

# The Neurobiology of EMDR: Exploring the Thalamus and Neural Integration

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Recent neuroimaging studies on posttraumatic stress disorder (PTSD) have revealed a consistent decrease in thalamic activity, relative to non-PTSD controls. Empirical studies of the past decade have shown the thalamus to be centrally involved in the integration of perceptual, somatosensory, memorial, and cognitive processes (thalamo-cortical-temporal binding). A theoretical model is proposed to suggest that one underlying mechanism of EMDR stimulation (dual-attention stimulation/bilateral stimulation [DAS/BLS]) is thalamic activation, specifically of the ventrolateral and central-lateral nuclei. It is hypothesized that this may facilitate the repair and integration of somatosensory, memorial, cognitive, frontal lobe and synchronized hemispheric functions that are disrupted in PTSD.

**Keywords:** EMDR; thalamus; neural oscillation; thalamo-cortical-temporal binding; 40-Hz gamma-band activity (GBA)

In the past decade, descriptive and empirical publications regarding posttraumatic stress disorder (PTSD) have yielded the impression of a disorder manifested by the inability to integrate the totality of a traumatic event into consciousness, thereby causing the intrusion into awareness of fragmented traumatic memories, primarily in sensory form. These intrusive sensory fragments tend to be visual, olfactory, auditory, kinesthetic, or visceral. Accordingly, PTSD manifests with dramatic symptoms of intrusive memorial recollections, nightmares, and various modalities of somatosensory flashbacks. Previous speculative EMDR models (noted in detail below) have addressed EMDR's facilitation of an investigatory reflex, frontal lobe activation, REM systems activation, reduction of emotional valence and electrodermal arousal, and synaptic depotentiation and reversal of reciprocal suppression in the anterior cingulate cortex.

Recent neuroimaging studies (noted below) on PTSD have revealed consistent decreases in thalamic activation, relative to non-PTSD controls, indicating that areas of the thalamus were operating at reduced functioning. Empirical studies of the past decade have shown the thalamus to be centrally involved in thalamo-cortical-temporal binding, a process wherein the activity of all functional networks of the brain,

each oscillating at their own different frequencies, is synchronized and coherently bound together in real time. Therefore, the thalamus and its binding functions have been shown to constitute the platform on which perceptual, somatosensory, cognitive, and memorial integration take place.

Accordingly, this discussion will explore and establish parallels between the research bases of the neurobiology of PTSD, neurobiological studies of EMDR, synchronous neuronal oscillation, perception, memory, and thalamic temporal binding. It will also introduce the proposed theory that EMDR stimulation (DAS/BLS) may facilitate the activation of the ventrolateral and central-lateral thalamic nuclei, thereby facilitating the repair and integration of somatosensory, memorial, cognitive, and synchronized hemispheric functions. Additionally, the activation of the ventrolateral nucleus will be shown to facilitate the activation of the dorsolateral prefrontal cortex, which is disrupted in PTSD.

## Speculative Models of EMDR's Mechanisms

Various models have been proposed to account for EMDR's underlying mechanisms of action. Denny

(1995) suggested an inhibition model in which an orienting reflex suppresses the disturbance of traumatic memories. Armstrong and Vaughn (1996) proposed an extinction model whereby the orienting reflex is seen to catalyze a new appraisal and change in the neuronal model of the unconditional stimulus. McCulloch and Feldman (1996) proposed that eye movements in EMDR facilitated a “reassurance reflex” as a result of the positive visceral components of an investigatory orienting response. Andrade, Kavanagh, and Baddeley (1997) suggested that eye movements interfere with the vividness of traumatic material in the visuospatial sketchpad of working memory. Bergmann (1998), in examining EMDR’s effect on hemispheric laterality, proposed that DAS/BLS facilitated the resetting of septal pacemaker cells, thereby resynchronizing the functional connectivity of the hemispheres. Bergmann (2000) suggested that DAS/BLS facilitated the activation of the lateral cerebellum, which acted as an association area, projecting to and activating the ventrolateral and central-lateral nuclei of the thalamus. The ventrolateral nucleus of the thalamus was shown to project to and activate areas of the dorsolateral prefrontal cortex, facilitating the integration of traumatic memory into general semantic and other neocortical networks. Stickgold (2002) suggested that EMDR stimulation mediated a sufficient surge of acetylcholine, thereby facilitating the activation of REM-like physiological systems, leading to the subsequent reduction in both the strength of hippocampally mediated episodic memories and the amygdaloid mediated negative affect of PTSD. Accordingly, this was seen to mediate the integration of traumatic memories into general semantic networks. Corrigan (2002) posited that auditory, visual, and tactile EMDR stimuli facilitate the bilateral stimulation of relevant thalamo-cingulate tracts, gradually stimulating and deactivating the affective (ventral) subdivision of the anterior cingulate gyrus, allowing, then, for the stimulation and activation of the cognitive (dorsal) subdivision and a reciprocal inhibition within the anterior cingulate. Consistent with McCulloch and Feldman (1996), Barrowcliff, Gray, MacCulloch, Freeman, and MacCullough (2003) demonstrated psychophysiological dearousal effects of eye movement following arousing stimulation. Barrowcliff, Gray, Freeman, and MacCullough (2004) noted reductions in measures of vividness and emotional valence of emotional memories during eye movements as compared to an eye-stationary condition. They suggested that these findings were consistent with and supportive

of McCulloch and Feldman’s (1996) reassurance reflex model. Rasolkhani-Kalhorn and Harper (2006), in exploring the process of limbic fear memory formation, noted that the depotentiation of these limbic synapses by the induction of low-frequency stimulation (LFS) leads to erasure or modification of these memories. The authors proposed that LFS could be induced by EMDR stimulation, leading to the depotentiation of these synapses, resulting in the quenching or modification of fear memory traces. Kaye (2007) isolated and explored eye movements in EMDR. The author suggests that the eye-to-finger tracking task may achieve its therapeutic effect by using error monitoring to reverse suppression of the upper (dorsal) cognitive subdivision of the anterior cingulate by the lower (ventral) affective subdivision, thereby facilitating the reversal of this reciprocal suppression. This model is consistent with Corrigan’s (2002) speculation.

## **Descriptive and Functional Anatomy**

In the past twenty years, a great deal of light has been shed in the area of neurobiology. Much of the research has focused on the components that mediate our emotional state of mind. In particular, the interrelationship between the structure and function of the amygdala, thalamus, left dorsolateral prefrontal cortex, and hippocampus has been articulated with increasing clarity. To understand the malfunctioning of these structures in PTSD, a brief description of their functional characteristics is required.

### **The Amygdala**

The amygdala is an almond-shaped cluster of interconnected structures perched above the brain stem. It is composed of two structures nestled in the temporal lobe, in the right and left hemispheres. The corticomедial amygdala, in the right hemisphere, is connected primarily with the olfactory bulb, the hypothalamus, and the visceral nuclei of the brain stem. The basolateral amygdala, in the left hemisphere, is connected with the thalamus and parts of the cerebral cortex (Brodal, 1980, 1992).

The amygdala is the central junction where information from all senses is tied together and endowed with emotional meaning. In the brain’s architecture, the amygdala is poised like an alarm. Incomplete or confusing signals from the senses let the amygdala scan experiences for danger. Sensory signals from the eyes, mouth, skin, and ears travel first in the brain to the thalamus and then across a single synapse to the amygdala. Sensory signals from the nose are routed

directly to the amygdala, bypassing even the thalamus (LeDoux, 1986, 1992, 1994). A second signal from the thalamus is routed to the neocortex—the thinking brain. This branching allows the amygdala to respond before the neocortex, which mulls over information through several layers of brain circuits before it fully perceives and initiates a response (LeDoux, 1986). As the amygdala becomes aroused, either from external stress or internal anxiety, a nerve running from the brain to the adrenal gland triggers a secretion of epinephrine and, from the locus coeruleus in the brain stem, norepinephrine, which then surge through the body, eliciting alertness. These neurotransmitters activate the receptors on the vagus nerve (LeDoux, 1986). While the vagus nerve carries messages from the brain to regulate the heart, it also carries signals back to the brain, triggered by epinephrine and norepinephrine. The amygdala is the main site in the brain where these signals are carried. They activate neurons within the amygdala to signal other brain regions to strengthen the memory of what just happened. This amygdaloid arousal seems to imprint in memory most moments of emotional arousal with an added degree of strength (Goleman, 1995). The more intense the amygdaloid arousal, the stronger the imprint (LeDoux, 1986).

### The Dorsolateral Prefrontal Cortex

The brain's damper switch for the amygdala appears to lie at the other end of a major circuit to the neocortex, in the left prefrontal lobe, just behind the forehead (Brodal, 1980). Some of this circuitry is also found in the temporal lobe. This dorsolateral prefrontal part of the brain brings a more analytic and appropriate response to emotional impulses, modulating the amygdala and other limbic areas (LeDoux, 1986). The presence of circuits noted above connecting the amygdala to the prefrontal lobes implies that the signals of emotion, anxiety, anger, and terror generated in the amygdala can cause decreased activation in the dorsolateral area, sabotaging the ability of the prefrontal lobe to maintain working memory and homeostasis (Selemon, Goldmanrakis, & Tamminga, 1995).

### The Hippocampus

The hippocampus has been referred to as the gateway to the limbic system (Winson, 1985). Like the amygdala, it is composed of two hippocampal structures, located in each of the hemispheres, adjacent to the respective amygdalas (Brodal, 1980, 1992). It is here that information from the neocortex is processed and

transmitted to the limbic system, where memory and emotion are integrated (Reiser, 1994). Accordingly, it appears to process memory in terms of perceptual patterns and contexts (LeDoux, 1992; van der Kolk, 1994). It is the hippocampus that recognizes the different significance of a bear in the zoo versus one in your backyard (Goleman, 1995). It also differentiates the significance of events that happened long ago from those that are recent (Reiser, 1994; Winson, 1985). Van der Kolk (1994) notes that when people are under severe stress, they secrete endogenous stress hormones that affect the strength of memory consolidation. He posits that “massive secretion of neurohormones at the time of the trauma plays a role in the long-term potentiation (and thus the overconsolidation) of traumatic memories” (p. 259). He cites LeDoux's (1986, 1992, 1994) work in noting that this phenomenon is largely mediated by the input of norepinephrine to the amygdala. This excessive stimulation of the amygdala interferes with hippocampal functioning, inhibiting cognitive evaluation of experience and semantic representation. Memories are then stored in sensorimotor modalities, somatic sensations, and visual images (van der Kolk & van der Hart, 1989).

In addition, the hippocampal neuronal network mediates the creation of a set of neurological “pointers”—or links—to the information mediated by the other neural systems when that information is needed for memorial recall. (McClelland, McNaughton, & O'Reilly, 1995). This will be illustrated in detail below, as will the relationship of this function to thalamic binding.

### The Cerebellum

Located behind the upper brain stem, within the enlarged, multifolded cerebellum, the number of nerve cells apparently exceeds the population in the cerebral cortex (Noback & Demarest, 1981; Shepherd, 1983; Zagon, McLaughlin, & Smith, 1977), making it the largest structure in the human brain (Williams & Herup, 1998). The cerebellum and its tremendous number of neurons is coupled with a high input-to-output axon ratio of 40:1 (Carpenter, 1991) and is reciprocally interconnected to parts of the brain stem, limbic areas, cerebral cortex, and the frontal lobes (Brodal, 1980; Larsell & Jansen, 1972; Llinas & Sotelo, 1992). Such a ratio of afferentation (input) to efferentation (output), combined with its comprehensive interconnectivity, suggests information processing, integration (Courchesne & Allen, 1997), and its consideration as an additional association area in the brain (Leiner, Leiner, & Dow, 1986, 1991).

Accordingly, evidence has emerged that the lateral cerebellum is strikingly activated when an individual performs information processing (Courchesne & Allen, 1997), semantic association (Peterson, Fox, Posner, Mintun, & Raichle, 1989), semantic memory (Andreasen et al., 1995), working memory (Awh, Smith, & Jonides, 1995), and declarative and episodic memory tasks (Andreasen et al., 1995). Based on its physioanatomical position, allowing it to affect known attentional systems, it has been shown that the cerebellum contributes to attention operations by allowing attention to be shifted rapidly, accurately, smoothly, and effortlessly (Akshoomoff & Courchesne, 1992, 1994; Courchesne et al., 1994; Courchesne & Allen, 1997). The importance of this will be illustrated below with respect to the relationship between attention and thalamic activation and function.

## The Thalamus

The thalamus has been described as the gateway to the cerebral cortex and, thus, to consciousness. Like almost all the major brain structures, the thalamus is bilateral. The two almond-shaped thalami are comprised of about 50 groupings of nerve cells, neural tissue, and fibers called nuclei. With the exception of olfaction (smell), which is projected first to the amygdala, all external sensory input is projected first to the thalamus. In addition, the thalamus is reciprocally interconnected with the prefrontal cortex, the basal ganglia, the somatosensory cortex, the association areas, the auditory cortex, visual cortex, motor cortex, cerebellum, brain stem, and limbic structures. The thalamus is, therefore, a relay center for top-down and bottom-up information processing. Its binding functions are activated by attentional aspects of arousal, alertness, or interest. As noted above and as will be discussed in detail below, its ability to synchronize the various neural assemblies throughout the brain, each oscillating at its own signature frequency, into infinite coherent combinations of functional networks may render it the cornerstone of perceptual, cognitive, memorial, and somatosensory integration.

## Neuronal Oscillation, Coherence, and Temporal Binding

To understand thalamic, frontal lobe, and hemispheric laterality malfunctions in PTSD, as well as their relationship to the functioning of the thalamus, their functional characteristics need to be explored in relation to neural oscillation, neural coherence, and temporal binding.

## Neuronal Oscillation

Oscillation, in general, is a rhythmic back-and-forth fluctuation within any aspect of natural phenomena. Neurons in the nervous system are endowed with particular types of intrinsic electrical activity that imbue them with particular functional properties (Llinas, 2001). Because neurons are similar to batteries, such electrical activity is manifested by variations in the minute voltage across the neuron's membrane (Llinas, 1988). These oscillations in voltage remain in the local vicinity of the neuron's body and dendrites and are measured in hertz (Hz.). Depending on the type of neuron, these frequencies range from less than 1 Hz to more than 40 Hz. Llinas (2001) notes that, on these voltage ripples—and in particular on their crests—much larger electrical events known as action potentials are evoked. These electrical firings are the powerful and far-reaching electrical signals that form the basis for neuron-to-neuron communication. What will become obvious as this exploration continues, is that oscillatory electrical activity, in all of its complex permutations, is not only paramount in neuron-to-neuron communication and whole network functioning, but is the electrical glue that allows the brain to organize itself functionally.

## Coherence and Rhythmicity

Neurons that display rhythmic oscillatory behavior entrain to each other via their action potentials (Llinas, 2001), becoming distinct, functionally coherent assemblies (Singer, 1993, 2001). The resulting, far-reaching consequences of this function is that of neuronal groups that oscillate in phase, thereby creating aspects (representations) of human perception and functioning as a result of their synchronized oscillations (Braitenberg, 1978; Edelman, 1987; Palm, 1990; Singer et al., 1997). For example, cardiac neurons synchronize their oscillations, becoming a local network of neurons that cause the heart to contract (Llinas, 2001). Hippocampal neurons synchronize their oscillations to perform their part in memorial functions (Montgomery & Buzaki, 2007). The same holds true for somatosensory (Murthy & Fetz, 1996; Steriade, Amzica, & Contreras, 1996), visual (Gray, 1994), motor (Kristeva-Feige, Feige, Makeig, Ross, & Elbert, 1993), auditory (DeCharms & Merzenich, 1996; Eggermont, 1992; Joliot, Ribary, & Llinas, 1994), amygdaloid (Collins, Pelletier, & Pare, 2001), cortical (Vaadia et al., 1995), and association (Jefferys, Traub, & Whittington, 1996) neuronal ensembles, as they synchronize their oscillations, respectively,

creating their specialized/function-specific fragments of consciousness.

As noted previously, these oscillations occur at different frequency ranges in different brain areas and have been related to particular behaviors (Basar, Basar-Eroglu, Karakas, & Schurmann, 2000; Fernandez et al., 1998). In the parietal and temporal cortices, neurons tend to comprise the following two networks: one network tends to oscillate in the delta range (0.1–3 Hz) and is related functionally to deep sleep and aspects of immune functioning; the other, oscillating in the theta range (3–8 Hz), is related functionally to aspects of REM sleep and mediation of memory functions (Basar, Basar-Eroglu, Karakas, & Schurmann, 2001; Klimesch, 1999). In the occipital cortex, neurons tend to oscillate in the alpha range (8–12 Hz) and are related functionally to aspects of visual processing (Klimesch, 1999). In the parietal and frontal cortices, neurons tend to comprise the following three networks (Hari, Salmelin, Makela, Salenius, & Helle, 1997): one network tends to oscillate at the low beta range (12–15 Hz) and is related to aspects of focus and attention; the second network oscillates in the mid-beta range (15–18 Hz) and is related functionally to mental ability, association, focus, and alertness; the third network, oscillates in the high beta range (above 18 Hz) and is related to full functional alertness. Throughout the brain, interneurons (neither sensory nor motor) oscillate in the gamma range (31–100 Hz). Predominantly, though, these neurons tend to oscillate at 40 Hz and mediate the synchronization, resonance, and binding of the various neuronal networks noted above (Collins et al., 2001; Jefferys et al., 1996; Joliot et al., 1994; Montgomery & Buzaki, 2007; Sederberg et al., 2007; Steriade et al., 1996; Vaadia et al., 1995).

## Neuronal Oscillation and Perception

Information from the external world passes first through specific unimodal sensory cortices that create separate internal representations of the observed stimulus in each sensory modality. Visual, auditory, tactile, and olfactory inputs are each processed by their respective regions of unimodal sensory cortex (Lyons, Sanabria, Vatakis, & Spence, 2006; Macaluso & Driver, 2005; Martin, 1991; Squire & Kandel, 1999). As discussed in detail below, these sensory representations are, then, combined as a result of the mediation of the thalamus and the thalamo-cortical neuronal network circuitry (Llinas, Grace, & Yarom, 1991; Llinas & Ribary, 2001; Singer, 1993; Steriade, Jones, & Llinas, 1990). By this time, some conscious but incomplete perception or trace of the sensations has

occurred (Schacter, Chiu, & Ochsner, 1993). As this information flows through the perceptual representation system, a memory of the sensation is formed within it (Squire & Kandel, 1999). From the sensory cortices, information flows into other neural networks. One set of pathways carries information to regions of the association cortex, where multimodal representations are constructed (Macaluso & Driver, 2005; Martin, 1991; Squire & Kandel, 1999). For visual perception, information is transmitted to the temporal lobe, where the object is identified and, through the mediation of language areas, is named (Ishai, Ungerleider, Martin, Schouten, & Haxby, 1999).

In order for stable memories to form, the neuronal network of the hippocampal complex is required. Simultaneously, information from both perceptual and semantic representational networks flow into the hippocampal complex. The hippocampal network mediates two major functions (McClelland, et al., 1995). Initially, while the memory traces formed in the perceptual and semantic memory networks in the cortex are too weak to support direct recall, the hippocampal neuronal network mediates the formation of a stable memory, combined with the associated effect mediated by the amygdaloid neuronal network, resulting in the capacity of long-term recall. However, memories are not stored in the hippocampus. For that matter, there appear to be no memory centers, or memory neurons, where complete memories are stored (Squire & Kandel, 1999). Accordingly, the hippocampal neuronal network mediates the creation of a set of neurological pointers or links to the information mediated by the other neural systems (McClelland et al., 1995). These pointers include links to all sensory modalities that were activated during the event, any semantic memories initially activated by the sensory input, material processed by the association area networks, and the emotional responses to it, mediated by the neuronal activation of the amygdaloid neuronal network (McClelland et al., 1995; Nadel & Moscovitch, 1998; Squire & Kandel, 1999; Stickgold, 2002). The sum total of synchronized activations of neuronal networks that were required to encode/create a unified perception is referred to as an *engram* (Squire & Kandel, 1999).

## Neuronal Oscillation and Memory

In a remarkable insight, Ramon y Cajal (1899) formulated the Synaptic Plasticity Hypothesis, which articulated the following: the strength of synaptic connections is not fixed, but plastic and modifiable; changes in synaptic strength can be modified by neural activity; learning produces prolonged changes

in the strength of synaptic connections by causing a growth of new synaptic processes; the persistence of these synaptic anatomical changes can serve as the mechanism for memory; neurons should be able to modulate their ability to communicate with one another; and the persistence of these alterations in basic synaptic communication, a functional property called synaptic plasticity, might provide the elementary mechanisms for memory storage. The testing and proof of these hypotheses would wait 75 years.

Equally insightful, Hebb's (1949) formulations foreshadowed current state-of-the-art conceptualizations and understanding of cognition, memory, and consciousness in general. His conceptualizations of the "Hebb-synapse" articulated that conditioned learning and memory were the result of changes in synaptic strength. His articulation of cell assembly described organization among assemblies of cells, mediated by their intrinsic properties and hypersynchrony of neural firing. Regarding memory, Hebb postulated that when an assembly was firing as a result of sensory input, its activity represented the perception of the stimulus. When it fired in the absence of the corresponding sensory input, the activity represented the concept of the stimulus. As will be evident below, the similarity between this conceptualization and our current understanding of the relationship between perceptual encoding and memory recall is remarkable. He also noted that "any two cells or systems of cells that are repeatedly active, at the same time, will tend to become 'associated' so that activity in one facilitates activity in the other" (p. 70)—foreshadowing our understanding of neuronal assembly dynamics, state-dependent memory, and memory triggers.

Studies on habituation (Bailey & Chen, 1983; Thompson & Spencer, 1966; Tigh & Leighton, 1976), the remembered unlearning of behavior, have shown decreases in synaptic strength in networks of interneurons when comparing postlearning to prelearning. Studies on sensitization and classical conditioning (Bailey & Chen, 1983; Hawkins, Abrams, Carew, & Kandel, 1983; Kandel, Brunelli, Byrne, & Castellucci, 1976), the remembered learning of behavior, have shown increases in synaptic strength in networks of interneurons when comparing postlearning to prelearning. Taken together, these studies illustrate that, at different times, the same set of synaptic connections can be modulated in opposite directions by different forms of learning, and, as a result, the same set of connections can participate in mediating different memories. Synapses that increase in strength serve as a memory mediation for certain kinds of learning—for example, sensitization and classical conditioning.

Synapses that decrease in strength serve as a mediation process for other kinds of learning—for example, habituation (unlearning).

Therefore, memories consist of and are stored as changes in synaptic strength within assemblies of neurons (Squire & Kandel, 1999). As noted above, there is no separate memory center where memories are permanently stored. A rather a long line of evidence (Herrmann, Munk, & Engel, 2004; Jokisch & Jensen, 2007; McClelland et al., 1995; Montgomery & Buszaki, 2007; Mulligan & Lozito, 2006; Nadel & Moscovitch, 1998; Osipova et al., 2006; Prince, Daselaar, & Cabeza, 2005; Sederberg et al., 2007; Slotnick, 2004) indicates that memories are stored in the same distributed assemblies of brain structures that were engaged in initially perceiving and processing what is yet to be remembered. Therefore, the brain regions in the cortex that are involved in the perceiving and processing of color, size, shape, and the various other object attributes are close to, if not identical to, the brain regions important for remembering. Ostensibly, remembering is the reactivation of the majority of the components (synchronized neuronal assemblies) of the engram that was used to encode the experience that one is trying to remember (Squire & Kandel, 1999). What is required to facilitate the reactivation of these engrams (i.e., to remember something) is the activations of the hippocampus and other areas of the parahippocampal region. Therefore, the hippocampal formation serves an integrative function (McClelland, 1994, 1996; Squire & Kandel, 1999), linking together the various neuronal assemblies that were established independently in several cortical regions throughout the brain, so that ultimately these assemblies are, again, activated as a synchronized network (Montgomery & Buzaki, 2007; Prince et al., 2005). It is the thalamus and the 40-Hz gamma-band activity (GBA), however, that is required to bind, in real time, the various neuronal assemblies mentioned above, each oscillating at its own respective frequency (Llinas, 2001; Llinas & Ribary, 2001; Singer, 1993, 2001).

### **Thalamo-Cortical Temporal Binding—The Basic Circuitry of Consciousness**

Given that sensory inputs generate a fractured representation of conscious experience, the issue of perceptual unity concerns the mechanisms that allow these different sensory components to be gathered into one global image or experience. In recent years, this has been described as binding—a process that is implemented by temporal conjunction wherein

simultaneous, real-time synchronization takes place (Bienenstock & von der Malsburg, 1986; Gray & Singer, 1989; Llinas, 1988; Llinas & Ribary, 2001; Singer, 1993). This dynamic interaction of neuronal assemblies based on temporal coherence appears to generate flexibly dissipative functional structures capable of as rapid change as the perceptions they generate. These give the brain the ability to constantly, and flexibly, remap and reconfigure its neural assemblies as needed. Therefore, the principle assumption in this discussion is that the intrinsic electrical properties of neurons, and the dynamic events resulting from their connectivity, result in global resonant states that we experience as integrated perception, cognition, and memory.

### Introspection and Reality Emulation

It has been suggested that the brain is essentially a closed system (Llinas, 2001; Llinas & Pare, 1996; Llinas & Ribary, 2001) capable of self-generated activity, based on the intrinsic electrical properties of the component neurons and their connectivity. In such a view, the central nervous system (CNS) is a “reality emulating system” (Llinas & Pare, 1996; Llinas & Ribary, 2001), and the parameters of this inner reality are delineated by the outer senses. The hypothesis that the brain is a closed system follows from the observation that the thalamic input from the cortex is markedly larger than that from the peripheral sensory system. In other words, the majority of cells in the CNS are thalamo-cortical interneurons (neither sensory nor motor), which do not communicate with the outside world. These studies indicate that neurons with intrinsic oscillatory capabilities that reside in this complex synaptic network, allow the brain to self-generate dynamic oscillatory states, which shape the functional events elicited by sensory stimuli, suggesting that this thalamo-cortical activity may be the basis for consciousness (Llinas, 2001; Llinas & Pare, 1991; Llinas & Ribary, 2001).

### 40-Hz Gamma-Band Activity (GBA)

Recent research in neuroscience utilizing magnetoencephalography indicates that 40-Hz oscillatory activity generated by thalamic and cortical interneurons is prevalent throughout the activated mammalian CNS, displaying a high degree of spatial organization (Collins et al., 2001; Jefferys et al., 1996; Joliot et al., 1994; Montgomery & Buzaki, 2007; Steriade et al., 1996; Vaadia et al., 1995). Therefore, interneurons at various cortical levels, and particularly those of the reticular thalamic nuclei, would be responsible for the

synchronization of gamma oscillations in distant thalamic and cortical sites.

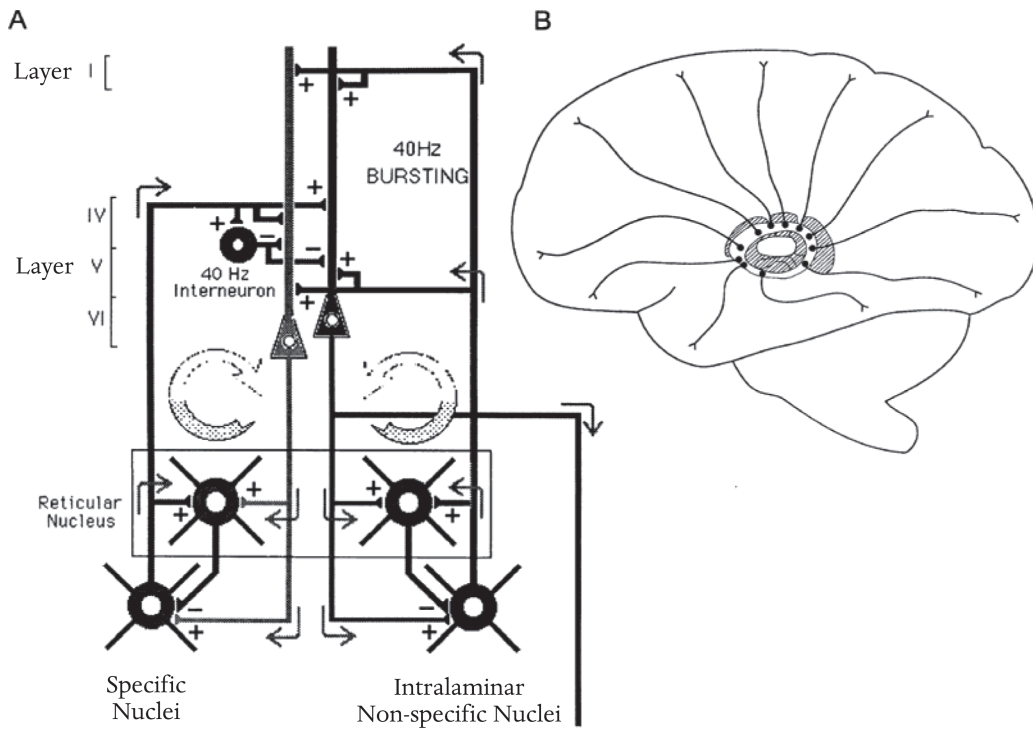
It has been suggested that this 40-Hz activity reflects the resonant properties of the thalamo-cortical system, which is itself endowed with intrinsic 40-Hz oscillatory activity. Consequently, the 40-Hz gamma-band oscillatory activity serves to mediate global temporal mapping by scanning for, targeting, and synchronizing the activity of the various neuronal assemblies, each oscillating at its own respective frequency, creating network resonance and binding them into a coherent and integrated outer and/or inner perceptual consciousness (Llinas, Leznick, & Urbano, 2003; Steriade, CurroDossi, & Contreras, 1993).

### Thalamo-Cortical Temporal Binding of Specific and Nonspecific Thalamic Nuclei

A schematic of the thalamo-cortical circuitry that appears to subserve temporal binding is presented in Figure 1.

In response to inner and outer attention, arousal, or perception, gamma-band oscillations (in the circuitry on the left side of Figure 1) in neurons in the specific, ventrolateral thalamic nuclei project to and establish cortical resonance and direct activation of 40-Hz interneurons in the reticular thalamic nucleus and in layer IV of the cortex. By projecting to cortical layer IV, the specific ventrolateral thalamo-cortical system encodes the sensory and motor activity that impact on the brain. The specific system is seen to comprise those nuclei, whether sensory or motor, that exist mainly, if not exclusively, in layer IV of the cortex. These specific ventrolateral oscillations reenter the thalamus via layer VI pyramidal cell collaterals (in the thalamus), producing thalamic feedback to the reticular thalamic nucleus and ventrolateral nucleus (Llinas et al., 1991; Llinas & Ribary, 2001; Singer, 1993; Steriade et al., 1990).

In the circuitry (shown on the right side of Figure 1), gamma-band oscillations in neurons of the nonspecific, central-lateral thalamic nuclei project to and establish cortical resonance and direct activation of 40-Hz interneurons in the reticular thalamic nucleus and in cortical layers VI and I. By projecting to cortical layers VI and I, the nonspecific central-lateral thalamo-cortical system scans for and encodes interoceptive aspects (memorial, attentional, emotional, and other inner subjective phenomena) within the CNS. These nonspecific, central-lateral oscillations reenter the thalamus via layer V or VI to the reticular thalamic nucleus and to the central lateral nucleus. The combined return of gamma-band



**FIGURE 1.** Thalamocortical circuits proposed to serve temporal binding. (A) Diagram of two thalamocortical systems. (Left) Specific sensory or motor nuclei project to layer IV of the cortex, producing cortical oscillation by direct activation and feed-forward inhibition via 40-Hz inhibitory interneurons. Collaterals of these projections produce thalamic feedback inhibition via the nucleus reticularis. The return pathway (circular arrow on the right) reenters this oscillation to specific and reticularis thalamic nuclei via layer V pyramidal cells. (Right) Second loop shows nonspecific intralaminary nuclei projecting to the most superficial layer of the cortex and giving collaterals to the reticular nucleus. Layer of the specific and nonspecific loops is proposed to generate temporal binding. (B) Diagram of the intralaminar nuclear complex, seen as a circular neuronal mass (stippled shading). Other nuclei in the thalamus are shown as hatched shading. The intralaminar nuclear complex projects throughout neocortical layer I.

*Note.* Adapted from Llinas, R., & Ribary, U. (1993). Coherent 40 Hz oscillation characterizes dream state in humans. *Proceedings of the National Academy of Sciences, USA, 90*, 2078–2081. Copyright 1993 National Academy of Sciences, USA.

oscillations (from both circuits) into the reticular nuclei produces a concurrent summation (a binding) of the specific and nonspecific encoded information.

These studies conclude that the summation of these two overarching and global thalamo-cortical circuits produces the temporal binding that is required to generate coherent cognitive, perceptual, emotional, somatosensory, memorial, and synchronized hemispheric functioning. Therefore, the specific thalamocortical circuitry provides the content that relates to the external world, while the nonspecific thalamocortical circuitry provides the context (interoceptive subjective alertness) of any given moment of consciousness. Llinas et al. (2003) note that, “In this fashion, the specific and nonspecific oscillatory inputs, by synchronously summing the distal and proximal activity . . . along the full extent of the cortex, provide a mechanism for temporal global binding” (p. 454).

## The Cerebellum and the Thalamus

The multifolded cerebellum is reciprocally connected with to-and-fro projections to the brain stem, frontal lobes, thalamus, hypothalamus, and limbic area (Leiner, Leiner, & Dow, 1991; Schmahmann & Pandya, 1997). Two direct routes from the cerebellum to the cerebral cortex have been traced anatomically. One route connects the cerebellum to the thalamus and, thence, to the prefrontal cortex. The other route connects the cerebellum to the hypothalamus and, thence, to the older, limbic structures of the brain (Heal & Matthews, 2002; Leiner et al., 1991; Middleton & Strick, 1997).

It is generally agreed that each of the two output dentate nuclei of the cerebellum sends its fibers to and activates two main regions of the thalamus: the ventrolateral and central-lateral nuclei (Chan-Palay,

1977; Leiner et al., 1991; Steriade et al., 1990). These two thalamic nuclei differ in their projections to the cerebral cortex. The central-lateral nucleus, which is an intralaminar nucleus, has a more diffuse projection and may provide a substrate through which the cerebellum can contribute to cortical arousal, alertness, attention, and other interoceptive aspects of consciousness (Leiner et al., 1991). In contrast, the thalamic ventrolateral nucleus provides a more specific route for conveying information to and activating particular columns of the cerebral cortex; most specifically, the frontal lobes (Heal & Matthews, 2002; Leiner et al., 1991; Middleton & Strick, 1997; Steriade et al., 1990). As noted above, the complementary activity of both thalamic nuclei appears to provide the platform for temporal binding in the brain.

The cerebellum receives input and is activated from virtually every sensory system (Parsons et al., 1997; Welker, 1987), including vestibular, proprioceptive, visual, auditory, tactile and somatosensory (Donga & Dessen, 1993; Naito, Lee, Beykirch, & Honrubia, 1995; Welker, 1987).

## Thalamic Neuroimaging

### Thalamic Function and Levels of Arousal

As noted above, the binding functions of the thalamus are activated by aspects of attention and arousal. Arousal and attention rely on distinct anatomical systems (Luria, 1973); the system for arousal is mainly subcortical, and the one for attention is mainly cortical. However, the two systems share an important anatomical substrate represented by the thalamus. Portas et al. (1998) utilized functional magnetic resonance imaging to examine the relationship between the thalamus, arousal, and attention. The results showed that thalamic activity (particularly the ventrolateral nucleus) was least activated (most down-regulated) when attentional performance was required in high states of arousal. Therefore, high arousal levels, though markedly below the levels of trauma, have been shown to reduce thalamic functioning.

### Thalamic Function and PTSD

Lanius, Williamson, and Densmore (2001) and Lanius, Williamson, and Hopper (2003) identified reduced thalamic activation in patients who suffer from PTSD, replicating similar findings by Bremner, Staib, and Kaloupek (1999) and Liberzon, Taylor, and Amdur (1999). Given the thalamus's pivotal role in binding, the consequences of this lowered

thalamic activation are impairments in the functional connectivity of various dynamic neuronal networks, evidenced by failures of the following:

1. Somatosensory integration, manifested by fragmentation with respect to olfactory memories, auditory memories, oral (taste) memories, visual flashbacks, and disturbing kinesthetic (bodily) sensations.
2. Cognitive integration, manifested by distorted self-blame and shame.
3. Memory integration, manifested by overconsolidated episodic memory, coupled with impaired semantic memory.
4. Hemispheric dynamic integration, manifested by marked hemispheric laterality and its concomitant kindled and hyperemotional state of the nervous system.

## Laterality Neuroimaging and Electroencephalogram Studies

Hemispheric laterality studies have consistently shown (in patients with PTSD as contrasted with controls) marked increased activation in the right hemisphere as compared to the left hemisphere (Lanius et al., 2004; Pagani et al., 2005; Schiffer, Teicher, & Papanicolaou, 1997; Teicher et al., 1997).

Propper, Pierce, Geisler, Christman, and Bellorado (2007) and Propper and Christman (this issue) report on the effects of eye movements on interhemispheric electroencephalogram (EEG) coherence, noting that bilateral eye movements lead to decreased interhemispheric gamma EEG coherence in the anterior prefrontal cortex. With regard to interpreting these results in relation to EMDR, the following should be noted: this was a study on the effects of eye movements on episodic memory and interhemispheric gamma-band coherence in a non-PTSD population; since this was not a study on PTSD, the presenting neurobiology (gamma-band activity, hemispheric coherence) would, presumably, be different; therefore, the examination of the effects of eye movements, when utilized in EMDR in the treatment of PTSD, might very well yield a completely different neurobiological outcome.

There has been a growing consensus (Aboitz, Lopez, & Montiel, 2003; Engel, Konig, Kreiter, & Singer, 1991; Quigley et al., 2003) that interhemispheric coherence (balance) is mediated by the corpus callosum as a result of its ability to facilitate the synchronous oscillation of bilateral neural networks and their dynamic functional connectivity. As noted above, it is the thalamus that is central in the mediation of synchronous oscillations throughout the

brain and must, therefore, be critically involved in the synchronous oscillatory functioning of the corpus callosum.

### **Additional PTSD Imaging Studies**

A number of studies on PTSD imaging that relate to the hypothesis of this article require brief description. The most replicated findings in PTSD include decreased activation (as contrasted to nontraumatized controls) of the dorsolateral prefrontal cortex (BA 9 and 10) (Bremner et al., 1999; Lanius et al., 2001, 2003; Shin, McNally, & Kosslyn, 1999; Shin, Whalen, & Pitman, 2001). These structures are known to serve several key emotion modulating processing functions (Selemon et al., 1995).

The emotional activity that the frontal lobes are required to modulate are proposed to be partly mediated by an overactive amygdala (in PTSD), which has been noted in neuroimaging studies (Liberzon et al., 1999; Rauch et al., 1996; Shin et al., 1999; Shin, Wright, & Cannistraro, 2005). Lowered activation of the left dorsolateral prefrontal cortex in cases of PTSD is consistent with the hyperarousal response that is noted. Studies have found a negative correlation in PTSD between the dorsolateral prefrontal cortex and amygdala activation (Frewen & Lanius, 2006; Gilboa, Shalev, & Laor, 2004; Shin et al., 2005).

### **Neuroimaging Studies of EMDR**

A number of neuroimaging studies of EMDR that relate to the hypothesis of this article require brief description. Neuroimaging studies of successful EMDR treatment have consistently shown increased frontal lobe activation (Lansing, Amen, Hanks, & Rudy, 2006; Levin, Lazrove, & van der Kolk, 1999; Oh & Choi, 2007; Pagani et al., 2007).

### **EMDR and the Thalamus**

As noted previously, the cerebellum receives input and is activated from virtually every sensory system, including vestibular, proprioceptive, visual, auditory, tactile, and somatosensory. Accordingly, it has been suggested that EMDR stimulation (visual auditory and tactile) constitutes a constant and marked stimulation of the cerebellum (Bergmann, 2000).

Output fibers to the hypothalamus and to reticular nuclei in the brain stem allow the cerebellum to transmit information to the limbic structures. Input fibers from the hypothalamus and from the reticular nuclei in the brain stem allow it to receive information from the limbic lobe. The concomitant activation of each

output dentate nucleus of the cerebellum, then, sends its processed information through output fibers, activating two main regions of the thalamus: the ventrolateral and central-lateral thalamic nuclei.

As indicated above, recent high-resolution neuroimaging studies have shown reduced thalamic activation in patients suffering from PTSD. The consequences of this thalamic reduction are impairments in the functional connectivity of various dynamic neuronal networks, evidenced by failures of: somatosensory integration (fragmentation and flashbacks in all sensory modalities); cognitive integration (distorted self-blame); memory integration (overconsolidated episodic memory coupled with impaired semantic memory); and hemispheric dynamic integration (marked hemispheric laterality and its concomitant kindled and hyperemotional state of the nervous system).

Therefore, EMDR's ability to mediate the activation of the specific ventrolateral and nonspecific central-lateral thalamic nuclei allows for the repair of synchronous neuronal oscillation and, therefore, of thalamo-cortical temporal binding. Clinically, this mediates repair in the integration of somatosensory, cognitive, and memorial function.

It was noted above that interhemispheric coherence (balance) appears to be mediated by the corpus callosum as a result of its ability to facilitate the synchronous oscillation of bilateral neural networks and their dynamic functional connectivity. Neurobiological studies of PTSD have consistently shown marked right-sided lateralization. It would seem more than plausible, then, that repair to the thalamus and its central function of synchronous oscillation and binding would mediate the repair of the corpus callosum and of the lateralization.

Additionally, the ventrolateral thalamic nucleus projects to and activates the dorsolateral cortices (the most consistent finding of EMDR neuroimaging), further facilitating the integration of traumatic memories into semantic and other neocortical networks.

### **Suggestions for Future Research**

To examine this model empirically, several lines of research may prove to be productive. High-resolution PTSD neuroimaging—such as the research of Lanius, Williamson, and Densmore (2001) and Lanius, Williamson, and Hopper (2003)—can be utilized to examine thalamic function both prior and subsequent to EMDR treatment. The use of both script-driven symptom provocation and clinically standardized concentration test paradigms, such as Lansing et al.

(2006), would investigate EMDR's effect on thalamic activation. The same techniques and paradigms could be used to examine EMDR's effects on hemispheric lateralization. Qualitative electroencephalography (QEEG) studies, such as Teicher et al. (1997), could be used as well. In addition, replication of previous QEEG EMDR studies (Kowal, 1998; Nicosia, 1994) could further examine and document EMDR's effect on laterality repair.

Magnetoencephalography studies could be used to explore 40-Hz gamma-band activity in PTSD both prior and subsequent to EMDR treatment. Various aspects of thalamic and thalamo-cortical functioning could be examined utilizing symptom provocation, standardized concentration, and cognitive paradigms.

## Conclusion

Previous models of EMDR have posited neural mechanisms of action with respect to reduction in affective disturbances, frontal lobe activation, REM system activation and memorial repair, reciprocal suppression/inhibition of the anterior cingulate, and depotentiation of limbic circuits. This exploration has suggested four additional mechanisms of repair with respect to hemispheric laterality, memorial integration, somatosensory integration, and cognitive integration resulting from synchronization of dynamic neuronal networks. Finally, by illustrating the thalamus and thalamo-cortical temporal binding as the platform on which perceptual, somatosensory, cognitive, and memorial integration take place, an overarching framework of consciousness is offered that can integrate and dovetail with previous and future EMDR models.

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