Bringing Unconscious Choices to Awareness: 'Default Mode', Body Rhythms, and Hypnosis

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Abstract: Psychotherapy, and in particular hypnotherapy, affects the brain, as well as the thoughts and beliefs processed by the brain, through the process of neuroplasticity and neurogenesis. The brain is constantly adapting to new information and new circumstances, e.g., modifying patterns of connection between different parts of the brain and reorganizing neural pathways and functions (neuroplasticity), as well as developing new neurons (neurogenesis). We will be looking at very specific ways that hypnosis and hypnotherapy influence these changes, and at some of the dysfunctional conditions that they can be helpful in managing or repairing, such as addictions, ADHD, autism, chronic pain, depression, and sleep disorders. First we begin with an overview of the unconscious functions at work, day and night, in our physical, emotional, and mental life: resting-state networks, especially the default mode network; vagal nervous system and heart-rate variability; ultradian and circadian rhythms; reward and stress networks; and the mirror neuron network. The default mode network is vital to our sense of self and sense of agency, moral sensitivity, organizing memory to reconstruct the past, simulating the future such as inner rehearsal and daydreaming, and imagination such as free association, stream of consciousness, and taking other people's perspective. We provide suggestions for "brain-friendly" psychotherapy.

"[M]ost people do not know that most mental processes are autonomous."1

"All mental states have correlates in the brain and any change in our attitudes, beliefs, cognitive styles, preferences and modes of interaction must therefore be accompanied by changes in the brain."²

"Whether it is called symptom relief, differentiation, ego strength, or awareness, all forms of therapy are targeting dissociated neural networks for integration."³

A fundamental assertion of this article is that "hypnosis can change the nervous system for the better."⁴ Hypnosis affects the brain, as well as the thoughts and beliefs processed by the brain, through the process of *neuroplasticity* and *neurogenesis*. The

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brain is constantly adapting to new information and new circumstances, e.g., modifying patterns of connection between different parts of the brain and reorganizing neural pathways and functions (neuroplasticity), as well as developing new neurons (neurogenesis).

An example of modifying connections is using psychotherapy to develop new neural pathways within the corpus callosum, the major highway between the two hemispheres of the brain, which is reduced through the effects of trauma.⁵ Another example is found with patients who suffer chronic fatigue syndrome, which has the symptoms of persistent fatigue and a decrease in cortical gray matter volume. After successful psychotherapy which addresses faulty thoughts and beliefs about the condition, patients not only feel better but also show a significant increase in gray matter volume localized in the lateral prefrontal cortex (an area related to the speed of cognitive processing).⁶ A depressed person usually suffers from an overall lowering of energy and excessive rumination and selfcriticism. The rumination and negative self-talk is due to an overactive prefrontal cortex, and that needs to be deactivated and calmed, perhaps through hypnosis or meditation. Low affect needs to be stimulated by activating the emotional limbic areas of the brain.

Another brain structure, the hippocampus, offers examples of both: modifying connections and developing new neurons. Connectivity in the hippocampus is implicated in a number of conditions related to deficits in mental processing, such as autism, Alzheimer's disease, drug addiction, and PTSD; hypnosis and other forms of psychotherapy can help to reestablish these interrupted connections. An example of developing new neurons is the regeneration of the hippocampus in the brain. The volume of the hippocampus is found to be as much as 18% smaller in adult survivors of trauma, in individuals presenting currently with severe and chronic PTSD, or with borderline personality disorder.⁷ That damage results in corruption of thought process and learning, particularly deficits of encoding short-term into long-term memory.⁸ Through the intervention of appropriate psychotherapy and social support, the full volume of the hippocampus can be restored, and with it the full functionality.9

brain changes Those structural both between are hemispheres and within each hemisphere. There is some evidence that in traumatic states, Broca's area (a circuit of interaction with other brain areas that plays a vital role in the production of language, located in the left hemisphere) may be inactive, whereas structures in the right hemisphere are highly activated.¹⁰ These findings point to the experience in trauma of overwhelming emotion without the mediating effect of cognitive discernment and the ability to communicate verbally. People who have suffered damage to the Broca's area of the brain lose their ability to speak although they can still understand what is said to them. This provides a key to understanding the importance in trauma resolution for the individual to speak, formulating words and expressing themselves verbally. Doing so rewires the brain's damaged connections between and within hemispheres. In fact, dysfunctional communication between cortical and subcortial structures within each brain hemisphere may be implicated in PTSD¹¹, attachment disorders¹², and psychosomatic disorders.¹³¹⁴

We will be looking at very specific ways that hypnosis and hypnotherapy influence these changes, and at some of the dysfunctional conditions that they can be helpful in managing or repairing, such as addictions, ADHD, autism, chronic pain, depression, and sleep disorders. First we begin with an overview of the unconscious functions at work, day and night, in our physical, emotional, and mental life.

The conscious mind and the unconscious share one body. And both have a definite effect on it. There are many functions of the body that are best managed by the unconscious: breathing, monitoring and dispatching hormones, regulating the heart beat, and a thousand others. One group of such functions is called *proprioception*: the unconscious perception of movement and spatial orientation arising from stimuli within the body itself. The body is constantly monitoring its own state and making adjustments to achieve optimal operation. This process is highly individual, of course; what is assessed to be too much adrenaline or too low blood sugar varies from one moment to the next, as well as from one individual to the next.

Another category of these unconscious functions is *neuroception*: the unconscious detection of environmental

influences, without awareness. This requires a neural circuit that evaluates risk in the environment from a variety of cues. Our nervous system detects these cues and reacts by automatically shifting us into different states. Something in the environment assessed to be a threat or danger immediately activates the fight or flight response of the sympathetic branch of the autonomic nervous system. Of course, what one person's unconscious determines to be a threat may to another person be benign or may even be desirable, depending on previous experience. Now for some people, or in certain situations, the body's response to threat in the environment is the opposite of fight/flight; namely, it is to freeze (withdraw, become despondent, feel stuck and paralyzed) through activation of the parasympathetic branch of the nervous system. When the unconscious neuroception network assesses safety instead of danger, the body responds with an activation of the "Social Nervous System," a third branch of the autonomic nervous system which allows letting one's guard down without the defense of freezing.

These are all cases of *bottom-up* processing: the body in its primitive systems of maintenance and defense dictate to the whole being a response in a given moment in time. This primitive response aspect of a human being relies on both our reptilian (parasympathetic capacity for freezing, or death feigning) and mammalian (sympathetic capacity for fight or flight) heritage. Here the body subjugates the brain and the mind. But, being human, we also have the potential for top-down processing which enables us to use our conscious cognitive functions to restructure the body's prescriptive conditioning even though we may have experienced traumas or disruptions in our normal development. We can recognize the old behavior patterns as an adaptive survival strategy, perhaps no longer needed or useful. Our brain can reorganize how our body feels. We can deconstruct the old belief system and reinterpret it, see things in a different way, and choose to recondition ourselves to a new set of responses.

The body responds to beliefs held either consciously or unconsciously, and in fact there is an intricate connection between conscious mind, the unconscious mind, and the body. This is obvious in the placebo and the nocebo effects. The placebo effect is well-known. The mind's expectations largely determine the body's experience when a drug is administered. Some patients in a double-blind clinical trial who are actually receiving a sugar-pill rather than the actual medication experience relevant physiological effects as if they had received the actual medication. This is a demonstration, of course, of the miraculous power of the human mind and imagination: mind over matter. Not so well known or studied is the *reverse* placebo effect, however: patients' expectations that an actual medication will *not* work leads their body to cancel out any physiological effect of the medication. In fact, research documents that the mind can make a placebo "real" and render a real drug useless.

A new study documents this nocebo effect.¹⁵ This particular experiment involved causing pain to the legs of test subjects, then adding a painkiller medication to an IV drip while assessing the subjects' pain levels. When the painkiller drug was present, the test subjects were told about it, and just as expected their pain scores significantly dropped. But when test subjects were told the pain medication had been stopped, their pain levels returned back to the original, non-medicated levels even though the pain medication was secretly still being dripped into their IVs. The expectation of poor or no results became a selffulfilling prophecy. "Doctors shouldn't underestimate the significant influence that patients' negative expectations can have on outcome," says Professor Irene Tracey of the Centre for Functional Magnetic Resonance Imaging of the Brain at Oxford University, who led the research. "For example, people with chronic pain will often have seen many doctors and tried many drugs that haven't worked for them. They come to see the clinician with all this negative experience, not expecting to receive anything that will work for them. Doctors have almost got to work on that first before any drug will have an effect on their pain." Subjects in the study were in an MRI scanner during the experiment, and the researchers used brain imaging to confirm the participants' reports of pain relief. MRI scans showed that the brain's pain networks responded to different extents according to their expectations at each stage, and matching their reports of pain. This showed that they really did physiologically experience different levels of pain when their

expectations were changed, although the administration of pain relief remained constant.

The miraculous power of the human mind and imagination is here at work; and hypnosis and hypnotherapy are especially well-suited to accessing the belief systems embedded in the mind, and to modifying the expectations of a treatment outcome.

The placebo effect is actually the ability, indeed the inclination, of the unconscious mind to accept suggestions and employ the innate human resource of imagination. One of the ways that the medical field can work together with others in integrative medicine is to become acutely aware of the principles of suggestion, how the unconscious mind works, and how what medical professionals say affects patients. A dramatic example is how the unconscious mind is processing all that happens in an operating room under anesthetic. Because patients are "asleep" does not mean they can't hear or are oblivious to their environment.

Anesthetized consciousness is ungrounded, but with "remote" awareness and memory. Milton Erickson¹⁶ and David Cheek¹⁷ discovered that anesthetized patients could perceive conversation at some level of awareness. Research with anesthetized surgery patients reported by Halfen¹⁸ and Bennett¹⁹ shows that a surprising amount of awareness does exist during anesthesia, for bad but also for good. The beneficial effects of positive suggestions to patients during anesthetized surgery have been well-documented.²⁰ Hypnosis has proven effective in patient recall of events that occurred under anesthesia.²¹ The famous neurosurgeon Penfield concluded that the mind is active during anesthesia. He discovered that patients consistently recalled exact details of incidents during surgery.

The question arises, what is the brain doing when engaged in unconscious mental processing such as proprioception, neuroception, anesthesia, placebo or nocebo?

"If we think in terms of symptomology, whether we are talking about psychiatric symptoms, behavioral problems, or even just physical health symptoms, most of the symptoms are actually in the periphery. The nervous system is not solely a brain independent of the body, but a brain–body nervous system."²²

Recent analysis produced by neuroimaging technologies has revealed something quite remarkable about this brain-body nervous system: a great deal of meaningful activity is occurring in the brain when a person is sitting back and doing nothing at all.²³ In fact, some brain regions are more engaged during rest than during constrained cognitive activity, and the network of these regions is referred to as the *resting-state networks*. Actually the resting individual is engaged in some form of mental process – of fantasy, imagination, daydreams, reconstructing the past, or reverie – which is not "doing nothing at all." Yet these mental activities occur when the person is "at rest," leaving most people with the impression that most brain areas stay pretty quiet until called on to carry out some specific task.

It turns out that when your mind is at rest – when you are daydreaming, contemplating (but not planning) tonight's dinner, asleep in bed, or anesthetized for surgery – dispersed brain areas are busy communicating with each other behind-the-scenes. And the energy consumed by this ever active messaging, known as the brain's *default mode network*, is about 20 times that used by the brain when it responds consciously to an outside stimulus. Another way to say this is that 60 to 80 percent of all energy used by the brain occurs in circuits unrelated to any external event.²⁴

Several discreet resting-state networks have been identified. At the highest hierarchical level, there are two anticorrelated systems in charge of intrinsic and extrinsic processing, respectively. They are the default mode network, a network of regions that show high metabolic activity and blood flow at rest but which deactivate during goal-directed cognition; and an attention system which attends to a specific task at hand but deactivates during periods of rest.²⁵ Both the default mode network and the attention system are associated with top-down processing, a unique collaboration evident in creative problemsolving, insight, and mental simulations, with the default mode network furnishing control of the attentional system.²⁶ The intrinsic default mode system appears to be partitioned in three modules: generation of spontaneous thoughts; inner maintenance and manipulation of information; and cognitive control and switching activity. The extrinsic system is made of two distinct modules: one including primary somatosensory and auditory areas and the dorsal attentional network, and the other encompassing the visual areas.²⁷

Regions of the brain termed resting-state networks mainly include the primary sensory, motor, language, attention and default-mode networks.²⁸ Regions included in these networks show a synchronized activity in absence of any specific cognitive activity, that is, at rest, while they are known to be engaged during sensory-, motor-, language- or attention-related tasks, respectively. As for the default mode network, it includes brain areas associated with multiple high-order functions that are stimulus-independent generally and thus self-referential processes. These can be related to organizing memory such as reconstructing the past; simulating the future such as fantasy, inner rehearsal and daydreaming; and imagination such as free association, stream of consciousness, and taking other people's perspective.

A recent meta-analysis²⁹ has identified various areas of the brain as default mode network components, including the posterior cingulate cortex (PCC), anterior cingulate cortex (ACC), middle temporal gyrus, inferior parietal cortex, medial prefrontal cortex (mPFC), and hippocampus.

The brain's default network may be acting as a "sentinel", supporting a broad low-level focus of attention to monitor the external environment for unexpected events and preparing ahead of time to respond to the outside world when the need arises.^{30 31} This preparation can take various forms, for example retrieving and organizing autobiographical memories, envisioning the future, and empathetically conceiving the perspectives of others. Among its jobs may be running life simulations, providing a sense of self and maintaining crucial connections between brain cells. In short, the default network is responsible for selfprojection-mentally transporting oneself into alternate times, locations, or perspectives-as manifested in episodic memory, navigation, prospection (i.e., anticipating future events), and theory of mind (taking another's perspective).³² Self-projection into alternative pasts and futures actually serves a vitally important function: survival. Default mode processes run continuously in response to external and internal cues. Incoming information activates associations and then expectations based

on past experiences. These associations are updated by new information and are used to generate predictions about future events. When incoming information is incongruent with activated associations, default network processes generate analogies that can be used to make new predictions. Thus, the predictions continuously generated by the default network guide people's thoughts, behaviors, and perceptions.^{33 34}

The strength of default mode network connectivity contributes to important introspective psychological processes in everyday social life, such as the intuitive understanding of other people through inner representation of their emotional states. Our empathy for others is carried as a "memory of the future," a neuronal cache, storing "a priori scripts," which are recalled to deal efficiently with upcoming environmental events.³⁵ The default mode network influences an automatic response of empathy when we observe painful situations involving others.³⁶

We have all had the experience of trying to remember some specific fact, perhaps a name or vocabulary word that escapes memory. Episodic memories like these can be retrieved by an intentional search for the desired information, but if that mental search fails, sometimes the fact may enter our consciousness without any intention to retrieve it as if 'out of the blue'. We can infer that a gating mechanism exists which regulates incidental retrieval activity, since such 'out of the blue' retrieval does not occur every time we could be reminded of something. One study³⁷ found that failure to incidentally retrieve was selectively associated with reduced activation of brain areas implicated in the default mode network. This demonstrates that relative deactivation of the brain's default mode regions acts to gate consciousness from currently irrelevant memories. When you can't remember where you left your keys, allow the 'trying' part of your brain to deactivate and you may suddenly remember where they are.

In one interesting experiment³⁸, experienced Zen meditators and non-meditator controls were asked to approximate the meditation experience by focusing on the breath and allowing thoughts to pass by without attachment to them, and a mental discrimination task was randomly presented interrupting the "meditation" (itself challenging given the intrusiveness of fMRI neuroimaging equipment). All subjects showed a heightened prevalence of default mode mental activity during uninterrupted periods, and a sudden deactivation of the default mode during the task-oriented periods. However, meditators showed an ability to voluntarily contain the automatic cascade of thoughts triggered by the mental task, and to return more promptly to a default mode of non-task mentation. For the meditators, activity in the brain during default mode was lower than for non-meditator controls, showing an ability to voluntarily regulate the stream of thoughts occurring automatically in the absence of goal-directed activity. Additionally, research results suggest that the long-term practice of meditation may be associated with functional changes in brain regions related to internalized attention even when meditation is not being practiced.³⁹

This unconscious processing includes the sense of personal agency, i.e., the subjective experience of being the cause of our own actions or attributing agency to the actions of others in a real or imagined social situation. The sense of agency, intentionality, and perspective taking, are closely allied to the subjective experience of personal responsibility: without agency we would not feel, or could not be held, responsible for our actions. Our natural propensity to attribute cause to our and to other people's behavior allows us to make moment-to-moment predictions on the intentions of others and to update them continuously as our experience of others changes.

The sense of personal agency drives "moral emotions" which might include guilt, anger, indignation, shame, regret, gratitude, pride, embarrassment, envy, pity, contempt, awe, and jealousy. Moral emotions are linked to the interest either of society as a whole, or at least of other people. For example, guilt is experienced when we recognize ourselves as the cause of another person's misfortune, whereas compassion is experienced instead if we witness someone being accidentally hurt. We may feel indignation when we get hurt by someone intentionally and also if we witness someone else being hurt intentionally by a third-party. One might feel embarrassed rather than indignant at a faux pas in which a witty remark intended to provoke amusement at a dinner party instead causes someone to feel ridiculed These reactions are generated automatically, unconsciously, and can be called moral sensitivity, the mechanism by which moral significance is automatically

attributed to ordinary events. And current brain research reveals that "moral sensitivity is at the core of spontaneous, or 'default-state', mental activity."⁴⁰

Clearly, the functioning or dysfunction of the default mode network affects an individual's sense of agency and moral sensitivity, and does so normally at an unconscious level.

We examine several ways in which the current research on default mode networks has application to our clinical work in healing. One is that some dysfunctions or malfunctions of an individual's body-mind can be explained as erroneous functioning of his/her default mode network. Two is that understanding the default mode network can help to identify and explain, and potentially access, preconscious and unconscious mental activity such as moodiness, prejudice, irrational fears, or uncontrollable anger. And three is that some default mode functions may be brought under conscious control and direction, potentially through the interface of mental processes that allow access to otherwise unconscious mentation, such as altered states, meditation, or hypnotic trance states.

Default mode network's access to preconscious and unconscious mental activity

Interestingly, the default mode network activates during retrieval of past events from memory but deactivates during encoding of novel events into memory.⁴¹ Furthermore, studies have found that these regions show less activity during encoding for items that are later remembered than for those that are forgotten.^{42 43} However, the brain's hippocampus region normally associated with the default mode network⁴⁴ – is activated in both memory retrieval and encoding. An explanation of this phenomenon is that retrieving memories is a mental process oriented toward internal events. activating the hippocampal regions coupled with the default mode network, whereas laying down memories is a mental process oriented toward external events, in which the activated hippocampus is decoupled from the default mode network.

The distinction has potential relevance to the efficacy of utilizing the hypnotic trance state for accessing traumatic memories, and for encoding newly contextualized memories following a therapeutic corrective emotional experience. The default mode network is activated in a traditional hypnotic trance state, as it is in any state of mental and physical relaxation. This explains the relative ease of access to memories in the hypnotic trance. The default mode network deactivates during encoding of *external* events into memory, but encoding newly contextualized *internal* events into memory may in fact continue to engage the hippocampal regions now decoupled from the default mode network.

The hippocampus can, under certain conditions, generate new growth, i.e., grow new neurons. One example is that taxicab drivers, who navigate their way through unfamiliar streets everyday, show measurable growth in their hippocampus.⁴⁵ We know that specific actions in the therapeutic experience contribute to new hippocampal regeneration as well. New measurable growth of neurons takes place in the hippocampus when something is learned or remembered, two basic components of effective therapy.

Bringing default mode functions under conscious control and direction

David Abram⁴⁶ relates his experience with traditional, indigenous shamans in rural Asia, where he learned that their commitment was primarily "to the earthly web of relationships" in which the community is embedded. The shaman serves first and foremost as an intermediary between the human and the nonhuman; healing is secondary. His or her primary task is to help maintain the balance between the human community and the nonhuman environment. That function in our modern societies can no longer be reserved for a select few specialists; it is now up to all people to participate in this project. And most people's participation is accomplished unconsciously, through the behind-the-scenes regulation by the default mode network and neuroception, tides in body fluids and rhythms of body energy, and the workings of the central nervous system through established reward and stress pathways. But, like the shamans of indigenous cultures, we too are capable of bringing to conscious control these functions of maintaining balance between the human community and the nonhuman environment. Ancestral people consider awareness or mind not as something in their *head*, but as something they themselves are *inside of*. The body

and conscious awareness are vehicles for the balancing of the Great Forces of the World. Abram refers to this as a "turning inside out." Once we come to recognize the relationship between our interiorized, psychological selves and the natural world around us, "we begin to turn inside-out, loosening the psyche from its confinement within a strictly human sphere, freeing sentience to return to the sensible world that contains us. Intelligence is no longer ours alone but is a property of the earth; we are in it, of it, immersed in its depths."⁴⁷

Daniel Siegel⁴⁸ explains this way of looking at ourselves and others as night vision. He likes to describe a walk he took on a deserted Oregon beach late one night, with his flashlight turned off. It took his eyes a while to pick out of the darkness the vague outlines of rocks, the billions of stars, the serpentine border of the sea and sand. Unlike the familiar reality that we see by the light of day, the world revealed to us by this kind of night vision can be disorienting, even surreal, possibly menacing, but often magical. We feel our capacities for perception sharpened and transformed as we become more attuned and attentive to the smallest, most subtle, variations of shape and shadow. In some way, by seeing less, we see more. Night vision is a metaphor for the world of subtle processes that interpersonal neurobiology opens up for our investigation. It awakens us to the everyday marvels of the human brain--our ability to transcend the boundaries of space and time, to "see" through the barrier of the physical body into the invisible precincts of our own or another's mind.

And so we expand our awareness beyond the strictly consciously knowable, beyond the strictly visible world, beyond the strictly human sphere, bringing to awareness the natural rhythmic patterns within our body-mind continuum that otherwise operate as they were programmed to in early life experience – which can too often be unresolved traumatic experience. There exists a transitional phase between activation of one pole of any of our body's systems and the other pole, when sympathetic activation is surrendering to parasympathetic, midway as executive functioning is dimming to allow the default mode to ascend to dominance. "The brain is always offering undifferentiated possibilities in the moments between activations and deactivations, before patterns are formed."⁴⁹ These are

"empty moments" of undefined potential, "like a clearing in a forest, an opening in which something new can be experienced. It is not and is, as the Zen master would say."⁵⁰ In this opening, brought to conscious awareness, we can make a choice of which way to go. And one of the available choices is, of course, to activate both at the same time. Doing so with the sympathetic-parasympathetic branches allows for extraordinary states of consciousness. A mindfulness meditative state or a hypnotic trance state allows access to both the focused attention of executive function and the relaxed openness of default mode.

Default mode functions may sometimes be brought under conscious control and directed in a top-down manner.⁵¹ Evidence from neuroscience⁵² further indicates two pathways that are involved in accessing memories: a top-down search process initiated in the left frontal cortex and a bottom-up spread of activation from the medial temporal cortex. The bottom-up spread of activation is outside of awareness or conscious direction, is automatically and continuously generated, and is what may be called mind-wandering or daydreaming. The topdown search process is generated through awareness and can be consciously directed; it is observable in activation of the orbitofrontal cortex, an area strategically located behind the eyes, between the "higher," thinking areas and the "lower," emotional areas. This region integrates and coordinates cognitive and emotional processes, helping us regulate emotional arousal and control our impulses. Sometimes called the "center of free will," it enables us to think before we act.

Mindfulness is the key to clients becoming more and more acutely aware of internal sensorimotor reactions and in increasing their capacity for selfregulation. Mindfulness is a state of consciousness in which one's awareness is directed toward here-and-now internal experience, with the intention of simply observing rather than changing this experience. Therefore, we can say that mindfulness engages the cognitive faculties of the client in support of sensorimotor processing, rather than allowing bottom-up trauma-related processes to escalate and take control of information processing. To teach mindfulness, the therapist asks questions that require mindfulness to answer, such as, "What do you feel in your body? Where exactly do you experience tension? What sensation do you feel in your legs right now? What happens in the rest of your body when your hand makes a fist?" Questions such as these force the client to come out of a dissociated state and future- or past-centered ideation and experience the present moment through the body. Such questions also encourage the client to step back from being embedded in the traumatic experience and to report from the standpoint of an observing ego, an ego that "has" an experience in the body rather than "is" that bodily experience."

Early brain development is adversely affected by traumatic experiences in the earliest relationships. Current research has shown that the impaired connections between the right and left hemispheres, that the fibre tract known as the *corpus callosum*, i.e., the major highway between the two hemispheres, may be reduced through the effects of trauma in those who have experienced childhood sexual abuse. However, the brain can develop new neural pathways in the brain, and in particular can develop the *corpus callosum*.⁵⁴ Further, we know that early trauma and abuse damages the hippocampus, and again that it can regenerate.

The vagal nerve system

Our nervous system has more than one defense strategy: a mobilized fight/flight strategy (activated sympathetic response) and an immobilization shutdown defense strategy (activated parasympathetic response). The selection of whether we use a mobilization or an immobilization strategy is not a voluntary decision; it is made outside the realm of our conscious awareness. Our nervous system is continuously evaluating risk in the environment, making judgments, and setting up priorities for responses to possible threats or nonthreatening elements. Our brain is always in the mode of threat detection without awareness, and that function is called *neuroception*.

According to the Polyvagal Theory⁵⁵ two branches of the vagus nerve serve different adaptive behavioral strategies. The phylogenetically older dorsal vagal complex is a slow unmyelinated responding vagus nerve that supports immobilization in response to threat (e.g., the freezing response, or death-feigning). The phylogenetically younger ventral vagal complex originates in the nucleus ambiguus and is a fast-acting myelinated vagus that functions as an active vagal brake. It can rapidly mobilize or calm an individual via its fast-acting inhibitory influence on the activity of the heart without mobilizing the slower-acting sympathetic nervous system. This branch of the vagus nerve is suggested to be involved in the modulation of respiratory-related heart rate variability.⁵⁶

The vagus nerve regulates the activation of the parasympathetic nervous system as a counterbalance, or brake, on the sympathetic nervous system, through communication with the heart. When the environment is appraised as being safe, the defensive limbic structures are inhibited, enabling social engagement and calm visceral states. In contrast, some individuals experience a mismatch and the nervous system appraises the environment as being dangerous, when it is safe. This mismatch results in physiological states that support fight, flight, or freeze behaviors, but not social engagement behaviors. Functionally, the impact of the vagal brake produces a baseline or resting heart rate substantially lower than the intrinsic rate of the pacemaker. When the vagal brake is removed, heart rate can approximate the intrinsic rate of the pacemaker without recruiting sympathetic influences. When cardiac vagal tone via the myelinated vagus is high, the vagus acts as a restraint or brake limiting the rate the heart is beating. When vagal tone to the pacemaker is low, there is little or no inhibition of the pacemaker. The vagal brake provides a neural mechanism to change visceral states by slowing or speeding heart rate. Neurophysiologically, the influence of the vagal brake is reduced or removed to support the metabolic requirements for mobilization (e.g., fight/flight behaviors) and maintained or increased to support social engagement behaviors.⁵⁷

From a clinical perspective psychopathology would be either the inability to inhibit defense systems in safe environments (ranging in severity from hypervigilance to Anxiety Disorders to PTSD) or the inability to activate defense systems in risk environments (e.g., codependency, Williams Syndrome). Many mental health or emotional disorders, such as autism, social anxiety, PTSD, borderline personality disorder, depression and schizophrenia, have an underlying state regulation disorder, an underlying flatness of affective tone expressed on their faces, an underlying lack of expressiveness in their voices and they also tend to have higher heart rates and less vagal activity. Low vagal tone correlates with higher emotional defensiveness, and with a diminished ability to calm down following fear or anger arousal.

Treatment of these disorders involves creating safety to allow the social engagement system to become activated. Functionally, when the environment is perceived as safe, the bodily state is regulated in an efficient manner to promote growth and restoration (e.g., visceral homeostasis). This is done through the vagal pathways to slow the heart, inhibit the fight/flight mechanisms of the sympathetic nervous system, dampen the stress response system (e.g., cortisol), and reduce inflammation by modulating immune reactions (e.g., cytokines). That is, we cannot embark on the adventure of honestly evaluating the efficacy of our habitual life patterns until we can self-regulate to a calm state through applying the vagal brake to our natural fight/flight response to stress.

Swinging or rocking in a head-to-toe direction stimulates the vagal receptors involved in blood pressure regulation and helps organize the whole vagal system. It is extremely calming for an individual. Rocking on an exercise ball may provide an efficient method of stimulating the sacral afferents of the parasympathetic nervous system. These afferents transmit information to the brainstem and increase parasympathetic tone.⁵⁸ Sounds that invite relaxation of defense and experience of safety include music and the human voice which has a wide range of modulation and expressiveness.

Heart Rate Variability

Basically the heart rate of a healthy person at rest is going up and down with his/her breathing. If a person has a flat, unvarying heart rate without this oscillation, he/she is at risk for serious complications including death. Overactivation of the vagus, applying too much parasympathetic influence too suddenly, can lead to massive slowing of the heart rate and cessation of breathing. This is analogous to the death feigning of an animal of prey who has reached the point of terminating a fight/flight response to threat.

Social engagement, deployment of emotion regulation strategies, and perceived security in attachment relationship have been shown to be accompanied by elevated output of the ventral vagal complex (as indexed by heart rate variability). On the contrary, social isolation has been related to attenuated heart rate variability, thus supporting the key assumptions of the Polyvagal Theory.

The autonomic nervous system responds to hypnosis with a shift toward parasympathetic control, and that response can be measured by heart rate variability.⁵⁹ An identical response is found with meditation.⁶⁰ The autonomic status in hypnosis is associated with a change towards parasympathetic activity, but

not necessarily with deactivation of sympathetic activity.⁶¹ The parasympathetic influence, as measured by heart rate variability, corresponds precisely with an individual's self-report of depth of hypnotic trance and with other physiological phenomena (EEG, pulse, blood pressure, breathing pattern). Further, there is a positive correlation between hypnotic susceptibility and autonomic responsiveness during hypnosis, with high hypnotizable subjects showing a trend toward a greater increase of vagal activation than do low hypnotizables.⁶²

According to current research,⁶³ high hypnotizables may be emotionally hypersensitive to the perception of threat, and therefore to be at greater risk of dysregulation of the sympathetic nervous system. That is, they have higher baseline arousal and slower recovery following stress than controls. Low hypnotizable subjects, on the other hand, are hyposensitive to emotional threats, and therefore are at greater risk of dysregulation of the parasympathetic nervous system. They have a lower baseline arousal level and faster recovery following stress. Both high and low hypnotizables tend to reduce or block from consciousness negative affectivity, as expressed in their verbal reports, but not their physiological effects. This could result in an incongruence between what they are aware of and what their bodily responses reveal.⁶⁴ Although results are not conclusive, there is some evidence that patients diagnosed with Posttraumatic Stress Disorder (PTSD) are more likely to be highly hypnotizable.⁶⁵

Acupuncture reliably activates the sympathovagal system, increasing connection between the pain, memory, and affective regions within the default mode network, most notably the anterior cingulate cortex (ACC) as measured by heart rate variability.⁶⁶

Ultradian and Circadian rhythms

It is well-established that the body, including mental processing, has rhythmic alternating cycles of activity and rest. This can be seen in sleep, with more active rapid eye movement (REM) states alternating with deeper non-rapid eye movement (NREM) states of restfulness. The body follows the same rhythmic cycle during waking hours as well.⁶⁷ There are a number of different ultradian cycles that seem to recur at

approximately 90–120 minute intervals in human physiology.⁶⁸ The basic rest–activity cycle includes phase changes in a wide range of psychophysiological, motor, cognitive, and perceptual functions, and more complex social behavior. These ultradian changes in mood, cognition, and behavior are managed unconsciously, but once brought to conscious awareness can be intentionally influenced.

Rossi⁶⁹ recognized this pattern as offering a periodic "ultradian healing response" approximately every two hours throughout the day, a kind of natural self-hypnosis state of restful openness. When it is not respected, it leads to an "ultradian stress syndrome" that offers resistance rather than openness. The need for a restful interlude every 90 minutes or so is evident in any classroom, business meeting, church service, or study group. This cyclical pattern regulates the alternating dominance of either the reception of information coming from external sources (the environment and the body, neuroception), or from internal sources of stimulation (the mind itself, selfreflection, proprioception). The rest phase manifests with a passive state of mind, increased spontaneous imagination, and contemplation; the active phase is characterized by reactivity to environmental stimuli and goal-directed activity. In the rest phase of ultradian rhythm, the brain's right hemisphere is more active and left nasal airflow is superior, while in the active phase the brain's left hemisphere is more active and right nasal airflow is superior.⁷⁰ Brought to conscious awareness, the difference between nostrils in one's breathing offers an opportunity to selfregulate the rest-activity cycle by deliberately breathing through the nostril that will activate rest or activity.

The ultradian rhythm synchronizes with the longer 24-hour circadian rhythm, creating unconscious patterns for individuals over time. Some people are day people, whose energy levels (and incidentally, susceptibility to hypnotic induction) peaks at 10 am and 2 pm. Night people are those whose energy levels and hypnotic susceptibility peak at 1 pm and between 6 and 9 pm.⁷¹ "The circadian rhythm controlling our sleep-wake cycle has farreaching effects on every aspect of life, from behavior, attention, and alertness, to hormone production, body temperature, appetite, and digestion."⁷² Sleep is obviously vital to all mammals for restoration of energy that is used and depleted

during the day. Another important benefit of sleep is the brain's opportunity to slow down its task-oriented cognitive processing involving mostly fast-paced beta frequencies, and to operate at the slower frequencies of theta and delta. In deep NREM sleep, delta frequencies are dominant. Also, we need sleep to consolidate memories. Memory consolidation of experiences during the day is enhanced when the brain functions at theta frequency, which is dominant during REM sleep and occurs in the hippocampus.⁷³ Theta brain waves are increased during hypnosis as well, indicating why it is so fortuitous to correct old beliefs, release old perseverating memories, and construct new paradigms within the hypnotic trance.

Reward and Stress networks

Much of an individual's behavior is determined through the unconscious operation of the *reward pathway*. This pathway is a convenient way to conceptualize the interconnected activities of the areas in the brain where dopamine flows. Dopamine is released when we experience pleasure, happiness and love, and when life-enhancing needs such as food and sex are met. Upon experiencing pleasure, dopamine is produced in the midbrain and distributed to relevant parts of the brain: the nucleus accumbens which activates motor functions, the amygdala which activates emotion, and the prefrontal cortex which focuses attention. The hypothalamus and pituitary regulate the level and duration of pleasure so that when the body experiences satiation, it stops the pleasure-seeking activity. When we have eaten enough food, or drunk enough water, or experienced enough enjoyment, we reach gratification and stop. An addict's compulsive use of a substance or behavior eventually compromises the reward pathway in two ways: the body develops a tolerance for the dopamine produced requiring ever higher dosages to achieve the feeling of pleasure, and the self-regulation of satiation stops working.

The *fear and stress pathway* is made up of the hypothalamus, pituitary, and adrenal glands, and controls how the body responds to threat by releasing hormones and neurotransmitters to initiate either the freeze response (parasympathetic dominance) or the fight/flight response (sympathetic dominance). When the experience of threat is repeated or sustained, or is anticipated even when the threat no

longer exists, then the fear pathway produces stress. This network of autonomic nervous system and endocrine system in constant activation produces anxiety, phobias, and posttraumatic stress. However, this hyperactivation can be reduced through pharmacological treatment or psychotherapy.⁷⁴ The compulsive activation of pleasure or fear "brings about a physiological change in the biochemistry of the reward pathway and the stress pathway. Hypnosis, by altering the neurochemistry, can help shift the balance back to the healthy, natural homeostatic balance that is wired in."⁷⁵

Mirror neuron network

The brain contains a network of neurons that respond directly to the intentional actions and emotions of others. The areas of my brain that would activate when I am serving a tennis ball or feeling poignant sadness are activated when I observe you serving a tennis ball or feeling poignant sadness. This phenomenon is called *embodied cognition* by today's neuroscience, i.e., understanding others' experience through my own body response and somatic experience.⁷⁶

There is evidence⁷⁷ that mirror neurons play a role in a number of interactive processes, including the intergenerational transfer of trauma,⁷⁸ the development of childhood depression as a result of interacting with a depressed parent, and the continuation of an abuse cycle — by producing non-conscious learning by mirroring of the implicit affective state of the perpetrator as well as that of the victim.

Hypnosis and bringing to awareness what has been unconscious

Altered states of consciousness, such as hypnotic trance, meditation, somnambulism, the stages of sleep, coma, and deep anesthesia, provide a privileged way to study the relationships between unconscious spontaneous brain activity and behavior.⁷⁹ Analysis of all of these is beyond the scope of this article; we will focus on hypnosis and mindfulness meditation.

"We know that the neuroscience of mindfulness and hypnosis is parallel, causing changes in brain activation of the same magnitude. Both feature cortical inhibition as revealed by slowed EEG theta waves, and both show higher levels of activity in areas where theta is prominent, such as the frontal cortex and especially the anterior cingulated cortex."⁸⁰

Hypnosis is a psychological technique that induces a temporarily altered consciousness, the hypnotic state, that is characterized by deep relaxation, focused attention, vivid imagery and increased receptivity to suggestion.81 Functional brain imaging⁸² and EEG⁸³ differentiate the hypnotic state physiologically from sleep, relaxed rest, and alert awareness. Recent EEG research has found a distinctive time sequence for the brain's peak response to a stimulus after hypnotic induction; the Event Related Potential is characteristically 300 milliseconds.⁸⁴ This seems to be distinct to someone in a hypnotic trance, because non-hypnotized individuals given the same stimulus do not respond in the same time frame. When people first go into hypnosis the left hemisphere is more active, but as they go deeper the left hemisphere is inhibited,⁸⁵ allowing for more mental processing in the nonverbal, intuitive, unconscious right hemisphere.

Hypnosis can

allow for intense absorption in both inner reverie and the environment, as well as rewarding engagement in tasks analogous to 'flow' experiences (Csikszentmihalyi, 1991). . . . recent data demonstrate a 'default mode' network of activation during self-referential rest, which is inhibited during cognitive and perceptual tasks. It involves activity in the ventral portion of the anterior cingulate gyrus and the posterior cingulate cortex. The ability to inhibit this default mode network may also be a neural component of hypnotic performance. These data linking hypnosis to modern genetic and neuroimaging methods make it clear that hypnosis is not some arcane idiosyncratic phenomenon, but rather a window into aspects of brain function that have important implications for learning, development, stress response and neural control over somatic processes.⁸⁶

One part of the brain is particularly important as an interface switch between executive function and default mode function, and is referred to as the salience network.⁸⁷ It is the anterior cingulate cortex (ACC) which has been linked to monitoring task performance and the modulation of arousal during cognitively demanding tasks.⁸⁸ In other words, this part of the brain decides when to pay attention to the outside world (task-oriented) and when to focus on the internal world (introspection). Both hypnosis and mindfulness meditation states feature higher levels of activity in areas where theta frequency brain waves are prominent, especially the ACC^{89 90} and the hippocampus, source of these theta rhythms.⁹¹ These altered states of consciousness, then, offer unique access to the mind's higher-order control of awareness and focused attention. And it is precisely these functions that are disrupted or debilitated in PTSD, ADHD, Alzheimer's disease, and others we will be discussing.

The ACC has been associated with monitoring for competition among potential responses or processes. Such conflict monitoring can signal the need for top-down cognitive control, which facilitates the switching of attentional focus between external/ internal focus, or task/ rest.⁹² Increased ACC activity is followed by increased top-down control through suppressing irrelevant

thoughts.⁹³ An example of this is that hypnosis-induced reduction of pain perception, a common function of hypnosis that has been validated in many many studies, is related to an increased functional modulation of the ACC.⁹⁴ Hypnotic suggestion provides the brain with a mechanism of intervening in a bottom-up autonomous pain response by generating a top-down alternative experience.

Recently, positron emission tomography (PET) research has confirmed the involvement of the anterior cingulate cortex (ACC) in the production of hypnotic states.⁹⁵ Hypnotic states are characterized by a decrease in cortical arousal, described as the brain's attentional system, and a reduction in cross-modality suppression (disinhibition). This accounts for the experience of "flow" and insight so common in the hypnotic trance state.

At the same time, hypnosis also brings about an inhibition of the left dorsolateral prefrontal cortex, the part of the brain activated when we engage in deliberate conscious planning, organizing, and regulating.⁹⁶ Conscious everyday thinking is quieted to make way for more creative, inspired, nonlinear connections.

Clinical research documents that during the hypnotic state, the capacity to access and influence functions beyond conscious control is increased.⁹⁷ For example, heart rate variability provides a tool to evaluate the hypnotic state through acute cardiac autonomic alterations.⁹⁸ The heart rate variability may even be useful in measuring the depth of hypnotic trance state.

Most states of consciousness carry an anticorrelation between 'rest' and goal-directed behavior, between self- and external-awareness networks, between openness and focused attention; the more awareness is focused on internal processing (introspection, or self-awareness), the less it is available for attention to sensory input (external awareness) and goal-directed focus, and vice-versa. The hypnotic trance state is an exception; parts of the brain that are normally activated with an opposite on/off switch can be dissociated from each other to allow both to activate at the same time. Under hypnosis, the anterior cingulated cortex (ACC) is activated which narrows attention. But, unlike in the waking state of narrowed attention, the posterior attentional system which stimulates vigilance is *de*activated during hypnosis.⁹⁹ Thus hypnosis creates a state of dual effect: relaxation yet responsiveness.

In a similar dual effect, meditation has been shown through fMRI and EEG studies to activate both the sympathetic and parasympathetic nervous systems simultaneously, creating a calm state with enhanced alertness.¹⁰⁰ There is, in addition, increased activity in the reward pathway, particularly the hippocampus and the amygdala during meditation,¹⁰¹ with increased levels of dopamine,¹⁰² as there is under hypnosis. However, there is a marked neural difference between hypnosis and meditation. In hypnosis, a decrease occurs in functional connectivity across the hemispheres, measured by EEG gamma band coherence,¹⁰³ while in meditation there is an increase in this coherence between and within hemispheres.¹⁰⁴ EEG coherence normally means more of the brain is being used, with an associated improvement in quality of attention. In the case of hypnosis, the decrease in coherence indicates a dissociation, or decoupling, of attention to more than one thing rather than a decrease in mental processing. This dissociation allows one to attend to apparently incongruous thoughts; for example a person can experience being a child of seven in age regression and at the same time experience being a healthy adult available to nurture that child.

Typically in meditation, the frontal cortex becomes active, while activity decreases in the posterior superior parietal lobe, known as the Orientation Association Area (OAA). According to SPECT imaging studies, the left OAA serves to create one's spatial sense of self, while the right side creates the physical space in which that self exists.¹⁰⁵ The OAA, for example, is

related to a lack of sense of self in patients with depersonalization disorder.¹⁰⁶ In meditators, the OAA demonstrates minimal activity levels during peak meditation times, correlating with the subjective experience of blurred distinction between the self and external reality, that is the unitary nature of many transcendental states.

In a detailed examination of the process by which meditation activates both the sympathetic (arousal) and parasympathetic (quiescent) nervous systems simultaneously, studies have shown that maximal stimulation of either component may induce a "spillover" effect which, rather than inhibiting the activation of the other, results in the simultaneous activation of both systems.^{107 108} For example, the "hyperquiescent" state achieved through meditation, experienced as oceanic bliss, may trigger the activation of the arousal system, resulting in a "burst of energy." That may lead one to feel the profound excitation of kundalini rising, or to be absorbed by an outside object, an experience of boundarylessness and mystical merging that Buddhists refer to as Appana samahdi. Conversely, the researchers found that a state of "hyperarousal" interrupted by a parasympathetic breakthrough may contribute to the trancelike condition achieved in many religious and shamanic rituals, often resulting from the rhythmic driving of drumming, chanting, dancing, etc.

Rarely both sympathetic and parasympathetic systems are stimulated maximally, resulting in the most intense forms of mystical experience, near-death experience, and other types of peak experience.¹⁰⁹ This phenomenon is created utilizing cognitive/emotional activity to drive the sympathetic and parasympathetic systems to maximal activation. This state can be produced by mentally focusing on an object so intently that the meditator's absorption into the object of meditation results in a total blocking of input to the OAA region of the brain, obliterating the self/other dichotomy, and creating a sense of union between the self and something outside the self (the object of meditation, usually God or some aspect of divinity). Actually, this occurs with a blocking of the *left* posterior superior parietal lobe. Blocking of the *right* posterior superior parietal lobe results in loss of the usual orientation to space and time, and thus in an experience of orientation toward nothing, a sense of complete nothingness, or union with the formless. With no information

from the senses arriving, the left orientation area cannot find any boundary between the self and the world. As a result, the brain seems to have no choice but to perceive the self as endless and intimately interwoven with everything. The right orientation area, equally bereft of sensory data, defaults to a feeling of infinite space. The meditators feel that they have touched infinity. When both hemispheres are synchronized and both halves of the OAA are non-activated, the meditator has the sense of self merging with all that is, with the vast Absolute.¹¹⁰

Hypnosis and meditation states differ, yet each offers a characteristic dual activation signature. "Hypnosis produces state-like shifts in consciousness, yet participants still respond in a goal-directed manner."¹¹¹ The brain's default mode network engages under hypnosis, yet brain structures associated with the executive system that are often engaged in cognitive tasks in everyday life involving focused attention, imagination, absorption, and reduced analytical thought also come into play during hypnosis.¹¹² The hypnotic trance state allows access to extraordinary mental functioning such as pre-experiencing future possibilities through simulation or rehearsal; autonoetic consciousness; a bias toward field (first-person) perspective rather than observer (third-person) perspective during retrieval of past events; and a facility for temporally extended self-awareness which allows for expanded clarity of lifelong behavior patterns. Brain networks activated in the default mode, and thus in hypnosis, are involved with personal goal processing; they are more activated when imaging future events that are relevant to one's personal goals than when imagining future events that are unrelated to personal goals. Their activation is also detected when individuals are engaged in self-referential processing, e.g., reflecting on their own personality traits. In other words, the default mode is involved in coding and evaluating the selfrelevance of mental representations, and in putting those representations in a broader personal context by relating them to autobiographical knowledge.¹¹³ These usually abstract unconscious mental operations are brought into conscious awareness in the hypnotic state.

Under certain conditions, there is a parallel recruitment of executive and default network regions of brain function, unlike the way that these two brain systems usually work in opposition. "Mind wandering" may evoke a unique mental state that allows these otherwise opposing networks to work in cooperation.¹¹⁴ Default network recruitment of brain resources often occurs during the precise moments when the mind wanders away from the task at hand. Individuals fluctuate in their explicit awareness of the contents of their own thought, a phenomenon termed meta-awareness or metaconsciousness. Recent research indicates that task performance is more disrupted by unaware than by aware mind wandering episodes. Brain recruitment in both default and executive network regions was strongest when mind wandering occurred in the absence of meta-awareness; mind wandering is most disruptive to concurrent task performance when it goes unnoticed. The condition that most supports this unique mental state is one that contains "conflict within the content of mind wandering itself, i.e., thoughts and memories from the stream of consciousness that pertain to discrepancies between one's more general personal goals and the current state of affairs.... the content of mind wandering is closely related to current personal concerns and unresolved matters."¹¹⁵ These are precisely the conditions established in a typical hypnotherapy session, setting up a cooperative sharing of mental processing between the task-oriented executive and the introspective default.

This parallel recruitment of brain resources in hypnosis is vividly demonstrated in a study that compared what happens when two competing processes are paired together.¹¹⁶ In the Stroop test, a list of color words (red, green, blue, etc.) are shown to subjects. In the control no-conflict situation, the word red is colored in red ink, whereas in the conflict situation the word *red* is presented in a different color, such as green or blue. Subjects are asked to state the color of the letters and to disregard the meaning of the word. Highly hypnotizable subjects who are simply told to relax are wrong (state the color meaning of the word) more often than controls. But subjects who were directed through suggestion to disregard the meaning and just see the color of the ink performed much better than the controls. The hypnotic trance state created a functional dissociation between conflict monitoring and cognitive control processes, but "hypnosis combined with suggestion made it possible for people to disregard the conflict altogether, and thus produce superior

conflict resolution, to bring about a higher level of performance.^{117,, 118}

Another illustrative example of this parallel recruitment of brain resources comes from pain research. Hypnosis can focus attention on one mental experience dissociating attention away from other experiences. In this way by distracting attention away from pain sensation, hypnosis is useful in reducing pain. Current research shows that hypnosis also can dissociate brain areas that are responsible for sensing pain from those that are responsible for evaluating pain.¹¹⁹

There is additional support for the perspective that this unique mental state matches a hypnotic trance or meditative state: the anterior medial prefrontal cortex, a prominent part of the default mode network which has previously been implicated in meta-awareness of one's own internal mental contents,¹²⁰ is significantly more active when subjects are unaware of their own mind wandering than when meta-awareness is present. This finding suggests that subjects may be aware of the contents of their consciousness, without being aware of the fact that they are mind wandering. Again, this is an accurate description of the hypnotic trance state. This dual activation pattern is also reminiscent of the neural recruitment observed during creative thinking, where executive regions and default network regions are activated before solving problems with insight. ¹²¹ ¹²² Also, a similar parallel recruitment of executive and default regions has been observed during naturalistic film viewing,¹²³ which is related to immersive simulation mental experience.¹²⁴

Kirsch and his colleagues¹²⁵ determined that highly hypnotizable individuals experienced a decrease in the default network after a hypnotic induction without increasing activity in other cortical regions. These same decreases were not observed in low hypnotizable participants. These decreases in the default mode signify the presence of goal-directed activity within the hypnotic trance state, meaning that hypnosis produces state-like shifts in consciousness toward resting state non goal-directed mental activity, yet participants still respond in a goal-directed manner.¹²⁶ The researchers interpret these data to indicate that the induction of hypnosis "creates a distinctive and unique pattern of brain activation in highly suggestible subjects."¹²⁷ Research data further shows that hypnotic depth ratings are

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correlated with reduced activation in the default mode, so that depth of trance actually increases goal-directed mental activity within the context of the altered state.¹²⁸

To summarize the unique state that hypnosis offers for changing individuals' behavior through changing and repairing connectivity in the brain:

Hypnosis activates an array of neurological processes. During trance, the usual conscious processing in the prefrontal cortex is deactivated and separated from other processing. This releases the unconscious processes from conscious direction and allows for a more free flow in creative, non-conscious pathways. In hypnosis, attention is focused and absorbed, and often directed inwardly, without the usual monitoring and vigilance that we usually turn to the outside world when we are paying conscious attention to our surroundings. When combined with suggestion, attention can be directed in a particular direction, bypassing conscious limits, defenses, and problems, thus providing an opportunity for something new to take place.¹²⁹

Disturbed functioning of the default mode network occurs in two ways: (1) diminished connectivity of the default mode network during rest, interfering with the restorative tasks that require default mode network activation; and (2) failure to suppress default mode activity during executive functioning, interfering with the ability to focus attention on a specific mental processing task. For individuals challenged with these conditions, increased connectivity with the hippocampal area (part of the default mode network) results in a dysfunctional failure to suppress default mode activity during executive function tasks, which in turn has been linked to lapses in attention and decreased performance. Healthy normal individuals successfully suppress the default mode network during executive functioning, allowing for focused attention to be maintained for an extended period of time. Studies also have connected PTSD symptomatology with diminished connectivity of the default mode network during rest.

Recent research suggests that the reactivity of the parasympathetic branch of the autonomic nervous system reflected in heart rate variability could become part of a realtime, quantitative measure of hypnotic depth.¹³⁰ The changes in heart rate variability following hypnotic induction are due to parasympathetic activation through the vagal nerve system. It is not due to slower or deeper breathing, since the breathing rate and depth remain similar before hypnotic induction and after. Also, heart rate remains unchanged under hypnosis, indicating that hypnosis prevents the sympathetic autonomic responses expected during stimulation.¹³¹

The importance of recognizing that hypnosis and hypnotherapy increase vagal modulation, i.e., parasympathetic activation, is that they reduce high blood pressure and other cardiac risks, among other benefits.

Body-mind malfunctions and neural network misconnections

Brain-imaging studies have found altered connections among brain cells in the default mode network regions of patients with Alzheimer's disease, depression, autism, PTSD, attention deficit/ hyperactivity disorder, Tourette syndrome, amyotrophic lateral sclerosis, epilepsy, multiple sclerosis, and even schizophrenia.¹³² ¹³³ Following is a brief summary of physical and mental pathologies whose etiology is related to neural malfunctions or misconnections.

Alcoholism

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In alcoholics, brain scans reveal that the default mode network areas of the brain are less synchronized than in nonalcoholics, indicative of compromised functional connectivity.¹³⁴ Further, greater efficiency of connections correlates with longer sobriety in alcoholics. There is research support for mindfulness meditation as a treatment for preventing alcohol and substance abuse relapses;¹³⁵ it helps patients feel better by increasing dopamine levels, and activates the discernment process in the prefrontal cortex.

Alzheimer's Disease

Alzheimer's, in fact, may one day be characterized as a disease of the default mode network. A projection of the brain regions affected by Alzheimer's fits neatly over a map of the areas that make up the default mode network.¹³⁶ And perturbation of resting-state activity in particular within the hippocampus is found in patients with Alzheimer's disease compared to controls.¹³⁷ These findings, consistent with a less dramatic process in normal aging, reflect a difficulty to switch from a resting-state to a task-related mode of brain function, which would mainly be due to a failure of default mode network

brain regions to show rapid and efficient synchronization in their activity.¹³⁸ Changes in resting-state activity or functional connectivity within the default mode network may well be an accurate and early marker of Alzheimer's disease. Hippocampal atrophy seems to induce episodic memory impairment, and this dysfunction that may be at work in some of the other pathologies mentioned above, including PTSD.

Attention-deficit Hyperactivity Disorder

Attention-deficit hyperactivity disorder (ADHD) likely represents a neurologic failure to properly deactivate the default mode network in order to devote brain processing resources to a goal-directed brain function. This has been demonstrated in ADHD across tests of sustained attention, attentional control, motor sequencing and control, inhibitory control, and time perception.¹³⁹ Studies of ADHD children and adults suggest possible abnormalities within the brain's default mode network itself, namely grey matter volume reductions and decreased cortical thickness in several specific default mode network sites in the brain.¹⁴⁰ ¹⁴¹ ¹⁴²

Functioning properly, the hippocampus is a center for comparing input with stored data, and, as such, a center to filter out irrelevant (that is, distracting) stimuli that might lead to maladaptive arousal responses.¹⁴³ The hippocampal circuitry locks onto only one set of inputs at a time, thereby facilitating selective attention to only one set. However, when dysfunctional, the hippocampus, rather than filtering out irrelevant stimuli, becomes fixated on them. Studies of damage to the prefrontal cortex and hippocampus reveal symptoms reminiscent of ADHD, i.e., hyperactivity, distractibility, and a tendency toward preoccupation with certain activities that verge on pathological "undistractibility-distractibility" reminiscent of ADHD children's tendency to become mesmerized by television or video games.144

Yoga, which helps people learn to direct their attention to breathing and somatic experience, has been applied successfully to treat ADHD as well as Obsessive-Compulsive Disorder, depression, addictions, and sleep disorders.¹⁴⁵

Autism

Recent research has documented deviations from normal default mode network activity in autism,¹⁴⁶ failing to deactivate when it should and activating when it shouldn't. The autistic individual's default mode network does not deactivate during an attempt to engage in a task, leading to distracting task-unrelated thoughts such as rumination or daydreaming. However, autistic individuals' default mode network is also dysfunctional at rest, failing to activate normally. This leads to a reduced capability for self-referential thoughts or introspection. In one study of adults with Asperger's syndrome, many were unable to describe their inner experience and thoughts when asked, although they were of normal intelligence and could describe the immediate physical environment.¹⁴⁷ One node of the default mode network is most prominently affected in autism: the anterior cingulate cortex (ACC).¹⁴⁸ The magnitude of disruption in connectivity is inversely correlated with the severity of patients' social and communication deficits.¹⁴⁹ This part of the brain is particularly involved in self-awareness, and is, interestingly, one that is very clearly affected by the induction of hypnosis.

In addition to activating when the brain is at rest, similarly high activity of the default mode network is also seen when people engage in tasks of a social, emotional or introspective nature,¹⁵⁰ ¹⁵¹ the very tasks which are most difficult for individuals with autism spectrum disorder.¹⁵² Autism spectrum disorder comprises a range of developmental disorders, including disorder. Asperger's disorder. autistic and pervasive developmental disorder not otherwise specified/ atypical autism. These disorders are characterized by impairments in social interaction and also by impairments in one or both of the domains of communication and behavioral flexibility, as manifested in a restricted and repetitive repertoire of behaviors and interests (DSM-IV).

Recent research reveals that individuals with autism spectrum disorder recall fewer specific past events (episodic memory) than normal individuals, and that they imagine significantly fewer possible future experiences (episodic future thinking or *prospection*) than comparison individuals; participants with autism spectrum disorder demonstrate impaired episodic memory and episodic future thinking. Autonoetic consciousness—a type of self-consciousness that involves becoming aware of past, present, or future states of self-is considered to be one of the key hallmarks both of episodic memory and of episodic future thinking. In line with this finding, participants with autism spectrum disorder are less likely than comparison participants to report taking a field (first-person) perspective and are more likely to report taking an observer (third-person) perspective during retrieval of past events (but not during simulation of future events).¹⁵³ The default mode network is considered to be instrumental in an individual's experience of autonoetic consciousness, and default mode disruption in autism, as well as in individuals with depression or schizophrenia, show reduced self-perspective and degree of autonoetic awareness.¹⁵⁴ A related concomitant attribute is diminished temporally extended self-awareness, i.e., awareness of one's continuing existence through time.

Recent research has explored the correlation of the deficit in empathy in individuals with autism spectrum disorder with an aberrant mirror neuron system.¹⁵⁵ People with autism spectrum disorder have trouble spontaneously imitating someone else, whether they are attempting to imitate something emotional or nonemotional. And fMRI studies document that when they are attempting (unsuccessfully) imitate another's facial to expression, they show little or no activation in typical mirror neuron areas of the brain.¹⁵⁶ In other words, the impairment in social orienting and attention is due to disrupted neural circuitry of the mirror neuron system. This research has lead to the development of a neurofeedback treatment to teach autistic children to retrain mirror neurons to respond appropriately to stimuli and integrate normally into wider circuits, which can reduce social symptoms of autism.¹⁵⁷

Bipolar Disorder

Default mode network connectivity is a marker for bipolar disorder, distinguishing individuals diagnosed as bipolar from controls as well as from schizophrenics.¹⁵⁸ Studies using fMRI have found abnormalities in the ventral medial prefrontal cortex in bipolar disorder and abnormalities in the dorsal medial prefrontal cortex in schizophrenia.¹⁵⁹ Further, bipolar disorder

subjects showing abnormal activation of the parietal cortex correlated with mania symptom severity.

Medication alone is most often inadequate as a treatment: "less than one-third of patients treated with lithium achieved remission."¹⁶⁰ Psychotherapy in conjunction with medication has been shown to be more effective, utilizing Gestalt Therapy¹⁶¹ and cognitive behavioral therapy.¹⁶²In one recent study, bipolar patients who had undergone cognitive behavioral group therapy presented fewer symptoms of mania, depression and anxiety, as well as fewer and shorter mood change episodes; cognitive behavioral group therapy sessions substantially contributed to the improvement of depression symptoms.¹⁶³

Borderline Personality Disorder

Several features of borderline personality disorder are related to difficulties in regulating behavioral state and emotional reactivity, functions regulated by the autonomic nervous system. The sympathetic component of the autonomic nervous system, which supports fight/flight behaviors, has been determined not to be hyperaroused in those with borderline personality disorder.¹⁶⁴ Rather, the parasympathetic component, which supports calm visceral states and social engagement behaviors, has been determined to be depressed, so that it is unable to effectively apply the "brake" to the sympathetic activation.¹⁶⁵ Also, with borderline personality disorder exhibit individuals anomalies in limbic structures implicated in emotion regulation, such as smaller hippocampal and amygdala volumes.¹⁶⁶ These volume reductions, especially in the hippocampus, are thought to be caused by the excessive stress that borderline personality disorder patients experience.¹⁶⁷ Borderline personality disorder might be associated with difficulties in regulating the vagal brake in social settings; in response to social stimuli, these individuals rapidly shift from a calm state to a state of agitation.

A similar maladaptive vagal brake has been found in individuals diagnosed with PTSD,¹⁶⁸ and perpetrators of violence.¹⁶⁹

Psychotherapy is considered to be the primary treatment for BPD.¹⁷⁰ Recent research has shown that about half of the symptoms of BPD are acute in nature (e.g., self-mutilation and help-seeking suicide attempts) and half are temperamental in

nature (intense anger and profound fears of abandonment).¹⁷¹ According to this research, acute symptoms resolve relatively rapidly, are specific to BPD, and often result in psychiatric hospitalizations. In contrast, temperamental symptoms are relatively slow to resolve, are not specific to BPD, and are associated with ongoing psychosocial impairment. Therapy includes forms of the cognitive behavioral approach, including Dialectical Behavioral Therapy (DBT),¹⁷² and tends to be most effective at reducing self-destructive acts.

Brain damage

Many patients surviving prolonged cardiac arrest or severe motor vehicle accidents are left with severe brain damage, leading to the presence of disorders of consciousness. Among disorders of consciousness, *coma* is defined by unarousable unresponsiveness; and *vegetative state* by preserved behavioral sleep–wake cycles and reflexive but not purposeful behaviors; *minimally conscious* patients, though unable to communicate, show inconsistent non-reflexive behaviors, interpreted as signs of awareness of self or environment; and the *locked-in syndrome* describes patients who are awake and conscious but have no means of producing speech, limb or facial movements.

Default network connectivity is decreased in severely braindamaged patients, in proportion to their degree of consciousness impairment, ranging from healthy controls and locked-in syndrome to minimally conscious, vegetative then coma patients.¹⁷³ The integrity of this resting-state connectivity pattern in the default network differs in different pathological alterations of consciousness. Connectivity is significantly stronger in minimally conscious patients as compared with unconscious patients.

Although the default mode network can still be identified in unconscious patients, connectivity strength within default mode network may be a reliable indicator of a patient's level of consciousness, differentiating unconscious patients such as those in a coma or vegetative state from minimally conscious and locked-in syndrome patients.

Other types of brain damage can result from stroke or multiple sclerosis, for example. Results from one recent study support the efficacy of self-hypnosis training for the management of chronic pain in persons with multiple sclerosis.¹⁷⁴ The types of issues that may need to be dealt with in addition to pain management include coming to grips with the reality of the current new situation, with the prognosis, with the uncertainty of what the future holds, with changing relationships due to the disability, with suicidal thoughts, with isolation.¹⁷⁵

Chronic fatigue syndrome

Mention has already been made of the effectiveness of psychotherapy which addresses faulty thoughts and beliefs about the condition for patients who suffer chronic fatigue syndrome. Patients not only feel better but also show a significant increase in gray matter volume localized in the lateral prefrontal cortex.¹⁷⁶ Cognitive behavioral therapy has been shown to be useful to patients with chronic fatigue syndrome and fibromyalgia.¹⁷⁷

Chronic pain

Chronic pain patients suffer from more than just pain; depression and anxiety, sleep disturbances, and decision-making abnormalities also significantly diminish their quality of life.¹⁷⁸ Chronic pain harms cortical areas unrelated to pain, including the functional connectivity of cortical regions known to be active at rest, i.e., the components of the default mode network. In one study,¹⁷⁹ chronic back pain patients, despite performing a cognitive task equally well as controls, displayed reduced deactivation in several key default mode network regions, indicating an underlying preoccupation with the internal somatic experience. "In conclusion, these findings suggest that the brain of a chronic pain patient is not simply a healthy brain processing pain information, but rather is altered by the persistent pain in a manner reminiscent of other neurological conditions associated with cognitive impairments."¹⁸⁰

Depression

Patients with depression exhibit decreased connections between areas of the default mode network and brain regions involved with emotion, motivation, and reward processing,¹⁸¹ leading to the conclusion that "deficits in default mode network connectivity with the caudate may be an early manifestation of major depressive disorder."¹⁸² The caudate is involved in the

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processing of rewarding stimuli in healthy controls and is therefore intricately involved in pleasure and motivation.

Major depressive disorder has been characterized by excessive default-network activation and connectivity. These hyperconnectivities are often interpreted as reflecting rumination, where individuals perseverate on negative, selfreferential thoughts, i.e., repetitively and passively focus on symptoms of distress and on the possible causes and consequences of these symptoms.¹⁸³ The research shows that rumination and brooding occur during off-task periods when the default network is activated, e.g. idle moments at work or nonengaged and unguided time periods at home. Consistent with common experience, the relationship between ruminative psychological processes and connectivity is mitigated by engaging in a task, decreasing the time spent brooding. Decreased activation of the default-mode network in patients with major depressive disorder correlates with depression severity and feelings of hopelessness.¹⁸⁴

There is considerable evidence that depressive mood is related to lower parasympathetic control of the heart, which of course decreases ability to relax and increases cardiovascular risk. When depressed individuals are alone they evidence lower heart rate variability and higher negative affect, but not when they are engaged in social interactions with a partner, family members, or friends.¹⁸⁵ The quality of the relationships is crucial to this outcome, however; social interactions with strangers or interactions) colleagues (potentially insecure are not accompanied by higher heart rate variability in these individuals. This affirms our commonsense expectation that social interaction in an intimate relationship improves the emotional state of someone afflicted with depressive mood. Furthermore, the frequency of spousal interaction has been related with less progression of heart disease among older men with elevated depression scores. However, this effect only operates for individuals with depression, not necessarily for non-depressed individuals 186

Hypnotherapy combined with cognitive behavior therapy has been shown to be effective in treating major depressive disorder,¹⁸⁷ including depression in children and adolescents.¹⁸⁸

Drug addiction

Research findings suggest that drug addicts (chronic heroin users) have an abnormal functional organization of the default mode network, including the hippocampus, resulting in abnormally increased memory processing but diminished cognitive control related to attention and self-monitoring.¹⁸⁹ These findings may underlie the addict's hypersensitivity toward drug-related cues, and weakened strength of cognitive control, i.e., compulsive patterns.¹⁹⁰

The hippocampus is the main brain structure involved in learning and memory, and thought to primarily contribute to the acquisition, consolidation and expression of learning of the drug-related cues that drive relapse to drug-seeking behaviors.¹⁹¹ ¹⁹² The hippocampus is also, as has been clearly determined, a prominent node within the default mode network.

There is growing evidence that hypnotically enhanced treatment for addictions is effective,¹⁹³ particularly by using the addictive urge to locate and process underlying trauma. Addiction uses three basic neuropathways in the brain. The arousal neuropathway concerns arousal and intensity, the numbing neuropathway produces a calming, sedative process and the fantasy neuropathway provides escape through a trance state.¹⁹⁴

Early-onset pregnancy-induced hypertension

Pregnancy-induced hypertension is contributed to by both sympathetic overactivity and vagal withdrawal, especially in early-onset type, SVI is mainly due to vagal inhibition. One recent study shows that hypnosis is effective in reducing blood pressure in the short term but also in the middle and long terms.¹⁹⁵

Multiple Sclerosis

Relapsing-remitting multiple sclerosis patients show a consistent dysfunction of default mode network.¹⁹⁶ ¹⁹⁷ The dysfunction is more evident in cognitively impaired patients than in those who are cognitively preserved. Cognitive impairment occurs in up to 70% of multiple sclerosis patients, sometimes starting early in the disease course.¹⁹⁸ The degree of dysfunction of default mode network appears to be a marker for severity of

symptoms for multiple sclerosis patients. In one study, hypnotic imagery and posthypnotic suggestion were accompanied by significantly improved control of pain, sitting balance, and diplopia in a 30-year-old female with multiple sclerosis.¹⁹⁹

Nicotine use

Nicotine is known to improve cognitive performance, in part by improving attention. Nicotinic decreases activity in regions within the default mode network, which then allows for more mental processing resources to be available for being attentive to task-orientation.²⁰⁰ Nicotine use that is so constant that it interferes with the brain's normal default mode processing may deprive the individual of aspects of that processing, such as selfreferential introspection, organizing memory and imagination. Hypnosis has been used successfully to assist in overcoming addictive nicotine use. In a metaanalysis on 633 studies of smoking cessation, with 48 studies in the hypnosis category, encompassing a total sample of 6,020 participants, hypnosis fared better than virtually any other comparison treatment (e.g., nicotine chewing gum, smoke aversion), achieving a success rate of 36%.²⁰¹

Obsessive-compulsive Disorder

People with OCD have a distinctive brain pattern: excessive activity in the orbitofrontal cortex and the right caudate nucleus, areas of the brain that, when activated, signal that something is wrong. This overstimulation interferes with the ability to accurately assess a situation and plan appropriately. Both psychotherapy and medication can reduce activation in this area of the brain, relieving the symptoms of perseverating thoughts which lead to obsessive attempts to control anticipated outcomes.²⁰² Interestingly, positive emotionality has been shown to be experienced when the orbitofrontal cortex is active during resting conditions (introspection, mind wandering).²⁰³

In one recent study,²⁰⁴ OCD symptoms, depression, anxiety and overall functioning improved robustly with brief intensive cognitive-behavioral therapy (CBT) treatment. In as little as four weeks, there was significant activation of the dorsal anterior cingulate cortex, a region involved in reappraisal and suppression of negative emotions. Studies of cognitive

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behavioral therapy effects in obsessive-compulsive disorder (OCD) are consistent in showing decreased metabolism in the right caudate nucleus as symptoms decrease.²⁰⁵

Schizophrenia

There is increasing evidence of default mode network dysfunction in schizophrenia, as well as brain structural changes in schizophrenia in the territory of the default mode network. On a task requiring identification or labeling of facial emotions, schizophrenic patients show failure to deactivate the default mode network in order to attend to the task. This failure is more extensive than that seen during performance of working memory tasks. Schizophrenics also show reductions of grey matter volume in cortical regions that overlap with the same parts of the default mode network.²⁰⁶

Seizures

The default mode network has been shown to be selectively impaired during epileptic seizures associated with loss of consciousness; decreased activity has been confirmed during these three seizure types: complex partial, generalized tonic-clonic, and absence seizures.²⁰⁷

Sleep deprivation

It is well known that one night of sleep deprivation significantly disrupts cognitive task performance; it is now clear that it also disrupts task-related deactivation of the default mode network.²⁰⁸ Interestingly, perturbations in default mode activity during wakefulness are observed in a number of disorders that display co-occurring abnormalities of sleep, including schizophrenia, autism spectrum disorders, anxiety disorders, attention deficit disorder and Alzheimer's disease characteristic signature of the sleep-deprived brain may be the dysregulation not only of on-task brain activity but also of offtask resting-state modes of brain activity, and may prevent the ability to sustain attention and to maintain stable task engagement, resulting in concentration lapses and slowed response times. Such an outcome has obvious implications for the disorders mentioned above, not to mention such conditions as drowsy driving.²⁰⁹ It is safe to say that at least some aspects of

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the default mode network are dependent on prior sleep for their stability.

Perhaps this provides new evidence for the advice to "sleep on it" in making an important decision. One prominent hypothesis suggests that²¹⁰

resting-state activity supports off-line processing of recently acquired information within the context of preexisting knowledge.²¹¹ Such processing may allow for the testing of unique memory associations, and by doing so importantly make flexible predictions about the future.²¹² In this regard, the role of sleep, and a lack thereof, becomes particularly relevant. For example, a recent report has demonstrated that sleep not only strengthens individual item memories but can actually facilitate the off-line building of distant relational associations between them.²¹³ Moreover, following initial practice on a problem-solving task, a night of sleep significantly increases the ability to gain insight was not evident immediately upon awaking. Instead, it emerged only following substantial additional waking (re)engagement on the task. Furthermore, participants who were sleep deprived and retested in the deprivation state failed to gain such creative insight.

During sleep, the default mode network connectivity and arousal level is at its lowest during deep non- rapid eye movement (NREM), while during rapid eye movement (REM) sleep it is as high as during wakefulness. This difference is true for subsystems of the default network, but connectivity among the core regions – the posterior cingulate cortex, rostral anterior cingulate cortex, and inferior parietal lobule – remain consistent across sleep states. The core network may serve to integrate brain regions throughout the sleep cycle, while certain subsystems perform functions specific to the level of arousal.²¹⁵

Social Phobia

Default mode network connectivity is a marker for social phobia.²¹⁶ Default mode activation increases the ability to predict another person's behavior, taking his or her perspective. Impairment in the default mode network in social phobia patients may be relevant in the development of the feeling of wariness of others' judgment and may be related to the so-called self-focused attention. Self-focused attention may prevent individuals from observing external information that could disconfirm their fears of being judged. Moreover, the abnormal modulation of activity in the default mode network may reflect persistent rumination or

anxiety-related thoughts that are not modulated by the switch from rest to task.

Hypnosis is an effective and powerful intervention for most types of stress and anxiety because hypnosis exploits the intimate connection between mind and body, provides relief through improved self-regulation, and also beneficially affects cognition and the experience of self-mastery.²¹⁷

Traumatic brain injury (TBI)

Sustained attention impairments in patients with traumatic brain injury (TBI) are associated with an increase in default mode network activation, and the degree of structural disconnection within the default mode network correlates with the level of sustained inability to attend. Abnormalities in default mode network function are a sensitive marker of impairments of attention and suggest that changes in connectivity within the default mode network are central to the development of attentional impairment after TBI.²¹⁸

Trauma and PTSD

The integrity of the default mode network is compromised in posttraumatic stress disorder (PTSD).²¹⁹ And the alteration of brain function is not restricted to a years- or decades-long process of deterioration. Physically healthy survivors of the magnitude 8.0 earthquake in Wenchuan, China who experienced severe emotional trauma, were assessed within 25 days after the disaster. These trauma victims had a reduced temporal synchronization within the default mode network of resting state brain function.²²⁰ The increased regional activity and reduced functional connectivity occurred in areas known to be important for emotion processing.²²¹ Atypical function in these areas has been implicated in previous studies of patients with chronic stress-related disorders.²²²

One recent study investigated relationships between default mode network connectivity and the severity of PTSD symptoms in a sample of subjects recently exposed to a traumatic event.²²³ Individuals with the most impaired connectivity developed the most severe and most persistent PTSD symptoms, even controlling for comorbid major depressive disorder. "The present study certainly suggests that examination of connectivity patterns of brain networks predict persistence of PTSD symptoms or related post-traumatic symptomatology."²²⁴

Altered default mode network connectivity in individuals with PTSD has now been related to prolonged childhood maltreatment, suggesting that early-life trauma may interfere with the developmental trajectory of the default mode network and its associated functions.²²⁵ Moreover, deficient default mode network connectivity in adults with childhood maltreatment-related PTSD appears similar to patterns of default mode network connectivity observed in healthy children aged 7 to 9 years.²²⁶ For example, default mode network connectivity observed in healthy children aged 7 to 9 years.²²⁶ For example, default mode network connectivity observed in women with severe chronic PTSD due to prolonged maltreatment during childhood closely paralleled that observed in children age 7 to 9 years.²²⁷

Healthy, normal default mode network structures begin to develop by age 1, but integration and connectivity between the default mode network nodes remains immature until about age 9 years. Impairment of the normal development of default mode network in childhood has far-reaching consequences over the lifespan. Typically, adults with chronic PTSD due to early-life trauma exhibit remarkable deficiencies in functions reliant upon self-referential processing such as emotion recognition and emotional awareness. Alexithymia, the inability to appropriately recognize one's own emotions, is known to be widespread in early-traumatized PTSD populations. It is also widely acknowledged that repeated exposure to traumatic events can affect one's sense of an adaptive and agentive self. This is illustrated by altered posttraumatic cognitions and disrupted selfreferential processing in patients with PTSD, arguably the most severe example being dissociative symptoms that may include depersonalization and identity disturbance. Neuroimaging studies suggest that dissociative experiences involve brain regions also implicated in the default mode network. Finally, although results are conflicting, a number of studies also point toward impoverished recollection of episodic events in survivors of early-life trauma, particularly among those in whom PTSD develops as a result of this exposure.²²⁸

PTSD patients who have suffered a trauma such as a serious car accident show lower activity in processing empathy in social situations. Following therapy, there is a greater activation in the left middle temporal gyrus, related to the experience of empathy, and increased posterior cingulated gyrus activation, related to the ability to forgive.²²⁹

The brain's theta rhythm circuitry is involved in memory retrieval, survival behavior, navigation including virtual reality tracking, wellbeing, and the integration of emotion and cognition. These processes are all implicated in the pathology and treatment of PTSD. Hypnosis, which elevates the brain's theta rhythm, has been found effective in contributing to the treatment of PTSD.²³⁰ Hypnosis has obvious affinities with the symptoms of PTSD. In fact, trauma may be viewed as a hypnotizing agent; Spiegel²³¹ has articulated three clusters of symptoms of PTSD in parallel with hypnosis as follows: intrusive flashbacks and nightmares with hypnotic absorption; dissociation with hypnotic dissociation; exaggerated response to disturbing stimuli with hypnotic automaticity. Hypnosis can enable the access of these symptoms and facilitate their reprocessing. Hypnotherapy assists in memory revivification and the integration of fragmented episodic memories, against a background of anxiety reduction, empowerment and psychic integration.²³²

Brain-friendly psychotherapy

"People come to therapy with a strong set of beliefs, entrenched feelings, and redundant behavior patterns. One aspect of the therapeutic process involves sensitively attuning to this apparently filled mind and brain, and look for the spaces between. Here we find the opportunities for movement and change."²³³

effective psychotherapy The most promotes brain connectivity; in other words accessing different parts of the brain at the same time and activating them so that they engage each other in new and productive ways. We design our interventions to trigger different structures and sense modalities in the brain, whether visual or kinesthetic or verbal. We allow the client's unconscious to select what material to work on, and then allow her to approach that material as directly as she is capable of. Initially that may mean approaching it tangentially, talking about a traumatic event, narrating the experience removed from actually re-experiencing it. We know that eventually deep

trauma healing will come with facing the original traumatic moment directly, experientially, and creating a corrective experience to replace the original defensive behavior pattern.

There are parts of the brain, and therefore mental processing, that you can't reach with language. These areas of the brain, too, must be accessed in order to simultaneously activate dissociated networks in the brain - fear circuits, language circuits, reward circuits, default mode - in ways that enable clients to reorganize their neural connections.

The hypnotic trance provides fertile interconnection between the cortex and the midbrain which is the requirement for repair of the damage done to the brain's hippocampus in childhood abuse. Trauma resolution focuses on time-stamping experiences to a discreet circumstance rather than a globally generalized reexperience of the original trauma. One of the core aspects of treating PTSD is to route the original traumatic memory through the brain's hippocampus region to time-stamp it, which did not occur during the original trauma.

Early trauma through abuse or neglect results in a significant decrease in the volume of the hippocampus structure in the midbrain. "The integrative failure that is characteristic of traumatized individuals may also relate to structural brain changes, notably in the hippocampus."²³⁴ The hippocampus is a brain structure instrumental in the synthesis of experiences, providing a conscious structure, context and a time stamp to the experience in the process of memory encoding, storage and retrieval. Smaller hippocampal volumes have been reported in female adult survivors of childhood sexual abuse.²³⁵ ²³⁶ That damage consists of a loss of neurons and synapses (a loss of up to 18%), and results in corruption of thought process and learning, particularly deficits of encoding short-term into long-term memory.²³⁷

fMRI images reveal that when people are emotionally distressed (anxious, angry, depressed), the most active sites in the brain are circuitry converging on the amygdala, part of the brain's emotional center, and the right prefrontal cortex, a brain region important for the hypervigilance typical of people under stress.²³⁸ By contrast, when people are in positive moods (upbeat, enthusiastic and energized) those sites are quiet, with the heightened activity in the left prefrontal cortex.

Activation of the amygdala is a manifestation of a neurobiological fear reaction, and enhanced amygdala activation is found in individuals with Borderline Personality Disorder²³⁹, posttraumatic stress disorder²⁴⁰, and obsessive-compulsive disorder²⁴¹ ²⁴² during fMRI scanning of their provoked symptoms. Further, the amygdala is activated by perseverative thoughts and memories.²⁴³

These two systems (amygdala and hippocampus) represent separable memory systems as well as separable processing systems: the amygdala as a "hot" memory system, and the hippocampus as a "cool" memory system. Psychopathology develops when there are dissociations between these two memory systems. Elevated cortisol levels - produced by fear, anxiety and stress - for chronic periods are associated with increased activity in anxiety-related brain regions, especially the amygdala.²⁴⁴ Chronically elevated cortisol levels are also damaging to hippocampal neurons.²⁴⁵

Under traumatic stress, emotional memories are encoded without a significant contribution of context from the hippocampal system. "This produces a pool of stimulus-bound emotional memories that have been encoded without a coherent event-specific spatiotemporal frame to organize them. This pool is, essentially, a population of traumatic memory fragments. Upon retrieval, traumatic memories cannot be experienced as a memorial event а beginning. end. and internal with spatiotemporal structure. Instead, each emotional memory is experienced as fragmented, disorganized, and intrusive²⁴⁶.²⁴⁷

The traumatic experiences, etched in procedural memory but not converted into long-term memory, interfere with current working memory. Past threats are perceived to be present threats, suggested by intrusive thoughts, flashbacks, and hypervigilance. Not only does PTSD obscure the ability to distinguish between past and present, but the "repertoire of survival skills remains confined to those skills that were acquired up to the time of the trauma, and they lack the resilience to learn new strategies."²⁴⁸ An aspect of this individual is frozen in the past, or perhaps more accurately that frozen dissociated part of the person is carried like deadweight in the ever-present – a "primitively organized alternative self." Fortunately, however, the hippocampus is a unique region of the human brain in that it can replicate new neurons as well as new synapses. Treatment of PTSD and resolution of early childhood trauma can reverse the damage to the hippocampus, and there is evidence that the hippocampal volume actually increases along with a decrease of PTSD symptoms and significant improvements in verbal declarative memory.²⁴⁹ ²⁵⁰

The age regression capability within hypnotherapy is an ideal means of access to mend the damage to the hippocampus, allowing the brain to return to the origins of the traumatic loss of neurons and synapses, and "rewire" the processing of experience and laving down of memory. Some of the relevant factors that apply to the state of hypnosis (as well as meditative states) are a proclivity to dissociation, tolerance of ambiguity, experiencing states of nonordinary reality, boundarylessness, merging, and fluid ego boundaries.²⁵¹ Further, the state of hypnosis is known to promote a state of being deeply engrossed in imaginative activities, to produce vivid imagery, and to engage in "holistic information-processing styles."252 The panoramic bird's-eye view common in the state of hypnosis, or meditation, activates the brain's lateral network circuits, associated with mindful, open, spacious awareness. Activating these lateral networks contributes to steadiness of mind, the capacity for deep concentration, and resilience.²⁵³

Theta frequency brain waves are dominant with unconscious mental processing, and the source of these theta rhythms is the hippocampus.²⁵⁴ The theta is especially pronounced during rapid eve movement (REM), such as during dreaming or hypnosis. A person can emerge into conscious thinking (beta brain waves) from unconsciousness (theta) either rapidly or more gradually. When the transition occurs abruptly, little of the experience can be brought back to be accessible to the conscious mind. However, when that same transition occurs more gradually, more of the theta-level experience can be accessed consciously. And the difference within the brain is that gradual transition allows alpha brain wave patterns to participate in the crossover. Alpha waves are associated with right brain processing, aesthetics, intuition, sensuality. So the hypnotic trance state allows us to experience with a theta rhythm, and yet access the material with an alpha rhythm. Theta rhythms originate in the hippocampus, important for "time-stamping" memories, which must then be forwarded to the prefrontal cortex to be processed in conscious awareness utilizing alpha and beta waves.

Cozolino²⁵⁵ has a number of suggestions for how to facilitate neural integration in psychotherapy that are solidly grounded in neurobiology. He proposes that neural growth and integration in psychotherapy may be enhanced by:

- 1. The establishment of a safe and trusting relationship.
- 2. Gaining new information and experiences across the domains of cognition, emotion, sensation, and behavior.
- 3. The simultaneous or alternating activation of neural networks that are inadequately integrated or dissociated.
- 4. Moderate levels of stress or emotional arousal alternating with periods of calm and safety.
- 5. The integration of conceptual knowledge with emotional and bodily experience through narratives that are co-constructed with the therapist.

Following is a brief summary of the guidelines outlined in a 2006 article²⁵⁶ by the current authors for differential approaches to treatment of sympathetic and parasympathetic nervous system dysfunction. While accessing the hyperarousal or hypoarousal response, we must facilitate the person to stay present, feel safe, communicate their experience, and feel empowered. Following are some suggested guidelines for treatment of trauma, complex trauma, DESNOS, and shock.

1. **Approach trauma-related material gradually** to avoid intensification of the affects and physiologic states related to the trauma. With the advent of distinguishing between trauma and complex trauma, or PTSD and complex PTSD, we now know that certain prerequisites must be in place for the client before proceeding to confront the trauma directly, namely

> the capacity of our patients to modulate their affective arousal: whether they are able to be emotionally upset without hurting themselves, becoming aggressive, or dissociating. As long as they cannot do this, addressing the trauma is likely to lead to negative therapeutic outcomes. Similarly, as long as they dissociate when they feel upset they will be unable to take charge of their lives and will be unable to 'process' traumatic experiences.²⁵⁷

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2. Cultivate an acute awareness of inner body sensations (bodily feelings of a distinctly physical character, such as trembling lips, heaviness in the chest, twitching in the hands, clamminess in the feet, tightness in the throat, tension in the buttocks, pounding heart, numbness in the extremities, tingling in the right arm, shallow breathing, or vibrating sensations in the solar plexus area, etc.).

The therapist asks questions that require awareness (mindfulness) to answer, such as, "What do you feel in your body? Where in your body do you experience that? What happens in the rest of your body when your hand makes a fist?" This focusing is especially important when the client is accessing traumatic memories so that he/she can experience the content of that moment consciously embodied. Questions such as these encourage the client to come out of a dissociated state, and future- or past-centered ideation, and be really present in the body, experienced from the perspective of an observing ego whose intention is simply observing rather than controlling or changing the experience, or reacting to it.

3. Access enough traumatic material to process but not so much as to dissociate. The therapist must "hold" the client's arousal within the optimal limits, while encouraging the accessing, exploring, and expressing of the trauma memory. When arousal reaches either the upper or lower limit, ask the client to temporarily suspend attending to her feelings and thoughts and instead focus on observing the physical sensations and movements in detail until these sensations settle and the movements complete themselves.

Redirect the person's attention by asking him/her to refocus as well as in other ways. That may be with a touch or specific eye contact, a sip of water or inquiring about level of comfort. In addition to interrupting the dysfunctional sequence, such attending to the personal comfort and safety of the client is nurturing and elicits their own social engagement activation. This process is one of titrating the traumatic pattern, alternating between small pieces of the traumatic material and one of the client's resources, discharging the activation in the nervous system that - 52

emerges as the person slowly works through the traumatic event.

- Interrupt a client's unconscious, autonomic patterns 4. somatically. One of the most common patterns is some form of "bracing", a condition in which a person in stress resorts to a destructive habitual physiological response, such as muscle tension, vaso-constriction or dilation, or breath suppression. A timely reminder to breathe can be immensely empowering to the person's body. The vaso-constriction or dilation in the hands or feet, stomach, neck or forehead, or any particular body part, causes them to be too cold or too hot, respectively. Hence, the application of the reverse effect (heat or cold) brings the constriction to the body's attention (not to conscious awareness) and allows a relaxing of the hyperarousal. Encouragement to yell or scream when the individual is imploding with suppressed fear is permission and an instruction to the body to change a very old and deeply embedded habit. One of the most effective means of relaxing muscle tension is laughter. The therapist can insert humor to accomplish this, as long as it is properly timed and not used to dissipate emotion and thus avoid cathartic release
- 5. Work toward cathartic release, or other physical forms of release such as "unwinding" the paralyzed energy in limbs or head. Levine discusses the concept of "exchanging ... an active response for one of helplessness".²⁵⁸ Here we are coaxing the person to use will power, and the support of the therapist or group members, to initiate a sympathetic response (fight/flight) to replace the deeply embedded parasympathetic response (freeze). For the person to experience the active defense sequence (pushing with legs or arms, moving out of paralysis into proactive movement, yelling "No!") brings her out of dissociation, into her body, and begins a kinesthetic reframing process. The client may begin to experience the somatic pleasure of physical resistance and defense, and the emotional delight of a new experience of personal power. The sequence, produced through will power and trust, becomes a template for the same new response to occur spontaneously in the future.

- 6. Experience having personal needs acknowledged and responded to by a caring and safe support person. The therapist may offer a drink of water (which also flushes toxins from the system), offer heat or cold if it would be soothing, offer additional means of protection (such as something to cover the vulnerable umbilicus, the hand to hold of a supportive group member, or bringing into an age regressed scene a virtual resource person from the client's life at that time to ameliorate the terror), or establish direct contact nurturing (touch, open eye contact).
- 7. **Discover and develop personal resources**, real or imagined, that would be available so that the client is not facing the threat alone, isolated, in secret, or unsupported. The resources help to initiate a social engagement response and to discharge excess fight/flight or freeze messages. The resources should be experienced not just as an imagined creative visualization, but with awareness to the sensations experienced in their bodies.
- 8. **Speak the experience of victimization** in order to integrate the memory functions and lessen the immobilizing emotional/sensory reflex. The client is literally reconnecting the experience (implicit or body memory) with the cognitive context for it (explicit memory), and in the process is repairing the physical damage done to her hippocampus. Verbalizing reflectively in the age-regressed traumatized ego state, in the presence of the caring therapist, begins to repair the damage through regeneration within the hippocampus.
- 9. Reconnect memories and emotions to events. Bucci²⁵⁹ states the case succinctly: "One may be aware of the physiological activation, the painful physical arousal, associated with the activated schema of anger or fear, and also aware of aspects of one's history, including the trauma and abuse, but without connecting the two. We see patients telling the stories of their history without emotion, feeling physiological activation without recognizing the its connections " This emotional is dissociation The physiological or cognitive activation is not connected in memory to the source of the trauma because they were not

connected at the highly stressful time of originally laying down the memory. $^{260}\,$

- 10. **Reframe basic assumptions** about the self as secure and intrinsically worthy, and about the world as orderly and just. The person's most deeply embedded beliefs are accessible for review and reframing, because in the age regression process you have arrived at the very scene of their inception. These personal conclusions and beliefs about the self and the world are held by the immature, developmentally arrested inner child and carried by the physical body (Reich's character armor).
- 11. Release the anger and blame directed against the self for the inability to defend against the abuse. A common response among trauma survivors is to interpret her dissociation and freezing as a personal weakness.²⁶¹ This is a golden opportunity for re-establishing a loving, accepting relationship with the inner child, who until now the client has likely reviled and rejected. One of the most important truths that child ego state needs to hear and accept from the adult ego state is a reattribution of responsibility.²⁶² ²⁶³ "It was not your fault. There was nothing you could do to stop it from happening. The responsibility and blame belong to the perpetrator."
- 12. Locate the traumatic experiences in time and place, to start making distinctions between current life stresses and past trauma. During traumatic threat, it has been shown, the hippocampus becomes suppressed. Its usual function of placing a memory into the past is not active. The traumatic event is prevented from becoming a memory in the past, causing it to seem to float in time, often invading the present.²⁶⁴ ²⁶⁵ We must recognize, too, that traumatic memories are stored as emotions and senses (implicit memory), and are therefore often hazy, impressionistic, or kinesthetic. Understandably, survivors become haunted by feelings and senses they suspect are related to the trauma, but cannot clearly identify as explicit memories.

Sometimes it takes a lot of patience to eventually get to clear memories of what happened at the time of the trauma. It is common in therapy with trauma victims to observe their healing process begin with awareness of "body memories" and other procedural-implicit memories, and then gradually expand to include specific episodic memories of where, when, and how the trauma occurred.²⁶⁶

- 13. Learn to trust and surrender to a healthy interpersonal relationship with the therapist, who demonstrates an ability to understand the client's distress and tolerates the description and re-experience of her traumatic experience without as therapist withdrawing or becoming hyperaroused or hypoaroused. The therapist must overcome the client's mistrust and suspicion the old fashioned way: he/she must earn it.
- 14. Recognize that current life stresses tend to be experienced as somatic states, and accept physical symptoms as ally rather than enemy. Physical symptoms are the body's way of communicating an unmet need to the person; they are not a statement of defiance, mutiny, hostility, or weakness. Developing an awareness of inner body sensations and a precise sensation vocabulary are helpful steps toward restoring a sense of the body and its sensations as friend, not foe.
- 15. **Develop new outlets for discharging stress** and for creating a sense of well-being. The individual may have relied on reenactment of stress and trauma, through endorphin release and dissociation, to achieve relief, numbing of the pain, and what has passed as a sense of well-being. In other words the pattern is to seek re-exposure to stress for the same effect as taking pain-killing opiates, providing a similar relief from stress. Expression rather than repression of feelings may be a new option for the person. It is important to engage in the process of learning to discharge excess stress response in a healthy way. And that requires a clear understanding of one's own response patterns.

An individual who is aware of a tendency toward hyperarousal (fight/flight) response will discover that it is, in fact, the body's attempt to discharge excess parasympathetic load. The quick temper or incessant talking (dissociated sympathetic response) is *preceded by*, if only momentarily, a fear of rejection or sense of shame (parasympathetic response). Likewise, if a person tends toward hypoarousal (freeze) stress response, he/she will discover that it may be, in fact, the body's attempt to discharge excess sympathetic load. The sense of exhaustion or constant snacking (dissociated parasympathetic response) is *preceded by*, if only momentarily, a flash of rage or frustrated need (sympathetic response). With this knowledge, the therapist can encourage the client to look directly at the momentary initial response that needs discharging rather than at the dissociated defensive response, and to develop new outlets to accomplish that discharge.

16. Increase self-regulation and thereby prevent the escalation of arousal to the point of discharge through aggression or other undesirable behavior. Hyperactive defenses can take the form of uncontrollable rage or frenzy (directed at self or others), and learning to sense the physical precursors to full-blown aggressive outbursts will extinguish the conditioned response, and therefore increase the person's feeling of safety. When we help the client achieve biological completion, i.e., discharge following activation, and unlock the constriction of the nervous system, he/she can then integrate the experience for future use with triggering events, replacing the dysfunctional autonomic pattern of response.

The capacity to bring the automatic reaction into a state from which choice is possible, to internalize new cognitive information, depends on having portions of the cortex activated, which, in turn, requires a state of *mindfulness*. Some effective techniques to increase this state are increasing conscious awareness to current emotions; having the vocabulary and the self-sensitivity to identify and label emotions; creating, acknowledging and anchoring positive emotional events; and using support and other resources to improve tolerance for stressful situations.

17. **Increase the sense of personal safety** through increasing the sense of internal locus of control and greater reliability of external forces.

Clients find that they can gradually slow down and maintain an integrated awareness from start to finish throughout the experience of re-living a traumatic incident, including the most

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invasive aspects. Then perceptually, the incident can move from seeming to be ever-present, or fixated in the future, back into the past where it belongs. Symptoms diminish. Triggers of fear, panic and anger are extinguished as continuity of self is reestablished and it is experienced as truly over.

References

- Abarbanel, A. (1995). Gates, states, rhythms, and resonances: The scientific basis of neurofeedback training. *Journal of Neurotherapy*, 1(2), 15-38.
- Abram, D. (1996). The Spell of the Sensuous: Perception and Language In a More-Than-Human World. Vintage Books.
- Aggleton, J. (1992). The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction. New York: Wiley-Liss.
- Alladin, A. (2010). Depression. In Arreed Franz Barabasz, Karen Olness, Robert Boland, & Stephen Kahn (Eds.), *Medical Hypnosis Primer: Clinical and Research Evidence*, (pp. 73-81). New York, NY: Routledge/Taylor & Francis Group.
- American Psychiatric Association. (2001). Practice guideline for the treatment of patients with borderline personality disorder. Washington, DC: American Psychiatric Association.
- Anderson, M. C., Ochsner, K. N., Kuhl, B., Cooper, J., Robertson, E., Gabrieli, S. W., Glover, G. H., & Gabrieli, J. D. E. (2004). Neural systems underlying the suppression of unwanted memories. *Science*, 303, 232–235.
- Apkarian, A. V., Sosa, Y., Krauss, B. R., Thomas, P. S., Fredrickson, B. E., Levy, R. E., Harden, R., Chialvo, D. R. (2004). Chronic pain patients are impaired on an emotional decision-making task. *Pain*, 108, 129–136.
- Assaf, M., Jagannathan, K., Calhoun, V. D., Miller, L., Stevens, M. C., Sahl, R., & Pearlson, G. D. (2010). Abnormal functional connectivity of default mode subnetworks in autism spectrum disorder patients. *NeuroImage*, 53(1), 247-256.
- Aubert, A. E., Verheyden, B., Beckers, F., Tack, J., & Vandenberghe, J. (2009). Cardiac autonomic regulation under hypnosis assessed by heart rate variability: Spectral analysis and fractal complexity. *Neuropsychobiology*, 60(2), 104-112.
- Austina, M. A., Riniolob, T. C., & Porges, S. W. (Oct 2007). Borderline Personality Disorder and emotion regulation: Insights from the Polyvagal Theory. *Brain Cognition*, 65(1), 69–76.
- Badre, D., & Wagner, A. D. (2004). Selection, integration, and conflict monitoring: Assessing the nature and generality of prefrontal cognitive control mechanisms. *Neuron*, 41, 473–487.
- Balbernie, R. (2007). The move to intersubjectivity: A clinical and conceptual shift of perspective. Journal of Child Psychotherapy, 33, 308–324.
- Baliki, M. N., Geha, P. Y., Apkarian, A. V., & Chialvo, D. R. (Feb 6, 2008). Beyond feeling: Chronic pain hurts the brain, disrupting the default-mode network dynamics. *The Journal of Neuroscience*, 28(6), 1398-1403.
- Bar, M. (2007). The proactive brain: Using analogies and associations to generate predictions. *Trends in Cognitive Sciences*, 11, 280–289.
- Bar, M., Aminoff, E., Mason, M., & Fenske, M. (2007). The units of thought. *Hippocampus*, 17, 420–428.
- Barabasz, A. F., & Barabasz, M. (2008). Hypnosis and the brain. In M. R. Nash, A. J. Barnier, M. R. Nash, & A. J. Barnier (Eds.), *The Oxford Handbook of Hypnosis: Theory, Research, and Practice*, pp. 337-364. Oxford, United Kingdom: Oxford University Press.
- Baxter, L. R., Schwartz, J. M., & Bergman, K. S. (1992). Toward a neuroanatomy of obsessive-compulsive disorder. Archives of General Psychiatry, 49, 681-689.
- Benedict, R. H., Cookfair, D., Gavett, R., Gunther, M., Munschauer, F., Garg, N., et al. (2006). Validity of the minimal assessment of cognitive function in multiple sclerosis. *Journal of the International Neuropsychology Society*, 12, 549–558.
- Benham, G., & Younger, J. (2008). Hypnosis and mind-body interactions. In Michael R. Nash & Amanda J. Barnier (Eds.), *The Oxford Handbook of Hypnosis: Theory, Research, and Practice*, (pp. 393-435). New York, NY: Oxford University Press.

- Bennett, H. L. (1988). Perception and memory for events during adequate general anesthesia for surgical operations. In H. M. Pettinati (Ed.), *Hypnosis & Memory*, pp. 193-231. New York: Guilford.
- Bennett, H. L., Davis, H. S., & Giannini, J. A. (1985). Nonverbal response to intraoperative conversation. *British Journal of Anaesthesia*, 57, 174-179.
- Berman, M. G., Peltier, S., Nee, D. E., Kross, E., Deldin, P. J., & Jonides, J. (September 19, 2010). Depression, rumination and the default network. *Social Cognitive and Affective Neuroscience*, 1-8.
- Bingel, U., Wanigasekera, V., Wiech, K., Mhuircheartaigh, R. N., Lee, M. C., Ploner, M., & Tracey, I. (Feb 2011). The Effect of Treatment Expectation on Drug Efficacy: Imaging the Analgesic Benefit of the Opioid Remifertanil. *Science Translational Medicine*, 16.
- Bluhm, R. L., Williamson, P. C., Osuch, E. A., et al. (2009). Alterations in default network connectivity in posttraumatic stress disorder related to early-life trauma. *Journal of Psychiatry Neuroscience*, 34, 187-194.
- Bluhm, R., Williamson, P., Lanius, R., Théberge, J., Densmore, M., Bartha, R., Neufeld, R., & Osuch, E. (2009). Resting state default-mode network connectivity in early depression using a seed region-of-interest analysis: Decreased connectivity with caudate nucleus. *Psychiatry and Clinical Neurosciences*, 63, 754–761.
- Boly, M., Phillips, C., Tshibanda, L., Vanhaudenhuyse, A., Schabus, M., Dang-Vu, T. T., Moonen, G., Hustinx, R., Maquet, P., & Laureys, S. (2008). Intrinsic brain activity in altered states of consciousness: How conscious is the default mode of brain function? *Annals of the New York Academy of Sciences*, 1129, 119–129.
- Bonavita, S., Gallo, A., Sacco, R., Della Corte, M., Bisecco, A., Docimo, R., Lavorgna, L., Corbo, D., Di Costanzo, A., Tortora, F., Cirillo, M., Esposito, F., & Tedeschi, G. (2011). Distributed changes in default-mode resting-state connectivity in multiple sclerosis. *Multiple Sclerosis Journal*, 17(4), 411-422.
- Bonnelle, V., Leech, R., Kinnunen, K. M., Ham, T. E., Beckmann, C. F., De Boissezon, X., Greenwood, R. J., & Sharp, D. J. (Sept 2011). Default mode network connectivity predicts sustained attention deficits after traumatic brain injury. *The Journal of Neuroscience*, 31(38), 13442-13451.
- Bowler, D. M., Gardiner, J. M., & Gaigg, S. B. (2007). Factors affecting conscious awareness in the recollective experience of adults with Asperger's syndrome. *Consciousness and Cognition*, 16, 124–143.
- Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., et al. (1996a). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron*, 17, 875-887.
- Breiter, H. C., Rauch, S. L., Kwong, K. K., Kennedy, D. N., Savage, C. R., Olivares, M. J., et al. (1996b). Functional magnetic resonance imaging of symptom provocation in obsessive-compulsive disorder. *Archives of General Psychiatry*, 53, 595-606.
- Bremner, J. D., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., Capelli, S., McCarthy, G., Innis, R. B., & Charney, D. S. (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse--a preliminary report. *Biological Psychiatry*, 41(1), 23-32.
- Brown, P. (1991). Ultradian rhythms of cerebral function and hypnosis. *Contemporary Hypnosis*, 8(1), 17-24.
- Bryant, R. A., et al. (2008) Enhanced amygdala and medial prefrontal activation during nonconscious processing of fear in posttraumatic stress disorder: An fMRI study. *Human Brain Mapping*, 29, 517–523.
- Bucci, W. (2003). Varieties of dissociative experiences: A multiple code account and a discussion of Bromberg's case of 'William.' *Psychoanalytic Psychology*, 20(3), 542-557.
- Buckner, R. L., & Carroll, D. C. (2007). Self-projection and the brain. Trends in Cognitive Sciences, 11, 49–57.

- Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network: Anatomy, function, and relevance to disease. *Annals of the New York Academy of Sciences*, 1124, 1–38.
- Calhoun, V. D., Maciejewski, P. K., Pearlson, G. D., et al. (2008). Temporal lobe and 'default' hemodynamic brain modes discriminate between schizophrenia and bipolar disorder. *Human Brain Mapping*, 29, 1265-75.
- Cardena, E. (2000). Hypnosis in the treatment of trauma: A promising, but not fully supported, efficacious intervention. *International Journal* of *Clinical and Experimental Hypnosis*, 48, 225-238.
- Carhart-Harris, R. L., & Friston, K. J. (2010). The default-mode, ego-functions and freeenergy: A neurobiological account of Freudian ideas. *Brain*, 133, 1265–1283.
- Carmona, S., Vilarroya, O., Bielsa, A., et al. (2005). Global and regional gray matter reductions in ADHD: A voxel-based morphometric study. *Neuroscience Letters*, 389, 88–93.
- Carnes, P. (2001). Facing the shadow. Wickenburg, AZ: Gentle Path Press.
- Castellanos, F. X., Kelly, C., & Milham, M. P. (October 2009). The restless brain: Attention-deficit hyperactivity disorder, resting-state functional connectivity, and intrasubject variability. *The Canadian Journal of Psychiatry*, 54(10), 665-672.
- Cavanna, A. E., Danielson, N. B., Guo, J. N., & Blumenfeld, H. (2011). The default mode network and altered consciousness in epilepsy. *Behavioural Neurology*, 24(1), 55-65.
- Chanraud, S., Pitel, A., Pfefferbaum, A., & Sullivan, E. V. (2011). Disruption of functional connectivity of the default-mode network in alcoholism. *Cerebral Cortex*, 21(10), 2272-2281.
- Cheek, D. B. (1959). Unconscious perception of meaningful sounds during surgical anesthesia as revealed under hypnosis. *American Journal of Clinical Hypnosis*, 1, 101-113.
- Christoff, K., Gordon, A. M., Smallwood, J., Smith, R., & Schooler, J. W. (May 26, 2009). Experience sampling during fMRI reveals default network and executive system contributions to mind wandering. *Proceedings of the National Academy of Sciences*, 106(21), 8719–8724.
- Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *National Review of Neuroscience*, 3, 201–15.
- Costa, R. T., Cheniaux, E., Rosaes, P. A. L., de Carvalho, M. R., Freire, R. C., Versiani, M., Rangé, B. P., Nardi, A. E. (June 2011). The effectiveness of cognitive behavioral group therapy in treating bipolar disorder: A randomized controlled study. *Revista Brasileira de Psiquiatria*, 33(2), 144-149.
- Cozolino, L. (2002). The Neuroscience of Psychotherapy: Building and Rebuilding the Human Brain. New York, NY: W. W. Norton.
- Crawford, H. (1994). Brain dynamics and hypnosis: Attentional and disattentional processes. *International Journal of Clinical and Experimental Hypnosis*, 42(3), 204– 32, page 223.
- Critchley, H. D., Wiens, S., Rotshtein, P., et al. (2004). Neural systems supporting interoceptive awareness. *Nat Neurosciences*, 7, 189-95.
- Csikszentmihalyi, M. (1990). Flow: The psychology of optimal experience. New York: Harper Collins.
- Dabbs, J. M., & Hargrove, M. F. (1997). Age, testosterone, and behavior among female prison inmates. *Journal of Psychosomatic Medicine*, 59, 477-480.
- Dane, J. R. (Jul 1996). Hypnosis for pain and neuromuscular rehabilitation with multiple sclerosis: Case summary, literature review, and analysis of outcomes. *International Journal of Clinical and Experimental Hypnosis*, 44(3), 208-231.
- Daniels, J. K., Frewen, P., McKinnon, M. C., & Lanius, R. A. (2011). Default mode alterations in posttraumatic stress disorder related to early-life trauma: A developmental perspective. *Journal of Psychiatry Neuroscience*, 36(1), 56-59.
- Daniels, J. K., McFarlane, A. C., Bluhm, R. L., Moores, K. A., Clark, C. R., Shaw, M. E., Williamson, P. C., Densmore, M., & Lanius, R. A. (2010). Switching between

executive and default mode networks in posttraumatic stress disorder: Alterations in functional connectivity. *Journal of Psychiatry & Neuroscience*, 35(4), 258-66.

- D'Argembeau1, A., Stawarczyk, D., Majerus, S., Collette, F., Van der Linden, M., Feyers, D., Maquet, P., & Salmon, E. (2009). The neural basis of personal goal processing when envisioning future events. *Journal of Cognitive Neuroscience*, 22(8), 1701–1713.
- Daselaar, S. M., Prince, S. E., & Cabeza, R. (2004). When less means more: Deactivations during encoding that predict subsequent memory. *Neuroimage*, 23, 921–927.
- Davies, J. M., & Frawley, M. G. (1992). Dissociative processes and transference/ countertransference paradigms in the psychoanalytically oriented treatment of adult survivors of childhood sexual abuse. *Psychoanalytic Dialogues*, 2, 5-36, page 16.
- Debenedittis, G., Cigada, M., Bianchi, A., Signorini, M. G., & Cerutti, S. (1994). Autonomic changes during hypnosis: A heart rate variability power spectrum analysis as a marker of sympatho-vagal balance. *International Journal of Clinical* and Experimental Hypnosis, 42(2), 140-152.
- Deeley, et al. (2007), as described in Oakley, D. A. (2008). Hypnosis, trance and suggestion: Evidence from neuroimaging. In M. R. Nash, A. J. Barnier, M. R. Nash, & A. J. Barnier (Eds.), *The Oxford Handbook of Hypnosis: Theory, Research, and Practice*, pp. 365–392. Oxford, United Kingdom: Oxford University Press.
- de Lange, F. P., Koers, A., Kalkman, J. S., Bleijenberg, G., Hagoort, P., van der Meer, J. W. M., et al. (2008). Increases in prefrontal cortical volume following cognitive behavioral therapy in patients with chronic fatigue syndrome. *Brain*, 13(8), 2172-2180.
- De Pascalis, V. (Dec 1998). Brain mechanisms and attentional processes in hypnosis. INABIS '98. Fifth Internet World Congress on Biomedical Sciences. McMaster University, Hamilton, Ontario, Canada. Retrieved January 30, 2010, from http://www.mcmaster.ca/inabis98'woody/de pascalis0311/index.html.
- De Pascalis, V., & Perrone, M. (1996). EEG asymmetry and heart rate during experience of hypnotic analgesia in high and low hypnotizables. *International Journal of Psychophysiology*, 21, 163–175.
- Dhond, R. P., Yeh, C., Park, K., Kettner, N., & Napadow, V. (2008). Acupuncture modulates resting state connectivity in default and sensorimotor brain networks. *Pain*, 136(3), 407-418.
- Diamond, S. G., Davis, O. C., & Howe, R. D. (2008). Heart-rate variability as a quantitative measure of hypnotic depth. *International Journal of Clinical and Experimental Hypnosis*, 56(1), 1–18.
- Divino, C. L., & Moore, M. S. (May, 2010). Integrating neurobiological findings into psychodynamic psychotherapy training and practice. *Psychoanalytic Dialogues*, 20(3), 337-355.
- Doucet, G., Naveau, M., Petit, L., Delcroix, N., Zago, L., Crivello, F., Jobard, G., Tzourio-Mazoyer, N., Mazoyer, B., Mellet, E., & Joliot, M. (June 2011). Brain activity at rest: A multiscale hierarchical functional organization. *Journal of Neurophysiology*, 105(6), 2753-2763.
- Drevets, W. C. (2000) Neuroimaging studies of mood disorders. *Biological Psychiatry*, 48, 813–829.
- Duchniewska, K., & Kokoszka, A. (2003). The protective mechanisms of the basic restactivity cycle as an indirect manifestation of this rhythm in waking: Preliminary report. *International Journal of Neuroscience*, 113, 153-163.
- Ebner, K., Wotjak, C. T., Landgraf, R., & Engelmann, M. (2000). A single social defeat experience selectively stimulates the oxytocin, but not vasopressin, within the septal area of male rats. *Journal of Brain Research*, 872, 87-92.
- Egner, T., Jamieson, G. A., & Gruzelier, J. (2005). Hypnosis decouples cognitive control from conflict monitoring processes of the frontal lobes. *Neuroimage*, 27, 143-149.
- Ehling, T., Nijenhuis, E. R. S., & Krikke, A. (Nov 2-4, 2003). Volume of discrete brain structures in florid and recovered DID, DDNOS, and healthy controls. *Proceedings*

of the 20th International Fall Conference of the International Society for the Study of Dissociation. Chicago, IL.

- Ellenbogen, J., Hu, P., Payne, J. D., Titone, D., & Walker, M. P. (2007). Human relational memory requires time and sleep. *Proceedings of the National Academy of Sciences*, 104, 7723–7728.
- Erickson, K., Drevets, W., & Schulkin, J. (2003). Glucocorticoid regulation of diverse cognitive functions in normal and pathological emotional states. *Neuroscience & Biobehavioral Reviews*, 27, 233-246.
- Erickson, M. H. (1963). Chemo-anaesthesia in relation to hearing and memory. *American Journal of Clinical Hypnosis*, 6, 31-36.
- Erickson, M. H., Rossi, E. L., & Rossi, S. I. (1976). Hypnotic realities: The induction of clinical hypnosis and forms of indirect suggestion. New York, NY: Irvington.
- Evans, C., & Richardson, P. H. (1988). Improved recovery and reduced postoperative stay after therapeutic suggestions during general anesthesia. *Lancet*, 2(8609), 491-493.
- Fair, D. A., Cohen, A. L., Dosenbach, N. U., et al. (2008). The maturing architecture of the brain's default network. *Proceedings of the National Academy of Sciences*, 105, 4028-32.
- Fleshner, M., Laudenslager, M. L., Simons, L., & Maier, S. F. (1989). Reduced serum antibodies associated with social defeat in rats. *Physiology and Behavior*, 45, 1183-1187.
- Freeman, T. W., & Kimbrell, T. (2001). A "cure" for chronic combat-related posttraumatic stress disorder secondary to a right frontal lobe infarct. *Journal of Neuropsychiatry and Clinical Neuroscience*, 13, 106–109.
- Friedberg, F. (June 2010). Chronic fatigue syndrome, fibromyalgia, and related illnesses: A clinical model of assessment and intervention. *Journal of Clinical Psychology*, 66(6), 641-665.
- Furmark, T., Tillfors, M., Marteinsdottir, I., Fischer, H., Pissiota, A., Langstrom, B. et al. (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Archives of General Psychiatry*, 59, 425-433.
- Gallese, V. (2009). Mirror neurons and the neural exploitation hypothesis: From embodied simulation to social cognition. In J. A. Pineda (Ed.), *Mirror Neuron Systems: The Role of Mirroring Processes in Social Cognition* (pp. 163-184). New York: Humana Press.
- Garnham, J., et al. (Dec 2007). Prophylactic treatment response in bipolar disorder: Results of a naturalistic observation study. *Journal of Affective Disorders*, 104(1-3), 185-90.
- Gay, M. (Jan 2007). Effectiveness of hypnosis in reducing mild essential hypertension: A one-year follow-up. *International Journal of Clinical and Experimental Hypnosis*, 55(1), 67-83.
- Gellhorn, E. (1969). Further studies on the physiology and pathophysiology of tuning of the central nervous system. *Psychosomatics*, 10, 94–103.
- Gellhorn, E., & Keily, W. F. (1972). Mystical states of consciousness: Neurophysiological and clinical aspects. *Journal of Nervous and Mental Disease*, 154, 399-405.
- Gemignani, A., Santarcangelo, E., Sebastiani, L., Marchese, C., Mammoliti, R., Simoni, A., et al. (2000). Changes in autonomic and EEG patterns induced by hypnotic imagination of aversive stimuli in man. *Brain Research Bulletin*, 53, 105–111.
- Gentili, C., Ricciardi, E., Gobbini, M. I., et al. (2009). Beyond amygdala: Default mode network activity differs between patients with social phobia and healthy controls. *Brain Research Bulletin*, 79, 409-13.
- Goleman, D. (Feb 4, 2003). Behavior; Finding Happiness: Cajole Your Brain to Lean to the Left. *New York Times*.

- Golland, Y., et al. (2007). Extrinsic and intrinsic systems in the posterior cortex of the human brain revealed during natural sensory stimulation. *Cerebral Cortex*, 17, 766– 777.
- Greene, D. J., Colich, N., Iacoboni, M., Zaidel, E., Bookheimer, S. Y., & Dapretto, M. (Feb 18, 2011). Atypical neural networks for social orienting in autism spectrum disorders. *NeuroImage*.
- Greimel, E., Schulte-Rüther, M., Kircher, T., Kamp-Becker, I., Remschmidt, H., Fink, G. R., Herpertz-Dahlmann, B., & Konrad, K. (Jan 1, 2010). Neural mechanisms of empathy in adolescents with autism spectrum disorder and their fathers. *NeuroImage*, 49(1), 1055-1065.
- Grimm, S., Boesiger, P., Beck, J., et al. (2009). Altered negative BOLD responses in the default-mode network during emotion processing in depressed subjects. *Neuropsychopharmacology*, 34, 932–943.
- Gruzelier, J. H. (1998). A working model of the neurophysiology of hypnosis: A review of the evidence. *Contemporary Hypnosis*, 15, 3-21.
- Gruzelier, J. H. (2002). The role of psychological intervention in modulating aspects of immune function in relation to health and well-being. *Neurobiological Immune System*, 52, 383–417.
- Gruzelier, J. H. (2006). Theta synchronisation of hippocampal and long distance circuitry in the brain: Implications for EEG-neurofeedback and hypnosis in the treatment of PTSD. Novel Approaches to the Diagnosis & Treatment of Posttraumatic Stress Disorder, 6(1), 13-22.
- Gujar, N., Yoo, S-S., Hu, P., & Walker, M. P. (2009). The unrested resting brain: Sleep deprivation alters activity within the default-mode network. *Journal of Cognitive Neuroscience*, 22(8), 1637–1648.
- Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001) Medial prefrontal cortex and self-referential mental activity: Relation to a default mode of brain function. *Proceedings of the National Academy of Sciences*, 98, 4259–4264.
- Guye, M., Bettus, G., Bartolomei, F., & Cozzone, P. J. (2010). Graph theoretical analysis of structural and functional connectivity MRI in normal and pathological brain networks. *Magnetic Resonance Materials in Physics, Biology and Medicine*, 23(5-6), 409–421.
- Halfen, D. (March 12, 1986). What do "anesthetized" patients hear? *Anesthesiology News*, p. 12.
- Hanson, R. (April 2011). Self-directed neuroplasticity: A 21st-century view of meditation. *Noetic Now* - Institute of Noetic Sciences, Issue 9. Available online at http://www.noetic.org/noetic/issue-nine-april/.
- Hartman, D., & Zimberoff, D. (2006). Healing the body-mind in Heart-Centered therapies. *Journal of Heart-Centered Therapies*, 9(2), 75-137.
- Herpertz, S. C., Dietrich, T. M., Wenning, B., Krings, T., Erberich, S. G., Willmes, K., Thron, A., & Sass, H. (August 15, 2001). Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biological Psychiatry*, 50(4), 292-298.
- Hippel, C. V., Hole, G., & Kaschka, W. P. (2001 May). Autonomic profile under hypnosis as assessed by heart rate variability and spectral analysis. *Pharmacopsychiatry*, 34(3), 111-113.
- Hoagwood, K. (1990). Blame and adjustment among women sexually abused as children. Women & Therapy, 9(4), 89-110.
- Hugdahl, K. (1996). Cognitive influences on human autonomic nervous system function. *Current Opinion in Neurobiology*, 6, 252-258.
- Huijbers, W., Pennartz, C. M. A., Cabeza, R., Daselaar, S. M. (2011). The hippocampus is coupled with the default network during memory retrieval but not during memory encoding. *PLoS ONE*, 6(4), e17463.
- Hurlburt, R. T., Happe, F., & Frith, U. (1994). Sampling the form of inner experience in three adults with Asperger syndrome. *Psychological Medicine*, 24, 385–95.

- Hyman, S. E., Malenka, R. C., & Nestler, EJ. (2006). Neural mechanisms of addiction: The role of reward-related learning and memory. *Annual Review of Neuroscience*, 29, 565–598.
- Ingvar, D. H. (1985). Human Neurobiology, 4, 127-136.
- Jacobs, W. J., Laurance, H. E., Thomas, K. G. F., Luzcak, S. E., & Nadel, L. (1996). On the veracity and variability of traumatic memory. *Traumatology*, 2(2).
- Jang, J., Jung, W., Kang, D., Byun, M., Kwon, S., Choi, C., & Kwon, J. (2011). Increased default mode network connectivity associated with meditation. *Neuroscience Letters*, 487(3), 358-362.
- Janicki, D. L., Kamarck, T. W., Shiffman, S., Sutton-Tyrrell, K., & Gwaltney, C. J. (2005). Frequency of spousal interaction and 3-year progression of carotid artery intima medial thickness: The Pittsburgh Healthy Heart Project. *Psychosomatic Medicine*, 67, 889–896.
- Jensen, M. P., Barber, J., Romano, J. M., Molton, I. R., Raichle, K. A., Osborne, T. L., Engel, J. M., Stoelb, B. L., Kraft, G. H., & Patterson, D. R. (Apr 2009). A comparison of self-hypnosis versus progressive muscle relaxation in patients with multiple sclerosis and chronic pain. *International Journal of Clinical and Experimental Hypnosis*, 57(2), 198-221.
- Jorgensen, M. M., & Zachariae, R. (2002). Autonomic reactivity to cognitive and emotional stress of low, medium, and high hypnotizable healthy subjects: Testing predictions from the high risk model of threat perception. *International Journal of Clinical and Experimental Hypnosis*, 50(3), 248-275.
- Kahn, S. (2010). Stress and anxiety. In Arreed Franz Barabasz, Karen Olness, Robert Boland, & Stephen Kahn (Eds.). *Medical Hypnosis Primer: Clinical and Research Evidence*. (pp. 83-86). New York, NY: Routledge/Taylor & Francis Group.
- Kalivas, P. W., & Volkow, N. D. (2005). The neural basis of addiction: A pathology of motivation and choice. *American Journal of Psychiatry*, 162, 1403–1413.
- Kennedy, D. P., Redcay, E., & Courchesne, E. (2006). Failing to deactivate: Resting functional abnormalities in autism. *Proceedings of the National Academy of Sciences*, 103, 8275–8280.
- Kennedy, D. P., & Courchesne, E. (2008). Functional abnormalities of the default network during self- and other-reflection in autism. *Social Cognitive & Affective Neuroscience SCAN*, 3,177–190.
- Kirsch, I. (2011). The altered state issue: Dead or alive? International Journal of Clinical and Experimental Hypnosis, 59(3), 350–362.
- Kohen, D. P., & Murray, K. (2006). Depression in children and youth: Applications of hypnosis to help young people help themselves. In Michael D. Yapko (Ed.), *Hypnosis and Treating Depression: Applications in Clinical Practice*, (pp. 189-216). New York, NY: Routledge/Taylor & Francis Group.
- Koike, T., Kan, S., Misaki, M., & Miyauchi, S. (2011). Connectivity pattern changes in default-mode network with deep non-REM and REM sleep. *Neuroscience Research*, 69(4), 322-330.
- Kompus, K. (2011). Default mode network gates the retrieval of task-irrelevant incidental memories. *Neuroscience Letters*, 487(3), 318-321.
- Kounios, J., et al. (2006). The prepared mind: Neural activity prior to problem presentation predicts subsequent solution by sudden insight. *Psychological Sciences*, 17, 882–890.
- Kounios, J., et al. (2008). The origins of insight in resting-state brain activity. *Neuropsychologia*, 46, 281–291.
- Lanius, R. A., Bluhm, R. L., Coupland, N. J., Hegadoren, K. M., Rowe, B., Theberge, J., Neufeld, R. W. J., Williamson, P. C., & Brimson M. (2010). Default mode network connectivity as a predictor of post-traumatic stress disorder symptom severity in acutely traumatized subjects. *Acta Psychiatric Scandanavia*, 121, 33–40.
- Larson, J., & Lynch, G. (1986). Induction of synaptic potentiation in hippocampus by pattered stimulation involves two events. *Science*, 232, 985-988.

- Lazar, S. W., Kerr, C. E., Wasserman, R. H., Gray, J. R., Greve, M., Treadway, T., et al. (2005). Meditation experience is associated with increased cortical thickness. *NeuroReport*, 16(17), 1893-1897.
- LeDoux, J. E. (1999). Psychoanalytic theory: Clues from the brain. Neuro-Psychoanalysis, 1, 44-49.
- Levine, P. (1997). Waking the Tiger. Berkeley, CA: North Atlantic Books.
- Levine, S. (2002). Turning toward the Mystery: A Seeker's Journey. New York: HarperCollins.
- Lim, J. L., & Dinges, D. F. (2008). Sleep deprivation and vigilant attention. Annals of the New York Academy of Sciences, 1129, 305–322.
- Lind, S. E., & Bowler, D. M. (2010). Episodic memory and episodic future thinking in adults with autism. *Journal of Abnormal Psychology*, 119(4), 896–905.
- Linden, D. (Nov 2008). Brain imaging and psychotherapy: Methodological considerations and practical implications. *European Archives of Psychiatry & Clinical Neuroscience*, Supplement 5, Vol. 258, 71-75.
- Linden, D. E. J. (June 2006). How psychotherapy changes the brain the contribution of functional neuroimaging. *Molecular Psychiatry*, 11(6), 528-538.
- Linehan, M. M. (1993). Cognitive-behavioral Treatment of Borderline Personality Disorder. New York: Guilford Press.
- Lui, S., Huang, X., Chen, L., Tang, H., Zhang, T., Li, X. L. D., Kuang, W., Chan, R. C., Mechelli, A., Sweeney, J. A., & Gong, Q. (September 8, 2009). High-field MRI reveals an acute impact on brain function in survivors of the magnitude 8.0 earthquake in China. *Proceedings of the National Academy of Sciences*, 106(36), 15412-15417.
- Lutz, A., Gretschar, L. L., Rawlings, N., Ricard, M., & Davidson, R. J. (2004). Longterm meditators self-induce high-amplitude gamma synchrony during mental practice. *Neuroscience*, 101(46), 16369-16373.
- Lynn, S. J., & Green, J. P. (2011). The Sociocognitive and Dissociation Theories of hypnosis: Toward a rapprochement. *International Journal of Clinical and Experimental Hypnosis*, 59(3), 277-293.
- Ma, N., Liu, Y., Fu, X-M., Li, N., Wang, C-X., et al. (2011). Abnormal brain defaultmode network functional connectivity in drug addicts. *PLoS ONE* 6(1), e16560.
- Makris, N., Biederman, J., Valera, E. M., et al. (2007). Cortical thinning of the attention and executive function networks in adults with attention-deficit/hyperactivity disorder. *Cerebral Cortex*, 17, 1364–1375.
- Maquet, P., Faymonville, M. E., Degueldre, C., Delfiore, G., Franck, G., Luxen, A., & Lamy, M. (1999). Functional neuroanatomy of hypnotic state. *Biological Psychiatry*, 45, 327–333.
- Mar, R. A., & Oatley, K. (2008). The function of fiction is the abstraction and simulation of social experience. *Perspectives on Psychological Sciences*, 3, 173–192.
- Marwan, N., Baliki, Paul Y., Geha, A., Apkarian, Vania, & Chialvo, Dante R. (Feb 6, 2008). Beyond feeling: Chronic pain hurts the brain, disrupting the default-mode network dynamics. *The Journal of Neuroscience*, 28(6), 1398–1403.
- Maunder, R. G., & Hunter, J. J. (2001). Attachment and psychosomatic medicine: Developmental contributions to stress and disease. *Psychosomatic Medicine*, 63, 556–567.
- McEwen, B. S., Gould, E. A., & Sakai, R. R. (1992). The vulnerability of the hippocampus to protective and destructive effects of glucocorticoids in relation to stress. *British Journal of Psychiatry*, 160, 18–24.
- McGeown, W. J., Mazzoni, G., Venneri, A., & Kirsch, I. (2009). Hypnotic induction decreases anterior default mode activity. *Consciousness and Cognition: An International Journal*, 18, 848–855.
- McGuire, E. A., Gadian, D. G., Johnstude, J. S., Good, C. D., Ashburner, J., & Frackowiak, K. S. (2000). Navigation-related structural change in the hippocampi of taxi drivers. *Proceedings of the National Academy of Sciences*, 91(81), 4398-4403.

65

- McClenon, J. (1997). Shamanic healing, human evolution, and the origin of religion. Journal for the Scientific Study of Religion, 36(3), 345–354.
- Mevel, K., Chetelat, G., Eustache, F., & Desgranges, B. (2011). The default mode network in healthy aging and Alzheimer's Disease. *International Journal of Alzheimer's Disease*, Volume 2011, Article ID 535816.
- Miall, R. C., & Robertson, E. M. (2006). Functional imaging: Is the resting brain resting? *Current Biology*, 16, R998–R1000.
- Miltner, W. H. R., & Weiss, T. (2007). Cortical mechanisms of hypnotic pain control. In G. A. Jamieson (Ed.), *Hypnosis and Conscious States: The Cognitive Neuroscience Perspective* (pp. 51-66). Oxford, England: Oxford University Press.
- Miyashita, Y. (Oct 15, 2004). Cognitive memory: Cellular and network machineries and their top-down control. *Science*, 306, 435–440.
- Moll, J., de Oliveira-Souza, R., Garrido, G. J., Bramati, I. E., Caparelli-Daquer, E. M. A., Paiva, M. L. M. F., Zahn, R., & Grafman, J. (2007). The self as a moral agent: Linking the neural bases of social agency and moral sensitivity. *Social Neuroscience*, 2(3-4), 336-352.
- Morgan, T., & Cummings, A. L. (1999). Change experienced during group therapy by female survivors of childhood sexual abuse. *Journal of Consulting and Clinical Psychology*, 67, 28-36.
- Nadel, L., & Jacobs, W. J. (1996). The role of the hippocampus in PTSD, panic, and phobia. In N. Kato (Ed.), *Hippocampus: Functions and Clinical Relevance*. Amsterdam: Elsevier Science.
- Newberg, A. B., & d'Aquili, E. G. (1994). The near death experience as archetype: A model for 'prepared' neurocognitive processes. *Anthropology of Consciousness*, 5, 1-15.
- Newberg, A. B., & d'Aquili, E. G. (2000). The neuropsychology of religious and spiritual experience. In J. Andresen & R. K. C. Forman (Eds.), Cognitive Models and Spiritual Maps: Interdisciplinary Explorations of Religious Experience, 251-266. Charlottesville, VA: Imprint Academic.
- Newberg, A. B., & Iversen, J. (2003). The neural basis of the complex mental task of meditation: Neurotransmitter and neurochemical considerations. *Medical Hypotheses*, 61(2), 282-291.
- Newberg, A. B., Alavi, A., Baime, M., Pourdehnad, M., Santanna, J., & d'Aquili, E. (Apr 2001). The measurement of regional cerebral blood flow during the complex cognitive task of meditation: A preliminary SPECT study. *Psychiatry Research: Neuroimaging*, 106(2), 113–122.
- Nijenhuis, E. R. S., & Van der Hart, O. (1999). Forgetting and re-experiencing trauma: From anesthesia to pain. In J. Goodwin & R. Attias (Eds.), *Splintered Reflections: Images of the Body in Trauma*, 39-66. New York: Basic Books.
- Nijenhuis, E. R. S., van der Hart, O., & Steele, K. (2004). Trauma-related structural dissociation of the personality. Trauma Information Pages website. Web URL: http://www.trauma-pages.com/nijenhuis-2004.htm.
- Oakley, D. A., & Halligan, P. W. (2010). Psychophysiological foundations of hypnosis and suggestion. In S. J. Lynn, J. W. Rhue, & I. Kirsch (Eds.), *Handbook of Clinical* Hypnosis, pp. 79–118.Washington, DC: American Psychological Association.
- Ochsner, K. N., Knierim, K., Ludlow, D. H., et al. (2004). Reflecting upon feelings: An fMRI study of neural systems supporting the attribution of emotion to self and other. *Journal of Cognitive Neuroscience*, 16, 1746–72.
- Ogden, P., & Minton, K. (Oct 2000). Sensorimotor Psychotherapy: One Method for Processing Traumatic Memory. *Traumatology*, 6(3).
- Öngür, D., Lundy, M., Greenhouse, I., Shinn, A. K., Menon, V., Cohen, B. M., & Renshaw, P. F. (2010). Default mode network abnormalities in bipolar disorder and schizophrenia. *Psychiatry Research: Neuroimaging Section*, 183(1), 59-68.
- Otten, L. J., & Rugg, M. D. (2001). When more means less: Neural activity related to unsuccessful memory encoding. *Current Biology*, 11, 1528–1530.

- Otti, A. A., Guendel, H. H., Läer, L. L., Wohlschlaeger, A. M., Lane, R. D., Decety, J. J., & Noll-Hussong, M. M. (2010). I know the pain you feel—how the human brain's default mode predicts our resonance to another's suffering. *Neuroscience*, 169(1), 143-148.
- Overmeyer, S., Bullmore, E. T., Suckling, J., et al. (2001). Distributed grey and white matter deficits in hyperkinetic disorder: MRI evidence for anatomical abnormality in an attentional network. *Psychological Medicine*, 31, 1425–1435.
- Pagnoni, G., Cekic, M., & Guo, Y. (2008). 'Thinking about not-thinking': Neural correlates of conceptual processing during Zen meditation. *PLoS ONE*, 3(9), e3083.
- Peng, C. K., Henry, I. C., Mietus, J. E., Hausdorff, J. M., Khalsa, G., Benson, H., et al. (2004). Heart rate dynamics during three forms of meditation. *International Journal* of Cardiology, 95, 19–27.
- Pineda, J. A., et al. (2009). Positive behavioral and electrophysiological changes following neurofeedback training in children with autism. *Research in Autism Spectrum Disorders*, 2, 557-581.
- Porges, S. W. (Feb 2007). The Polyvagal Perspective. *Biological Psychology*, 74(2), 116– 143.
- Porges, S. W. (Autumn-Winter, 2010). The Global Association for Interpersonal Neurobiology Studies Interview. Connections & Reflections, The GAINS Quarterly.
- Porges, S. W. (2011). The Polyvagal Theory: Neurophysiolofical Foundations of Emotions, Attachment, Communication, and Self-regulation. New York: W. W. Norton & Co.
- Raichle, M. E. (Mar 2010). The Brain's Dark Energy. Scientific American, 302(3), 44-49.
- Rainville, P., Hofbauer, R. K., Bushnell, M. C., Dunca, G. H., & Price, D. D. (2002). Hypnosis modulates activity in brain structures involved in the regulation of consciousness. *Journal of Cognitive Neuroscience*, 14(6), 887-901.
- Rauch, S. L., van der Kolk, B. A., &Fisler, R. E. (1996). A symptom provocation study of posttraumatic stress disorder using positron emmission tomography and scriptdriven imagery. Archives of General Psychiatry, 53, 380–387.
- Raz, A., Shapiro, T., Fan, J., & Posner, M. I. (2002). Hypnotic suggestion and the modulation of Stroop interference. Archives of General Psychiatry, 59, 1155-1161.
- Revenstorf, D. (1999). Clinical hypnosis: theoretical and empirical state of the art. Psychotherapy & Psychosomatic Medicine in Psychology, 49, 5–13.
- Robbins, T. W., Ersche, K. D., & Everitt, B. J. (2008). Drug addiction and the memory systems of the brain. *Annals of the New York Academy of Sciences*, 1141, 1–21.
- Rocca, M. A., Valsasina, P., Absinta, M., Riccitelli, G., Rodegher, M. E., Misci, P, et al. (2010). Default-mode network dysfunction and cognitive impairment in progressive MS. *Neurology*, 20, 74: 1252–1259.
- Rossi, E. L. (1986). Altered states of consciousness in everyday life: The ultradian rhythms. In B. B. Wolman & M. Ullman (Eds.), *Handbook of States of Consciousness*, 97-132. New York, NY: Van Nostrand Reinhold.
- Saey, T. H. (July 18, 2009). You are who you are by DEFAULT. Science News, 176(2), 16-20.
- Sahar, T., Shalev, A. Y., & Porges, S. W. (2001). Vagal modulation of responses to mental challenge in posttraumatic stress disorder. *Biological Psychiatry*, 49, 637– 643.
- Saito, Y., & Sasaki, Y. (1997). The relationship between ultradian rhythm and cerebral dominance. *Japanese Journal of Hypnosis*, 42(1), 18-22.
- Salgado-Pineda, P. P., Fakra, E. E., Delaveau, P. P., McKenna, P. J., Pomarol-Clotet, E. E., & Blin, O. O. (2011). Correlated structural and functional brain abnormalities in the default mode network in schizophrenia patients. *Schizophrenia Research*, 125(2/3), 101-109.
- Saxena, S., Gorbis, E., O'Neill, J., Baker, S. K., Mandelkern, M. A., Maidment, K. M., Chang, S., Salamon, N., Brody, A. L., Schwartz, J. M., London, E. D. (Feb 2009). Rapid effects of brief intensive cognitive-behavioral therapy on brain glucose metabolism in obsessive-compulsive disorder. *Molecular Psychiatry*, 14(2), 197-205.

67

- Scaer, R. C. (2005). The Trauma Spectrum: Hidden Wounds and Human Resiliency. New York: W. W. Norton, page 67.
- Schacter, D. L., Addis, D. R., & Buckner, R. L. (2007). Remembering the past to imagine the future: The prospective brain. *Nature Reviews Neuroscience*, 8, 657–661.
- Schmahl, C. G., Elzinga, B. M., Ebner, U. W., Simms, T., Sanislow, C., Vermetten, E., McGlashan, T. H., & Bremner, J. D. (2004). Psychophysiological reactivity to traumatic and abandonment scripts in borderline personality and posttraumatic stress disorders: A preliminary report. *Psychiatry Research*, 126, 33–42.
- Schmahl, C. G., Vermetten, E., Elzinga, B. M., & Bremner, J. D. (2003). Magnetic resonance imaging of hippocampal and amygdala volume in women with childhood abuse and borderline personality disorder. *Psychiatry Research: Neuroimaging*, 122, 193–198.
- Schore, A. N. (2002). Clinical implications of a psychoneurobiological model of projective identification. In S. Alhanati (Ed.), *Primitive mental states: Psychobiology and psychoanalytic perspectives on early trauma and personality development* (pp. 1–65). London: Karnac.
- Schuff, N., Marmar, C. R., Weiss, D. S., et al. (1997). Reduced hippocampal volume and n-acetyl aspartate in posttraumatic stress disorder. Annals of the New York Academy of Sciences, 821, 516–520.
- Schwerdtfeger, A., & Friedrich-Mai, P. (2009). Social interaction moderates the relationship between depressive mood and heart rate variability: Evidence from an ambulatory monitoring study. *Health Psychology*, 28(4), 501–509.
- Scott, J., et al. (2006). Cognitive-behavioural therapy for bipolar disorder. The British Journal of Psychiatry, 188, 488-489.
- Seeley, W. W., Menon, V., Schatzberg, A. F., et al. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *Journal of Neuroscience*, 27, 2349-56.
- Segal, J., Baker, S., Körber, E., Arbiser, E. (March 2010). Working with brain damaged clients. *Therapy Today*, 21(2).
- Shannahoff-Khalsa, D. (2006). Kundalini Yoga Meditation: Techniques Specific for Psychiatric Disorders, Couples Therapy, and Personal Growth. New York: W. W. Norton & Co.
- Shulman, G. L., Corbetta, M., Fiez, J. A., et al. (1997). Searching for activations that generalize over tasks. *Human Brain Mapping*, 5(4), 317–322.
- Siegel, D. (1999). The Developing Mind: Toward a Neurobiology of Interpersonal Experience. London: Guilford Press.
- Simeon, O., Guralnik, E. A., Hazlett, J., Spiegel-Cohen, J., Hollander, E., & Buchsbaum, M. S. (2000). Feeling unreal: A PET study of depersonalization disorder. *American Journal of Psychiatry*, 157, 1782-1788.
- Simpkins, C. A., & Simpkins, A. M. (2010). Neuro-Hypnosis: Using Self-Hypnosis to Activate the Brain for Change. New York, NY: W. W. Norton.
- Spiegel, D. (1996). Hypnosis in the treatment of post-traumatic stress disorder. Casebook of Clinical Hypnosis. Washington, DC: American Psychological Association, 99-112.
- Spiegel, D. (2008). Intelligent design or designed intelligence? Hypnotizability as neurobiological adaptation. In M. R. Nash, A. J. Barnier, M. R. Nash, & A. J. Barnier (Eds.), *The Oxford Handbook of Hypnosis: Theory, Research, and Practice*, pp. 179-199. Oxford, United Kingdom: Oxford University Press.
- Spreng, R. N., Mar, R. A., & Kim, A. S. (2009). The common neural basis of autobiographical memory, prospection, navigation, theory of mind and the default mode: A quantitative meta-analysis. *Cognitive Neuroscience*, 21, 489-510.
- Sprick, U. (Jan 1995). Functional aspects of the involvement of the hippocampus in behavior and memory functions. *Behavioral Brain Research*, 66(1-2), 61-64.
- Squire, L. R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychological Review*, 99(2), 195-231.

- Supekar, K., Menon, V., Rubin, D., Musen, M., & Greicius, M. D. (2008). Network analysis of intrinsic functional brain connectivity in Alzheimer's disease. *PLoS Computational Biology*, 4(6), Article ID e1000100.
- Tanabe, J., Nyberg, E., Martin, L., Martin, J., Cordes, D., Kronberg, E., & Tregellas, J. (2011). Nicotine effects on default mode network during resting state. *Psychopharmacology*, 216(2), 287-295.
- Tebartz van Elst, L., Hesslinger, B., Thiel, T., Geiger, E., Haegele, K., Lemieux, L., Lieb, K., Bobus, M., Hennig, J., & Ebert, D. (2003). Frontolimbic brain abnormalities in patients with borderline personality disorder: A volumetric magnetic resonance imaging study. *Biological Psychiatry*, 54, 163–171.
- Teicher, M. (2000). Wounds time won't heal. Cerebrum, 2, 4.
- Thomas, K. G. F., Laurance, H. E., Jacobs, W. J., & Nadel, L. (1995). Memory for traumatic events: Formulating hypotheses and critical experiments. *Traumatology*, 1(2).
- Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: Looking up and down from the brain. *Psychoneuroendrocrinology*, 30, 1050–1058.
- Tramontana, J. (2009). Hypnotically Enhanced Treatment for Addictions: Alcohol Abuse, Drug Abuse, Gambling, Weight Control, and Smoking Cessation. Norwalk, CT: Crown House Publishing Limited.
- Umhau, J. C., George, D. T., Reed, S., Petrulis, S. G., Rawlings, R., & Porges, S. W. (2002). Atypical autonomic regulation in perpetrators of violent domestic abuse. *Psychophysiology*, 39, 117–123.
- Valent, P. (2007). Eight survival strategies in traumatic stress. Traumatology, 13(2), 4-14.
- van Baalen, D. (2010). Gestalt Therapy and Bipolar Disorder. Gestalt Review, 14(1), 71-88.
- van den Heuvel, M. P. & Hulshoff –Pol, H. E. (2010). Exploring the brain network: A review on resting-state fMRI functional connectivity. *European Neuropsychopharmacology*, 20(8), 519–534.
- van der Hart, O., & Nijenhuis, E. R. S. (2001). Generalized dissociative amnesia: Episodic, semantic and procedural memories lost and found. *Australian and New Zealand Journal of Psychiatry*, 35(5), 589-600.
- van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of post-traumatic stress. *Harvard Psychiatric Review*, Vol. 1.
- van der Kolk, B. A. (2001). The assessment and treatment of complex PTSD. In R. Yehuda (Ed.), *Traumatic Stress*. American Psychiatric Press.
- Vanhaudenhuyse, A., Boly, M., Laureys, S., & Faymonville, M. (2009). Neurophysiological correlates of hypnotic analgesia. *Contemporary Hypnosis*, 26(1), 15–23.
- Vanhaudenhuyse, A., Noirhomme, Q., Tshibanda, L. J. F., Bruno, M., Boveroux, P., Schnakers, C., Soddu, A., Perlbarg, V., Ledoux, D., Brichant, J., Moonen, G., Maquet, P., Greicius, M. D., Laureys, S., & Boly, M. (2010). Default network connectivity reflects the level of consciousness in non-communicative braindamaged patients. *Brain*, 133, 161–171.
- Vermetten, E, Vythilingam, M., Southwick, S. M., Charney, D. S., & Bremner, J. D. (2003). Long-term treatment with paroxetine increases verbal declarative memory and hippocampal volume in posttraumatic stress disorder. *Biological Psychiatry*, 54, 693-702.
- Vincent, J. L., Snyder, A. Z., Fox, M. D., Shannon, B. J., Andrews, J. R., et al. (2006). Coherent spontaneous activity identifies a hippocampal-parietal memory network. *Journal of Neurophysiology*, 96, 3517–3531.
- Viswesvaran, C., & Schmidt, F. (1992). A meta-analytic comparison of the effectiveness of smoking cessation methods. *Journal of Applied Psychology*, 77, 554-561.
- Volkow, N. D., Tomasi, D. D., Wang, G. J., Fowler, J. S., Telang, F. F., Goldstein, R. Z., & Alexoff, D. D. (2011). Positive emotionality is associated with baseline metabolism in orbitofrontal cortex and in regions of the default network. *Molecular Psychiatry*, 16(8), 818-825.

- Wagner, U., Gais, S., Haider, H., Verleger, R., & Born, J. (2004). Sleep inspires insight. *Nature*, 427, 352–355.
- Wallace, B. (1993). Day persons, night persons, and variability in hypnotic susceptibility. Journal of Personality and Social Psychology, 64(5), 827-833.
- Wickramasekera, I. (1998). Secrets kept from the mind but not the body or behavior: The unsolved problems of identifying and treating somatization and psychophysiological disease. Advances in Mind-Body Medicine, 14, 81-98.

Wilson, E. O. (1975). Sociobiology. Cambridge, MA: Belknap.

Winson, J. (Nov 1990). The meaning of dreams. Scientific American, 86-96.

- Witkiewitz, K., Marlatt, G. A., & Walker, D. (2005). Mindfulness-based relapse prevention for alcohol and substance use disorders. *Journal of Cognitive Psychotherapy*, 19(3), 211-228.
- Worthington, E. L. (2005). Handbook of Forgiveness. East Sussex, UK: Brunner-Routledge.
- Yapko, M. (Sept/Oct 2011). Suggesting mindfulness. Psychotherapy Networker, 29-33, 50-52.
- Zanarini, M. C., Frankenburg, F. R., Reich, D. B., et al. (2007). The subsyndromal phenomenology of borderline personality disorder: A 10-year follow-up study. *American Journal of Psychiatry*, 164, 929–935.

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Endnotes

- ¹ Erickson, Rossi, & Rossi. (1976).
- ² Linden. (Nov 2008).
- ³ Cozolino. (2002), p. 46.
- ⁴ Simpkins & Simpkins. (2010), p. 4.
- ⁵ Teicher. (2000).
- ⁶ de Lange, Koers, Kalkman, et al. (2008).
- ⁷ Bremner, Randall, Vermetten, et al. (1997).
- 8 Squire. (1992).
- ⁹ Ehling, Nijenhuis, & Krikke. (Nov 2-4, 2003).
- ¹⁰ Rauch, van der Kolk, & Fisler. (1996).
- ¹¹ Freeman & Kimbrell. (2001).
- ¹² Schore. (2002).
- ¹³ Maunder & Hunter. (2001).
- ¹⁴ Thayer & Brosschot. (2005).
- ¹⁵ Bingel, Wanigasekera, Wiech, et al. (Feb 2011).
- ¹⁶ Erickson. (1963).
- ¹⁷ Cheek. (1959).
- ¹⁸ Halfen. (March 12, 1986).
- ¹⁹ Bennett. (1988).
- ²⁰ Evans & Richardson. (1988).
- ²¹ Bennett, Davis, & Giannini. (1985).
- ²² Porges. (Autumn-Winter, 2010), p. 64.
- ²³ Raichle. (Mar 2010).
- ²⁴ Raichle. (Mar 2010).
- ²⁵ Corbetta & Shulman. (2002).
- ²⁶ Carhart-Harris & Friston. (2010).
- ²⁷ Doucet, Naveau, Petit, et al. (June 2011).
- ²⁸ van den Heuvel & Hulshoff –Pol. (2010).
- ²⁹ Spreng, Mar, & Kim, (2009).
- ³⁰ Buckner, Andrews-Hanna, & Schacter. (2008).
- ³¹ Shulman, Corbetta, Fiez, et al. (1997).
- ³² Buckner & Carroll. (2007).
- 33 Bar. (2007).
- ³⁴ Bar, Aminoff, Mason, et al. (2007).
- ³⁵ Ingvar, (1985).
- ³⁶ Otti, Guendel, Läer, et al. (2010).
- ³⁷ Kompus. (2011).
- ³⁸ Pagnoni, Cekic, & Guo. (2008).
- ³⁹ Jang, Jung, Kang, et al. (2011).
- ⁴⁰ Moll, de Oliveira-Souza, Garrido, et al. (2007), p. 350.
- ⁴¹ Huijbers, Pennartz, Cabeza, et al. (2011).
- ⁴² Daselaar, Prince, & Cabeza. (2004).
- ⁴³ Otten & Rugg. (2001).
- ⁴⁴ Vincent, Snyder, Fox, et al. (2006).
- ⁴⁵ McGuire, Gadian, Johnstude, et al. (2000).
- ⁴⁶ Abram. (1996).
- 47 Abram. (1996), p. 262.
- ⁴⁸ Siegel. (1999).
- 49 Simpkins & Simpkins. (2010), p. 238.
- ⁵⁰ Simpkins & Simpkins. (2010), p. 235.
- ⁵¹ McVay & Kane. (2010), p. 194.
- ⁵² Miyashita. (2004).
- ⁵³ Ogden & Minton. (Oct 2000).

- ⁵⁶ Porges. (2007).
- ⁵⁷ Porges. (2011).
- ⁵⁸ Porges. (Autumn-Winter, 2010).
- ⁵⁹ Gemignani, Santarcangelo, Sebastiani, et al. (2000).
- ⁶⁰ Peng, Henry, Mietus, et al. (2004).
- ⁶¹ Hippel, Hole, & Kaschka. (2001 May).
- ⁶² Debenedittis, Cigada, Bianchi, et al. (1994).
- 63 Jorgensen & Zachariae. (2002).
- ⁶⁴ Wickramasekera. (1998).
- 65 Cardena. (2000).
- 66 Dhond, Yeh, Park, et al. (2008).
- 67 Duchniewska & Kokoszka. (2003).
- ⁶⁸ Brown. (1991).
- 69 Rossi. (1986).
- 70 Saito & Sasaki. (1997).
- ⁷¹ Wallace. (1993).
- 72 Simpkins & Simpkins. (2010), p. 199.
- ⁷³ Larson & Lynch. (1986).
- ⁷⁴ Furmark, Tillfors, Marteinsdottir, et al. (2002).
- ⁷⁵ Simpkins & Simpkins. (2010), p. 196.
- ⁷⁶ Gallese. (2009).
- ⁷⁷ Divino & Moore. (May 2010).
- ⁷⁸ Balbernie. (2007).
- ⁷⁹ Boly, Phillips, Tshibanda, et al. (2008).
- ⁸⁰ Yapko. (Sept/Oct 2011), p. 31.
- ⁸¹ Revenstorf. (1999).
- ⁸² Maquet, Faymonville, Degueldre, et al. (1999).
- ⁸³ DePascalis & Perrone. (1996).
- ⁸⁴ Barabasz & Barabasz. (2008).
- ⁸⁵ De Pascalis. (Dec 1998).
- 86 Spiegel. (2008), p. 179.
- ⁸⁷ Seeley, Menon, Schatzberg, et al. (2007).
- ⁸⁸ Critchley, Wiens, Rotshtein, et al. (2004).
- 89 Barabasz & Barabasz. (2008).
- ⁹⁰ Oakley & Halligan. (2010).
- 91 Winson. (Nov 1990).
- 92 Badre & Wagner. (2004).
- ⁹³ Anderson, Ochsner, Kuhl, et al. (2004).
- ⁹⁴ Vanhaudenhuyse, Boly, Laureys, et al. (2009).
- ⁹⁵ Rainville, Hofbauer, Bushnell, et al. (2002).
- ⁹⁶ Gruzelier. (1998).
- ⁹⁷ Gruzelier. (2002).
- ⁹⁸ Aubert, Verheyden, Beckers, et al. (2009).
- ⁹⁹ Spiegel. (2008).
- ¹⁰⁰ Hugdahl, K. (1996).
- ¹⁰¹ Lazar, Kerr, Wasserman, et al. (2005).
- ¹⁰² Newberg & Iversen. (2003).
- ¹⁰³ Egner, Jamieson, & Gruzelier. (2005).
- ¹⁰⁴ Lutz, Gretschar, Rawlings, et al. (2004).
- ¹⁰⁵ Newberg, Alavi, Baime, et al. (Apr 2001).
- ¹⁰⁶ Simeon, Guralnik, Hazlett, et al. (2000).
- ¹⁰⁷ Gellhorn. (1969).

⁵⁴ Teicher. (2000).

⁵⁵ Porges. (2011).

¹¹¹ Lynn & Green. (2011), p. 287.

- ¹¹³ D'Argembeau, Stawarczyk, Majerus, et al. (2009).
- ¹¹⁴ Christoff, Gordon, Smallwood, et al. (May 26, 2009).
- ¹¹⁵ Christoff, Gordon, Smallwood, et al. (May 26, 2009), p. 8722.
- ¹¹⁶ Egner, Jamieson, & Gruzelier. (2005).
- ¹¹⁷ Raz, Shapiro, Fan, et al. (2002).
- ¹¹⁸ Simpkins & Simpkins. (2010), p. 49.
- ¹¹⁹ Miltner & Weiss. (2007).
- ¹²⁰ Gusnard, Akbudak, Shulman, et al. (2001).
- ¹²¹ Kounios, et al. (2006).
- ¹²² Kounios, et al. (2008).
- 123 Golland, et al. (2007).
- ¹²⁴ Mar & Oatley. (2008).
- ¹²⁵ McGeown, Mazzoni, Venneri, et al. (2009).
- 126 Kirsch. (2011).
- ¹²⁷ McGeown, Mazzoni, Venneri, et al. (2009), p. 848.
- ¹²⁸ Deeley, et al. (2007), as described in Oakley, D. A. (2008).
- ¹²⁹ Simpkins & Simpkins. (2010), p. 53.
- ¹³⁰ Diamond, Davis, & Howe. (2008).
- ¹³¹ Aubert, Verheyden, Beckers, et al. (2009).
- ¹³² Guye, Bettus, Bartolomei, et al. (2010).
- ¹³³ Saey. (July 18, 2009).
- ¹³⁴ Chanraud, Pitel, Pfefferbaum, et al. (2011).
- ¹³⁵ Witkiewitz, Marlatt, & Walker. (2005).
- ¹³⁶ Raichle. (Mar 2010).
- ¹³⁷ Supekar, Menon, Rubin, et al. (2008).
- ¹³⁸ Mevel, Chetelat, Eustache, et al. (2011).
- ¹³⁹ Castellanos, Kelly, & Milham. (Oct 2009).
- ¹⁴⁰ Makris, Biederman, Valera, et al. (2007).
- ¹⁴¹ Overmeyer, Bullmore, Suckling, et al. (2001).
- ¹⁴² Carmona, Vilarroya, Bielsa, et al. (2005).
- 143 Sprick. (Jan 1995).
- ¹⁴⁴ Abarbanel. (1995).
- ¹⁴⁵ Shannahoff-Khalsa. (2006).
- ¹⁴⁶ Kennedy, Redcay, & Courchesne. (2006).
- ¹⁴⁷ Hurlburt, Happe, & Frith. (1994).
- ¹⁴⁸ Kennedy & Courchesne. (2008).
- ¹⁴⁹ Assaf, Jagannathan, Calhoun, et al. (2010).
- ¹⁵⁰ Gusnard, Akbudak, Shulman, et al. (2001).
- ¹⁵¹ Ochsner, Knierim, Ludlow, et al. (2004).
- ¹⁵² Hurlburt, Happe, & Frith. (1994).
- ¹⁵³ Lind & Bowler. (2010).
- ¹⁵⁴ Bowler, Gardiner, & Gaigg. (2007).
- ¹⁵⁵ Greimel, Schulte-Rüther, Kircher, et al. (Jan 1, 2010).
- ¹⁵⁶ Greene, Colich, Iacoboni, et al. (Feb 18, 2011).
- ¹⁵⁷ Pineda, et al. (2009).
- ¹⁵⁸ Calhoun, Maciejewski, Pearlson, et al. (2008).
- ¹⁵⁹ Öngür, Lundy, Greenhouse, et al. (2010).
- ¹⁶⁰ Garnham, et al. (Dec 2007).
- ¹⁶¹ van Baalen. (2010).

¹⁰⁸ Gellhorn & Keily. (1972).

¹⁰⁹ Newberg & d'Aquili. (1994).

¹¹⁰ Newberg & d'Aquili. (2000).

¹¹² Oakley & Halligan. (2010).

¹⁶⁷ Schmahl, Vermetten, Elzinga, et al. (2003).

- ¹⁶⁹ Umhau, George, Reed, et al. (2002).
- ¹⁷⁰ American Psychiatric Association. (2001).
- ¹⁷¹ Zanarini, Frankenburg, Reich, et al. (2007).
- ¹⁷² Linehan. (1993).
- ¹⁷³ Vanhaudenhuyse, Noirhomme, Tshibanda, et al. (2010).
- ¹⁷⁴ Jensen, Barber, Romano, et al. (Apr 2009).
- ¹⁷⁵ Segal, Baker, Körber, et al. (March 2010).
- ¹⁷⁶ de Lange, Koers, Kalkman, et al. (2008).
- ¹⁷⁷ Friedberg. (June 2010).
- ¹⁷⁸ Apkarian, Sosa, Krauss, et al. (2004).
- ¹⁷⁹ Marwan, Baliki, Geha, et al. (Feb 6, 2008).
- ¹⁸⁰ Baliki, Geha, Apkarian, et al. (Feb 6, 2008), p. 1402.
- ¹⁸¹ Grimm, Boesiger, Beck, et al. (2009).
- ¹⁸² Bluhm, Williamson, Lanius, et al. (2009).
- ¹⁸³ Berman, Peltier, Nee, et al. (September 19, 2010).
- ¹⁸⁴ Grimm, Boesiger, Beck, et al. (2009).
- ¹⁸⁵ Schwerdtfeger & Friedrich-Mai. (2009).
- ¹⁸⁶ Janicki, Kamarck, Shiffman, et al. (2005).
- ¹⁸⁷ Alladin. (2010).
- ¹⁸⁸ Kohen & Murray. (2006).
- ¹⁸⁹ Ma, Liu, Fu, et al. (2011).
- ¹⁹⁰ Kalivas & Volkow. (2005).
- ¹⁹¹ Robbins, Ersche, & Everitt. (2008).
- ¹⁹² Hyman, Malenka, & Nestler. (2006).
- ¹⁹³ Tramontana. (2009).
- ¹⁹⁴ Carnes. (2001).
- 195 Gay. (Jan 2007).
- ¹⁹⁶ Bonavita, Gallo, Sacco, et al. (2011).
- ¹⁹⁷ Rocca, Valsasina, Absinta, et al. (2010).
- ¹⁹⁸ Benedict, Cookfair, Gavett, et al. (2006).
- ¹⁹⁹ Dane. (Jul 1996).
- ²⁰⁰ Tanabe, Nyberg, Martin, et al. (2011).
- ²⁰¹ Viswesvaran & Schmidt. (1992).
- ²⁰² Baxter, Schwartz, & Bergman. (1992).
- ²⁰³ Volkow, Tomasi, Wang, et al. (2011).
- ²⁰⁴ Saxena, Gorbis, O'Neill, et al. (Feb 2009).
- ²⁰⁵ Linden. (June 2006).
- ²⁰⁶ Salgado-Pineda, Fakra, Delaveau, et al. (2011).
- ²⁰⁷ Cavanna, Danielson, Guo, et al. (2011).
- ²⁰⁸ Gujar, Yoo, Hu, et al. (2009).
- ²⁰⁹ Lim & Dinges. (2008).
- ²¹⁰ Gujar, Yoo, Hu, et al. (2009).
- ²¹¹ Miall & Robertson. (2006).
- ²¹² Schacter, Addis, & Buckner. (2007).
- ²¹³ Ellenbogen, Hu, Payne, et al. (2007).
- ²¹⁴ Wagner, Gais, Haider, et al. (2004).
- ²¹⁵ Koike, Kan, Misaki, et al. (2011).

¹⁶² Scott, et al. (2006).

¹⁶³ Costa, Cheniaux, Rosaes, et al. (June 2011).

¹⁶⁴ Schmahl, Elzinga, Ebner, et al. (2004).

¹⁶⁵ Austina, Riniolob, & Porges. (Oct 2007).

¹⁶⁶ Tebartz van Elst, Hesslinger, Thiel, et al. (2003).

¹⁶⁸ Sahar, Shalev, & Porges. (2001).
- ²¹⁶ Gentili, Ricciardi, Gobbini, et al. (2009).
- ²¹⁷ Kahn. (2010).
- ²¹⁸ Bonnelle, Leech, Kinnunen, et al. (Sept 2011).
- ²¹⁹ Daniels, McFarlane, Bluhm, et al. (2010).
- ²²⁰ Lui, Huang, Chen, et al. (Sept 8, 2009).
- ²²¹ Drevets. (2000).
- ²²² Bryant, et al. (2008).
- ²²³ Lanius, Bluhm, Coupland, et al. (2010).
- ²²⁴ Lanius, Bluhm, Coupland, et al. (2010), p. 39.
- ²²⁵ Bluhm, Williamson, Osuch, et al. (2009).
- ²²⁶ Daniels, Frewen, McKinnon, et al. (2011).
- ²²⁷ Fair, Cohen, Dosenbach, et al. (2008).
- ²²⁸ Daniels, Frewen, McKinnon, et al. (2011).
- ²²⁹ Worthington. (2005).
- ²³⁰ Gruzelier. (2006).
- ²³¹ Spiegel. (1996).
- ²³² Spiegel. (1996).
- ²³³ Simpkins & Simpkins. (2010), p. 240.
- ²³⁴ Nijenhuis, van der Hart, & Steele. (2004).
- ²³⁵ Bremner, Randall, Vermetten, et al. (1997).
- ²³⁶ Schuff, Marmar, Weiss, et al. (1997).
- ²³⁷ Squire. (1992).
- ²³⁸ Goleman. (Feb 4, 2003).
- ²³⁹ Herpertz, Dietrich, Wenning, et al. (August 15, 2001).
- ²⁴⁰ Rauch, van der Kolk, & Fisler. (1996).
- ²⁴¹ Breiter, Etcoff, Whalen, et al. (1996a).
- ²⁴² Breiter, Rauch, Kwong, et al. (1996b).
- ²⁴³ Aggleton. (1992).
- ²⁴⁴ Erickson, Drevets, & Schulkin. (2003).
- ²⁴⁵ McEwen, Gould, & Sakai. (1992).
- ²⁴⁶ Thomas, Laurance, Jacobs, et al. (1995).
- ²⁴⁷ Jacobs, Laurance, Thomas, et al. (1996).
- ²⁴⁸ Scaer. (2005).
- ²⁴⁹ Ehling, Nijenhuis, & Krikke. (Nov 2-4, 2003).
- ²⁵⁰ Vermetten, Vythilingam, Southwick, et al. (2003).
- ²⁵¹ McClenon. (1997).
- ²⁵² Crawford. (1994).
- ²⁵³ Hanson. (April 2011).
- ²⁵⁴ Winson. (Nov 1990).
- ²⁵⁵ Cozolino. (2002), p. 27.
- ²⁵⁶ Hartman & Zimberoff. (2006).
- ²⁵⁷ van der Kolk. (2001).
- ²⁵⁸ Levine. (1997), p. 110.
- ²⁵⁹ Bucci. (2003), p. 548, italics added.
- ²⁶⁰ LeDoux. (1999).
- ²⁶¹ Nijenhuis & Van der Hart. (1999).
- ²⁶² Hoagwood. (1990).
- ²⁶³ Morgan & Cummings. (1999).
- ²⁶⁴ Nadel & Jacobs. (1996).
- ²⁶⁵ van der Kolk. (1994).
- ²⁶⁶ van der Hart & Nijenhuis. (2001).