Neuromechanisms of Action in EMDR Therapy

Amy Serin, PhD
The Serin Center
4/30/2016
The “Controversy” about EMDR Therapy

- The “controversy” about EMDR Therapy’s effectiveness is outdated and ignores 26+ years of research.

- There is controversy about the mechanisms of action,
- Aspirin first used in 1897; the mechanism of action was discovered in 1971.

Guidelines for the Management of Conditions Specifically Related to Stress (2013)

- trauma focused CBT and EMDR Therapy are the **ONLY** recommended PTSD treatments for children, adolescents, and adults.
- Benzos and SSRIs were not recommended and Benzos were **contraindicated**.

“Like CBT with a trauma focus, EMDR therapy aims to reduce subjective distress and strengthen adaptive cognitions related to the traumatic event. Unlike CBT with a trauma focus, **EMDR does not involve:**

- detailed descriptions of the event
- direct challenging of beliefs
- extended exposure, or
- homework.”

http://apps.who.int/iris/bitstream/10665/85119/1/9789241505406_eng.pdf /
Off Label Use

- AETNA: EMDR Therapy “medically necessary” for PTSD
- “Experimental and investigative for everything else”

Off-Label

- Prozac: Generalized Anxiety, Premature Ejaculation, Migraine Headaches
  [link](http://www.aafp.org/afp/2003/0801/p498.html)

- EMDR: Phantom Limb Pain, Addictions, Headaches, Chronic Pain, Generalized Anxiety, Phobias, Paranoia, etc.

- Contraindications: none when EMDR Therapy is conceptualized as the 8-phase process. Pacing phases is appropriate. If eye movements are difficult, other forms of Bi-lateral stimulation/Dual Attention stimulation may be used.
Contraindications

- No unique contraindications when EMDR Therapy is conceptualized as the 8-phase process and delivered with fidelity. Pacing phases is appropriate. If eye movements are difficult, other forms of Bi-lateral stimulation/ Dual Attention stimulation (BLS/DAS) may be used.
We Use EMDR Therapy For

- Post Traumatic Stress Disorder
- Physical/Sexual Abuse
- Phobias/Fears/Panic Attacks
- Anxiety Disorders
- Complicated Grief
- Substance/Behavioral Addictions
- Reactive Behaviors
- Chronic Pain
- Encopresis/Eneuresis
- Attachment Issues
- Eating Disorders
- Performance Anxiety
- Sexual Addictions
- Conduct Disorders
- Disruptive Behavior Disorders
- Acute Stress
- OCD
What is EMDR Therapy

- An 8-phase comprehensive psychotherapy treatment originally designed to alleviate traumatic distress (Shapiro, 1989a, 1989b).

- Shapiro’s (2001) Adaptive Information Processing (AIP) model:
  - EMDR facilitates the accessing and processing of traumatic memories & adverse life experiences resulting in an adaptive resolution.
  - Distress is relieved, negative schemas are changed, and physiological arousal is reduced/eliminated.

- Shapiro (1995, 2001) hypothesis:
  - EMDR facilitates traumatic memory network access
  - information processing is enhanced
  - new associations forged resulting in complete information processing, learning, emotional distress elimination, and cognitive insight development.
Three Pronged Approach

- past events contributing to dysfunction are processed, and new associative links with adaptive information are created

- current distress triggers are targeted, and internal and external triggers are desensitized

- Imagined future events are incorporated to build adaptive functioning skills regarding the situation
Advantages of EMDR Therapy

- Verbalization of details is unnecessary
- No homework
- Spontaneous behavioral change
- Children process incredibly quickly
- Better dropout rate shown in some studies
- Complete resolution of distress is possible
- Faster resolution of panic/anxiety/distress
  - Single incident PTSD = 6 hours of treatment
Phase I: History and Treatment Planning

- Thorough history
- Develop treatment plan
- Defines specific EMDR targets
  - Events from past
  - Present distressing situations
  - Key skills/behaviors for future well-being

Note: Patient DOES NOT have to disclose specifics.
Phase 2: Preparation

- Establish relational trust
- Explain EMDR theory
  - How it is done
  - What person can expect for results
  - Teach calming/relaxing techniques
Phase 3: Assessment

- Access each target in controlled/standardized way for effective processing
- Patient selects a specific picture scene that best represents the memory (Image)
- Chooses an associated negative self-belief (NC)
- Picks a positive self-belief she would rather believe (PC) and rates it on Validity of Cognition (VoC) scale of 1-7
- Identifies feelings and body sensations
- Rates level of disturbance (SUD) 0-10
Assessment Example

- **Target**: A parent told me my kid was a bully and I snapped at him.
- **Image**: Standing there yelling and noticing other people were looking at me
- **Negative Cognition**: “I’m out of control” “I should have handled things differently”
- **Positive Cognition**: “I can learn from this” “I can stay calm in the future”
- **Emotions**: embarrassed, anger towards the parent, shame
- **Body Sensation**: tight chest  
  SUD = 7, VOC = 4* (has had CBT)
Phase 4: Desensitization

- Begin eye-movements and other stimulation to resolve disturbance until SUD = 0.
- “Notice the image, that it’s a 7/10, the anger, embarrassment, tight chest, and the thought “I was out of control and should have done something different”

- Note: if SUD=0 does not occur by the end of the session, the therapist stops, contains the target, and helps patient relax to close session.
Phase 5: Installation

- Increase PCs “I can learn from this” and “I can stay calm” to VOC 7/7
- Note: the therapist cannot get patient to believe something that is untrue or that involves actions of others. So “I am safe” is not valid if someone is in an unsafe environment. And “he won’t bother me again” cannot be a PC either.
Phase 6: Body Scan

- Patient re-imagines the target event and if any body sensations occur, these are desensitized by using eye movements until they remit completely.
Phase 7: Closure

- Occurs after Phase 6 or if the target is unfinished, at the end of the session.
- Closure ensures that the patient leaves feeling some relief and contains unfinished targets so they are less distressing in between sessions.
Phase 8: Re-evaluation

- At the beginning of each session, the therapist ensures that positive results have been maintained, identifies any new targets, and starts a new target if the last one is completed.
- Spontaneous behavioral change is also assessed to help the patient link improvements with the therapeutic process.

(It’s not the essential oils!!!  Sheesh!!!)
CAUTION!! WARNING!! THEORIES ABOUT TO BE PRESENTED!!
Common Questions

- What do the eye-movements do?
- Isn’t EMDR Therapy just CBT with eye movements?
- Can eye-movements just be a sham addition?
- Is it only good for PTSD?
- Isn’t EMDR Therapy the same as desensitization?
- Is the “resolution” different than in other therapies?
- Is there something special about EMDR Therapy that “unblocks” one’s ability to heal?
- How do you explain the euphoria when a target is complete?
Research/Current Theories

- Working Memory (WM)
- Interhemispheric Connectivity
- Orienting Response
- Desensitization/Inhibition
- Eye-Movements Mimic REM
- "Investigatory Reflex"
- It’s All A Bunch of Hooey
Limitations of Current Theories

- Theories stymied by the localization approach
- Rich clubs, DTI, nodes and networks are not considered in terms of how they interact with each other
- Research is flawed (of course)
- The theories aren’t mutually exclusive but are fragmented conceptually
- EMDR community clumps BLS/DAS into same categories at times
- Focus is on trauma and memories, not distress and the entire system
- Comparing EMDR Therapy to other approaches can leave out critical differences depending on research design
If Psychologists Studied Cars

- Car A vs. Car B both got participants from point A to point B x % of the time.

- Therefore, there is no difference.
Car A = $3k  Car B = $1k
Eye Movements

- Eloffsson et al. 2008 found heart rate, skin conductance, LF/HF ratio, finger temperature, breathing frequency, carbon dioxide, and oxygen level changes during EMDR eye movements.

- Conclusion: EMDR eye movements inhibits sympathetic symptoms that is similar to pattern of reactivity in REM sleep.
Eye Movements

- Kapoula et al. (2010) found EMDR during moderately distressing worries increased the “smoothness” of ocular pursuit which may have reflected an improvement in the use of visual attention needed to follow the target accurately.

- Conclusion: perhaps EMDR reduces distress and activates a cholinergic effect known to improve ocular pursuit
Eye Movements

- Hornsveld et al. (2010) participants reported less negative emotionality and concentration after eye movements vs. recall only and recall with music playing.

- Conclusion: eye movements are effective with loss/grief memories.

- Does this finding contradict the WM hypothesis?
REM

• Stickgold (2002) Posits that repetitive attentional redirecting induces a neurobiologic state like REM that supports cortical integration of traumatic memories, reduces the strength and the associated negative affect.
Investigatory Reflex

http://www.emdria.org/?page=Mechanism

- Barrowcliff et.al (2004) “investigatory reflex” results in relaxation response due to reciprocal inhibition when a threat is not detected and processed as a threat over time.
  - Neurobiological (Bergmann, 2008, Stickgold, 2002)
  - Behavioral (Armstrong & Vaughan, 1996)
Orienting Response

Maybe the “orienting response” disrupts the traumatic memory network and interrupts previous associations with negative emotions and new information can be integrated into cortical semantic memory (Stickgold, 2002)
**Thalamus/Information Processing**

- Sensory stimulation may repair thalamic and thalamo-cortical function and facilitates the repair of maladaptive neural information linkages for information processing (Bergman, 2008).
Blocks To Information Processing

- Richardson et al. (2009) fMRI study. Concluded: ventromedial activation, intensified by ABS can overcome the block to information processing that prevents natural healing from occurring spontaneously in the female patient with PTSD.

- Other research suggests that the ventromedial activation triggers dopamine release which can explain the “high” achieved when a target is processed.

- Note: auditory bi-lateral stimulation was used in this study.
Support For Current Theories

- Varies and some conclusions may be a stretch
- Studies have shown decreased image vividness and associated emotionality (Andrade et al., 1997) along with changing the somatic perceptions accompanying retrieval that leads to decreased emotionality. (van den Hout et al., 2013)

- For more, visit http://www.emdria.org/?page=EMDRResearch
Deeper Down The Rabbit Hole
Amygdala Inhibitory Circuits and the Control of Fear Memory

Ingrid Bohn1,2, *, Yann Humeau2, François Grousset3, Stéphane Giochi4, Cyril Henry5,5,6 and Andreas Lüth1,5,6
1Division Masliah Institute for Behavioral Research, 4098 Baselland, Switzerland
2Institut des Neurosciences Cognitives et Intégratives, Université Louis Pasteur and CNRS, UMR 7185, F-67084 Strasbourg, France
3Present address: INSERM U 865, Neurocentre Magnétique, 146 Rue Léo-Saignet, 33077 Bordeaux, France
4Present address: Max Planck Institute for Clinical Brain Research, 72076 Tübingen, Germany
5Correspondence: andreas.luth@insERM.fr
DOI: 10.1016/neuron.2009.05.020

Classical fear conditioning is a powerful behavioral paradigm that is widely used to study the neural substrates of learning and memory. Previous studies have clearly identified the amygdala as a key brain structure for acquisition and storage of fear memory traces. Whereas the majority of this work has focused on principal cells and glutamatergic transmission and its plasticity, recent studies have started to shed light on the intricate roles of local inhibitory circuits. Here, we review current understanding and emerging concepts of how local inhibitory circuits in the amygdala control the acquisition, expression, and extinction of conditioned fear at different levels.

Introduction

Classical fear conditioning is one of the most powerful models for studying the neural substrates of associative learning and the mechanisms of memory formation in the mammalian brain (Davis, 2000; Fanselow and Poulos, 2005; LeDoux, 2000). In unmasking the substrates of memory storage in fear conditioning and related learning paradigms, the major focus has been the study of excitatory elements of the brain. However, interneurons are critical components of neuronal networks, and inhibition plays an important role in shaping network activity. The idea is that little is known about the involvement and function of inhibitory circuits in learning and memory. This situation is starting to change as recent studies point to key roles of inhibitory interneurons within the amygdala during fear and extinction memory acquisition and expression. Here, we review some of these results and point out how inhibitory circuits contribute to both acquisition and expression of memory traces by multiple mechanisms and at multiple levels in the amygdala.

In classical fear conditioning, the subject is exposed to a neutral conditioned stimulus (CS), such as a tone or a light. As a result of the training, the tone acquires aversive properties, and when subsequently presented alone, will elicit a fear response. In rodents, such responses include freezing behavior, alterations in autonomic nervous system activity, release of stress hormones, analgesia, and facilitation of place aversion. Subsequently, conditioned fear can be suppressed when the conditioned stimulus is repeatedly presented alone, a phenomenon called fear extinction. Behavioral studies in animals have demonstrated that fear extinction is not simply the forgetting of previously learned fear but rather a new, active learning process (Bouton et al., 2006; Myers and Davis, 1997; Rescorla, 2001). Fear extinction is context dependent; that is, fear responses can still be expressed if the CS is presented in a different context than the one in which extinction was acquired. Moreover, fear extinction is generally not permanent, as the original CS-evoked fear behavior can spontaneously recover over time or can be prevented by exposing animals to US presentations alone (Myers and Davis, 2007). Thus, fear and extinction memory traces coexist and can be retrieved depending on the behavioral state of the animal.

The amygdala plays a key role in the neural structure and neural circuitry that are involved in fear memory acquisition and storage, a notion commonly supported by a large number of studies using different experimental paradigms and measures of conditioned fear responses (Davis, 2000; Fanselow and Poulos, 2005; LeDoux, 2000; Marun, 2001). In addition, the amygdala also modulates fear-related learning in other brain structures, such as the cortex and the hippocampus (McGaugh, 2000). The amygdala consists of several anatomically and functionally distinct nuclei, including the lateral (LA) and basolateral (BLA) nuclei, together referred to as the basolateral amygdala (BLA) and the central nucleus (CEA) (Kandel and Schreiber, 1978) (Figure 1A). The CEA can be further divided into a lateral (CEL) and a medial (CEM) part (McDonald, 1993). While the CEA has been subdivided on anatomical and immunohistochemical grounds into a lateral capsular division (CEL), an intermediate division (CEI), and a lateral division proper (CEM) (Casella et al., 1988; Johansen and Pitkänen, 1998; McDonald, 1982), from a functional perspective it is often considered as a whole (e.g., Samson et al., 2005). The cytoarchitecture and organization of the amygdala are similar to that of other parts of the telencephalon. The lateral structures (BRA) are corticopontine, consisting of a majority of glutamatergic projection neurons and a minority of local GABAergic interneurons (McDonald, 1982) (Figure 1B). The medial structures (CEA) are striosome-like, with the vast majority of neurons being GABAergic (Figure 1B) and exhibiting medium spiny-type morphology (Casella et al., 1986; McDonald, 1982; Swanson and Petrovich, 1998). The interneuron projections generally follow a dorsal-ventral and dorsal-medial direction (Imbert and Price, 1978) (e.g., from LA to BLA and from BLA to CEA and, within CEA, from the CEA to the CEM) (Figures 1A and 1C). An interesting addition to the cortex- and striatum-like...
Amygdala Inhibitory Circuits and The Control of Fear Memory

- “Information can be processed both by mechanisms intrinsic to amygdala networks as well as modified by interactions with other brain structures to integrate sensory inputs, generate fear response outputs, and modulate fear responses according to circumstances, such as fear extinction” (Pitkanen et al., 1997; Sah et al, 2003 cited by Erlich, et al. 2009)

- Basolateral (BLA) and central nucleus (CEA) of amygdala can process information in series and in parallel. Inhibitory circuits can be either cortex or striatum like in the amygdala. Maybe this serves to optimize speed, signal to noise and signal processing and the enabling of the integration of excitatory and disinhibitory signals that could act in an “instructive and permissive manner” to set CEA output (p. 766). So the pathways here act as FEAR GENERATORS and FEAR EXTINCTORS.

- “Neuromodulation of inhibitory activity and gating of long term potentiation (LTP) in this pathway are attractive candidate mechanisms in line with the requirement for neuromodulatory input for fear conditioning in vivo at the physiological and behavioral level (Rosenkranz and Grace 2002b, cited in Erlich et. al 2009)
Amygdala Inhibitory Circuits and The Control of Fear Memory

- “Evidence from a multitude of studies using pharmacological manipulations, electrical stimulation, or lesions suggests diminishing CEm output attenuates fear and anxiety responses.” (p. 762) while increasing it leads to stronger fear responses.
- “The CEI may function as an inhibitory interface, gating CEm output by integrating sensory cortical and subcortical inputs.” (p. 762)
  CEI: latero-capsular subdivision of the central amygdala
  CEm: medial subdivision of the central amygdala

Conclusion: “Addressing the functions of local inhibitory circuits may be the key in achieving understanding of amygdala circuit function” (p. 765)
Controlling output appears to be involved in modulating fear expression as well as establishing a new, perhaps competing memory trace following inhibitory learning, such as extinction. (p. 766) This may be achieved by inhibition but also disinhibitory processes.
Emotion Processing and the Amygdala

- The amygdala is a “coordinator” of functional cortical networks that serves to evaluate the biological significance of visual stimuli.
- The amygdala has broad connectivity with the cortex and other subcortical structures.
Let’s Take It a Step Further

• Maybe EMDR Therapy elicits the inhibitory circuits during fear processing to change the amygdala’s response to the activating memory.

• Can’t, then, alternating BLS/DAS theoretically evoke the inhibitory response during everyday events or as a real-time desensitization method to keep the fear response at bay?

• Also can BLS/DAS re-train and strengthen the inhibitory pathways in the same way that prolonged and repetitive exposure to stress increases the excitatory pathways that set off fight-flight-freeze?
The LC

Roles in the Regulation of Arousal and Autonomic Function Part II: Physiological and Pharmacological Manipulations and Pathological Alterations of Locus Coeruleus Activity in Humans

E. R. Samuels and E. Szabadi

Psychopharmacology Section, University of Nottingham, Division of Psychiatry, Queen’s Medical Centre, Nottingham NG7 2UH, UK

Abstract: The locus coeruleus (LC), the major noradrenergic nucleus of the brain, gives rise to fibres innervating most structures of the neuraxis. Recent advances in neuroscience have helped to unravel the neuronal circuitry controlling a number of physiological functions in which the LC plays a central role. Two such functions are the regulation of arousal and autonomic activity, which are inseparably linked largely via the involvement of the LC. Alterations in LC activity due to physiological or pharmacological manipulations or pathological processes can lead to distinct patterns of change in arousal and autonomic function. Physiological manipulations considered here include the presentation of noxious or anxiety-provoking stimuli and extremes in ambient temperature. The modification of LC-controlled functions by drug administration is discussed in detail, including drugs which directly modify the activity of LC neurones (e.g., via autoreceptors, storage, reserupte) or have an indirect effect through modulating excitatory or inhibitory inputs. The early vulnerability of the LC to the ageing process and to neurodegenerative disease (Parkinson’s and Alzheimer’s diseases) is of considerable clinical significance. In general, physiological manipulations and the administration of stimulant drugs, $\alpha$-adrenoceptor antagonists and noradrenaline uptake inhibitors increase LC activity and thus cause heightened arousal and activation of the sympathetic nervous system. In contrast, the administration of sedative drugs, including $\alpha$-adrenoceptor agonists, and pathological changes in LC function in neurodegenerative disorders and ageing reduce LC activity and result in sedation and activation of the parasympathetic nervous system.

Key Words: Locus coeruleus, arousal, autonomic function, noxious stimuli, anxiety, Parkinson’s disease, Alzheimer’s disease, aging.

Abbreviations: 6-OHDA, 6-hydroxydopamine; AD, Alzheimer’s disease; BF, Basal forebrain; CR, Caudal raphe; CS, Conditioned stimulus; DMV, Dorsal motor nucleus of the vagus; DR, Dorsal raphe; EDS, Excessive daytime sleepiness; EEG, Electroencephalogram; EMG, Electromyogram; FWN, Edinger-Westphal nucleus; fMRI, Functional magnetic resonance imaging; GABA, Gamma-aminobutyric acid; LC, Locus coeruleus; LDT, Laterodorsal tegmental nucleus; MPTP, 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine; PD, Parkinson’s disease; PPT, Pedunculopontine tegmental nucleus; PST, Papilliform sleepiness test; PVN, Paraventricular nucleus; REM, Rapid eye movements; RVL, Stellarvemilateral medulla; SWS, Slow wave sleep, US, Unconditioned stimulus; VLPo, Ventrolateral preoptic area; VTA, Ventral tegmental area.

1. BRIEF OUTLINE OF THE ROLE OF THE LOCUS COERULEUS IN THE MAINTENANCE OF AROUSAL AND REGULATION OF AUTONOMIC FUNCTIONS

The LC, the largest group of noradrenergic neurones in the central nervous system, is a major nucleus involved in the neural pathways controlling arousal and autonomic function. These physiological functions are inseparably linked, largely due to the central role of the LC in controlling these functions. The LC projects extensively to widespread areas of the neuraxis (see Part I) and these projections can result in both excitatory effects via the activation of $\alpha$-adrenoceptors and inhibitory effects via the stimulation of $\alpha$-adrenoceptors [206]. Therefore, complex changes in the neural circuitry

1.1. Arousal

The LC is recognised as a major wakefulness-promoting nucleus [204, 205], where the activity of the LC closely correlates with level of arousal [16, 17, 18, 152, 153, 355, 360]. This wakefulness-promoting action results from the dense projections from the LC to most areas of the cerebral cortex [208] and from the multitude of projections from the LC to alertness-modulating nuclei (see Part I). The LC exerts an excitatory influence on wakefulness-promoting neurones such as cholinergic neurones of the BF [111, 156, 203, 265] and of the PPT and LDT nuclei [26], cortically-projecting neurones of the thalamus [280, 281] and serotonergic neurones of the DR [219, 290, 372], and an inhibitory influence on sleep-promoting GABAergic neurones of the III [268].
Functional Neuroanatomy of the Noradrenergic Locus Coeruleus

- LC regulates arousal and autonomic activity with dense excitatory projections to the majority of the cerebral cortex, cholinergic neurons of the basal forebrain, cortically-projecting neurons of the thalamus, serotonergic neurons of the dorsal raphe, and cholinergic neurons of the pedunculopontine and laterodorsal tegmental nucleus.
- LC has substantial inhibitory projections to sleep-promoting GABAergic neurons of basal forebrain and ventrolateral preoptic area.
- LC can enhance alertness.
- LC can control autonomic functions from direct spinal cord projections and autonomic nuclei (including the amygdala).
- LC can increase sympathetic activity, decrease parasympathetic activity via those projections.
Functional Neuroanatomy of the Noradrenergic Locus Coeruleus

- LC is the **sole source** of cortical Noradrenaline.
- Increases in LC activity increase EEG signs of cortical arousal (see p. 236).
- **Electrical stim of LC in a human = reduction of slow wave and rapid eye movement sleep** (Kaitin et al. 1986 cited in Samuels and Szabadi 2008).
- LC “densely innervates” the amygdala, in particular the central and basal nuclei.
- The adrenoreceptors expressed in amygdala are involved in the autonomic response to stressful stimuli. The noradrenergic influence on the amygdala is likely largely excitatory.
Functional Neuroanatomy of the Noradrenergic Locus Coeruleus

- LC projection to amygdala may also play a role in forming and retrieving emotional memories (59, 340)
- “The level of arousal, highly correlated with LC activity, determines the likelihood of a memory being encoded and subsequently retrieved” (p. 236)
- Alpha1 and beta adrenoceptors in the basolateral amygdala have been implicated in memory storage (100, 101).
Functional Neuroanatomy of the Noradrenergic Locus Coeruleus

- LC innervates the hippocampus projections to the hippocampus may contribute to memory formation.
- There may be a negative feedback circuit from LC to lateral hypothalamus/perifornical area (LH/PF) that likely prevents excessive activity in the arousal pathway during wakefulness (p. 237).
- LC has an alerting effect and is COMPLETELY quiescent during REM sleep. It’s activity also seems to vary depending on how calm someone is.
LC + Saccadic Eye Movements

- LC activates with surprising events
- Studied LC activation in monkeys go/no go saccadic eye movements
- LC+ phasic activation encodes sensory and motor events related to decisions to execute, but not withhold, movements, implying a functional role in goal-directed actions, but not necessarily more covert forms of processing (Kalwani et al. 2014)
On the Neural Basis of EMDR Therapy: Insights From qEEG Studies

Melvin L. Harper, Tasha Basolkhani-Kalhorn, and John F. Drozd

Eye movement desensitization and reprocessing (EMDR) therapy has been shown by empirical studies to be effective in relief from psychological trauma including posttraumatic stress disorder (PTSD). Several logical concepts regarding the origin of the EMDR effect have been presented, but no detailed neural explanation is available. This lack of a widely accepted scientific explanation for the EMDR effect has led to skepticism about the therapy by many therapists and potential clients. The authors present evidence based primarily on quantitative electroencephalogram studies that the neural basis for the EMDR effect is depolarization of fear memory synapses in the amygdala during an evoked brain state similar to that of slow wave sleep. These studies suggest that brain stimulation during EMDR significantly increases the power of a naturally occurring low-frequency rhythm in memory areas of the brain, binding these areas together and causing receptors on the synapses of fear memory traces to be disabled. This mechanical change in the memory trace enables it to be incorporated into the normal memory system without the extreme emotions previously associated with it. EMDR is a medical procedure because it changes the physical structure of the brain to modify problematically stored memories.

Keywords: EMDR therapy; memory; PTSD; EEG; neuronal response frequency

Much study has been devoted to determine the neural basis of the positive effects of eye movement desensitization and reprocessing (EMDR) therapy. Bregman (1999) proposed that EMDR therapy with bilateral brain stimulation results in the resetting of pacemaker cells in the septum. These cells are known to pace the firing of principal cells in the greater hippocampal system (the GHFS, consisting especially of the amygdala and the hippocampal proper). Resetting of the pacemaker cells to delta rhythm (from the usual waking state theta rhythm) may increase the synchronization of the hemispheres and improve functional connectivity. Bregman (2000) also discusses evidence that EMDR stimulates the cerebellar processing center, which results in activation of the dorsolateral and orbitofrontal cortices. He believes this leads to further integration of traumatic memory into general semantic networks, as well as other neocortical areas. Corrigan (2002) suggested that the EMDR effect is centered on the anterior cingulate cortex (ACC), and he hypothesized that EMDR promotes the disconnection between the affective and cognitive subdivisions of the ACC. This disconnection would lead to relief from the affective portion of the memory. MacCulloch and Feldman (1996), Martin, Hofmann, Wenzelmann, and Lempa (2008), and Sander and Elefson (2008) describe beneficial changes in psychophysiological...
The neural basis for EMDR Therapy’s effectiveness is “depotentiation of fear memory synapses in the amygdala during an evoked brain state similar to slow wave sleep.”

EMDR may increase the power of low-frequency rhythm in memory while disabling the synapses of fear memory. Memories are then incorporated into the normal memory system.

Negative emotions accompanying a conversation are recorded by both hippocampus and amygdala. During emotional memory recall, only the amygdala activation occurs without the cognitive portion from the hippocampus. “I know logically I’m enough but I FEEL this way when I think about....”
Re-thinking BLS’s role in EMDR

- In rats, somatosensory and auditory stimulation activated the dorsal amygdala
- ONLY somatosensory stimulation activated the ventrolateral amygdala
- Ventromedial amygdala did not respond to either
- The lateral nucleus is essential in auditory fear conditioning

- Conclusion: different stimulation can effect different parts of the amygdala in rats, with tactile stimulation affecting a discrete part that auditory stimulation does not affect
- (Adolfs et al. 2000)
Anecdotal Data on BLS/DAS

- Case studies; 67 year old man with IBS recalled SUD = 3 memory. After 30 seconds of BLS, SUD = 3 but his stomach ache that had preceded memory recall had partially remitted. After 30 additional seconds, stomach ache was completely resolved.
- One patient “this is like taking a Xanax!” can I have one of these?
- N = 3 parents of gifted children aged 3-5 report that their children can sleep through the night if the BLS continue through the sleep cycle.
- N = 7 female patients age 37-49 report SUD decreases by an average of 1.6 after 2-4 minutes of BLS during history recall of traumatic events.
- Patients report that BLS seem to prevent panic or “tone down” the acute effects of stress. However, if they use BLS after panic, their panic continues. This supports the notion that BLS are inhibiting the excitatory response that creates F3 and sympathetic nervous system arousal but does not reverse it once arousal is too high.
Back on Solid Ground
Case Studies

- 30 yr. old prison worker
- ACES score 2; positive childhood; good coping skills; secure attachments
- Injured in prison riot, saw a guard get stabbed to death 2 months prior
- PTSD; hypervigilance, almost quit, sleep problems, panic
- TX: 3, 2.5 hour sessions. Complete resolution of symptoms during last session after 1 hour including new insights and hope about marriage. SBC was remission of all PTSD symptoms, some weight loss, and more connection with husband
You May Ask

- Why did I only schedule 3 sessions?

- Lee et. al 2002, Marcus et al. 1997 and Rothbaum, 1997 all showed ELIMINATION of PTSD diagnosis in 77-90% of patients after 3-7 sessions.

- Prior clinical experience with other patients is in line with the research
Case Studies

- 5 yr. old girl; ACES =0, securely attached
- Separation anxiety/panic/stomach aches every day before school
- Fixated/obsessed about conflict with a friend in class

- After 1st session, separation anxiety/panic/stomach
- 15 minutes into 2nd session, resolved distress over friend. Spent remainder of the time educating mom on general parenting issues.
Case Studies

- 4 year-old with encopresis
- Dr. Choi’s first patient
- Identified the child’s distress: sitting on the toilet
- Followed 8-phase protocol

- Complete resolution in 2 sessions
Case Studies

- 12 yr. old girl refusing to eat after anaphylactic shock; Lost 6 pounds in 10 days
- Taking max Benadryl; daily body sensations of throat closing; panic/fear of death; MD advising $3000 of invasive testing
- 1\textsuperscript{st} session: education re phantom limb pain and started processing
- 2\textsuperscript{nd} session: eating some foods without panic, returned to school for lunch
- 3\textsuperscript{rd} session: new fear- soy allergy after testing confirmed a reaction- new avoidance emerged, after processing she stopped avoiding soy
- By 5\textsuperscript{th} session we had processed earlier traumas, her weight had stabilized; she re-joined cheer squad
The 4-minute mile

- Sir Roger Bannister, a budding English neurologist ran the first recorded mile under 4 minutes in the 1952 Helsinki Olympics.
- The Daily Telegraph quoted it as being “sport’s greatest goal”, and something “as elusive and seemingly unattainable as Everest”.
- In his lifetime, he dismissed the four minutes as no more than an “interlude” that delayed his high-powered career as a neurologist.
- He trained hard but in short bursts “fartlek” (speedplay)

http://www.telegraph.co.uk/sport/othersports/athletics/10803219/I-gave-it-everything-Sir-Roger-Bannister-marks-60-years-since-his-record.html
The 4-minute mile

- Bannister’s achievement was yoked together with Everest, which had been conquered the year before.
- Since then, over 4,100 people have climbed Everest.
- By 2014, 1,305 people had run a sub-4 minute mile.

- In his book, *The Four Minute Mile*, Bannister states: “No one can say, 'You must not run faster than this, or jump higher than that.' The human spirit is indomitable."
A Word About What Is Possible
Miles and Mountains

- What Everests is our field denying are climbable?
- What 4-minute miles are we saying can’t be run?

- The idea that mental health diagnoses are permanent may be damaging. “How do I cope or manage” should not be the main treatment question.
- Our in-group/out-group bias may be holding us back
- “If I didn’t study it, it doesn’t work” is a problem.
- We must balance our skepticism with evidence and be willing to contradict our current beliefs in the future.
Strive For This!

© Copyright 2015 Amy Serin, PhD
www.TheSerinCenter.com
Constantly Seek Improvement
Thank You!

- Questions
Amy Serin, Ph.D.
The Serin Center

*Neuropsychology Ages 3 - Adult
*Psychological Assessment
*EMDR Therapy, CBT
*Children, Adolescents, Adults
*School Consultations
*Forensic Evaluations
*Executive Consulting Services
*Concierge Consulting Services
*19 Channel Neurofeedback

© Copyright 2014 Amy Serin, PhD
www.TheSerinCenter.com
Mark Ruggiero, MD, FAAP
The Serin Center

Developmental Pediatrician

* Medication Management
* Evaluation/Treatment ages 0-18
* Holistic Treatments
* ADHD, Autism, Mood Disorders, Developmental Disorders, Genetic Disorders

© Copyright 2010 Amy Serin, PhD
www.TheSerinCenter.com
Dr. Meena Choi, Ph.D.
The Serin Center

* Child and Family Therapy
* Psychological Assessment
* Cognitive Behavioral Therapy
* Play Therapy, EMDR Therapy
* Parent Training Groups
Jennifer Montgomery, LCSW
The Serin Center

*Child and Family Therapy
*Autism Spectrum Disorders
*Play Therapy
*EMDR Therapy
Jamie Dana, MC, LAC
The Serin Center/Capstone Learning Solutions

* Individual Counseling for Adolescents and Adults
* Psychological Assessment
* EMDR and CBT Therapy
* Phobias, Anxiety, Depression, Gifted, Trauma, Women’s Issues
* Cogmed Working Memory Training

© Copyright 2014 Amy Serin, PhD
www.TheSerinCenter.com
Kim Van Roekel, BA, SpEd.
Director of Academic Rehabilitation

*Specialized Programs for Dyslexia, Dyscalculia, Dysgraphia
*Lindamood-Bell Trained
*Individualized Reading Programs
*IEP and 504 Assistance
*Twice Exceptional Assistance

© Copyright 2014 Amy Serin, PhD
www.TheSerinCenter.com
Other Specialists/Programs

- Summer Reading Intensive: six highly trained master’s level/special education teachers deliver Lindamood-Bell Programs and Handwriting Without Tears.

- We have two 19-channel z-score Neurofeedback technicians using Arizona’s only Cognionics dry-cap technology.
References


References

References


References


PTSD Studies


- Tested the working memory theory. Eye movements were superior to control conditions in reducing image vividness and emotionality.


- Tested the working memory theory. Eye movements were superior to control conditions in reducing within-session image vividness and emotionality. There was no difference one-week post.


• **Van den Hout, M., Muris, P., Salemink, E., & Kindt, M. (2001).** Autobiographical memories become less vivid and emotional after eye movements. *British Journal of Clinical Psychology, 40*, 121-130. - Tested their theory that eye movements change the somatic perceptions accompanying retrieval, leading to decreased affect, and therefore decreasing vividness. Eye movements were superior to control conditions in reducing image vividness. Unlike control conditions, eye movements also decreased emotionality.


