receptors. Endorphin receptors are found in brain areas that influence mood and certain aspects of consciousness, but not in the *substantia*. When the endorphins “hook up” at their receptors, they cause the release of dopamine in those specific areas.

Endorphins in the brain cause dopamine release in the areas that regulate mood and consciousness. In the *substantia*, no endorphins get in and no dopamine release will be triggered. Thus an injured animal can have a dopamine release in the brain that causes heavy sedation or coma. *And* he will have the pain-blocking benefits of dopamine on the spinal nerves. *But* he will not have the dopamine-induced hunger, curiosity, or imagining of muscle function that leads to movement. The *substantia*-area dopamine, which triggers seeking behaviors, is only released

when an animal feels safe – a signal that comes from the heart after the brain stops sending signals of danger.

The high level of dopamine release in the consciousness area of the brain is not without risk: as footnoted earlier, brain scans of abused children who use dissociation to deal with frequent assault show that the frequent instances of unnaturally high levels of dopamine in the consciousness-focusing frontal lobe of the brain set in motion brain changes that are similar to the changes seen in the brains of addicts who frequently use strong dopamine-enhancing drugs such as methamphetamine. However, as a once-in-a-great-while method for escaping from imminent death, the automatic dissociation mechanism has definite benefits.

In times of near-death injury, a person who has automatically dissociated can have the benefit of localized dopamine-based pain relief and even the ability to appear as if dead, while *inhibiting* the potentially dangerous mid-brain dopamine-based behaviors such as hunger, curiosity, or self-expression. In other words, during trauma, dopamine is still being used, but its use is limited to certain highly specific areas and functions: local pain relief and alteration of consciousness in the brain. During these traumatic times, dopamine release and receptor activity for *other* dopamine-based functions such as imaging movement, hunger, and curiosity are inhibited.¹

Three situations that can trigger dopamine inhibition

During automatic dissociation, dopamine inhibition keeps the severely injured animal from making any incautious moves. Dopamine inhibition can also be beneficial during times of severe danger, as in the example of frequently abused children: a person who appears lifeless may be less likely to excite the interest of a predator.

¹ The mechanism for pain relief from opium-derived drugs and from internally produced endorphins is this: the endorphins latch onto endorphin-specific receptors on the nerves that carry pain messages from the body to the brain. When these receptors are activated (hooked up to an opiate or an endorphin), the release of GABA is inhibited. GABA is a regulatory neurotransmitter. When GABA is inhibited, dopamine release in the area of the endorphin receptors becomes uninhibited. This means that, when the endorphin receptors are activated by either endorphins or opiates, dopamine floods the area, hooking up to the nerves. When dopamine attaches to the nerves that carry pain signals to the brain, the pain signal is blocked.

This pain blocking is automatic and occurs when endorphins activate the area. “Runner’s high,” the rush of well being that floods the body of a person who overexerts himself in sports, is thought to be set in motion by the release of endorphins. Sometimes this flood of good feeling is called an “endorphin high.” Whatever it’s called, the sense of well-being is actually caused by the flood of dopamine that results from the surge of endorphin activity. In nature, the mild sedation of consciousness induced by intense exertion, the “runner’s high,” is probably the mechanism that tells the animal that he has run enough and he can now *stop* running. We can hypothesize that, when he’s run long enough, the animal feels calmed by the endorphin “high”: he feels *safe*. Unless danger is still imminent, in which case the animal’s adrenaline will override the endorphin-induced feelings, the “runner’s high” will cause him to relax, to stop running. Animals cannot “decide” to stop running, nor do they use logic: they rely on innate mechanisms to determine whether situations are “safe” or “not safe,” or if enough running has been done. The “runner’s high” may serve animals by telling them that it is now time to stop running.

Interestingly, prior to manifesting obvious symptoms of Parkinson’s, some PDers have learned that, by overexerting themselves into an endorphin rush and its accompanying safe feeling, they can obtain a moderate level of easy motor function for a short while: but the easy movement only lasts until the endorphin rush wears off. This method of staging a false metabolic surge in order to stimulate endorphins in order to stimulate a short burst of safe feeling followed by a short burst of midbrain dopamine is not actually a healthy mechanism. Over the long run, it may reinforce the habit of relying on overexertion in order to move: just the opposite of learning how to feel safe regardless of circumstances.

Also, during the initial healing stages of a severe injury, midbrain dopamine is electrically inhibited so that the injured person will sleep a lot, without much movement, and will not even notice sensations such as hunger. This electrical inhibition of midbrain dopamine allows the body to focus its energy on mending and rebuilding.

Again, although this section was primarily about automatic dissociation, I want to drive home the larger point that midbrain dopamine-release inhibition is a *healthy*, automatic body function during times of danger, trauma, or severe injury.¹



¹ The following is just an interesting aside. There are many types of dopamine receptors, and depending on mood and levels of alertness, these dopamine receptors activate various mental and physical behaviors. These types of receptors are named, cleverly, D1, D2, D3, D4, etc. I find it even more interesting that, according to cutting edge research, *thoughts* play a role in determining which dopamine receptors will be active at any given moment. For example, if a person *thinks* about getting up off the sofa, dopamine receptors in the activity-imaging area are opened up and ready for dopamine. As “getting up off the sofa” is imaged, neurons in this section activate the muscles necessary to perform the imaged activity. (The word “image” is *not* a misspelling of the word “imagine.” A person *imagines*, and his brain imaging area processes the *imagining*.)

Pathologies of receptor activity can occur. These errors allow dopamine to hook up to the wrong receptors at the wrong times. Sleep-walking is an example of what happens if motor-function-imaging dopamine receptors are receptive during sleep – a time when these receptors are supposed to be off and dopamine release is supposed to be minimal – if the person happens to dream about or imagine walking.

Although the idea that thoughts can determine chemical function may seem like science fiction and many neurologists are blissfully unaware that this concept has been proved, scientific research over the last decade increasingly backs up this new (and ancient) idea. In Dr. Candace Pert’s *Molecules of Emotion*, she explains some turn of the twenty-first century research that proved neurotransmitters and other chemicals throughout the body change shape and change function in response to thoughts. This shouldn’t actually be so surprising; *electrical* forces create the bonds that determine the *configurations* of atoms in the smaller molecules in the body. Brain waves and the much stronger waves generated by the heart create electromagnetic fields that must necessarily exert influence over these electrically determined, easy to “flip” molecular configurations.

Another good book on the subject of thoughts directing brain changes is *Train Your Mind, Change Your Brain*, by Sharon Begley. This book describes brain scan-using studies that revealed that focused thinking modified or changed brain wave patterns and neural connections, and that brain structures and zones changed or grew larger or smaller in response to repetition of focused thoughts. Other books, including *The Brain That Changes Itself*, by Norman Doidge, M.D., and *The New Brain*, by Richard Restak, M.D., are included in the suggested reading bibliography in the appendix.