

The Neurobiology of Freud's Repetition Compulsion

Denise K. Shull

The paper develops a hypothesis regarding how the processes of neurological development could underlie the enactment of Freud's Repetition Compulsion. Drawing largely on Allan Schore's work in infant neurodevelopment as it relates to the mother and Joseph LeDoux's study in fear and implicit emotional memory, the paper outlines the mechanisms of neurological growth and operation. It builds a case for the importance of a right brain socio-emotional circuit in the incarnation of compulsions to repeat.

Rita Mae Brown, in *Starting from Scratch: A Different Kind of Writer's Manual*, defines insanity as “doing the same thing over and over and expecting a different result.” Can we say that the experience of unwanted repetitive circumstances is precisely what motivates people to seek help? Isn't this phenomenon precisely what we aim to resolve? “Patients come to us hoping to escape the repetition. ... Understanding the repetition in the transference will eventually lead to understanding the patient's perception of the past” (Meadow, 1989, p. 161).

Freud originally spoke of undesirable repetitions in his 1920 treatise *Beyond the Pleasure Principle*. He described how some people behave not according to the “pleasure principle” but apparently in response to a “compulsion to repeat:”

“...people all of whose human relationships have the same outcome: such as the benefactor who is abandoned in anger after a time by each of his protégés, however much they may otherwise differ from one another, ...or the man whose friendships all end in betrayal by his friend; or the man who time after time in the course of his life raises someone else into a position of great private or public authority and then after a certain interval, himself upsets that authority and replaces him with a new one; or, again, the lover each of whose love affairs with a woman passes through the same phases and reaches the same conclusion” (p. 16).

He went on to say that impulses continually press for discharge and satisfaction, thereby imbuing the compulsion with a biological energy and giving it the life of a demonic force (1920). Patients speak of this force every day. What is this “biological energy” and where does it come from?

A pattern posits a prototype. At some point, the pattern must have been established. Its parameters are learned and remembered on some level, then repeated. Human brains, with their complex substructures and chemistry, develop in a way that stores a detailed image of an infant's earliest experiences. Subsequently, the stored characteristics of the pattern act as a filter and a benchmark for adult adjustment patterns and responses.

This paper surveys today's research in neuroscience to propose how the brain could support undesirable and destructive repetitive behaviors. My thesis is that the compulsion results from neurological artifacts representing one's earliest experiences. Because the central needs of tension reduction in the infant rely on another person, the interactions within that dyad powerfully influence the developing brain. In the adult, these remnants direct behavior by tilting responses in accordance with a pre-ordained neurological architecture. The artifacts reveal themselves in what approach behavior is later conducted, in how it is conducted, in the subjective feelings that arise around the choice of approach or withdrawal, and in the perceptual results of processing stimuli. The past exists in the form of "malignant memories" (Schwarz & Perry, 1994) stored in neural networks that involve the hemispheres, neurotransmitters, and sub-cortical structures like the amygdala. The process involves specific mechanisms of learning and memory combined with homeostatic systems of emotional reactivity.

This story begins with genes and experience combining to shape neurological development through the processes of synaptogenesis, pruning and neurotransmitter distribution. Critical periods, developmental processes such as resonance, and the learning mechanisms of Hebbian plasticity and long-term potentiation can indeed create a brain that operates under the direction of implicit social and malignant memories.

Freud had hoped to accomplish a similar objective, but abandoned his "Project for a Scientific Psychology" by admitting that the neurological information was just not yet available. Spotnitz, trained in neurology and "very interested in brain research" (Bershatsky and Bershatsky with Spotnitz, 2002, p. 17) also approached the question of neurological mechanisms underlying psychological phenomena. In *A Neurobiological Approach to Communication*, (1985, p. 88), he says: "...The mind or psychic apparatus, with which the analyst is concerned, appears to be the product of a specific neural organization.... The concomitants of the

psychic patterns that operate as obstacles to personality maturation are patterns laid down by early learning in the organ of the mind..."

Today's neuroscience supplies the information Freud lacked. Researchers know a great many details about the specific neural organization to which Spitz referred. Of course the hypothesis cannot yet be proven empirically, but the literature convincingly indicates a direction. Even the neuroscientists see it. Weinfeld and colleagues thus comment on a proposal by Allan Schore, a neuroscientist at UCLA: "[I]t is possible that the experiences within the early attachment relationship influence the developing brain, resulting in lasting influences at a neuronal level (Schore 1994)." "This possibility... [is] compelling (Weinfeld et al., 1999, p. 75)." "Environmental experience is now recognized to be critical to the differentiation of brain tissue itself" (Cicchetti and Tucker, 1994, p.538).

What Is The Compulsion To Repeat?

LaPlanche and Pontalis, respected arbiters of psychoanalytic terminology, define the experiential and psychological "Repetition Compulsion" as follows. "At the level of concrete psychopathology, the compulsion to repeat is an ungovernable process originating in the unconscious. As a result of its action, the subject deliberately places himself in distressing situations, thereby repeating an old experience, but he does not recall this prototype; on the contrary, he has the strong impression that the situation is fully determined by the circumstances of the moment." (1993, p. 78).

Webster's New Collegiate Dictionary defines "compel" as: "to drive or urge forcibly or irresistibly," "to cause to do or occur by overwhelming pressure" and "to drive together." (p. 229)

Freud (1914) described its presence in the analytic relationship. The patterns acquired in childhood are forgotten and are instead acted out and repeated in life. They are reproduced not as memories but as actions. The patient thus surrenders to the compulsion to repeat. The analyst's main tool for working through and resolving this resistance is the transference. In *The Dynamics of Transference* (1912) he said that as a result of innate predispositions and environment, each person comes to develop his own way of gratifying his impulses. These patterns become embedded and stored in the unconscious and are repeated throughout the person's life. In particular, these patterns are reactivated in the patient's relationship with the analyst through the transference.

The discovery of the repetition compulsion led Freud to develop the concept of the death drive. Freud (1920) observed that a child who experienced an unpleasant experience was likely to re-enact this in his subsequent play. He found this curious because it seemed to violate his understanding of the pleasure principle. Hence, Freud updated the original pleasure principle to include the presence of an additional force, the death drive (1920). He noted its presence in the widespread forms of aggression exhibited in behavior. A century later, Guterl (2002) wrote that “unconscious drives, similar to libido and aggression, have now been located in the most primitive part of the brain.” (p.50). Indeed, as this survey outlines, Freud’s prescience is now formidably borne out by modern neuroscience.

How do these repetitions become virtually ubiquitous? What embodies these patterns with such perseverance? The question becomes not one exclusively of the pattern but also of its power. How do brain cells made of protein, communicating with each other via electrochemical messages, exert such a pervasive yet covert influence?

Brain Basics

Neurons, Glia & Synapses –The Brain’s Infrastructure

Brains consist primarily of neurons, glia, synapses, and neurotransmitters. A neuron, the basic cellular unit, consists of dendrites, a cell body, and an axon. Dendrites receive messages and forward them, via electrical signals, through the cell body. They contain numerous branches and appear under the microscope like the spindly branches of a tree. Axons project a single stem away from the cell body and function by passing on messages to the next dendrite or cell. Axons vary in length. Some extend over three feet. By most estimates, the human brain consists of 100 billion neurons (Stahl, 2000).

Glia outnumber the neurons by a factor of ten. They perform administrative functions in the brain like waste management and repair. Glia also appear to play a role in guiding neurons to their intended destinations (Kalat, 1992).

Synapses are the microscopic spaces between the ends of axons and the beginnings of dendrites. Neurons “synapse” onto thousands of other neurons. What happens across these tiny clefts comprises the essential operation of the brain. LeDoux goes so far as to call synapses the seat of the self (2002).

Neurochemicals, Vesicles and Receptors – The Neurological Pony Express

In order for a message to cross the synapse, a neurotransmitter, a chemical substance, must be present and active. Neurotransmitters excite or inhibit the cells they contact. Cell bodies produce these neurotransmitters, which travel along axons and are released after an electrical signal travels down the axon. This release is called the action potential. Release communicates a message to the next cell. These messages form the basis of neurological functioning.

At one time, neurologists thought that electrical signals themselves did all the communicating. Otto Loewi, a German physiologist, discovered that by transferring the fluid surrounding one frog's heart to a second heart, he could produce in the second frog the same response seen in the first—for example, increasing or decreasing the heart's rate (Kalat, 1992). He showed therefore that since free electricity was not transferred within the fluid, the active ingredient must be chemical.

Pre-synaptic neurons store substances in vesicles and post-synaptic neurons possess specific receptors to receive a given transmitter. Together the neurotransmitter and the receptor operate like a lock and key. The chemical wafts into the synapse and meets the receptor on the next dendrite.

Several dozen neurochemicals are known or suspected (Stahl, 2000). Cells in the brainstem, called nuclei, manufacture the best-known ones: serotonin, adrenaline, and dopamine. Called amines, they play key roles in learning, memory, attention and emotions. They can arrive rapidly—within 50 nanoseconds—in response to emotionally sensitive events (Derryberry & Tucker, 1992).

Lastly, neurochemicals play multiple roles. The hormones oxytocin and vasopressin do double duty as neurotransmitters and hormones. They work in the brain and in the body both in psychological constructs like love and physical events like labor and lactation. Both have been shown to be integral to the process of pair-bonding in certain animals (Insel, 2001).

The Basement, Mezzanine and Penthouse of the Human Brain

The brain and the body connect through the brainstem which sits in the lower back of the head. Within the skull, sub-cortical structures such as the amygdala and the hippocampus sit more or less on top of the brainstem. Some apparent specialties include the amygdala in emotions and the hippocampus in working or factual memory. Surrounding these sub-cortical structures, the cortex looks like wrinkled sausage. Neurologists speak of the forebrain and the

pre-frontal cortex which sits approximately in the area of the forehead. This area receives and processes information from all external and internal sensations. It develops more fully in humans than in any other species.

Hemispheres – The Neurological East & West

The cortex also splits into the right and left hemispheres. Extensive work supports the tendency for one of the hemispheres to dominate processing of a given task (Hellige, 1993). Evidence exists of a left/right split between the frontal lobes regarding positive and negative emotions (Davidson, 2003). Today an emphasis on the right brain arises in work on the neurological substrate for unconscious events. “The experience-dependent expansion of the right brain is reflected in the growth of the unconscious over the life span.” (Schoore 1999).

Amygdala – The Almonds inside Your Head

Anatomically, the amygdalae sit in the temporal lobe, a bit forward but to the sides of the head. Originally they were named for their resemblance to almonds. Extensively connected with both lower brainstem and higher cortical brain centers, the amygdalae perform somewhat like Grand Central station. Rolls noted that these connections imply that the amygdala receives highly processed sensory information and in turn influences autonomic, motor, and some cortical processing (1992). Research in the last ten years repeatedly indicates that the amygdala is central to unconscious processing, emotions and social behaviors. Schoore indicates that parts of the amygdala experience a critical period of growth during the last trimester before birth and in the first two months of life (2001a). This suggests a pivotal position in the creation of both a repetition and the compulsion associated with it.

Neural Circuits

Networks for Emotion

Ten years ago neural networks—and the idea that responses to stimuli involve multiple areas of the brain—were still conjecture in the scientific community. The indicators that such networks operated came from classic studies of lesions in particular brain areas. One example showed how injuries in the cortices handling sight or hearing failed to disrupt processing of previously-conditioned behavior (LeDoux, et.al., 1992). Given that the behaviors occurred particularly in response to sensory stimuli, it meant that the conditioned behavior arose from more brain centers than just the

cortex. Today, neuroscientists accept the existence of circuits and networks in the brain.

The “limbic” system refers to an emotional network within the brain. Some researchers reject the concept, but it remains prevalent. Current thinking depicts it as a group of sub-systems of vertically organized circuits. Traditional elements include the sub-cortical structures the amygdala, hippocampus, hypothalamus, septum, and stria terminalis. Both the amygdala and hippocampus connect to the brainstem regions involving hormonal, motor, and autonomic behaviors. In fact, research on emotion networks indicates involvement of all levels of the brain—cortical, neocortical, “limbic, paralimbic”—and brainstem elements (Derryberry & Tucker, 1992). This includes a possible circuit of emotion consisting of the orbital frontal cortex, the anterior cingulate and the amygdala (Davidson, Putnam, & Larson, 2000).

A number of examples of such subsystems appear in the literature. In discussing anger, for example, Davidson reported a focus within both the right brain and the amygdala. He also found that infants crying “in frustration” demonstrate right-sided activation (2003). A huge body of work revealing the role of the amygdala and its interaction with the prefrontal cortex in the emotion of fear comes from Joseph LeDoux’s lab at New York University (1996, 2002).

As I will show, this proposed emotional circuit could play the key role in facilitating a pattern of repetition by virtue of its ability to bias perception and emotion.

Neurological Development

Stages

The brain develops hierarchically from brainstem to cortex. According to Schore, (2001a), the amygdala is active at birth; the cingulate becomes active within three to nine months and the orbitofrontal cortex at 10-12 months. This pattern of brain growth reveals the process that underlies how psychological realities become unconscious. It also explains how early experiences maintain such power: each lower structure of the brain modifies development of the next higher one.

Ultimately, “The orbital cortex matures in the middle of the second year, a time when the average child has a productive vocabulary of less than 70 words. The core of the self is thus nonverbal and unconscious, and it lies in patterns of affect regulation” (2001a, p. 37). Is Schore a closet modern analyst?

Nature meets Nurture

A “Nature vs. Nurture” debate has long raged within and between the fields of religion, philosophy, biology and psychology. From geneticists to people in the park, everyone has an opinion. So many studies about identical twins separated at birth have appeared that one begins to wonder exactly how so many twins got separated!

Thankfully, the debate graduated to how nature and nurture cooperate. No neuroscientist seriously thinks that either nature or nurture trump the other. Richard Davidson, prominent neuroscientist at The University of Wisconsin, commented “What’s particularly exciting about these findings is that the impact of environment on brain development has been traced down to the level of actual gene expression. This has, only so far, been done in animals, but we have every reason to believe it applies to humans, too. For a person raised in a nurturing environment, there are actually demonstrable, objective changes in gene expression. For example, there are genes for certain molecules that play an important role in regulating our emotions and which respond to nurturing” (2003, p. 189).

Critical Periods

The “critical period” concept, now firmly established in biology (Katz 1991), states that “specific critical conditions or stimuli are necessary for development and can influence development only during that period” (Erzurumlu & Killackey, 1982, p.207). In order to understand this, look at a clear example from the days before human neuroimaging. When a bird is not exposed to its specific song during a certain very early period, it will never be able to sing that song as an adult (LeDoux, 2002).

Schore articulates a case for the neurobiology supporting a critical period of infant-mother attachment. He notes that attachment experiences “specifically impact development of the infant’s right brain” (2001a, p, 15). One supporting pillar is that by sixty days, infants experience a change in their emotional and social capabilities via the changes in visual processing at eight weeks (Yamada et al, 1997, 2000). This allows eye contact between mother and infant and thereby facilitates emotional exchanges. Supporting his thesis is work showing that the visual information about faces is of utmost interest to the infant at the same time that areas of the right hemisphere are in their most receptive state (Deruelle & de Schonen, 1998; de Schonen, Deruelle, Mancini & Pascalis, 1993).

Critical periods provide the next rung on the ladder, after hierarchical brain construction, explaining how early experiences exert such dramatic control.

Building the Brain's Infrastructure

Neurogenesis

Historically, neuroscientists claimed that no new neurons emerge after birth. Perry and Pate (1994) indicated that full term infants arrive with all of their 100 billion neuronal bodies in existence.

Stahl (2000) says that neurons settle by birth but that their axons can grow. Glia and other adhesion formulas provide the scaffold for neurons to find their destination.

New research also indicates that new neurons can grow in adult humans (Gould et.al., 1999). As Cozolino indicates, this discovery remains controversial, yet the evidence is growing (2002). If proven, adult neurogenesis provides an interesting possible explanation of how analysis succeeds.

Synaptogenesis and Pruning

This process of creating synapses, synaptogenesis, requires axonal, dendritic, and neurotransmitter cooperation. An explosion in the creation of synapses occurs in the months following birth. Supporting the aforementioned turning point for eye contact between mother and infant, Peter Huttenlocher found that synaptic density in the visual cortex almost doubles between two and four months of age.

However, more synapses develop than ultimately are used. Neuroscientists believe that the synapse must be fired across to survive.

Scientists call loss of a synaptic connection *apoptosis*, or colloquially, *pruning*. Ultimately, 100 trillion synapses exist. Some neurons work with up to 10,000 synapses each (Stahl, 2000). The choice of synaptic connections for the 100 billion neurons leaves unimaginable room for change. This process therefore presents the prime opportunity for outside stimuli to modify the resulting structure. "...neurodevelopmental experiences and genetic programming lead the brain to select wisely which connections to keep and which to destroy. If this is done appropriately, the individual prospers during this maturational task and advances gracefully into adulthood. Bad selections theoretically could lead to neurodevelopmental disorders such as schizophrenia or even ADHD" (p.29).

Neurotransmitter Development

Neurochemical differentiation also transpires during development. This refers to the process whereby neurotransmitters, their storage vesicles, and receptors emerge. Variations in type and

number of receptors emerge to accommodate the types and amounts of an expected neurotransmitter. Interaction apparently takes place causing changes in the amount of an available neurotransmitter, which in turn produce concomitant changes in the receptor population. More or fewer receptors may survive, or the existing receptors may become more or less sensitive to the appropriate transmitter.

Specifically, the secretion of adrenaline surges in response to a threat. If excess adrenaline floods the synapse regularly, the receptors on the post-synaptic cell may alter their receptivity to adrenaline in an attempt to maintain a baseline amount (Schwarz & Perry, 1994). As a clinical example, fewer receptors for adrenaline have been found in non-medicated patients with borderline personality disorder (Southwick, Yehuda, Giller, & Perry, 1990). It has been proposed therefore that borderline disorders result from early childhood trauma and overstimulation which sets up the continual production of excess adrenaline (Herman, Perry, & van der Kolk, 1989 in Southwick et. al. 1990). Early exposure to frustration and tension affects the level of adrenaline release in the infant's system. This in turn may set up a certain pattern of neurochemical reactivity, via amounts or receptors or both, in response to later stimulation. Later in life, the adult may thus be sensitive to situations reminiscent of the early experience. According to the repetition compulsion, he will even set out inadvertently to create a similar situation.

Wiring by Firing

“Hebbian Plasticity,” proposed by Donald Hebb in 1949, says that if a pre- and post-synaptic neuron are simultaneously active as a result of unrelated firing, then the connection between them will become stronger. This occurs in the case where an anatomical connection exists but not necessarily a very strong synaptic communication. This concept is colloquially expressed in the neuroscientific community as “cells that fire together, wire together” (LeDoux, 1996).

We can imagine how such a process assists in associating new or previously unrelated information. To use a simple example, think of sitting in the park looking at the blue sky. A popsicle vendor walks by. You look at the flavors and order a “sky blue” popsicle. The color, sky, and popsicle neurons most likely didn't fire together initially, but now they are firing at the same time. Hebb says that the next time they will be more likely to fire together, and that perhaps you will ask for the blue one without needing to be shown it.

“Today, so called Hebbian plasticity is everyone’s favorite idea about how learning and memory work at the level of individual cells in the brain” (LeDoux, 1996, p. 215).

Long Term Potentiation (LTP)

When the neuron receives a series of very rapid stimuli over a brief period of time, the subsequent reaction to a single weaker stimulus increases (Cozolino, 2002). In other words, once the neuron gets pounded, it only takes a light tap to produce subsequent firing. This phenomenon is referred to as *Long Term Potentiation* or LTP.

More importantly, the change endures. In other words, the synapse doesn’t forget (LeDoux, 2002). A tap years later can produce the same response.

Clearly this suggests how memories might occur. It also relates to how a repetition could be enacted. Effectively, it could produce a neurological “path of least resistance.” If one event of intense firing causes the neuron to later fire in response to relatively weak stimuli, this could account for how the brain can effortlessly invoke previous experience—either as it relates to using a skill or to an assessment of an interpersonal interaction.

Resonance

“In physics, a property of resonance is sympathetic vibration, which is the tendency of one resonance system to enlarge and augment through matching the resonance frequency pattern of another resonance system” (Schore, 2001a, p. 12). According to Schore, in the process of brain building and operation, resonance “refers to the ability of neurons to respond selectively to inputs at preferred frequencies and ‘amplified resonance’ or ‘amplifying currents’ serve as a substrate for coordinating (synchronizing) patterns of network (circuit) activity” (p. 17). He sees this working in the way an infant comprehends and absorbs its mother’s emotions.

In other words, getting the best signal on a radio is analogous to how neurons fire: certain frequencies optimize the process of firing. In the context of the repetition compulsion, this could again be a “path of least resistance” scenario. Possibly resonance uses LTP as its mechanism.

Study of the mechanism of resonance in the brain could also shed light on how the compulsion to repeat comes about. During a person’s conscious evaluation of new acquaintances, his neurons may be searching behind the scenes for just the right frequency to match a previous experience. Once they find it, they in lock in on it and color the person’s perceptions—which leads to altered behavior,

which in turn recreates in actuality what the brain had hidden all along.

In relevance to this discussion, Schore asserts that resonance “tunes” the right brain circuits to process socio-emotional information (1994, 1997b, 2000d).

The Proposed Neurobiology Of Freud’s Repetition Compulsion

Memory - The Link between Doctor Freud and Donald Hebb

Without memory, we cannot learn. Without memory, we cannot recognize our own faces. Hebbian plasticity, LTP and resonance may explain the specific operations causing the encoding of our neurons with pictures of events, but how do we get from these mechanisms to the psychological reality of the repetition compulsion? Memory links the biological to the psychological.

Day-to-day memory, the stuff of lists and bills, is called *declarative* or *explicit*. Researchers believe this type of memory to be primarily supported by the hippocampus. While this is interesting in and of itself, the memories underlying the repetition compulsion lie elsewhere.

Perry and Pate, working on post-traumatic stress disorder (PTSD), coined the term “malignant memory” to explain the source of the hallmark flashbacks and intrusive thoughts involved in PTSD (1994). They described how malignant memories become the preferred approach for integrating information based on a prolonged reaction to a past threat. In the case of a sudden traumatic event, the brain’s reaction mechanisms fire so intensely that an overpowering memory is created. The scenario of PTSD resembles the theoretical substance of a repetition in that something repeatedly stressful, albeit unknown, creates an ongoing reaction to a historical event. We can see how the repetition compulsion essentially embodies the existence of a malignant memory.

This happens through what neuroscientists call *implicit* memory. Formerly termed *procedural*, it includes such things as the muscular details of riding a bike, or writing in cursive, but more importantly it subsumes what we consider “the unconscious.” In today’s neuroscience, countless studies refer to implicit memory’s unconscious properties and seem to include no debate over the power of unconscious processing. The amygdala appears to be the primary structure involved. Ironically, Joseph LeDoux describes implicit memories as those that show up more in the way we act than in what we consciously know (2002). In other words, even “pure”

neuroscience indicates that implicit memory is the assumed source of the behavior of acting out.

LeDoux expands his argument by declaring the indelibility of sub-cortical emotional memories (LeDoux et. al., 1992). He reviews research showing that stimuli which are associated with highly charged emotional situations will cause a persistent conditioned response. He shows that conditioned responses persist for very long periods of time, even with unreinforced trials. In other words, the arrival of a stimulus-provoking emotion can produce the same result it initially caused even when much time has passed.

Where & How Do Electro-chemical Signals Create a “Malignant Memory?”

How could the abstract concept and ephemeral experience of a repetition compulsion be correlated with neurons and electrochemical signals? Perhaps Descartes was right when he split the brain and the mind. Ten years ago, the idea of defining these unconscious forces had hardly been explored. At that time I proposed that synaptogenesis, pruning and long-term potentiation could combine to build a neural substrate that would predispose its owner to patterned reactions (Shull, 1995). Today, we know more. In turn, I suggest the following.

The “compulsion to repeat” originates from the development of patterns of neurochemicals, particularly dopamine, noradrenalin and oxytocin, which send their messages across preferred synapses. Resistance to change occurs due to resonance and long-term potentiation driving firing across a neural circuit that connects the brainstem, amygdala, and the right orbitofrontal cortex. This circuit mediates implicit emotional memories from the earliest months of life that filter perceptions in order to create emotional homeostasis. By operating in this manner, the brain compels the behaviors that coalesce into an adult repetitive experience.

Here is the evidence.

The Neural Network – Pattern Storage

Mesulum indicates that the circuit from brainstem to right cortex fosters “a highly edited subjective version of the world” (1998, p.1013). Brothers (1995, 1997) describes the circuit (including the temporal pole) as a social “editor” that is “specialized for processing others’ social intentions” by appraising “significant gestures and expressions” (1997, p.27). Clearly, appraising others relates to the instigation of relationships. The studies of Price, Carmichael and Drevets show that “the mature orbitofrontal cortex

acts in the highest level of control of behavior, especially in relation to emotion” (1996, p. 523).

In contrast, Baxter and colleagues report that the orbital-amygdala circuit gives the individual the ability to avoid making bad choices (2000). This would be true when the circuit experiences the opportunity for full development. In a situation with less than optimal experiential input, incomplete development may prevent the most advantageous choices. Furthermore, while typically the compulsion to repeat implies a negative outcome, in reality that is not always the case. It is just that we only notice it when it causes problems. Healthy individuals also repeat. They just repeat a different pattern. Perhaps the circuit causes choices that steer towards replication because, as far as the brain knows, the pattern that developed is the one that supported survival.

The Right Brain

Elliott and Dolan found that “pre-exposing subjects to visual stimuli is sufficient to establish a subsequent preference, even when previous exposure is subliminal” (1998 p. 1). This “mere exposure effect” results from activation in the right prefrontal area during retrieval of implicit memory. “Retrieval ...was implicit and unconscious, yet it influenced behavior as expressed in preference judgments. We suggest that this region of the right lateral prefrontal cortex may subsume behavioral guidance functions, even in the absence of conscious awareness” (p. 10). This study evaluated the simple act of choosing an item that to which one had previously been exposed. This was done through subliminal presentation of pictures of objects. After the presentation, participants were presented with a group of items and asked to choose those they liked best. They chose the ones which had been subliminally presented, despite asserting that they had no prior knowledge of the items. The bearing of this finding on repetition compulsion in relationships is obvious. We could extrapolate from this to say that people chose someone who matches what they previously saw—even when they weren’t conscious of the previous exposure. This could mean choosing to replicate the first relationship we knew, even when we don’t remember it, because the hippocampus, where explicit memory operates (LeDoux, 2002) was not yet fully developed.

Looking at it from another perspective and finding further agreement, Bechara speaks about the systems managing the brain’s overall processing. The regulatory mental systems are a “product of the experience-dependent maturation of the orbitofrontal system

which generates nonconscious biases that guide behavior before conscious knowledge does" (Bechara et. al., 1997).

The Sub-Cortex

Repeatedly, the amygdala gets credit for emotional assessments and processing. Although much of the work studied elements of fear, Davidson reported that felt but unexpressed anger shows up in a pattern of right frontal side and amygdala activation (2000).

With emotion defined "as the process by which the brain determines or computes the value of a stimulus" (2002, p.201), LeDoux's work repeatedly identifies the amygdala as the "brains" behind integrating stimuli and generating reactions. Historically, animal studies confirmed the role of the amygdalae in maintaining these stimulus-reward associations. Bilateral amygdalaelectomy in animals results in reduced response to normally stimulating objects. Kluver and Bucy (1939 in Rolls, 1992) first identified this syndrome of changes. The "Kluver Bucy Syndrome" includes a lack of fear in response to what would normally be threatening stimuli, such as mating with other species, in test animals which have undergone amygdalaelectomy.

This poses an interesting idea relevant to the repetition compulsion in which participants often lack concern when beginning a relationship with a person evaluated by others as potentially troublesome. This behavioral pattern could be accounted for through incomplete development of the amygdala stemming from the lack of proper experience during insufficient early nurturing. Indeed, Bachevalier indicates that damage to the amygdala in early infancy leads to "profound changes in the formation of social bonds and emotionality" (1994).

Specific support for the amygdala's role in unconscious evaluation of danger comes from a study showing that it "lights up" in response to subliminally perceived fearful faces. The blood oxygen level-dependent signal in an FMRI spiked significantly higher during viewing of the masked fearful faces. This does not happen with masked happy faces suggesting that the amygdala tunes into danger signals (Whalen et. al.1998). The study showed that this activity occurs completely unconsciously. Again, it raises the question of a deficient amygdala possibly missing signals that could be used to avoid a negatively charged repetition.

A decade ago, Halgren stated that the evidence clearly implicates the amygdala in the evaluation of complex stimuli "long before they are analyzed cognitively and probably long before they enter awareness" (1992, p. 194). The right brain uses its nonverbal

encoding and retrieval functions (Wagner et al., 1998) to rapidly assess a stimulus based on input from the amygdala. Zald and Kim (1996) indicate that this is what we call “spontaneous gut feelings to others.” Hasn’t many a patient in a repetition said “but I had such a good feeling about them?” Much research remains to be done, but it does appear plausible that the amygdala generates unconscious assessments encouraging approach behavior, while it may be failing to catch a helpful danger signal.

The Neurochemical Links – Compelling Forces

With a bird’s-eye view of the neurological landscape, we can examine the neurochemical highways to understand more detail. The neurotransmitter systems extend from brainstem to cortex and in turn form the glue within a neural circuit.

Again, Allan Schore’s work on infant mental health offers specific direction. Connecting the brain centers so far discussed with the network of neurons providing the pivotal chemical messengers, Schore summarizes the situation as follows “...the right limbic system is more directly [than the left] connected with subcortical neurochemical systems associated with emotion...” (Schore, 2001a, p. 44). Contrasting a scenario of secure attachment with an inadequate one, he illuminates the developing neurotransmitter stress/coping systems. “Severe attachment problems with the caregiver negatively impact the postnatal development of these [the infants’] biogenic amine systems” (Kraemer & Clarke, 1996). Remember that amines include adrenaline, noradrenalin, serotonin and dopamine. First, in addition to their own resulting systemic deficiencies, a deficiency of these transmitters can prevent the amygdala and the cortex from developing to their optimal states. These deficiencies result in “the individual’s inability to regulate affect” (p.9).

In practice, the failure to manage emotions usually plays a pivotal role in repetitive relationship scenarios. Whether it is a habit of being too quick to anger or one of being hypervigilant for abandonment, the behavior that results from the overpowering feelings in turn causes a cascade of reactions resulting in similar experience.

Adrenalin and Noradrenalin

Perry et.al. (1995), speaks of the two responses of an infant to trauma: hyperarousal and dissociation. Hyperarousal causes what we call a typical “fight or flight” neurochemical reaction. It involves adrenaline and other stress chemicals kindling the emotional circuits

of the infant. Stresses incumbent to this original attachment process may modulate the production of noradrenalin. By doing so, the experience may impact the final configuration of noradrenergic axons in the cortex. While active throughout the body, the cellular source of noradrenalin is the *locus coeruleus* (LC), a brainstem structure involved in overall regulation of physiological function. Axons from the LC ascend to all brain centers with some clustering appearing to occur, not surprisingly, in the right hemisphere (Tucker & Williamson in Derryberry & Tucker, 1992).

Because this occurs during a critical period, "characterological styles of coping" (Schoore, 2001b, p. 11) result. In other words, the need for excessive arousal occurs and in turn shapes the individual's typical style of reacting to maintain this state. Weinstock says that trauma in the infant's life, in the form of chronic or inescapable stress, may lead to a hyperactivity of the circuits managing adrenaline. In doing so, the overactivity of the alarm circuits could lead to excessive anxiety, feelings of hopelessness and defeat and depression (1997).

Not surprisingly, this is the same neurochemical mechanism supposed in PTSD and "malignant memories" (Perry & Pate, 1994).

Calcium

The severity may further arise from the following. The stress-induced neurotransmitters "selectively induce neuronal cell death" (Kathol et.al., 1989). Excess dopamine and adrenaline cause increased production of receptors at post-synaptic neurons. This causes a long-lasting alteration in how the neurotransmitter systems operate. During development, the excess excitatory chemicals dopamine and glutamate cause increased calcium in the neurons which leads to cell damage and death. In this extreme case, the effects would go beyond pruning synapses, which have a built in mechanism to regenerate (in LTP and Hebbian plasticity.) Cell death, barring the aforementioned neurogenesis research, would leave very little room for recovery because the neurons themselves would no longer exist.

Oxytocin and Dopamine

The May 2003 issue of *Discover* magazine included an article entitled "Love." Its author, Steven Johnson, discusses the hormone and neuropeptide oxytocin which is being studied for its likely role in love and attachment. Ferguson and her colleagues found that oxytocin works specifically in the amygdala in mice during the acquisition of a new category of memory. "Social memory may be a unique form of memory, critical for reproduction..." (Ferguson,

et.al., 2002, p.200). Blocking oxytocin prevents certain rodents from forming pair-bonds even when that is their natural inclination (Johnson, 2003). Translating this spectrum of reports into the idea that the repetition compulsion may arise from the architecture of early experiences, Johnson describes a paradigm shift to “the idea that brain circuitries devoted to affiliation and social bonds may well be as sophisticated as our fear mechanisms” (p.74). Oxytocin “links molecular, cellular and systems approaches” (Insel and Young, 2001, p.7).

Dopamine, likewise, can be thought of as the “feel good” neurotransmitter. This appears to be its function from the beginning. Schore suggests (1994) that when an infant sees its mother’s face during play experiences, a high level of dopamine-driven arousal and elation occurs in the infant’s right brain. Effectively this means that a baby’s first experiences of satisfaction outside the realm of food have to do with the mother’s behavior. Schore goes on to describe the effect as “critical right brain events by which psychobiologically attuned attachment communications generate amplified resonance that tunes reward circuits to certain forms of human visual and auditory patterns of stimulation” (p. 18). In other words, the reward circuits are built according to the sights and sounds associated with the interaction with the mother. This be why we try to recreate that very scenario.

When oxytocin and dopamine are put together, Insel and Young find that “it [oxytocin] seems to link with “social stimuli to dopamine pathways associated with...reward and euphoria – critical elements in the process of attachment” (Insel & Young, 2001, p.7). In other words, the neuropeptide most associated with attachment appears to speak to the one most associated with feeling good.

Hence, it could be that the only path the brain knows to generate what may have been fleeting good feelings is to recreate the original circumstances, and this may drive compulsive behavior.

What Have We Learned?

In summary, both brain tissues and chemistry are built with the influence of early experiences. The resulting constructions create filters for subsequent development and for learning, perception, and behavior. Early experiences have a particularly powerful influence over neurological formation due to the explosive brain growth which occurs in infancy. The placements of synapses are influenced, the neurocommunicators calibrated and the elements of implicit and emotional memories laid down.

Theoretically, as subsequent experiences occur, the initial experiences, embedded in the physical substrate, color the individual's feelings and perceptions. Choices are influenced through these echoes. The right hemisphere, informed by the neurotransmitters and amygdala assessment, processes stimuli and directs responses. This transaction happens so quickly, on the basis of LTP and possibly resonance, that the participant cannot be aware of it. Ultimately, the operation of this circuit between the amygdala and right brain, with help from adrenaline, dopamine and oxytocin, may secretly guide the individual into situations that mirror the characteristics of one's early life.

Implications For Treatment

Kindling – A Neurological Antidote?

Kindling refers to a mechanism usually spoken of in regard to how neurons might fire in preverbal disorders. It means exactly what its name implies: lighting little fires eventually causes bigger ones to burn. Small excitations across synapses may eventually burgeon. First observed in epilepsy, kindling was found through studies in which a small amount of current was passed to the relevant brain area of an experimental animal. Initially, nothing happened, but with continued small bursts of current, a small seizure resulted. When intermittent bursts were applied, eventually less and less current was required to generate more widespread seizures. It is possible to continue this process to the point where seizures occur spontaneously. Kindling appears to modify the nerve pathways. Cells downstream from those which receive the current change anatomically. These changes take place prior to the onset of the seizures. According to Peter Kramer, the altered cells produce a rewiring of the brain. The modified cellular chemistries induce modifications in the synaptic configurations (1993).

This paper introduces a possible physiological basis for the compulsion to repeat. We should also ask "What happens neurologically as the unconscious becomes conscious? What underlies the analysis and resolution of resistance?" Clearly, neural networks change as the patient says everything. Could kindling be this biological mechanism? Its description in seizures, although a disease process, resembles the experiential process of therapy. It is reasonable to assume that kindling could also be employed to create a positive outcome for the patient.

Furthermore, resonance is involved in induction and its transmission of feelings between therapist and patient. Clearly, the analyst's work tunes into the neurological activity of the patient.

Could this tuning incite kindling? In this way resonance could lead to firing across a spectrum of neurons which in turn might make conscious something previously unconscious.

The recent glimpses into possible neurogenesis also hold promise. Could saying everything coax brand new neurons to synapse into the old networks, thereby diluting unconscious patterns? Hopefully, as research uncovers the construction of the compulsion to repeat, we can also anticipate learning how analysis deconstructs it.

A Final Note – The Even More Difficult Question

Exploring the neurological basis of the unconscious is challenging. In fact, Damasio wrote *The Feeling of What Happens* (1999) to address an even greater enigma: the explanation of consciousness.

Ironically, he asserts that consciousness arises from a mental pattern built to describe the relationship between and the self and the object. He goes on to say that this pattern is built through feelings. Lastly, he believes that homeostasis provides the key. One way he characterizes this homeostasis is as “a dispositional arrangement for regulation of internal states that subsumes a mandate to maintain life” (p.136). It only takes a small leap to transpose this seminal idea onto the neurology of the compulsion to repeat—it maintains a steady state of emotions regarding oneself and an object because, however debilitating those emotions originally were, they kept the organism alive.

He tells the story of David, who suffers from one of the most severe defects in learning ever recorded. David cannot learn any new facts: no new people, sounds, places, or words. Hence he cannot remember anyone or anything he has ever done. If he speaks to you and walks away, he will remember neither what he said nor that he said it to you even a few minutes later. David has damage to both the hippocampus and the amygdala. It was noticed, though, that David always went to the same people for cigarettes or a cup of coffee and that there were certain people to whom he would never go. He didn't know their names, didn't know he had gone to them before, and didn't know he had ever seen any of them, yet approached and avoided the same people each time. Clearly, something was up. Somehow, somewhere in him, he “remembered” something.

Damasio and a colleague decided to test this scientifically. They set up a “good guy/bad guy” experiment in which, under controlled circumstances, they exposed David to three different people: a

pleasant and welcoming one, a neutral one, and a brusque person who always said no. They did this for five days. When later asked to look at photographs of the three to say whom he would consider his friend and to whom he would go for help, he picked the “good guy” over 80% of the time! This was in spite of the fact that the “brusque guy” was actually a pretty young female doctoral student.

Somehow, despite the lack of all conscious memory, the *emotional* message was communicated and it changed David's behavior—a heartening discovery for the challenging moments spent trying to help patients rearrange the resilient architecture within their minds.

References

- Bachevalier, J. (1994). Medial temporal lobe structures and autism: a review of clinical and experimental findings. *Neuropsychologia*, 32, 627-648.
- Baxter, M.G., Parker, A., Lindner, C.C.C., Izquierdo, A.D., & Murray, E.A. (2000). Control of response selection by reinforcer value requires interaction of amygdala and orbital prefrontal cortex. *Journal of Neuroscience*, 20, 4311-4319.
- Bechara, A., Damasio, H., Tranel, D., & Damasio, A.R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science*, 275, 1293-1295.
- Bershatsky, D., & Bershatsky, C. (2002). Interviews with Dr. Hyman Spotnitz, *Annals of Modern Psychoanalysis*, 1(1): 1-19.
- Brothers, L. (1995). Neurophysiology of the perception of intention by primates. In M.S. Gazzaniga (ed.), *The cognitive neurosciences*, (pp. 1107-1115). Cambridge, MA: MIT Press.
- _____ (1997). *Fridays footprint*. New York: Oxford Univ. Press.
- Brown, Rita Mae. *Starting From Scratch: A Different Kind of Writers Manual*. NY: Bantam, 1988.
- Cicchetti, D. & Tucker, D. (1994). Development and self-regulatory structures of the mind. *Development and Psychopathology*, 6, 533-549.
- Cozolino, L. (2002). *The Neuroscience of Psychotherapy, Building and Rebuilding the Human Brain*. New York: W.W. Norton & Co.
- Damasio, A. (1999). *The Feeling of What Happens: Body and Emotion in the Making of Consciousness*. New York: Harcourt

- Davidson, R. (2003) in *Destructive Emotions, How Can We Overcome Them? A Scientific Dialogue with the Dalai Lama*. New York, Bantam Dell.
- Davidson, R.J., Putnam, K.M., & Larson, C.L. (2000). Dysfunction in the neural circuitry of emotion regulation a possible prelude to violence. *Science*, 289, 591-594.
- Derryberry, D. & Tucker, D.M. (1992). Neural Mechanisms of Emotion. *Journal of Consulting and Clinical Psychology*, 60:329-338.
- Deruelle, C., & de Schonen, S. (1998). Do the right and left hemispheres attend to the same visuospatial information within a face in infancy? *Developmental Neuropsychology*, 14, 535-554.
- de Schonen, S., Deruelle, C., Mancini, J., & Pascalis, O. (1993). Hemispheric differences in face processing and brain maturation. In de Boysson-Bardies, S. de Schonen, P. Juszyk, P. McNeilage, & J. Morton (Eds.), *Developmental neurocognition: Speech and face processing in the first year of life* (pp. 149-163). Dordrecht: Kluwer Academic Publishing.
- Elliott, R., & Dolan, R. (1998). Neural Response during Preference and Memory Judgments for Subliminally Presented Stimuli: A Functional Neuroimaging Study. *The Journal of Neuroscience*, 18(12):4697-4704.
- Erzurumlu, R.S., & Killackey, H.P. (1982). Critical and sensitive periods in neurobiology. *Current Topics in Developmental Biology*, 17, 207-240.
- Ferguson, J., Young, L., & Insel, T., (2002). The Neuroendocrine Basis of Social Recognition. *Frontiers in Neuroendocrinology* 23, 200-224.
- Freud, S. (1912). *Papers on technique. The Dynamics of Transference*. Standard Edition, 12:97-109.
- _____ (1914). *Papers on technique. Remembering, Repeating and Working Through*. Standard Edition, 14:145-157.
- _____ (1920). *Beyond the Pleasure Principle. Traumatic Neurosis and Childrens Play are Repetitious*. Standard Edition, 18:12-18.
- _____ (1920). *Beyond the Pleasure Principle. Revision of the Theory of the Instincts*. Standard Edition, 18:34-43.

- Gould, E., Reeves, A.J., Graziano, M.S.A., & Gross, C.G. (1999). Neurogenesis in the neocortex of adult primates. In Cozolino, L. (2002). *The Neuroscience of Psychotherapy*. New York: W.W. Norton & Co.
- Guterl, F (2002). What Freud Got Right. *Newsweek*, November 11: 50-51
- Halgren, E., (1992). Emotional Neurophysiology of the Amygdala Within the Context of Human Cognition. In John P. Aggleton (ed.), *The Amygdala*, (pp.191-228). New York: Wiley-Liss.
- Hellige, J.B., (1993). Hemispheric asymmetry across the lifespan. In *Hemispheric Asymmetry Whats Right and Whats Left*, (pp. 260-291) Cambridge: Harvard University Press.
- Huttenlocher, P.R., (1990). Morphometric study of Human Cerebral Cortex Development. *Neuropsychologica*, 28, 517-527.
- Insel, T., & Young, L. (2001). The neurobiology of attachment. *Nature Reviews/Neuroscience*. 2:1-7.
- Johnson, S. (2003). Emotions and The Brain: LOVE. *Discover*, 24(5): 70-76.
- Kalat, J. (1992.) *Biological Psychology* (4th ed.), Belmont, CA: Wadsworth Publishing Company.
- Kathol, R.G., Jaeckle, R.S., Lopez, J. F., & Meller, W.H. (1989). Pathophysiology of HPA axis abnormalities in patients with major depression: An update. *American Journal of Psychiatry*, 146, 311-317.
- Katz, L.C. (1999). Whats critical for the critical period in visual cortex? *Cell*, 99, 673-676.
- Kraemer, G.W., & Clarke, A.S. (1996). Social attachment, brain function, and aggression. *Annals of the New York Academy of Sciences*, 794, 121-135.
- Kramer, P. (1993). *Listening to Prozac*. New York: Penguin Books.
- LaPlanche, J. & Pontalis, J.B. (1973). *The Language of Psychoanalysis* (D. Nicholoso-Smith, Trans.) New York: Norton. (Original work published 1967).
- LeDoux, J. (1996). *The Emotional Brain, The mysterious underpinnings of emotional life*. New York: Touchstone.
- _____ (2002). *Synaptic Self, How our brains become who we are?* New York Viking Penguin.
- LeDoux, J., Romanski, L., & Xagoraris, A. (1992). Indelibility of Subcortical Emotional Memories. *Journal of Cognitive Neuroscience*, 1, 238-243.

- Meadow, P. (1989). How We Aim To Be With Patients. *Modern Psychoanalysis*. 14: 145-162.
- Mesulam, M. (1998). From sensation to cognition. *Brain*, 121, 1013-1052.
- Perry, B. & Pate, J.E. (1994). Neurodevelopment and the psychobiological roots of post-traumatic stress disorder. In L. Koziol & C. Stout (Eds.), *The Neuropsychology of Mental Disorders, A Practical Guide*. (pp. 205-217). Springfield, IL: Charles C. Thomas.
- Perry, B.D., Pollard, R.A., Blakely, T.L., Baker, W.L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation, and "use-dependent" development of the brain. How "states" become "traits." *Infant Mental Health Journal*, 16, 271-291.
- Price, J.L., Carmichael, S.T., & Drevets, W.C. (1996). Networks related to the orbital and medial prefrontal cortex; a substrate for emotional behavior? *Progress in Brain Research*, 107, 523-536.
- Rolls, E. (1992). Neurophysiology and the functions of the Primate amygdala. In John P. Aggleton (Ed.), *The Amygdala*, (pp. 143-166). New York: Wiley-Liss.
- Schore, A.N. (1994). *Affect regulation and the origin of the self: The neurobiology of emotional development*. Mahwah, NJ: Erlbaum.
- _____ (1997). A century after Freuds Project: Is a rapprochement between psychoanalysis and neurobiology at hand? *Journal of the American Psychoanalytic Association*, 45, 841-867.
- _____ (1999). Psychoanalysis and the development of the right brain. Unpublished address, The First North American International Psychoanalytic Association Regional Research Conference, "Neuroscience, Development & Psychoanalysis." New York, December, 1999.
- _____ (2000). Early relational trauma and the development of the right brain. Unpublished invited presentation, Anna Freud Centre. London, England, March, 2000.
- _____ (2001a). The Effects of a Secure Attachment Relationship on Right Brain Development, Affect Regulation, and Infant Mental Health. *Infant Mental Health Journal*, 22, 7-66. (Reprinted in www.trauma-pages.com.)

- (2001b). The Effects of Early Relational Trauma on Right Brain Development, Affect Regulation, and Infant Mental Health. *Infant Mental Health Journal*, 22, 201-269. (Reprinted in www.trauma-pages.com.)
- Schwarz, E. & Perry, B. (1994). The post-traumatic response in children and adolescents. *Psychiatric Clinics of North America*, 17, 311-327.
- Shull, D. (1995). The Neurobiology of Freud's Repetition Compulsion. Unpublished Masters Thesis, The University of Chicago: Chicago.
- Southwick, S., Yehuda, R., Giller, E., & Perry, B. (1990). Altered Platelet alpha2-adrenergic Receptor Binding Sites in Borderline Personality Disorder. *American Journal of Psychiatry*, 147, 1014-1017.
- Spotnitz, H. (1985). A Neurobiological Approach to Communication. In *Modern Analysis of the Schizophrenic Patient*, 2nd ed., New York: Human Sciences Press, Inc. (Original work published 1969)
- Stahl, S (2000) *Essential Psychopharmacology, Neuroscientific Basis and Practical Applications*, 2nd ed., Cambridge: Cambridge University Press.
- Wagner, A.D., Poldrack, R.A., Eldridge, L.L., Desmond, J.E., Glover, G.H., & Gabrieli, J.D.E. (1998). Material-specific lateralization of prefrontal activation during episodic encoding and retrieval. *NeuroReport*, 9, 3711-3717.
- Websters New Collegiate Dictionary (1973), 1st printing, Springfield, Massachusetts. G.& C. Merriam Company
- Weinfeld, N.S., Sroufe, L.A., Egeland, B., & Carlson, E.A. (1999). The nature of individual differences in infant-caregiver attachment. In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: theory, research, and clinical applications* (pp. 68-880). New York: Guilford Press.
- Weinstock, M. (1997). Does prenatal stress impair coping and regulation of hypothalamic-pituitary-adrenal axis? *Neuroscience and Biobehavioral Reviews*, 21, 1-10.
- Whalen, P., Rauch, S., Etcoff, N., McInerney, S., Lee, M., & Jenike, M. (1998), Masked Presentations of Emotional Facial Expressions Modulate Amygdala Activity without Explicit Knowledge. *The Journal of Neuroscience*. 18(1): 411-418.

Yamada, H., Sadato, N., Konishi, Y., Kimura, K., Tanaka, M., Yonekura, Y. & Ishii, Y. (1997). A rapid brain metabolic change in infants detected by fMRI. *NeuroReport*, 8, 3775-3778.

_____ with Itoh, H. (2000). A milestone for normal development of the infantile brain detected by functional MRI. *Neurology*, 55, 218-223.

Zald, D.H., & Kim, S.W. (1996). Anatomy and function of the orbital frontal cortex, II: Function and relevance to obsessive-compulsive disorder. *Journal of Neuropsychiatry*, 8, 249-261.

561 10th Ave. #43J
NY, NY 10036

Annals of Modern Psychoanalysis
Volume II, Number 1, 2003