

# ***NEUROPLASTICITY: THE BIOLOGY OF PSYCHOTHERAPY***

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## **Introduction**

Some time in the 1980s, the Dalai Lama was visiting a university hospital in the United States (Begley, 2007) observing a surgery to remove a tumor from the brain of a patient. At the conclusion of the operation, the surgeon announced that the patient would be fully functioning when he recovered. The Dalai Lama voiced his understanding that a tumor in the brain would impair mental functions. However, he asked the surgeon whether or not a mental activity, a thought, could affect the physical structure of the brain. The prompt response was a definite "No! There is no way mental activity can have physical effects in the brain." Some 20 years later, that "no" has been shown to be wrong. Years of neuroscience research support what H. H. the Dalai Lama was implicitly suggesting: Mental activity effects physical, neuroplastic changes in the brain.

In just over two decades, developments in a branch of biology—the discipline of neuroscience—promise to enhance our understanding of mental illness and mental health, and expand our treatment strategies and tools. They are leading to a major shift in how we understand the relationships between brain biology and effects of experience. Although the impact on clinical practice has been quite limited thus far, we can actually point to more than just "promise." There have been some direct applications of neuroscience findings in psychotherapy practice, and these examples are described later in this book. I am referring to three major developments. One is the advance of neuroimaging technologies allowing us to see electrical and metabolic activities in a living working brain. The second development involves the concept of neuroplasticity; imaging shows that the brain exhibits durable physical, biological changes as a result of learning. When we observe behaviors that change and persist as a result of experience, that is what we refer to as

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learning. What we know now, is that what we observe as learning, is accompanied by predictable biological changes that have occurred in the brain.

The third discovery is what can be called self-directed neuroplasticity. Conscious voluntary directing of attention and practice with voluntary behaviors can produce durable biological changes in the brain. The processes of psychotherapy always involve this self-directed neuroplasticity. In the following pages you will see detailed discussions of the ideas, the evidence and what the findings of neuroscience mean for the practice of psychotherapy. I will provide a detailed review of a major university applied research project for treating Obsessive-Compulsive Disorder (OCD), making use of the brain's neuroplastic capacities. This study is a concrete example of how neuroscience findings can enhance psychotherapeutic benefit. It involves a two-pronged set of objectives: reducing behavioral symptoms identified in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*, while normalizing metabolic activity in specific brain structures. These were achieved by exploiting the brain's plasticity potentials—the capacity for durable physical biological changes resulting from interaction of the brain with its environment.

The point of the following pages is to articulate the benefits of supplementing the DSM system of behavior clusters with systematic identification of relevant brain structures and processes. The closing section will review interventions that are applicable for reducing/eliminating symptoms in other Axis I disorders, in ways that also correct brain impairments. I have found this perspective, and the knowledge that comes with it, most helpful in my own professional practice. Understanding principles of how the brain learns is a most important resource to guide how we enhance healthy behavior and thinking.

There was a time when we lacked tools to see the working, brain as we designed and used therapies for treating mental illness. The data were not accessible. Today we use tools that yield images of working brains. We can benefit from systematic findings of neuroscience studies of twenty-some years. These advances provide a knowledge base from which to devise interventions that benefit from the plasticity potentials of the brain. Usually, when you see a reference to biological psychiatry, you expect to read about the effects of drugs and other medications on brain biology. You can also expect to see no biological measures of brain change. Typically you find no biological data defining either impairments or improvements in brain systems. This statement holds for both clinical assessments, and research addressing therapy outcomes. Researchers

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evaluate medications in terms of reductions of symptomatic behaviors. Major mental illness has been defined as biological brain disease. However, the biology of mental diseases remains steadfastly hypothetical through all the updated versions of the DSM. Diagnostic systems, per the DSMs, limit themselves to strictly behavioral observations. The closest conception of a biological brain disease is expressed in the term "chemical imbalance." This phrase is used to help patients with major psychoses to name what is causing their distressed emotions and behavior; it provides a rationale for pharmaceutical compliance. However, the chemical imbalance specification is still hypothetical. Diagnosis typically involves no actual biochemical assessment.

The definition of mental illness as brain disease involving chemical imbalance has expanded to define disorders beyond the major psychoses. These include anxiety and depressive spectrum disorders. A largely unquestioned implication of the biological hypothesis is that biological disease calls for primacy of one type of treatment intervention—biochemistry. From a limited conception of what brain biology means, neuropharmaceutical primacy makes sense. If the underlying disease is chemical imbalance, chemical agents might be the best route for restoring balance.

Part of the history of mental illness treatment is a long-standing conflict between those who assert biological malfunctioning as the foundation of mental illness, and those who argue that the pathologies in behavior and thinking stem from toxic learning experiences. The biological origin proponents argue that effective treatment must start with change in the biology of the brain (Torrey, 1983). They support this position with evidence of benefits from neuropharmaceuticals that arrived on the treatment scene in the mid-1950s. These medications arrived with very dramatic benefits for patients with long histories of major psychoses, with diagnoses like schizophrenia, mania and major depression. There were amazing remissions of delusions and hallucinations. For these patients, psychotherapies had shown only meager benefits, if any.

Of course, with the passage of time and increased use of these medications, the iatrogenic effects surfaced in the form of severe and permanent neurological damage. There was only limited damping of enthusiasm for this form of biological intervention. Research expanded to develop agents that might benefit those who had not responded to earlier medications, and to avoid the adverse neurological effects. Newer agents created a much lower incidence of neurological damage. However, they did not totally eliminate them. Unfortunately, the newer atypical anti-psychotics also presented new adverse biological reactions. These included significant weight gain

as well as endocrine pathology, in the form of insulin-dependent diabetes. Nevertheless, this particular form of biological strategy has expanded to include those other disorders that are less severe, as medications have been developed that alleviate distressing symptoms of anxiety and depression. Biology proponents have also cited studies that describe biological brain differences between the "mentally ill" and the "mentally normal." Yet, none of these differences are used for diagnosing a specific disease in all the years of DSM updates. Contrary views, rejecting the biological brain disease model, argued that toxic learning experiences were the source of pathology in behavior and thinking, observed in mental illness. They contended that corrective learning could undo symptoms that resulted from toxic learning experiences.

Thomas Szasz (1974) questioned the very concept of brain disease. He criticized the biologizing of issues that he saw having to do with character rather than biology. Both proponents and opponents of the biological brain disease model shared an implicit crude concept of brain biology. Neither side showed the least understanding that brain biology involved more than biochemistry. Neuroscience discoveries of neuroplastic processes in the brain had not yet influenced the mental health arena. S. Peele's book, *Diseasing of America* (1989), makes repeated arguments that what are called "diseases" are really examples of failure on the patient's part to exercise self-control, and own up to responsibility for one's own voluntary behaviors. His biology target reflected a view that biology disease was almost some kind of incubus that makes people do things in spite of themselves. His critique implicitly accepts that issues of self-control and accepting responsibility are off-brain phenomena. These views reflect an ignorance of the brain as a living system, and of how our natural biological brainwork can produce unhealthy as well as healthy behavior.

Especially important is that later research in neuroplasticity supports the idea that humans have the power to correct biological brain glitches by retraining the brain with more positive learning experiences. An important note is that brain activity and organization are here-and-now phenomena. Although these are partly results of history, in the form of genetic blueprints and experiences from the past, neuroplasticity takes place in the present. We should be cautious, at a minimum, about deciding if an impairment is correctible, based on origin—e.g. genetic source, or brain injury source, or early life experience source, or long-term chronic source. We do better with prognosis by looking at evidence for if and how a particular impairment can be corrected. Assumptions about the origins of malfunctions don't lead to answers about how to correct the

condition. Issues framed in terms of genetics versus experience, for instance, will not lead to solutions for correcting a condition. The empirical question is whether or not the impaired biology can be corrected. The science of genetics has, of necessity, spawned an emergent field of epigenetics, with discoveries of how other life factors influence how a specific genetic blueprint will express itself (Jaenisch & Bird, 2003). Illness, medication, nutrition, and other life experiences can alter outcomes for identical genes. Obviously this does not hold for all genes. However, many genetic programs are not immutable predetermined fate; they are tendencies with more than one possible outcome. An interesting example is the work by Kagan (1992) with his studies of differences between timid and bold children.

Mothers had been bringing their toddlers to his laboratory at Harvard University for many years, for studies of developmental changes overtime. Studies of temperament differences followed children about age two, and in later years. Mothers brought their toddlers of about 21 months of age to the new experience of the lab. Observers noted sharp differences in the way different toddlers responded. Most of these children exhibited a robust attraction to the play materials and to the other children. They were outgoing and began readily playing with other children. However, about a fifth of these children reacted very differently, showing signs of fearfulness in these new surroundings. They would cling to their mothers and resisted engaging with other children, showing obvious signs of discomfort. These children had been timid about anything unfamiliar from the time they were infants. A most intriguing observation however, was noted several years later when the children entered kindergarten. About a third of the previously timid children no longer exhibited the earlier timid, fearful behaviors. They acted just like the other bold children. Research staff had also observed interactions in the homes of these children, and described differences in parent-child interactions in the homes of those children whose timidity had faded, from those of children whose timidity persisted.

It appears that learning experiences can modify how genes will express. The altering of early life temperament tendencies involved biological changes in the brain. None of the bold children had become timid by kindergarten age, suggesting some genetic tendencies are more durable than others.

Knowing what caused the observed behaviors initially does not tell you whether or not that behavior can be changed. Whether the source is genetics or experience, observed behavior is always an expression of how the brain is working now. We don't inherit a behavior control gene.

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We inherit a brain structure with certain propensities; at least some of these tendencies can be altered with neuroplastic changes in that structure. Whether the brainwork can be changed, or not, is an empirical question. It cannot be deduced from the initial source.

Although there is general acceptance of the idea of brain impairment, and of necessity for biochemical treatment, there is also agreement that some form of "talk therapy" is an essential adjunctive treatment to sustain healthy function, especially for stress reduction. The biology/experience controversies have been resolved in a kind of working compromise.

The resolution is a kind of division of labor. The medical profession treats the biology illness with medication regimens. Psychotherapy in the form of various talk therapies, is provided by psychologists, social workers, counselors and related professionals. These talk therapies focus on the experience component of pathological symptoms, and often include encouraging compliance with medication protocols. How these therapies serve to correct behavioral and emotional symptoms without changing brain biology does not seem to have troubled anyone. Did these interventions bypass the brain as they effected changes in thinking, behavior and emotion? Several decades of neuroscience research have yielded an answer to this question, and that answer is an unqualified "no." All learned behavior change involves the brain's capacity for neuroplasticity. Although the brain can learn without a deliberate intention to learn, many neuroplastic brain changes are products of deliberate choices to engage in a learning effort. "Choice" refers to deliberate conscious directing of attention, and practice with voluntary behavior options.

The division of labor between those who are competent to prescribe medication and those who are competent in psychotherapy skills still makes good sense. However only ignorance can support the belief that biological treatment is limited to chemical agents. If you interact with a patient, you have no choice over whether or not to effect biological brain change, sometimes in lasting ways. You can only choose *what* change you might help that person to achieve.

Paradoxically, in an era when we are learning more and more about the power of learning to effect biological brain change, there has been a major effort by a subgroup in the profession of psychology to acquire access to the prescription pad. This movement includes post-graduate training curricula, as well as lobbying for legislation to permit privileges to prescribe medication. The anachronistic rationale is that we can't provide effective treatment without treating the biological component of mental illness. This view continues the false logic of division between

those who treat the brain biology disease, and those who do this other stuff that floats outside the physical biology of the brain. It only changes the personnel assigned to the "biology" part of the labor. Biological treatment will no longer be restricted to M.D.s and O.D.s. Some people with Ph.D.s would now provide pharmaceuticals for biological brain disease.

However, the stubborn reality is that all interventions that promote learning alter brain biology (Zull, 2002). Unfortunately the false distinction between biological treatment and treatments that do not improve brain biology, influences both goals and efficacy of treatments, especially with major mental disorders. Medications are not expected to produce durable corrections in brain biology. This is understandable since there is no empirical specification of the actual biological impairment. Hence, long-term compliance with a medication regimen is essential to ward off symptoms, and control, not cure, is the goal for correcting mental impairment. Although biology in the brain is implicated, medications are prescribed and evaluated for change in behavior, not for correction of biological impairment. Hardly any treatment settings employ a biological assessment protocol for diagnosis, for applying medication, or for terminating a medication regimen. The implicit conclusion is that we have no way to correct the brain biology; we can only compensate for the defects.

A related assumption also affects treatment adversely. It is the idea that the brain, as a command/control system, determines behavior and thinking unilaterally. This assumption has no grounding in reality. Deliberate practice with new behaviors and thinking can alter brain biology in predictable, observable ways. The empirical world of scientific data shows that the brain is an open system designed for learning. Influence is a two-way process. The brain exhibits changes, even durable changes, as a result of experience in its environment.

Modern imaging technology has opened a visual window to specific physical, biological changes that occur in the brain when we learn and remember. The brain's transactions with its environment are a two-way process. There is no way even to study a living, working brain separate from its experience in an environment. Even more telling, you cannot find a living working brain with no history of social experience.

Until recently the practice of psychotherapy could not benefit from knowledge about how the brain was affected by experience. The inner workings of the brain were once conceived of as an unknowable black box. However, much of the early work in psychotherapy was remarkably congruent with principles of how the brain learns. Although practitioners had no inkling of the

brain's capacity for neuroplasticity, they devised interventions that resulted in durable, learned changes in behavior. Whether we consider Salter's (1961) conditioned reflex therapy, Roger's (1951) non-directive interventions, Gestalt interventions (Polster, 1973) like guiding patients to shift from talking about experience, to physical creation of experience, Cognitive therapy (Burns, 1981), Hypnosis, Psychodrama, or various other "schools," they offer many diverse tools to facilitate learning processes. All of these orientations involve actively and deliberately shifting attention, and practicing new, beneficial behaviors. Practitioners were not thinking that they were actively promoting biological change, yet this is what they were doing. It's a little like the old joke about the person who discovered he had been speaking prose all those years without knowing it.

Until very recently, the prevailing view in biological brain science was that the physical structure and architecture of the brain was fixed, at least by the adolescent years. The only changes, other than those resulting from trauma or disease, were natural losses from some of our initial 100 billion neurons, as we aged. Neuroscience findings have thoroughly contradicted this assumption. We know this from imagery of physical brain changes that accompany learning experiences. This kind of change has been observed in ages far beyond the adolescent years.

One pertinent study, sometimes referred to as the "taxicology" study (Maguire et al., 2000), illustrates this phenomenon. Researchers found that taxi drivers in the city of London had hippocampus sizes significantly larger than were found in bus drivers in that city. The taxi drivers were required to pass very rigorous tests involving places and routes, and their daily driving involved a lot of variation. Bus drivers usually drove the same routes every day and faced much less complexity in what they had to learn and retain. An area in the hippocampus is particularly involved in learning and remembering spatial locations and relationships. This area is also richly endowed with stem cells, which can transform to neurons when needed. The results in this study are congruent with other neuroscience findings: learning something new, at any age, appears as neuroplastic change in the brain. These changes can even include the creation of new neurons, a process termed neurogenesis.

Where are we going with these observations? What difference does this kind of information make for the practice of psychotherapy and for enhancing mental health? What these discoveries mean is that the practice of psychotherapy involves self-directed biological changes in the brain. Psychotherapists, like all teachers, will improve the benefits of treatment by understanding the principles of how the brain learns.

The following pages stray from the DSM diagnostic model. This does not mean that mental health practitioners should not know how to use the DSM as a diagnostic tool. DSM publications have provided coherent descriptions of symptom syndromes. These symptom clusters are generally what patients or clients present. The initial diagnosis that "something is wrong" stems from behavior observations by the client, or an observer (who might be a friend, family, teacher, police, or work associate). Initial diagnosis does not typically come from results of a brain scan. As therapists, we receive the client's perception of distressing behavior, thoughts and emotions. This is where we start our therapeutic work. We are further ahead in having more than a simple listing of disconnected symptoms, with the reliable patterns organized in the DSM. Some of these patterns exhibit identifiable biological correlates in the brain, observed with modern imaging technology (Schwartz, 2002). The fact that the DSMs do not include biological markers does not disconfirm the biological factors in mental illness. The first DSM in the 1950s predated even a hint of neuroplasticity, and had few resources for incorporating biological considerations in the form of neuroplastic processes. Even within the Biology community, the idea that the brain could undergo neuroplastic changes was regarded as unthinkable as late as the early 1980s.

Diagnosis following DSM guidelines does not preclude therapy practices guided by findings from neuroscience. The DSM system provides little guidance for a psychotherapy strategy. Assessments do not identify specific brain impairments that cause symptomatic behaviors, nor do they specify what corrections could improve biological processes in the working brain. Also, as Amen (1998) has demonstrated in his book discussing brain scans of six types of Attention Deficit Disorder, similar behavior symptoms can stem from quite different sets of brain impairments.

The dominant diagnostic theme for many years has been the hypothetical "chemical imbalance." This theme has been guiding many treatment strategies. For much of this time, there has been no alternative to the hypothetical. The not-so-new neuroimaging technologies now provide many actual data on the working brain. We are in a position to go beyond the hypothetical and to identify real goings-on in the working brain.

Dr. Daniel Amen, a faculty member at the University of California Irvine campus in the Department of Psychiatry, is among the relatively few practitioners who draw on this progress. He has identified one type of actual chemical imbalance associated with certain behavior pathologies; metabolic over-activity or under-activity in specified brain structures exhibits with specific

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behavior symptoms (1998). Treatments in his clinics include nutritional improvements, psychotherapy, medications, yoga, and exercise. Decisions about what treatments to use include assessments of brain activity and behavior. Dr. Amen has pioneered the use of Single Photon Emission Computed Tomography (SPECT) scans to assess metabolic activity in brain structures. Therapy benefits are assessed in terms of both reductions in behavior symptoms and normalizing metabolic activity in the brain. The Department of Psychiatry at UCLA, Irvine is still one of the few places training psychiatrists in using SPECT scans for diagnoses of mental illness.

Others have documented the same kinds of connections between brain activity and behavior symptoms of mental illnesses using Functional Magnetic Resonance Imagery (fMRI) and other imagery tools (Schwartz, 2002). Dr. Schwartz directed an applied research project for treating Obsessive-Compulsive Disorder with psychotherapy interventions to correct metabolic imbalances as well as reduce symptomatic behavior, thinking and emotion. I will describe more detail about this project later in these pages. We will look at advances in treatment from symptom control, to treating causes of observed pathological behavior. We might even dare to use the word "cure" as a goal of research in treatment of mental illness. I am not implying that we now have a cure for mental illnesses. However, research can focus on improving specific brain functions in order to reduce behavior symptoms. Addressing biological brain causes as well as symptomatic behavior holds promise for going beyond control to durable biological improvements in how the brain is doing its work.

Toward that end, below are a set of propositions that are discussed and supported in the following pages:

- There are universal goals guiding all brains (Zull, 2002);
- Brain goals are *surviving, seeking pleasure, avoiding harm*;
- Goals are inferred from structures and traffic pattern;
- Brain is an open system;
- Brain is a command/control and learning system;
- Brain learns from experience in its environment;
- Learning occurs whether intended or not;
- Learning is manifest as biological changes in the brain;
- "Neuroplasticity" refers to these learning changes;
- Thoughts and behaviors are products of neuronal firing networks;

- Neuronal networks grow stronger with repeated activation;
- Neuronal firing can be a product of conscious thinking/behavior;
- When networks compete, the stronger one is dominant;
- Intentional learning is self-directed neuroplasticity;
- Self-directed neuroplasticity means conscious willful action;
- Psychotherapists guide clients in a learning process;
- Psychotherapists provide guidance, instruction, support and coaching;
- Brain has two operating learning systems;
  - o One system is designed for rational empirical learning;
  - o The other system involves emotions and other autonomic events;
- Once something is learned with conscious effort, it goes on automatic;
- We have a mind which is more than the brain (Schwartz, 2002);
- Free will is a valuable concept for self-directed neuroplasticity;
- Will power can direct attention;
- Will power can activate voluntary muscle behavior;
- Learning new habits does not require undoing the old ones.

A recent book, Zull's *The Art of Changing the Brain* (2002), provides a thoughtful model for incorporating biology in the definition of mental illness and mental health. He discusses the brain as a system, an open system, and draws inferences about the goals of that system. He bases these inferences on neuroscience studies of the structures, architecture, communication pathways, and the results of the working brain. The goals he infers are *survival*, *seeking pleasure* and *avoiding harm*.

Although words like mental health and mental illness appear nowhere in his book, there is much that is relevant for the practice of psychotherapy. His academic work involves designing and testing teaching strategies and tools that are compatible with how the brain works. He is especially interested in how knowledge of the working brain can improve the academic teaching/learning experience. When we recognize the brain as a *system* that interacts with its environment, we think of impairment, or sickness, in terms of what hurts its ability to achieve goals. No systems engineer would set out to design a system, or trouble-shoot, without clarity about the goals of that system (Churchman, 1968). It makes sense to think of a healthy system as one that is working effectively

to achieve its goals. We might eventually define mental health or illness with inclusion of empirical measures of how well the system functions in achieving its goals, rather than relying solely on a DSM-style consensus model. Zull (2002) describes structures in the cortex that appear designed for rational experiential learning. It's almost like the brain is designed for practice of science. Sense organs bring information from the brain's environment. These data are transmitted to a cortical structure that Zull (2002) refers to as the back integrative cortex, which is activated as the person relates the information to an existing store of knowledge, to make meaning of it. Incoming stimuli never contact a passive blank slate. As a result of genetic programming and prior learning, the brain selects where to direct attention, and what to highlight for further processing.

My eight-month old grandson sees an object on the floor near where he is sitting. Color and shape are probably what catch his attention. He has seen and handled this object before. The next brain structure in this sequence is referred to by Zull as the front integrative cortex. This structure lights up metabolically and electrically as the child formulates abstractions and hypotheses about what might lead to what. This little fellow had been crawling to different areas of the floor for a little over a month. He has some inkling that this object falls into the category of appealing. He probably has some idea that crawling toward it will lead to the experience of touching it and even putting it in his mouth.

The next brain structure to activate is a motor cortex which triggers voluntary muscle activity, and he tests his hunch, finding he can reach the object and grasp it (though putting it in his mouth is another matter when daddy decides it might be too small to be safe—another learning bit—and takes it away). As we develop and learn language, testing can also involve discussion or argument with another person, or other forms of voluntary motor activity. In any event, the natural learning cycle includes some kind of reality testing about how things work.

Actions and results of actions provide new input to our sense organs. A learning sequence could involve a single cycle or multiple cycles. Although the picture above is overly simplified, the overall sequence of cortical processing produces realistic empirical learning about how things work in the world. We learn to form abstractions and practical theories and skills to help us survive, experience pleasure and avoid harm.

To summarize, this kind of learning involves four structures: a sensory system that moves information from the sense organs to the brain; a structure that integrates new input and derives meaning by comparisons with a store of previous learning; a structure that activates formulation of

abstractions and predictive theories; and a motor cortex which controls voluntary muscle activity and tests predictions actively.

This is the kind of learning that involves rational empirical processes for learning how the world works. It grows a realistic bank of cognitive and behavior skills. Realistic means in terms of achieving the goals of survival, pleasure and avoidance of harm. This is the kind of learning Mowrer (1960) referred to as solution learning. Although his work could not benefit from later findings in neuroscience, he was remarkably prescient in terms of a learning concept congruent with later discoveries of how the brain works.

Mowrer (1950) proposed a two-factor learning theory: solution learning, and a conditioning process which described learning in the emotional brain structures as well as in other autonomic systems. These older structures that govern emotional experience lie deep in the brain. Activity in these structures can either help or hinder the kind of cortical learning processes described earlier. A very important part of the emotional system is the tiny, almond-shaped unit called the *amygdala*. The amygdala is constantly scanning for threats from the environment. It responds by setting off emotions like dread, anger, or fear when it senses threats. This structure is not a cognitive organ.

For some time, scientists believed that it was activated in response to messages from cognitive areas of the cortex. When these areas produce a conscious identification of a threat, they signal the amygdala. Indeed, this is one possible source for activation of the amygdala. It is true that there are nerve pathways from the cortex to the amygdala; perceived sensible threats from the cortex can rouse the amygdala. However, there are even more pathways from the amygdala to the frontal lobes, and it can react to non-cognitive sources as well. Sensory stimuli are transmitted from sense organs to the sensory cortex which makes cognitive meaning of the data. However, raw sensory stimulation also travels a shortcut with a path of nerve fibers running directly to the amygdala, bypassing the sensory cortex (LeDoux, 1997). The amygdala can respond to such raw data as color, smell, shape and other basic sense stimulation as it is on the lookout for threat, with no conscious awareness of the nature of the threat. Signals from the amygdala to the hypothalamus can trigger a biochemical cascade which includes flow of adrenalin and cortisol into the bloodstream as well as other chemical stress responses. Its capabilities are fully in place long before we have developed the cortical capacities for language and rational thinking.

Native inborn distress responses in infants can be observed with reactions to loud sounds, sudden loss of support and other experiences long before they can verbalize or think about the

event. Although a non-cognitive structure, the amygdala is capable of learning. The learning process is different from what is involved in the cortical processes described earlier. It learns through the process of what is called classical conditioning. This kind of learning is not governed by reason, understanding or deliberate testing of predictions. The new connections result from presenting sensory stimuli that have been neutral, closely in time and place with stimuli that have a history of eliciting an autonomic response, a response that is not a product of the voluntary nervous system. These autonomic responses can include sweating, heart rate, salivation and other gastrointestinal activity, and even immune system activity. Everybody is familiar with Pavlov's famous experiments conditioning dogs to salivate to a bell by pairing the sound of a bell with the stimulus of a piece of meat. Dogs naturally salivate when meat is served to them; they do not naturally salivate to the sound of a bell. With repeated pairing, the bell comes to elicit the salivation response, even when the sound is no longer followed by food. This is just one illustration of a brain learning principle articulated by Zull (2002). Neurons that fire together wire together. Whatever native responses the dogs produce in response to the sound, have been accompanied repeatedly by salivation. What we call classical conditioning is a physical change in the form of a new neuronal network. Research has shown that repeated stimulation of a neuron, or a network of neurons, strengthens the neuronal response (Kilgard & Merzenich, 1998). Strength refers to increase in the number of neurons in the network. The electrical impulses crossing synapses do not vary in strength; it is the number of firings involved. Whatever the innate responses to sound, when the neurons are firing at the same time as those involved in salivation, the outcome is that the activation in the auditory cortex comes to trigger the neurons involved in salivation. In this example of the conditioning paradigm, the conditioned response will weaken after repeated bell sounds without the reinforcement of meat. After enough trials without the food, the bell sound ceases to elicit salivation and it appears that the conditioned learning has been extinguished. However, when the neutral stimulus has been paired with a stimulus that naturally elicits a strong distressing emotion like fear or anger, the learned connection does not usually dissipate as it does with salivation. For reasons we will examine later, conditioned fears do not typically dissipate, even when the previous natural trigger (usually referred to as the unconditioned stimulus) is no longer presented, or much time has passed since the original learning experience. The sound of a car backfiring can elicit anxiety that was part of an event with a lot of gunfire, smoke, bloody

bodies and other battlefield sights. Amygdala arousal is an inner response that can activate in a new safe setting.

I recall a training group in which I had asked managers to engage in an exercise to experience dependence, trust and responsibility. We were meeting in a very lush and peaceful mountain resort in West Virginia. I had asked the participants to form pairs and walk in the woods with one member blindfolded. The assignment to the member who could see was to provide the blindfolded person with a walk that was both interesting and safe. One requirement of this exercise was that it proceed in silence. They were to change roles after 30 minutes. During debriefing sessions the blindfolded person often would talk about the discomfort of so much reliance on another person, with some anxiety about not being able to see, and feeling disoriented. However, in one of the pairs, a member who was a veteran of combat in Viet Nam, spoke about the discomfort he felt when he was the person whose eyes were open, and he was the guide. He reported that he felt a lot of tension as he was looking at the bushes and trees. He said he knew there were no snipers in these woods, but was not able to shake the physical tensions and the kind of anxiety he had felt in the jungles of Viet Nam. His reactions were relatively mild, and he reported no fear of losing control. Sometimes, however, the amygdala can trigger very powerful emotional and behavioral responses. When these reactions are strong enough, they will override the rational thinking and reality-oriented behavior governed by the cortex. LeDoux (1997) has described what he calls "emotional hijacking" in his book, *The Emotional Brain*. When there is such a severe emotional reaction, the likelihood of any rational learning is very low, and the emotional reactions might upstage any reality-oriented behavior. Also, conscious recovery of memory might not be a possibility sometimes because there might have been no cognitive component in the original experience. Amygdala learning that occurs prior to cognitive capacity cannot be remembered cognitively. In situations of trauma, where the emotional temperature was so high that it overrode the cognitive processes, there would be no cognitive memory, as there was no clear cognitive experience; there might have been no suppression or repression of a cognitive memory if the experience didn't register cognitively.

For the psychotherapist it is also helpful to know that there are experiences that have a calming effect on amygdala reactivity. Seeing pleasant and peaceful images can lower amygdala reactivity. Dr. Zull (2002) also describes how engagement in an interesting and non-threatening cognitive activity has a calming effect on the amygdala. These kinds of experiences add options in

addition to methods like progressive muscle relaxation. There are times when directing attention to an interesting cognitive challenge can have immediate and powerful calming effects. A personal example involved a session with a patient in a hospital forensic unit. We met shortly after an incident when this person had attacked some of our hospital police, throwing rocks at them and their car. This patient was very irate, and while talking about how unfairly she felt she was treated, and what had led to her attacking the security people, was raising her voice volume. I noticed her voice was also rising higher and higher in pitch. I encouraged her to continue to tell me details of her complaints, but asked her to notice her pitch was getting higher, and that this higher pitch might be increasing level of stress. I just asked "if you speak with slightly lower pitch, notice any difference in how you are feeling—but keep telling me about your complaints, and I am going to suggest you speak with our ombudswoman." She grew visibly calmer as she focused attention and tried a lower voice pitch, and made a coherent statement of her case; she was also able to recognize that the behavior toward the officers was wrong and self-defeating. When she was later moved to another unit, she continued to make use of lowering voice pitch as a self-calming tool she could employ easily: she recognized the rising pitch as a signal that she was increasing her stress level. She could appreciate the increased control she felt with this simple step. Of course, there are times when expressing distressing emotions in safe ways and a safe context is self-limiting, and can be another path to a calmer state.

The emotional brain also includes pleasure centers. The relevant structures are more decentralized, but play an important role in survival. Our natural bent to eat foods which are fat and sweet has helped support this goal. In the wild, poisonous edibles tended to taste bitter. Fat and sweet foods were more likely to be nutritious. In our modern world, human ingenuity has found ways to create edibles that can taste sweet and fat with little or no nutrient value, and even some that are toxic to our bodies. So we now have to use our cognitive capacities to learn how to make healthy and tasty food choices. We learn to rely on more than innate sensory attraction in our choice of foods. It means we read labels, and gather information from trusted sources. It isn't true that if it tastes good, it is bad for you. We just have to be more picky about what foods we decide to eat.

The promise of sexual pleasure motivates behavior that promotes the survival of the human race as well as attraction to human relationships that enrich life. Here we also learn to rely on rational cognitive processes. People have been known to get into serious trouble when they seek

sexual satisfaction directed only by their innate sensory sexual arousal. We learn when, where and with whom to enjoy the pleasures safely. Some of the early research into the pleasure centers actually predated modern day imagery technology. Dr. James Olds (1956) carried out some of this groundbreaking research while a student at McGill University in Quebec, studying with Dr. D. O. Hebb. His studies involved the rat brain, but we have similar structures in the human brain. He found that rats would spend hours pressing a lever that delivered electric impulses through electrodes implanted in a specific brain structure. These rats would ignore usually attractive stimuli like foods or presence of a female in heat.

The pleasure centers support innate survival goals, and we are born with some innate attractions that promote survival. During infancy, and in a safe environment with nurturing adults, what is innate is likely healthful. As we grow and encounter other environments, our innate tendencies are not adequate to safely satisfy our wants and needs. However, we are endowed with capacities in the brain for learning to distinguish safe ways to find pleasurable experiences. The plasticity potentials in the brain allow for wiring changes as we learn how things work in our world, and what conditions and actions safely activate the pleasure structures in the brain. The brain is wired to provide emotional support and reinforcement for learning that leads to effective coping with the world. Effective means learning those thinking and behavior habits that further the goals of that brain. Nobody seeks to have electrodes poked into the brain to assure repeated pleasurable stimulation. However, the world does present drugs that bypass the inherent neural pathways by which certain learnings light up pleasure centers in the brain. These agents achieve experiences of pleasure more quickly and more certainly than do learning efforts. They can impair the natural capacity of the brain to participate in adaptive learning activities.

In summary, the brain functions as a command-control system designed for specific goals of promoting survival, avoiding harm and seeking pleasure. However, it is not a fixed system like a thermostat that simply processes input from the environment and issues a programmed response. That system converts information about temperature to a heating unit and signals turn-on or turn-off. The brain does much more than process incoming stimuli at one end and order a response output, without changing itself. The brain can make physical changes in its system as a result of interaction with its environment. Changes in wiring networks, firing patterns and even adding new wiring units are all capabilities of this system. The brain also develops a store of knowledge about what behaviors can produce these kinds of changes, in its own physical structure. In this elaborate

system the brain can, and does, accomplish a great deal of learning in the absence of conscious awareness or intent to learn. It is part of what the brain does to optimize its potential as a system. Some of this learning is healthful, but some is not. Experiments by Pavlov produced learning where the subject dogs had no intention to learn to salivate to a bell. Gantt (1944), the visiting student from England, caused a conditioned response in dogs who had no intention to learn to respond with fear to the sensory stimuli of a cage. A very early study extinguishing a fear response was conducted by Dr. Mary Cover Jones (1924). A pioneer in behavior therapy, she guided a protocol to extinguish a fear of rabbits in a three-year old child named Peter. She produced the fear extinction by gradually bringing the feared rabbit closer to Peter while he was given his favorite food. He progressed to where he could actually touch the rabbit with no sign of fear. Peter was too young to deliberately choose to collaborate in this brain changing process. Yet a stable learning outcome ensued.

A most dramatic example of plasticity is the outcome picture for children who have undergone hemispherectomy, a surgical procedure which involves removal of half the brain. Who would expect a child with half the brain removed to even survive? Yet, pediatric neurosurgeon Ben Carson, at Johns Hopkins hospital, has a record of over 100 such operations. This surgery is typically undertaken with children suffering life-threatening seizures that were not responsive to medications. Surprisingly, for many there appeared to be no losses in intellectual functioning despite loss of half the brain, in young children. Although there are some physical impairments in the side opposite the excised hemisphere, physical and occupational therapy and speech therapy support a rewiring of the remaining brain. The result in young children is that most are able to get around without wheelchair or walker assistance (Vining et al, 1997).

For the psychotherapist, conscious awareness and intent are an intrinsic feature of the learning process in our clients. Consciousness is a phenomenon *sui generis*. The experience of consciousness cannot be reduced to observed electric and biochemical activity in the brain. Consciousness is unique in that there is no way an observer can observe someone else's conscious experience. The only path to access another's consciousness is communication and trust. This is the stuff of psychotherapy. A client's engagement in psychotherapy can be described as self-directed neuroplasticity, with the help of a skillful and knowledgeable consultant. This engagement involves conscious deliberate decisions that switch on specific brain regions. When we set out to deliberately learn something and focus conscious attention, there are specific brain regions that

light up electrically (Passingham, et al., 1997). Once a level of learning has been reached, the brain can take note of the stimulus situation and set off the learned responses without conscious awareness. In Passingham's study, the subject had to learn a complex sequence of buttons to tap through a lengthy trial-and error process. He was asked to practice until he could effortlessly perform a correct sequence. During the learning process numerous brain structures were intensely active. After the subject had thoroughly learned the sequence, and could smoothly and repeatedly tap out the correct sequence, only the motor regions of the cortex were still active. However, when asked to pay attention to his learned behavior, the subjects PET scan showed renewed activation in the prefrontal cortex and anterior cingulate. The mindful focusing of attention yielded specific regional activation of electrical energy in the brain.

Earlier, I described examples of autonomic conditioned learning that can occur without conscious awareness or intent. However, they provide protocols that can be employed by a person who makes a deliberate conscious decision, using rational, empirical knowledge about how to reset the brain. Neuroplastic changes can affect autonomic emotional responses, reality-thinking about how things work, and the development of behavioral skills. Most clients come to therapy with complaints that have to do with autonomic matters, typically distressing emotions like anxiety, depression and anger, and a sense of losing control. Learning behaviors and thinking that reset brain functions, and help a client cope effectively with environmental challenges, empowers that client, and provides realistic confidence as well as healthier brain function.

Dr. Joseph Wolpe (1990), a behavioral psychiatrist, was a leading therapist in the U.S., applying a learning model to treat pathology. Although he was practicing and carrying out research long before widespread use of brain imaging tools, his theories were remarkably congruent with modern neuroscience findings. He argued that learned responses did not just fade away. He actually conducted experiments supporting the thesis that a response would only disappear when there was a stronger competing response. Translation would reflect Zull's (2002) observation that when there are two competing neuronal networks, the stronger response network will prevail. Wolpe was a major figure in applying a practical model for treating phobias and other anxiety disorders. He employed a process for "systematic desensitization" of anxiety-arousing stimuli. The competing responses he used were practices to produce deep muscle relaxation. He coached his patients with a guided progressive muscle relaxation exercise (Jacobson, 1938). His therapy, pairing deep muscle relaxation with anxiety-arousing stimuli produced more significant benefits,

than did either relaxation training alone or repeated exposure to the anxiety stimuli without activating the relaxation response. These effects confirmed earlier research with phobia treatment (Rachman, 1965). When the neuronal network triggering relaxation responses, competes with an anxiety network that is at a weaker level, the relaxed response will dominate. A network gets stronger with repeated stimulation. The neuronal network for relaxation strengthens with repeated stimulation practice. Strengthening refers to recruitment of increased numbers of neurons to the network.

One caveat to this principle is that only what stimuli the brain recognizes as important will cause these increases in neuronal numbers. The experiment by Kilgard (1998) demonstrated the effects of this principle with rats. Repeated presentation of a buzzer failed to produce noticeable increases in neuronal responses. It seemed that the animals simply got habituated to the stimulus. However, when radio waves were beamed to another structure, deep in the brain—the nucleus basalis—the repeated sound stimuli caused increases in network neurons. This brain structure signals importance when activated. Typical identification of an important stimulus does not come about via radio waves. However, attention is affected by whether or not a stimulus is seen as important. Relevance for survival, anticipation of pleasure or threat of harm will be seen by the brain as important. At birth the number of relevant stimuli is very limited. As we grow and develop, we learn to identify more stimuli as important. A teacher or coach or therapist can work with a student or client to help them appreciate the importance of a learning activity. Two ways neuroscience discoveries contribute to practice in psychotherapy are that they help us understand why some historical practices succeed in helping clients relieve distressing symptoms, and they provide a basis for development of new strategies and interventions in practice. They also provide clients with realistic confidence in mastering skills for more effective control of their lives. We increase our understanding of how learning happens and persists in both cognitive and emotional spheres.

Understanding how the physical brain can change itself, and how it responds to influences from the environment clarifies why some interventions are more likely to work in alleviating pathological symptoms. We can look at different "schools" as providing arrays of different teaching/learning tools, rather than competing theories about what is really wrong. All of the "schools" can be seen as providing guidance for focusing conscious attention, and for practice with different behaviors. This appears, whether it involves asking a patient to shift from talking about

an experience to physically acting that experience, or to participate in a psychodrama role-play episode, or to engage in a self-hypnosis activity, among others.

Neuroscience discoveries provide inspiration for both therapists and clients to approach psychotherapy as self-directed neuroplasticity; it provides them with practical tools to accomplish desired changes. This latter impact is illustrated in the work of Dr. Jeffrey Schwartz (2002) and his team in the Psychiatry Department of UCLA. The study is an applied research project for treating patients diagnosed with OCD. The design of strategy and tools for corrective learning drew heavily on neuroscience studies. So far as I know, this project reflects the most thorough set of applications of neuroscience findings in modern psychotherapy literature. Data from this biological discipline identified specific brain structures and how they are implicated in the behavior symptoms of OCD. These neuroscience studies also helped guide process interventions, with clues for activating and directing relevant neuroplastic brain change.

One brain structure involved in the symptoms of OCD is in the underside of the frontal cortex, called the orbital frontal cortex. It doesn't send cognitive messages, and works more like an error detector or warning that something important is not right. It probably plays an important part in survival and avoiding harm when it is working properly. You will find a brief discussion below of two neuroscience studies that illustrate this benefit. One of the studies was conducted with monkeys, the other with human subjects.

The study with primates (Thorpe et al, 1983) involved training them to lick the end of a tube whenever they saw a blue light. They were rewarded with a trickle of berry juice every time they responded to the blue light. After the learning was well established, the experimenters changed conditions. They proceeded to substitute brine for the berry juice. Monkeys really love the taste of berry juice; on the other hand, they find the taste of brine quite offensive. The behavioral reactions to the brine substitution were a great deal of agitation and other signs of distress. In the brain, the Orbital Frontal Cortex (OFC) showed a dramatic upsurge of metabolic and electrical activity. This level of activity persisted until the monkeys learned a new system when responding to a green light brought a berry juice reward. This OFC arousal did not occur when brine was offered to monkeys with no prior training linking blue light to berry juice. It clearly had to do with "something is not right," rather than with the distasteful quality of brine.

The study with human subjects involved two groups. Each group had to select playing cards from four decks. Neither group was informed that the four decks were "loaded," so that

selecting from two of the decks yielded average losses, while selecting from the other two decks averaged winning monetary outcomes. All of the participants eventually settled on the winning decks.

One group of subjects had intact OFCs. They shifted their strategies earlier, showing measurable signs of stress, as they continued to play, whenever they reached for a card from a losing deck. Although the subjects were shifting their choices, they could not verbalize any conscious awareness of why they were altering these choices. The other group of subjects in the study were missing an intact OFC and shifted their choices more slowly as they gained conscious awareness that there was a difference in winning odds between the decks (Bechara et al., 1997). In OCD patients, this brain structure is not functioning properly, is overactive, and is signaling strongly with many false alarms. A major interest in this study was to explore the effects of guided learning experiences on resetting electrical and metabolic activity levels, without the use of medication. Dr. Schwartz is a psychiatrist and does make use of medicines in his practice. In this study he wanted to determine how learning experiences alone could affect the brain. One of the ways Schwartz built on knowledge from neuroscience, was in framing the therapy goals and process in terms of a collaborative effort; he showed patients their (f)MRI scans, indicating the area in the underside of the cortex, the OFC, and explained that this area functions as a kind of error detector. When it is working properly, it sets off an emotional signal that can be a healthy asset. It signals that something important is not right, even before there is clear cognitive awareness of what is wrong. He showed them that in their brains, this structure exhibits above-average metabolic and electrical activity. It was hyperactive, signaling "HAZARD, HAZARD," with no way to figure out what had to be fixed. He pointed to another high activity area that is a kind of communication hub, the striatum, that receives and routes signals from the frontal lobes, the OFC and the deep limbic structures. He explained that this structure works like a kind of automatic transmission, allowing easy shifting of thoughts and behavior. With overly-high electrical activity, it starts acting like a sticky manual shift; their brains were exhibiting those high levels. Schwartz pointed to other elements in the brain images that added juice to the impact of the error signal. He showed participants activated pathways to brain regions that govern reactions in the gut, explaining this is why such gut-churning sensations persist until they act out the compulsive ritual. He oriented his patients with four assertions:

1. Your brain is driving your emotions and behavior, and is confusing your thinking;

2. If you try to cope by fighting your brain, you can't win for long;
3. Your mind is more than your brain—you can train your brain;
4. The good news is that a) you can train your brain and, b) the therapy team will guide you and coach you.

They were guided with four specific steps toward this re-training:

1. Re-label: "These obsessions are not my thoughts—what appear to be urges are really brain glitches"—faulty brain work;
2. Re-attribute: "What I am aware of is misfiring in my brain's worry circuit, not some urgent problem";
3. Re-focus: After some weeks in the program, patients were instructed to plan a time-limited activity they would engage, temporarily postponing the compulsive ritual. After this activity, they could allow themselves to act out the compulsive ritual if they wanted to. The activity was to have two qualities: it would be pleasurable and it would be healthy—the activity could be singing, dancing, knitting, gardening or any number of other options. They were to engage in the activity in a mindful way for 15 or 20 minutes (mindfulness meditation is discussed in the next section) then they were to give themselves permission to act out the compulsive ritual if they wished;
4. Re-valuing: My understanding of this step is appreciating the power to manage and train the brain. This goes beyond the issue of reducing distressing symptoms to appreciating new-found ability.

During the first two steps, for several weeks, Schwartz instructed patients to avoid fighting the compulsions. Success consisted of simply telling themselves what was going on in the brain. They were to verbalize understanding that the obsessive thought and the compulsive "urge" were indicators of misfiring in the brain rather than a real "must do." Most interesting is that a number of participants reported that putting that thinking into spoken words allowed them to refrain from the compulsive behavior without much difficulty. The cognitive act of re-framing the experience affected neuronal activities, lowered levels of tension, and allowed participants more leeway in responding to obsessive thoughts.

Early in the program Schwartz and his team introduced participants to the practice of mindfulness meditation. They provided ongoing coaching and troubleshooting for this practice.

This intervention yielded multiple benefits in a therapy process where patients would be consciously following a learning protocol for desired biological brain changes.

The practice with this kind of meditation involves taking on the attitude of an observer of products from the brain. It implicitly induces a degree of conscious emotional distance between the person and his brain. It reinforces a sense of deliberate conscious decisions and actions that redirect the brain's attention to a desired focus—the physical sensations of breathing. This focus is directed by the conscious decision to accept the therapist's suggestion. It does not emerge from a spontaneous brain urge to anchor attention. The practice is structured in such a way that there is no way to fail. Part of the instruction is to expect that the brain will drift its attention away from the breath to awareness of thoughts or sensations, away from the breath. They are advised that it is natural that this drifting will not be noticed immediately. They are also told not to try to prevent this natural wandering of the brain, and to refrain from struggling against the wandering. What they were asked to do was to note in their minds where the brain had wandered, before gently bringing attention back to the sensory stimuli of breathing. What the person learns is that he can use the power of his conscious will to direct attention to a desired focus. You cannot force a sustained attention focus by fighting the brain's tendency to wander. However, as you consciously and deliberately stimulate a shift to a desired focus, your brain recruits additional neurons to the relevant network. As that neuronal network becomes stronger it will upstage competing networks. The measure of successful practice is not how long focus stays with the breath. Success is repeated deliberate attention shifts after awareness that attention has wandered from physical sensations of breathing.

This practice was not presented as a weapon to fight obsessive thoughts or compulsive behavior. Rather, it freed an emotional load for the person who has been battling OCD distress, typically for many years. The practice of an unfamiliar, yet calming and non-threatening activity was also helpful for loosening the "brain lock" which is a feature of the OCD biology. The striatum, which was discussed earlier as a center of a rich and complex neuronal communication network, is receiving signals from rational cortical areas, from the OFC error detector, and from the limbic system, which plays such a huge role in emotion. This includes signals from that important structure, the amygdala, which elicits emotions of fear and dread. Neuronal signals also go out from this striatum. When it is not working right, its structures interfere with smooth shifts in behavior, emotion and thinking. With OCD patients, the old nemesis is metabolic and electrical

over-activity. Instead of smoothly shifting attention from the distressing obsession, the person gets stuck. A widely and glibly cited example of what causes obsessions is the injunction to try not to think of a purple elephant. Indeed if you try to "don't think of a purple elephant," you would get stuck focusing on the purple elephant that you don't want to notice.

The reality is that most of us are not likely to get stuck this way. Usually, most people would turn their attention to something more interesting. For some people, the brain is stuck, and not shifting easily. During meditation practice, the person is deliberately interrupting the natural tendencies of the brain's automated program. When they neither follow nor fight the neuronal activity, and simply shift attention, they interrupt the locked-in habit of complying with the brain's automated "trip-tic." And, although metabolic over-activity impairs voluntary deliberate attention shifts, Schwartz

(2002) found that the mindfulness protocol practice contributed to normalizing those metabolic activity levels. Causation works in both directions.

Paradoxically, the way the brain lets us perceive an object with richness and clarity, is not by locking eyes onto that object. In her book, *The Power of Mindful Learning*, Ellen Langer (1998) discussed how we perceive a clear visual image. The process of seeing a stable image involves a lot of shifting eye movements. In fact, if you force your eyes to lock onto an object, and prevent any eye movement, the image becomes blurry. The brain just seems to direct eye movements naturally with no need for conscious decision. However, this natural brain interaction with the environment can be enhanced by recruiting conscious willful choice. Langer used this potential in an experiment in which students in an art class were asked to look at a painting. One group sat in one place looking at the painting. Another group was asked to walk back and forth while looking at the same painting. The latter group noticed and remembered many more elements in the painting, than did those who sat in one place.

The simple experience of wandering awareness carries nowhere near the emotional load of obsessive thoughts and compulsive "urges" experienced by those struggling with OCD. Steering the brain's attention is not an emotionally loaded task in the mindfulness meditation. Instructions include the idea that you not try to exert your will to hold on to a pleasant thought or image, nor do you try to force negative images from your awareness. The process involves accepting and noting whatever your brain has brought to your conscious awareness, before gently shifting your attention to the here-and-now sense experiences of breathing. The process of repeated deliberate shifting of

attention appears to loosen the activity in the striatum. As behavior symptoms subside, electrical activity levels normalize in this area, as well as in other overactive structures.

Although the meditation experience does have a calming effect, this is not its main purpose. As a learning system, the brain is impaired by whatever obstructs the flexibility to shift attention or to provide exposure to new experiences. This is not a deficiency limited to OCD. It impairs the ability to self-correct when encountering new challenges. It is probably implicated for many people who suffer from other chronic pathological behaviors. The repeated practice with deliberate conscious shifting of attention appears to loosen the sticking effects of the overactive striatum, as metabolic activity levels normalize along with reduction of OCD behavior symptoms.

A unique feature in the study by Schwartz and his team was gathering data on changes in observed behavior symptoms, self-reports of emotional responses and thinking, and on the biological brain changes exhibited in imaging. The monkeys in Thorpe's (1983) experiment solved the problem of correcting the "something is not right," by learning to both refrain from licking the tube when seeing the blue light, and to get the reward by responding only when the light was green. Our OCD patients have locked in on a ritual act that reduces a very noxious emotional experience of anxiety. They are usually not aware of how this circuit started originally. It probably did not involve some "who took my berry juice" kind of crisis. It might have involved a non-cognitive amygdala reaction, and searching through memory would be fruitless. The good news is that we don't have to know this history in order to help the OCD victim restore healthy balance in the brain structures that have become impaired. In fact, going back into a patient's history could prove to be a distraction from the tasks involved in correcting the imbalance, which involves a here-and-now process in the brain. This process is especially responsive to a self-directed learning effort. With OCD patients, an impairment in normal brain function is blocking the kind of shift that could produce an adaptive learning outcome.

Anxiety can be a most aversive experience. A cycle of anxiety-avoidance-relief, or anxiety-ritual-relief can be self-perpetuating. Relieving anxiety can be a powerful reinforcer of a behavioral response. However, it is a mistake to think of an illness like OCD, or of phobia, as fear of anxiety, and of treatment as learning to tolerate anxiety. Our anxiety circuits play a vital role in survival. They can signal a variety of threats to vital goals of the brain. In addition to signaling physical dangers in the environment, anxiety sensations serve as a signal of acute hypoglycemia to an insulin-dependent diabetic. What is usually referred to as an insulin reaction, involves the same

adrenalin response we see in other events where a person describes sensations of anxiety. These usually include feeling shaky, sweating, and awareness of increased heart rate. These sensations abate shortly after ingestion of a high glucose food. Since the dangers of dropping blood sugar are so great, any insulin-dependent diabetes patient has learned to recognize signs of "insulin reaction." He knows the importance of a fast-acting snack like orange juice or glucose tablets.

A problem that surfaced during a major diabetes study of the benefits of tight control, was the number of participants who were not mounting the adrenalin reaction early enough to correct a hypoglycemic trend. In the absence of distress signals that blood sugar levels could be getting dangerously low, they can progress to severe reactions like coma and seizures. Impaired cognition and increased adrenalin can even lead to very combative behaviors.

Hand in hand with tight control comes a resetting of the hypothalamus. Blood glucose levels must drop much lower before anxiety is triggered. The delay of the anxiety reaction is not a blessing for the diabetes patient. The reaction is an alarm signal, and the drill is to eat something to raise your blood sugar, not to get rid of anxiety. The diabetes patient does not think of the candy bar as an antidote for anxiety. Eating something sweet raises blood glucose from dangerous to safe levels. Once the hazard is corrected, the alarm turns off. If the alarm delays until glucose levels are much too low, the diabetic is no longer able to think clearly and to seek needed sugar.

You have probably seen films of police training in simulated crisis situations. They suddenly confront figures of bad guys or innocents, like a mother and child. They have to make quick decisions like shoot or hold your fire. The idea, of course, is to act in a way that keeps you alive, disables the bad guys, and does not cause harm to innocent bystanders. In the FBI training course, they did find that too high a level of anxiety led to increased errors of commission, like gunning down a silhouette of a mother holding a baby. However, too low a level of anxiety arousal produced more errors of omission, like not noticing the sound of a gun being cocked. Anxiety is not a design error or defect. It is a valuable capability. Fear, like pain, and other unpleasant emotions and sensations can be a lifesaver, signaling danger.

It is true that alarm signals can sometimes mislead us. Fear response might trigger because of misperception of an event. It might be due to chronic habits of tensing muscles or poor breathing activity. In those situations the signal is not much help for identifying a threat in the environment, but corrective learning is not aimed at tolerating or dulling the capacity to feel

anxious. When fear is aroused because of poor habits and self-stressing, we can learn how to correct the habits that are causing the unhealthy stress.

Anxiety itself is not a disease. Some level of tension enhances performance. The Yerkes-Dodson Law (Yerkes) was proposed far back in 1908, but still holds validity to this day. Yerkes described an inverted U curve, in which some level of anxiety improves learning and performance, but beyond some level impairs performances like memory, attention and motor skills. For some people, problems from anxiety feelings are increased because they become anxious about feeling anxious. This is happening because the feelings make no sense to them. They feel the emotion when they see nothing that should make them afraid, but have a pervasive feeling of being out of control. If only someone were firing a gun at them, all would be well emotionally. For those who have suffered the extreme anxiety of panic, even low levels of anxiety trigger expectations of uncontrolled escalation.

Therapeutically, it can be beneficial for these folks to learn that the normal brain has structures that trigger emotional responses in ways that don't involve rational thinking, e.g., the amygdala. It is helpful to learn that this structure is not responsive to rational argument or to forceful exertions of willpower. It is also reassuring to learn that the emotional brain systems can be trained to change how, and to what they respond. The message is not that you don't have control over your emotional reactions; it is that you have not learned what kinds of control work. If you are first learning to fly a small plane and want to fly to a higher altitude, you will exhaust yourself, and actually stall your engine no matter how much, or how often, you point the nose of that plane up. You will also experience some definite emotional upset. After you learn that increasing acceleration takes the plane to a higher altitude, you will achieve a real sense of control, even if you don't understand all the details of aerodynamics.

The desensitization paradigm is one vehicle for successful retraining of the brain. It also builds confidence in the effects of using the right tools for emotional control. In this process the client learns how to deliberately arouse a targeted low level of anxiety, and how to reduce it. He learns through experience, that any level of anxiety is not automatically an express elevator to uncontrolled panic. With more conscious control of the experience, there is less fear of the emotion itself. We are wired for amygdala arousal when there is any perceived threat to control. At low levels of anxiety—feeling warm all over, increased heart rate, sweaty palms, slight tremors, feeling muscle tension—we can all do a wide range of tasks competently. When legs are wobbly, with

strong muscle tension, headache, dizziness, nausea, headache, visual distortion, feeling spacey and frantic, then it can become virtually impossible to do anything other than try to escape the threatening environment.

When we frame therapy as a learning process, we don't need to show brain scans to convey the notion that the brain might be running some automated drills that affect your behavior, thinking and emotions, and that the brain is not designed to change everything it is doing in response to force or rational argument. However, a client can empower himself or herself, using instruction and coaching on how to rewire and reset that brain. Analogy with a computer operating system is another helpful way to frame the process. You will feel, and for good reason, positively powerless, if you are trying to use a computer without some basic knowledge about how the operating system uses what is put into it. Knowing something about the brain's working principles, and using effective learning exercises reinforce a realistic sense of control.

Another example of brain re-training illustrates its practical applications. Although Dr. Arthur Hardy's (1981) self-help therapy program for treating panic and agoraphobia therapy protocol developed without explicit reference to modern neuroscience findings, it is highly compatible with what later neuroscience studies have learned about brainwork. His treatment program was geared to help patients learn ways to achieve healthy effective control of emotions with voluntary behaviors to identify and control stimuli that triggered distressing anxiety. He educated them about anxiety circuits and about the stimulus-response principle. He also advised that, although that first panic attack seemed to come out of the blue, there were both internal and external trigger stimuli; some stimulus triggered the attack. Most patients had been living for years without conscious awareness of habitually tensing muscles and impairing their breathing. Early in the program, participants were introduced to a Progressive Muscle Relaxation exercise (PMR). It's not that they were oblivious to general feelings of discomfort. Many were exquisitely aware of any signs of feeling anxious, with resulting fear of impending panic. The PMR practice had two benefits. The practice provided some quick feedback in the form of a calming experience. It also helped them to identify specific physical sources of the anxious feelings, along with voluntary physical actions that reduced the impact of those sources.

Deliberately tensing a striped muscle group, then letting go, leads to lower tension in that muscle group. A deliberate slow deep breath is not only calming, it enhances awareness of the contrast with chronic shallow breathing. Although not presented in those terms, these patients were

developing stronger neuronal networks for relaxing voluntary muscles and for more adequate breath work. At first this requires conscious attention to these behaviors. Eventually, these new behaviors, which are actually old natural behaviors, dominate without conscious effort.

In the desensitization protocol, participants are asked to provide a preliminary list of anxiety-arousing situations, and to rate them on a scale from zero to ten. They are provided some additional guidelines in the form of conscious sensory experiences. A very low level might include sweaty or clammy palms, or butterflies in the stomach. A panic level would include experiences like feeling spacey, disoriented, dizzy, nauseous, or suffering diarrhea. Instructions were to aim for a low level which could include sensations like increase of heart beat, or mild tremors. They learned to achieve this by combinations of muscle relaxation, breathing exercises, and stimulus control.

The desensitization protocol develops confidence in controlling anxiety level by controlling distance from the stimulus, rather than by blanket avoidance. It involves physically going *toward*, not to, what is anxiety arousing. It is not necessary, at first, to know any more than the general situation that arouses the fear. Clients need not know the specific stimulus, but they know where it happens. It might be a supermarket, a large crowd, riding in a car or being at a distance from home or from a "safe" person. This is enough for stimulus control, even if they do not know exactly what about the situation fires up the anxiety circuits. Instructions are to go in the direction of the feared situation until reaching a low level (three) of anxiety. Clients are advised that there is no learning benefit to tolerating a higher level of anxiety. They are not to try to win a contest with the brain. When reaching that level three, they are to retreat, physically stop, or slowly turn and walk away, just until some easing of the anxiety. They then follow the relaxation skill exercise. Patients are advised that there is a brief recovery period in ratcheting down from a spike of anxiety, with some small blips of anxiety increase as the overall move is down. They are asked to use this time to identify what stimulus triggered the rise in anxiety. They do this by asking what they were aware of just before they became conscious of the anxiety increase and to write their observation; it might be a thought or something they perceived. If they could not recall anything specific, they could write "nothing." There would be other opportunities, as they practiced several times a week. The last step is to repeat the cycle at least once. They are to turn back *toward the* stimulus situation. Success is defined as completing a learning cycle with at least one repetition, not by how

close they get to the feared stimulus. A key principle that enhances control is the idea of SLOW. Moving slowly lowers the risk of suddenly feeling a surge of anxiety.

With repeated practice patients were able to get closer and closer to the feared situation with increased comfort levels. In this treatment protocol, patients were provided a mnemonic RRRR: React, Relax, Recover, Repeat. This part of the learning program retrained autonomic responses. Cognitive exercises reduced the "what if ..." phobic thinking impact on amygdala activation.

President Franklin Delano Roosevelt's admonition that "The only thing we have to fear is fear itself" may have sounded good, but it really makes no sense. It makes about as much sense as asserting "The only thing to fear about your smoke alarm is the noxious sound of the alarm." People may argue about the origins of the brain—if it was created by an intelligent designer or emerged through an evolutionary process—but there is no question that the resulting brain conducts a complex and intelligent process. The brain is organized as a learning system guided by goals. It uses both emotional and cognitive information in pursuit of its goals.

### **Implications and Applications to Psychotherapy**

Integrating what we have learned from neuroscience discoveries is not a matter of looking for a good fit with Multi-axial symptom clusters. I suggest these findings are best used as a complement to the DSM system. The DSM gives us clusters of symptom targets for change. Neuroplastic processes can guide our strategies for effective learning experiences that apply in a wide range of syndromes. There are numerous benefits from the very idea of neuroplasticity:

- Real sense of empowerment for clients; importance of learning that you can change this brain that is not acting right;
- You can do this by engaging in retraining exercises. Three encouraging messages are:
  1. Your brain is not working right, and this is what is causing your distressing symptoms;
  2. You have the power to re-train your brain;
  3. We will provide instruction, coaching and guidance to help you use that power.

What Schwartz and his team applied to help patients suffering with OCD can benefit patients struggling with other distressing symptom patterns, and the therapists who are helping them. This orientation is helpful, of course, if the therapist does provide information and guidance that confirm the above three assertions. Diagnostically, it would help to have a system, complementing the DSM, to identify learning deficits and goals, as well as likely brain regions that are malfunctioning. The DSM presents pathological behavior syndromes for diagnostic assignment. We could benefit by making available some of the neuroscience findings about the working brain, to direct corrective psychotherapy interventions. At this time we have no widely available resources for administering or reading SPECT scans. It is not always necessary, or even desirable, that all psychiatric complaints be diagnosed with a brain scan to plan effective treatment. Although radiation levels are reported to be low we would always prefer to avoid any such exposure when possible. Such scans are likely to be particularly helpful when treatment seems to be going nowhere, and we might be making faulty assumptions about what's going on in the brain.

We are already several steps ahead when we think of observed behavioral symptoms in terms of how they relate to brain region malfunctions. We might consider impairments that could be interfering with the brain's potential for adaptive learning. Schwartz's description of how metabolic hyperactivity in the striatum (Amen refers to an overactive cingulate gyrus, which is part of the striatum, as the source of the kind of brain-lock Schwartz describes) interferes with the ability to shift thinking and attention smoothly. If you are working with a client who seems to have this kind of difficulty, it could be an indication for a specific learning intervention. With all the talk about evidence-based interventions, there is a danger of *a priori* elevating statistical findings of published studies above the evidence of your own senses or the senses of your clients, when you apply interventions. There are some assessment tools that I have found helpful for hypotheses about what brain structures might be involved, and in what way. Dr. Amen's clinic markets a diagnostic checklist, which is scored for impairments in different subsystems of the brain. This instrument has no validity studies to support the scoring as yet. However, UC-Irvine is in the process of conducting validation studies (personal phone communication). The instrument does infer specific brain system impairments from observation of behavioral patterns. It can suggest psychotherapy strategies that impact such impaired systems, strategies that are not evident from a DSM diagnosis alone. Although the instrument does not yet yield a definitive conclusion of specific system impairment, it can supplement clinical judgment that takes into account other data

and keeps us mindful of how the therapy process affects workings in the brain. I have found Lazarus' MultiModal assessment system (1981) helpful for choosing learning goals and interventions. His Behavior, Affect, Sensations, Imagery, Cognitions, Interpersonal Relations and Drugs (BASICID) system lists areas of experience that can be addressed in therapy. The term "Drugs" includes all physical health-related factors, including diet, exercise, diseases, medications, and street drugs. Each of these experience-arenas can be an identified problem or, except for Affect, an intervention focus. By intervention focus, I mean a person can effect a change with a conscious intentional act. Affect is always the result of intervention in one of the other elements. Even in the brain, when we talk about the amygdala—that non-cognitive structure in the old brain that generates fear or dread or rage, we have seen that some raw sensory stimulus can activate it, or it can be aroused in response to messages from the frontal lobes that have perceived a threat in the environment. Something has to physically connect with the emotional structure. Each of the other experiences in Lazarus' list can be activated or changed by conscious decisions to focus or shift attention, or to carry out some behavior. Focusing attention and practicing voluntary behaviors are the only assets we have for deliberately improving how our brains will work.

We engage in helping our clientele to consciously use their powers to direct attention, and their ability to practice behaviors that are in voluntary control, toward certain ends; we want to help them reduce stressful destructive autonomic responses, and learn to practice thinking and actions that support goals of the brain: survival, avoiding harm and finding pleasure.

### **A Partial Summary of How Awareness of Brain Plasticity Impacts Therapy Practice**

- Effective therapy is successful learning experiences;
- The brain is a goal-oriented system;
- Brain goals are survival, pleasure, and avoiding harm;
- Pathology results when brain ability to promote goals is impaired;
- Two kinds of learning impairment:
  1. Learning content deficit, e.g., lack of assertive skills;
  2. Impairments in the learning process, e.g., brain lock, false emotional alarms;
- The therapist supports learning with inquiry, instruction, and/or coaching;

- The patient applies knowledge about how the brain learns;
- The origins of impairment might be genetic or environmental;
- The critical question for therapy is not history, but the patient's capacity to learn;
- *Neuroplastic learning* can restore healthy functioning
- There are two kinds of learning systems:
  1. Cognitive—rational thinking and testing;
  2. Emotional/autonomic—conditioning and extinction;
- Corrective learning does not require revisiting disturbing origins, although perceptions of history can sometimes clarify present learning needs;
- Not remembering is not necessarily repression;
- The amygdala can learn without input from cortical cognitive brain structures;
- The past cannot affect the present, though memory does; memory activity is itself a present event;
- Episodic memory is not reliable. It is assembled, not retrieved;
- Neuroplasticity is a here-and-now learning process;
- Neuronal network responses can't be erased;
- When two networks compete, the stronger response dominates;

*There are only two assets for self-directed neuroplasticity:*

1. Directing focus of consciousness/attention;
2. Consciously directing voluntary muscles.

This book is offered as a vehicle for integrating neuroscience findings of how the human brain works with practice in psychotherapy. The overall premise is that the brain is a learning system with command/control functions over behavior, thinking and emotion. Learning involves biological structures and processes designed to further certain goals. Psychotherapy provides consultation for enhancing mental health. The benefits of all psychotherapy come from supporting and guiding an educational experience. Academic educators have been designing learning experiences with strategies and tools for improving academic learning. There is an abundance of research and website sources on which teachers can draw. Some of these are listed in the Reference section. There are also computer programs designed to improve the efficacy of how the brain works by exploiting its plasticity potentials. One commercial enterprise was cofounded by Dr. Michael Merzenich whose

work is cited earlier in this book, and who is one of the early pioneers in study of neuroplasticity. Some of his brain training programs have been applied in treatment to enhance cognitive performance of patients with schizophrenia. It seems like an appropriate time for psychotherapists to explicitly apply biology findings in our work to help clients learn healthier ways to function. In addition to the fund of biology research, we can draw on the kind of model found in Schwartz's work, for specific practical applications of brain—compatible learning interventions.

I deliberately avoided graphic anatomical pictures of the brain in this book. As psychotherapists, we do not physically touch brain parts. My focus has been to present a description of some important structures, their functions and how they both affect, and are affected by mental and behavioral events, and especially how the mental process of consciousness causes biological brain changes.

As therapists we can help our clients use their powers of consciousness to learn healthy behaviors and thinking that promote survival, experience of pleasure and avoidance of harm. Sometimes this learning involves new skills for achievement of desired goals; sometimes it involves undoing impediments to learning in the brain like the "brain lock" Schwartz describes.

### **Therapist Considerations: Connecting, Rapport, Trust**

- Relate in terms of prior experience, including expectations about therapy. Although learning is a here-and-now process, clients' pictures of therapy might include an opportunity to tell their personal stories.
- Helping clients learn effective controls builds confidence and trust. Threats to control are amygdala-arousing; bolstering a sense of control is calming.
- Acknowledge distressing feelings, and segue with guidance for shifting attention. This can rouse interest, curiosity, and calms the amygdala when the client can deliberately shift attention to a non-threatening action (such as shifting from talk about feeling edgy, keyed up, or nervous, to noticing specific body sensations which a client can deliberately manage, like consciously tightening a specific muscle group, then letting go. Choose a behavior you know the person can do successfully.

Coach in mindfulness exercise, an exercise which is a very effective experience for learning to deliberately steer attention, and free what Schwartz refers to as "brain lock."

### **Miscellany of Neuroscience Considerations**

- When emotional reactions seem to make no sense, consider the source might be amygdala arousal. This non-cognitive structure learns via a conditioning paradigm. This learning model appears to govern many autonomic processes, and is not limited to emotional systems. Goleman (1995) describes early research findings, dating from the 1970s, of conditioning immune system responses. Educate patients about this non-cognitive brain structure and provide coaching for effective calming skills.
- Psychotherapy benefits come from neuroplastic changes in the brain; these are durable physical, biological changes.
- Learning new healthy behavior does not involve understanding and reversing old maladaptive habits. Once a client has been helped to identify behaviors he or she finds unsatisfying, and wants to change, (sometimes clients are able to describe behaviors they want to change in their first therapy encounter) the therapist can help them describe what they would rather do. More often, clients identify feelings that are distressing rather than behaviors. This can be a good starting point for education about how the brain works and how conscious intentional behavior affects emotions, and electrical and chemical processes in the brain. A key consideration is that new behavior will be incompatible with the old habits. As with any teacher or coach, a critical contribution is providing a first step for change. An example is providing assertiveness training for a client who is both anxious and depressed about feeling at the mercy of other people in social settings. Assertiveness training groups provide a safe setting for learning to use and repeat assertive words and sentences, with increasing comfort. Relaxation skills might be an additional beneficial skill. Self-talk practice with Affirmation scripts is another intentional behavior that can upstage habits of negative pessimistic thinking. Giving a client an assertiveness script to

practice in a challenging situation will probably not be much use. Role play in a safe setting will still produce some anxiety initially, but will be less emotionally charged, and repeated practice will reduce the anxiety. Gradually raising the level of challenge increases both skill and comfort. Clarifying how or when or why maladaptive habits were learned is not much benefit for change, and can even reinforce a belief that one is strongly controlled by outside forces. This doesn't mean you refuse to listen to history the client believes is important. Actually some clients are relieved about not rummaging about in the past, and are happy for guidance in trying out new ways of acting. As Zull pointed out, the therapist *cum* teacher is not communicating to a blank slate. There is a structural store of past experiences. A lot of people do spend time thinking about past experiences and how they affected them. Having another person listen and understand builds trust. Although details about the past events might not be relevant to healthy functioning today, it is realistic to believe that some of what happened before might have lasting impact. Where the therapist can help, is explaining how past learning and genetic effects cause durable processes in the brain, but intentional behavior and thinking can also bring about desired brain changes. The client's history might provide clues to what he is experiencing today. Listening to a client's story is one way of getting to know how that person thinks about his experiences, and how he perceives his current problems. It also can be an important step in developing a trusting relationship. These interchanges are good sources to answer questions that are critical as, e.g., What feelings or behaviors would you wish happened less often and what would you want to be able to do or feel more often? Don't expect ready access to SPECT scans soon. However, observations of behavior, and reports of thinking and feelings can provide valuable clues about brain processes when the therapist is alert to brain issues. For instance, if a client appears to be blocked, with difficulty shifting attention or behaviors, the therapist might consider hyperactive striatum structures. Remedies include finding ways the client is able to do something different.

Healthy functioning does not involve triumph of reason over emotion. The emotional structures of the brain are not included to make life tougher. They are important regions that help the brain do what it is designed to do. When our reason gives us understanding of how our emotions work, we make healthy use of them. It is more than just adding up these two capacities. There is a synergy in this system such that emotion and reason interacting, yield outcomes that are richer than simply adding up what they produce separately.

## **Appendix**

This is a brief historic overview of what is often referred to as "Biological Psychiatry," yet is actually "Biochemical Psychiatry." Explicit formulation of major mental illness as a biological brain disease benefited from a chance occurrence. This surprising finding did not come about in the course of psychiatric research. It was not a product of research into brain biology (Rosenbloom, 2002). The origins involved a French surgeon, Henri Laborit. He was intent on finding a medication that would allow reductions in anesthesia dosing for surgery. Too many of his patients failed to survive surgery. The fatalities almost never resulted from the surgical procedures or from infection. The cause of death was often the anesthesia, and fatalities tended to be dose-related. Trials with a fairly new pharmaceutical, chlorpromazine, yielded impressive results. It allowed significant dose reductions of anesthesia, with marked increase in survival rates. Laborit also noted some additional and unexpected effects of this medication. Patients who had received the chlorpromazine exhibited much less anxiety in approaching surgery than did those who had not received this treatment. Further, some patients with histories of major psychiatric symptoms, including hallucinations and delusions, presented with significant reductions or remissions of these pathological symptoms. These were major events, impossible to ignore. Here was a pharmaceutical agent achieving such impressive benefits. No other interventions had even come close to this level of impact in psychiatric practice.

Laborit discussed his observations with a medical colleague. This colleague suggested they discuss their findings with his brother-in-law, Dr. Pierre Deniker, who was a practicing psychiatrist. Dr. Deniker was sufficiently intrigued, and agreed to test this new agent with some of

his patients. The outcomes generated considerable excitement for this group. They replicated the same dramatic findings that Dr. Laborit had encountered—major symptom reductions that had not been seen in response to any other therapy. The results looked so promising that the two physicians undertook a visit to the United States. They expected to find a fertile field for studying and expanding the use of this medication.

Their initial experience was not very encouraging, and actually quite disappointing. The dominant interest among U.S. psychiatrists was in psychoanalysis at that time. There was little receptivity to the use of drugs or other medications in treatment of mental illness. The French visitors persisted, and found a friendly audience in several state mental health agencies. These folks were especially attracted by the vision of emptying large numbers of state hospital beds, and realizing substantial dollar savings. The marked, dramatic impact on symptoms in a rather short term was most impressive. This was, of course, before the surfacing of equally impressive adverse neurological effects like tardive dyskinesia and extrapyramidal symptoms.

Benefits were so obvious in that initial period. Patients, who had failed to respond to any therapies for many years, were awakening and returning to the real world. Major pharmaceutical companies became interested in the possibilities of this treatment path. Over the years the industry funded a wealth of research projects. The search was on for medications that would effect symptom reductions, while avoiding the adverse neurological effects. Research was also spurred to develop agents that would benefit non-responders; not all patients responded with symptom remission. Theories evolved to identify which biochemicals were responsible for reducing disordered thinking, emotions and behavior, and what chemicals were involved in triggering symptoms.

A neuropharmaceutical industry burgeoned to multibillion-dollar enterprises to meet the growing therapeutic needs of the mentally ill, and this treatment mode has dominated the treatment arena to the present time.

A simplified summary of this research stream is the study of effects of various neuropharmaceuticals on moods, thought processes, and behaviors. This has not been a research flow addressing issues of how to improve the biology of the working brain. Improvement in the biology of the brain has been inferred from observations of reduced behavior symptoms. The implicit and unspoken assumption is that the biology workings of the brain are fixed. Treatment involves external control to affect biochemical levels or electrical activity. When patients are told

that they must comply with a medication regimen for a lifetime, they are often given the analogy to a diabetic's lifetime requirement for insulin. Of course, with Type 1 diabetes, there are no active beta cells producing insulin, and no evidence of biological means to grow or revive those cells. If the brain is not doing well in terms of levels of biochemicals or in terms of electrical activity, and its working processes are fixed, it would make sense that the only alternative for treatment is external control, i.e., neuropharmaceuticals and electric stimulation.

The particular explication of a biological brain disease derives from reasoning backward from the effects of chemical agents on behavior. There is no reference to a model of how a healthy brain works and little consideration of the brain as a system that sometimes self-corrects its structures and electrical firing patterns. Neuroplasticity has not been part of the orienting framework in the practice of Biochemical Psychiatry.

A paradoxical anachronism is the emergence of a sub-group in the discipline of Clinical Psychology to acquire competence and licensure for prescribing neurochemical treatment. With the growing knowledge of the phenomenon of neuroplasticity, it becomes ever more obvious that practices in psychotherapy provide support for self-directed neuroplastic brain change. Neuroscience discoveries are suggestive for therapy education innovations to benefit patients whose conditions appeared impervious in the past. Dr. Schwartz and his team exemplify one set of such innovations in the treatment of OCD.

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<http://www.positscience.com>

<http://www.sharpbrains.com>

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# Neuroplasticity – The Biology of Psychotherapy

## POST-TEST

1. The human brain is endowed early in life with:
  - a. 1,000,000 neurons
  - b. 10,000,000 neurons
  - c. 100,000,000 neurons
  - d. 100,000,000,000 neurons
  
2. Once we have our full complement of neurons, we lose neurons over the years through aging or trauma – we can't get any more.  
 True  
 False
  
3. One source of OCD patients' difficulty letting go obsessive thoughts is overactivity in a specific brain area known as:
  - a. the striatum
  - b. the amygdala
  - c. the orbital frontal cortex
  - d. temporal lobe
  
4. The term referring to the brain's capacity to change wiring and firing patterns is:
  - a. neurogenesis
  - b. hemispherectomy
  - c. neuroplasticity
  - d. homeostasis
  
5. The "taxicology" study found that London cab drivers were different from bus drivers in what way:
  - a. they were more flexible in their thinking
  - b. they had larger hippocampus size than did bus drivers
  - c. their amygdala reactivity was lower than was that of bus drivers
  - d. they exhibited overall higher intelligence
  
6. SPECT scans have been used extensively by Dr. Daniel Amen at U.C. Irvine. This imaging tool is especially important for detecting:
  - a. Dopamine and Serotonin levels in the brain
  - b. Volume increases in specific brain areas
  - c. Nutritional deficits in the brain
  - d. Metabolic and electrical activity levels

7. Stronger neuronal electrical response involves:
- a stronger electrical pulse firing across the synapse
  - increased number of neurons in a network
  - increased electrical transmission to a neuron
  - blocking electrical stimulation of a competing neuron
8. Kagan's study of timid and bold children yielded support for the following:
- genetics are relatively unimportant determinants of behavior
  - genetic behavior source precludes any significant change
  - some genetic tendencies can be markedly reversed
  - there is no way to identify specific genomes that guide behavior
9. Zull has inferred goals of the brain as a system in the list below:
- survival, avoiding harm and seeking pleasure
  - survival and avoiding harm
  - love, dominance and consumption
  - altruism, aggression and pleasure
10. Dr. Schwartz's team project for treating OCD was unique in that it:
- did not limit its goals to reducing pathological behavior
  - applied learning technologies for normalizing brain functions
  - assessed both biological brain change and remission of behavior symptoms
  - all of the above
11. The amygdala is a significant emotional brain structure which:
- triggers emotions like dread and anxiety,
  - responds to cognitive messages from the cortex
  - responds to raw sensory, non-cognitive signals
  - sends signals affecting cognitions in the frontal lobes
  - all of the above
12. Kilgard et al found that a neuronal network is strengthened by :
- repeated stimulation
  - properly selected drugs
  - enhanced perception
  - repeated stimulation only when stimulus is seen as important
13. Wolpe and others demonstrated that phobic behavior reduces more strongly when exposure to the feared situation is accompanied by relaxation learning, than it does with relaxation training alone or exposure alone. These findings support:
- the principle of a flooding intervention
  - Zull's principle that with competing networks, the stronger will dominate behavior
  - the benefits of relaxation training
  - the benefits of psychotherapy

14. All schools of psychotherapy employ interventions to promote learning, by asking clients to deliberately steer attention and practice new available behaviors.

\_\_\_\_\_ True

\_\_\_\_\_ False

15. The discovery of benefits of chlorpromazine came from:

- a. neuroscience brain studies of how the healthy brain functions
- b. search for an effective tranquilizer to treat major mental illness
- c. a chance finding as a French surgeon sought to decrease anesthesia doses
- d. all of the above

16. Anxiety is best seen as:

- a. a pathological symptom
- b. a valuable emotional signal of possible danger
- c. a natural biological reaction, but always impairs performance
- d. always a product of phobic thinking

17. With regard to the emotional structures of the brain, it is helpful to orient the patient

- a. these parts of the brain are not in your control but can't hurt you
- b. the best way to control undesired reactions is stay away from distressing stimuli
- c. planning to live with the reactions will lead to the least stress
- d. none of the above

18. The desensitization paradigm is a vehicle for a client to successfully retrain the emotional structures in the brain by:

- a. learning control of the stimulus e.g. controlling distance from the stimulus
- b. skills to relax the voluntary, striped muscle system
- c. skills to cope effectively with situation demands-e.g. learning to control an automobile and develop driving skills, or learning assertive communication skills to safely negotiate social needs
- d. all of the above

19. Some of the patients in the OCD project reported somewhat easier to resist compulsions successfully in the early weeks of the program, even though instructed that it was okay to act these out, but to label them as misfiring in the brain. This illustrated what important principle?

- a. the safety of the therapy group lowered the anxiety from refraining
- b. a voluntary cognitive act has effects on involuntary emotional reactions
- c. not everybody reacts the same
- d. none of the above

20. In psychotherapy, the therapist supports learning with:

- a. inquiry
- b. instruction
- c. coaching
- d. all of the above

21. Amygdala arousal can be reduced by:

- a. Relaxation practice
- b. Viewing peaceful scenes
- c. Non-threatening cognitive challenges
- d. All of the above

22. Healthy functioning involves :

- a. triumph of reason over emotion
- b. learning to trust your gut feelings
- c. avoiding emotionally distressing situations
- d. none of the above

23. With regard to memories of the past:

- a. episodic memories are notoriously fallible
- b. there is no way to change the past
- c. memory is a here-and now cognition that affects behavior and emotion
- d. all of the above

24. Not remembering

- a. might signify repression
- b. might signify non-cognitive amygdala reaction
- c. a and b
- d. neither a nor b

25. Origins of thinking or behavior don't confirm susceptibility to change, or what will be effective for change.

\_\_\_\_\_ True

\_\_\_\_\_ False

26. The Orbital Frontal Cortex acts as:

- a. a structure for diagnosing a problem or danger
- b. a non-cognitive "something is not right" error detector
- c. a source of brain lock
- d. a signaler of noxious experience

27. Studies of anxiety effects in FBI training exercises found that:

- a. performance increases with decreases in anxiety levels
- b. performance is heightened with anxiety increases
- c. anxiety very low produces more errors of omission
- d. too high anxiety produces more errors of commission
- e. c and d

28. Thorpe's study of OFC effects in monkeys found:

- a. They behaved like OCD patients
- b. The OFC was activated by an offensive stimulus
- c. OFC was activated when positive expectations violated by offensive stimulus
- d. all of the above

29. Bechara's study of OFC effects demonstrated:

- a. how it helped subjects make advantageous choices, prior to cognitive awareness of the advantage
- b. the activation of OCD symptoms in naïve subjects
- c. specific problems with this error detector
- d. none of the above

30. In Passingham's study, the subject was asked to learn a complex series of button pushing. During the learning brain structures involved in conscious experience were lit up. After he learned to effortlessly perform the task, except for motor cortex activity, many of the other areas turned off as he continued to perform. However, when asked to pay conscious attention, the other areas reactivated. This illustrates:

- a. A Conscious willful act to pay attention, switches on relevant neurons
- b. Once some tasks are well-learned, they can go on "automatic"
- c. Willful conscious attention can refocus on previously automated process
- d. All of the above

31. Emotional Hijacking involves:

- a. strong amygdala reaction that overwhelms frontal lobes preventing rational learning
- b. more nerve fibers running from amygdala to frontal lobes than those in the other direction
- c. strong amygdala reaction can block cognitive processing and memory
- d. all of the above

32. Classical conditioning illustrates what principle of neuroplasticity per Zull.

- a. a stronger neuronal network will block the weaker one
- b. neurons that fire together wire together
- c. non-cognitive learning is very ephemeral
- d. all of the above

33. The biology component of mental illness is treated with

- a. medication
- b. nutrition
- c. psychotherapy
- d. all of the above

34. Genetic control of behavior pathology is

- a. modified by illness, medication, physical trauma, learning
- b. unchangeable
- c. an indicator of alterability of behavior pathology
- d. all of the above

35. The learning exhibited in neuroplastic brain change can be a result of

- a. interaction with the environment without conscious awareness
- b. deliberate choice to engage in a learning activity
- c. neither a nor b
- d. a and b

36. Although physical electrical and metabolic activity are involved in turning consciousness on and off, the phenomenon of consciousness is not reduced to electricity and metabolism. Independent observers can see these activities. One's consciousness is only observed directly by the aware person. For others to know what that experience is, requires:

- a. trust
- b. communication
- c. a and b
- d. not relevant because not confirmable by independent observers

37. Conscious recovery of a memory will sometimes be impossible because

- a. repression sometimes is too powerful
- b. the event is too remote in time
- c. amygdala arousal overwhelmed cognitive cortical activity – no cognitive content to the experience
- d. a and b

38. Control is significant factor in amygdala arousal

- a. increase in conscious control calms amygdala
- b. threats to control trigger amygdala arousal
- c. psychotherapy involves learning healthful control skills
- d. all the above

I, \_\_\_\_\_ (name of participant) affirm that I am the person who completed this home study and am responsible for this post test.

Signature: \_\_\_\_\_

