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Melvin Harper¹

Abstract

Animal and human studies have shown that the emotional aspects of fear memories mediated in the lateral nucleus of the amygdala can be extinguished by application of low-frequency tetanic stimulation or by repetitive sensory stimulation, such as tapping the cheek. Sensory input creates a remarkable increase in the power of the low-frequency portion of the electroencephalogram (EEG) spectrum. Glutamate receptors on synapses that mediate a fear memory in attention during exposure therapy are depotentiated by these powerful waves of neuronal firings, resulting in disruption of the memory network. In this study, the role of sensory input used in the principal exposure therapies is examined through analysis of the raw EEG data obtained in clinical and lab tests. Nearly all sensory inputs applied to the upper body result in wave power sufficiently large to quench fear–memory networks regardless of input location and type and whether the sensory input is applied unilaterally or bilaterally. No power advantage is found for application of sensory input at energy meridians or gamut points. The potential for new or extended applications of synaptic depotentiation in amygdalar memory networks is discussed.

Keywords

EEG, PTSD, exposure therapy, brain stimulation, amygdalar evolution, cross-species therapy.

This article is a follow-up to a 2009 report by Harper, Rasolkhani-Kalhorn, and Drozd (2009) on a qEEG study of the reaction of the brain to EMDR therapy, based on a theory proposed by Rasolkhani-Kalhorn and Harper (2006). During the EEG study of EMDR, bilateral sensory input was provided by vibrating pads held in the palms of the hands of the participants. This clinical EEG study was supplemented by laboratory studies, which allowed for more control of input parameters. Following are the main findings:

Similarity of results of animal studies by others of depotentiation of glutamate receptors on fear memory synapses through direct tetanic (electrical) stimulation led us to conclude that EMDR and similar therapies cause a similar depotentiation through the creation of powerful waves of neuronal depolarizations.

Sensory inputs (vibrating pads in palms of hands) greatly magnify the power of the low-frequency portion of the neuronal firing spectrum (delta waves).

Frequency of the powerful waves created in the memory areas of the brain, between 1 and 2 Hz (1 to 2 repetitions per second), remained unchanged through all brain states investigated in the study.

A delta wave power increase of 50% to 100% occurring during exposure to the fear memory without sensory input suggested a reason for the success of

exposure therapies such as flooding in which no sensory input is included.

The frequency and power of waves created during these therapies are much like those generated during the natural memory editing system of slow-wave sleep.

The results of this study of EMDR therapy can be generalized to include all exposure therapies, which are those in which the client is exposed to a fear memory as part of the therapy. These therapies have this in common: They work by utilizing a natural mechanism of the brain to remove the material basis for the fear memory. This mechanism is depotentiation of glutamate receptors on synapses mediating the memory in the lateral nucleus of the amygdala as discovered by animal studies such as those by Lin, Lee, and Gean (2003) and Rubin, Gerkin, Bi, and Chow (2005).

Dr. Edna Foa and others developed exposure therapy itself in 1999, and use of this term as a general name may be misleading. For this reason, the term depotentiation therapy is suggested for this group of therapies. To save five syllables in each use of this term, dep. therapy is used instead in this report.

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Included in the dep. therapy group are the following:

- Those in which specific sensory input is not given during therapy
 - Behavioral therapy (Wolpe, 1958)
 - Flooding therapy (Keane, Fairbank, Caddell, & Zimering, 1989)
 - Exposure therapy (Foa et al., 1999)
- Those in which sensory input is given during therapy (fast dep. therapy)
 - Eye movement desensitization and reprocessing therapy (EMDR; Shapiro, 1989)
 - Thought field therapy (TFT; Callahan, 1996)
 - Emotional freedom technique (EFT; Craig, 2008)
 - Others being developed, such as havening therapy (Ruden, 2010)

Experiments carried out by Dr. Francine Shapiro in 1989 proved that mild brain stimulation created by sensory input is a necessary component of fast depotentiation therapies, in this case EMDR. Her PTSD (posttraumatic stress disorder) patients were instructed to keep a fear memory in mind while she performed EMDR therapy without using sensory input. They were then given a test for evidence of PTSD; no posttherapy change was found. Subsequently, she performed EMDR using sensory input to relieve the PTSD symptoms. Shapiro made the obvious deduction from these experiments that sensory input is an essential element of EMDR therapy. Nevertheless, many have since questioned the relevance of such input during therapy, including Davidson and Parker (2001) and Renfrey and Spates (1994). Others, such as Devilly (2005), Gaudiano and Herbert (2000), and Ost and Easton (2006), debate the effectiveness of exposure therapies in general.

Still other therapists believe that sensory input must be applied specifically to energy meridians and the gamut point on the back of the hand. (Thought field therapy developer Callahan in 1996 suggested the name *gamut point* for a specific location on the back of the hand because of the large number of treatments using this location.)

To examine these and other issues, analysis of EEG records from the EMDR clinical sessions and lab experiments was undertaken in the current study. Since clinical studies do not lend themselves to sufficiently controllable input conditions, over 50 experiments were carried out in an EEG lab to record waves created by the more common sensory stimulation methods used in dep. therapy. This allowed a more accurate analysis of the range of power and frequency of waves generated during baseline periods and during sensory stimulation of various kinds. The results and conclusions of these studies are reported here.

EEG Relevance in Neuropsychological Research

The EEG waves on records studied in this project are thought to originate primarily through the firing of principal neurons

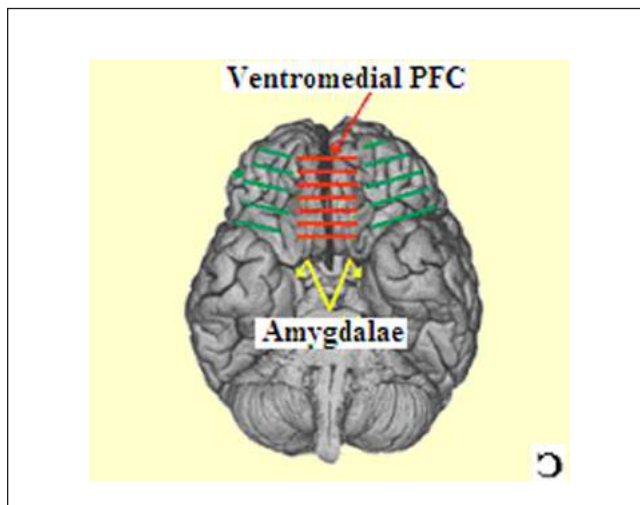


Figure 1. Photo of the ventral (lower) surface of the brain displaying ventromedial prefrontal cortex (VMPFC) and amygdalae; the hippocampi are located immediately below and in the same plane as the amygdalae
Source: Original image from Dubuc (2003) reprinted with permission.

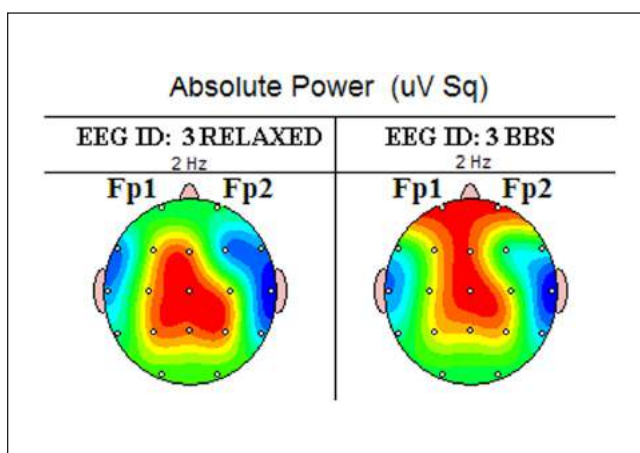


Figure 2. Topographic maps of areas of maximum intensity of electrical activity, Participant 3. Left: activity before brain stimulation while the participant was in a relaxed attentional state; right: activity during the final 5 min of EMDR Phase 4, with sensory input (BBS [bilateral brain stimulation], pads in hands). Fp1 and Fp2: locations of EEG electrodes principally used in this study

in the memory areas of the brain, particularly the hippocampus, amygdala, and the ventromedial prefrontal cortex (VMPFC, Figure 1). In general, memory is recorded by neurons in these areas firing at about 8 to 10 times per second (8 to 10 Hz, alpha waves), and memory erasure (extinction) by neurons firing at 1 to 4 times per second (1 to 4 Hz, delta waves). These two frequency ranges are ordinarily found to occur during the waking state and slow-wave sleep, respectively.

Information on locations within the brain most active in contributing to the EEG was obtained through two-dimensional mapping of the distribution of wave power intensity (Figure 2, from Participant 3). A LORETA mapping of the

EEG obtained during the EMDR session of Participant 2 confirmed and extended the interpretation of location data.

The raw EEG data contain detailed information about the activity of memory neurons during dep. therapy and the EEG protocols leading up to the therapy sessions. The recorded waves are sufficiently powerful to be manipulated mathematically for extraction of frequency and power, using advanced forms of wavelet analysis such as Fourier transforms (first extensively used in seismology). Although not as precise in location as fMRI, the EEG has the great advantage of being a continuous record over several minutes or hours. A large database builds as data are received and analyzed in the processing programs. The EEG tracks this process precisely in real time for subsequent quantitative analyses. Power is expressed in microvolts squared (μV^2 , millionths of a volt squared). Frequency of the waves of neuronal depolarizations is expressed in Hertz (one repetition per second is 1 Hz).

The quantitative analysis of the EEG in this study should not be confused with conventional qEEG (quantitative electroencephalogram) analysis. We used only the actual power and LORETA analyses from the qEEG processed data on the first three participants, and instead, carried out full analysis of the frequency and power information in the raw data to determine the following parameters for all participants: the firing frequencies and power of the principal neurons; the shape and amplitude of the peaks and troughs of the waves of depolarization, and thus, the likelihood of memory erasure or memory enhancement; and the presence of electrical activity from bioelectrical sources such as eye movements (artifacts).

Standard qEEG is more likely to be used to determine deviation of an individual EEG from large database norms rather than to investigate behavior of the brain during specific conditions such as those in this study, particularly the reaction to repetitive low-frequency sensory input. QEEG analysis is extended to precise timing of EEG event arrivals and the variations in response time and phase between various possible sources within each hemisphere or between the two hemispheres. Many, such as Schiff (2005), believe the qEEG interpretation is overextended and may lead to false conclusions. It is for this reason that we used raw data processing only in this study. However, we recognize that even this simpler approach to EEG interpretation can give ambivalent results because of the complexity of the brain itself and the variable emotional and cognitive responses to day-to-day life.

Equipment and Procedures

Equipment

For the clinical studies, two different sets of equipment were used, as detailed in an earlier paper (Harper et al., 2009). A Lexicor Neurosearch 24 EEG unit was used to provide EEGs of study Participants 1 through 3; the two electrode BrainMaster Atlantis II was used for the remainder of the participants and for the lab experiments. For the Lexicor unit, a 19-channel full-head electrocap was used; electrodes

were placed according to a standard location technique. The two electrodes of the BrainMaster unit were placed at standard locations in the temporal and frontal areas, most commonly at the Fp1 and Fp2 locations as shown in Figure 2. A Tac/Audioscan unit provided vibratory sensory input to the palm of the hand during Phase 4 of the EMDR protocol used in all the clinical trials. A digital metronome was used to help guide input frequency during some of the lab experiments.

Clinical and Lab Procedures

Three- and 5-min EEG recordings were made of each phase of the EMDR sessions. The EEG of relaxed states, with eyes open and then closed, were recorded to provide a baseline against which other attentional states could be compared. The next step was to record waveforms created while the participant was thinking of the fear memory with eyes closed, with no sensory input. This procedure was followed by 2 to 8 brief eyes-closed recordings of the EEGs of participants receiving Phase 4 of EMDR therapy. Sensory input during this part of the study was supplied by vibrating pads in the palms of the hands while the participant kept the memory in attention. The EEG protocol concluded with a brief recording of the relaxed state after the EMDR session. Thirty to 40 min of EEG records were obtained from each participant.

For the lab experiments, the participant was instructed to relax and think of neutral, nonemotional subjects. An EEG baseline value was obtained for each separate experiment, followed by recordings of the EEG resulting from sensory input such as tapping or eye movements.

Participants

Volunteer participants for this study were chosen through a clinical interview and a battery of tests designed to assess their suitability for the procedure. A fear memory target was agreed beforehand with each participant. The actual EEG recording session followed 2.5 to 4 hr of participant contact. Follow-up sessions were held with participants to determine their ongoing experience after the therapy. Finally, each participant was contacted 9 to 18 months following the EEG-EMDR sessions. All clinical results were positive. The lab experiments provided most of the data used in this study.

Data Processing

EEG records from the first three participants were processed conventionally, including subjecting the data to a z-score analysis (comparisons of power, timing, and frequency records with average base levels for these parameters). As noted previously, we found that this kind of data processing is not relevant to the questions we wished to answer, and so we were unable to use most of these processed data. Instead, we performed wavelet analysis of the raw data from the EEG records. Programs developed by the NeuroGuide and

BrainMaster companies were used in the analysis. Average power and frequency were calculated at intervals to adequately characterize the various groups of EEG responses after filtering to eliminate extraneous artifacts such as muscle (EMG) or cable noise. Simple mathematical programs were used to obtain frequency statistics. Artifacts, primarily from muscle contractions (EMG) or cable noise, were removed from the records prior to power and frequency analysis. Sensory input during the therapy and during the lab experiments often resulted in development of high-amplitude wave trains similar to artifacts. However, this input was generally given at a lower frequency than the natural firing frequency of the neurons (about 1.78 Hz) and so can be reliably distinguished from extraneous artifacts. Electrode pop (bounce) as illustrated in Rowan and Tolunsky (2003, p. 138) was found only when tapping within about 4 cm of the electrode (see also supplementary backup data). Records used are available for inspection, downloading, or further analysis at the website <http://www.mindspaces.org>, or by contacting the author.

Synaptic potentiation and depotentiation. A fear memory is formed through a process called synaptic potentiation, which is thought to result in strengthening of a specific circuit through the lateral amygdala (LeDoux, 2003). Reversal of potentiation occurs through synaptic depotentiation, which causes closure of AMPA (alpha-amino-3-hydroxy-5-methyl-4-isoxazole) glutamate receptor channels on the synapses involved. Research by Lin et al. (2003) indicates that this closure is caused by the action of calcineurin released when calcium ions enter the synapse (mainly through NMDA, N-methyl-D-aspartate receptors) during the minimum phase of the depolarizing wave (Figure 3); the depotentiated receptors are subsequently internalized within the synapse (Earnshaw & Bressloff, 2006), and the material basis of the fear memory has been removed.

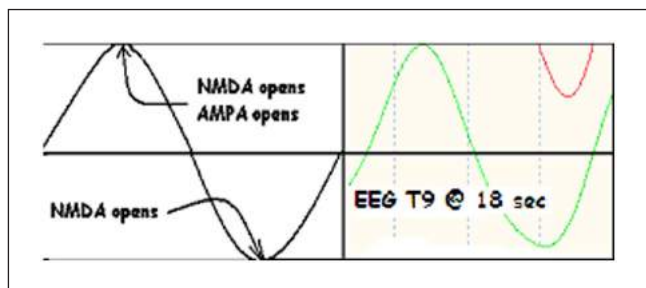


Figure 3 .Theoretical waveform required for depotentiation, left curve (from data derived by Hölischer, Anwyl, & Rowan, 1997). Actual waves recorded during sensory input from tapping the shoulder are displayed on the right. One wavelength is shown. AMPA (alpha-amino-3-hydroxy-5-methyl-4-isoxazole) and NMDA (N-methyl-D-aspartate) are synaptic receptors
Note: All EEG identifiers for this and subsequent figures are available at the website <http://www.mindspaces.org>. All figures are from eyes-closed data, except Figure 10.

Results

Frequency

The firing frequency of the neurons contributing to the EEGs recorded during clinical and laboratory studies in this project fall in the range of 1.5 to 2 Hz (they fire 1.5 to 2 times per second); average frequency calculated from 69 measurements of EEG samples from EEGs of the 6 participants is 1.78 Hz with a standard deviation of 0.13. This delta frequency pattern was substantially the same in all records examined, regardless of participant, the mental state of the participant during the recordings, or of sensory input (Figure 4).

A power variation with a frequency of 0.2 to 0.33 Hz can be seen on many of the records; that is, a wave-like change in power occurs over intervals of about 3 to 5 s.

Power

Maximum spectral power occurring in the delta range (from near 0 to 4 Hz) was determined for each type of sensory input in numerous separate computations. Each was found to have a characteristic magnitude that usually varied only with changes in recording conditions, including sensitivity of the electrodes used during the experiment. In general, power of sensory inputs such as tapping decreases as the distance from the sensory cortex increases (e.g., neck taps were usually more powerful than shoulder taps). This decrease is possibly the result of two factors: decay of sensory input signals with distance, and a general decrease in sensitivity of the skin away from the head. The former is unlikely since conduction loss is minimal (Ehrenstein & Lecar, 1972), and the variation of power is thought to result from variation in receptor density and type. Mann (1981), partially on the basis of data from Woodworth and Schlosberg (1965), lists the number of sensitive points per square centimeter on certain areas of the upper body: 100 at the tip of the nose, 50 on the forehead, 29 on the chest, 15 on the inside (volar area) of the forearm, and 14 on the back of the hand. The skin area with the greatest density of touch receptors is on the palm of the hand, with 120 per square centimeter, an increase relative to the back of the hand by a factor of 8.6 fold. This difference in sensitivity to touch is directly reflected in the power of the EEG recorded at these two sites: $2.2 \mu\text{V}^2$ on the back of the hand compared to $20 \mu\text{V}^2$ on the palm of the hand, a 9-fold power increase (Figure 11).

Delta wave power expressed as a multiple of that of the relaxed state for the more frequently used sensory input sites used during dep. therapy was found to be: cheek, up to X90; neck, X19; shoulder, X5 to X38; palms of hands, X5; back of hands, X1.1; knee, X1.0 (no increase). These are only examples of a wide range of values determined. A larger set of power values noted in this study is shown in supplementary data at the website <http://www.mindspaces.org>.

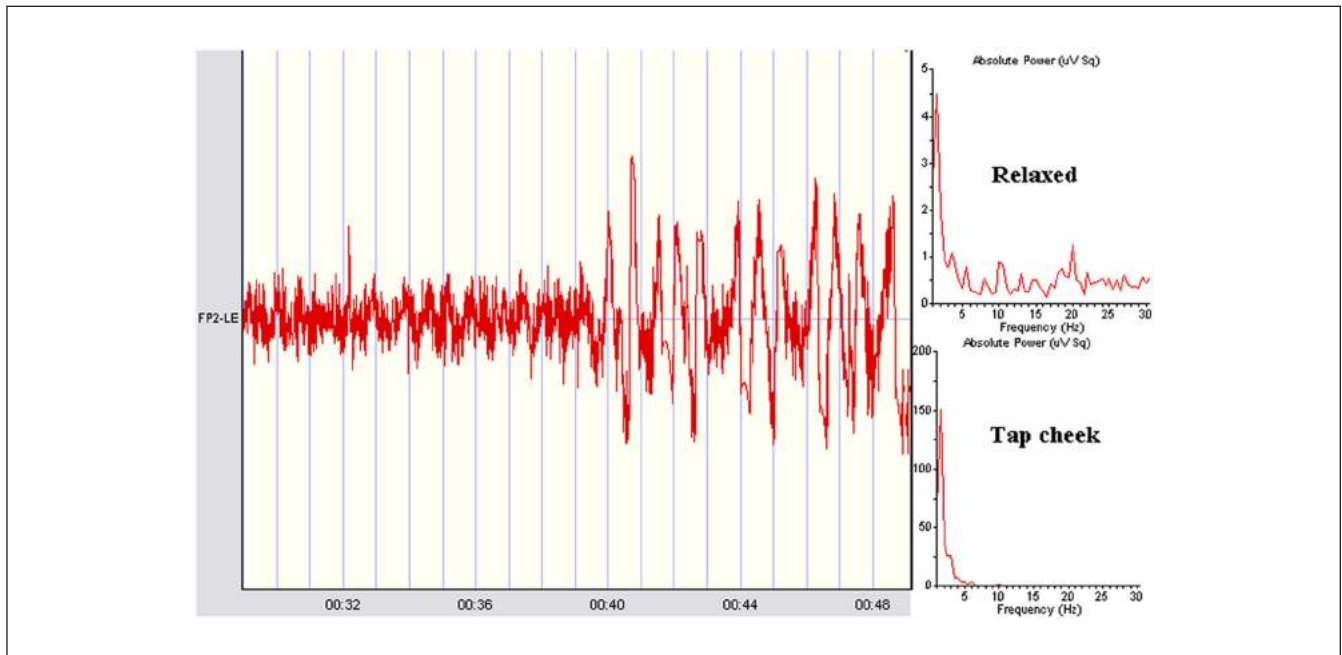


Figure 4. Junction at 40 s between relaxed state and tapping cheeks. No change in wave frequency (about 1.4 Hz) occurs at the junction. Graphs on right show that power at the Fp1 electrode, recording mainly from the left hemisphere, changes within 2 s from 4.3 to 150 μV^2 , a 35-fold increase (from lab experiment, eyes closed)

Vibrating pads in the palms of the hands were found to create waves of neuronal firing with power usually 3 to 4 times greater than that of the relaxed state (using a Tac/Audioscan unit set at 1 Hz and maximum intensity). Lateral eye movements cause an increase of 12 to 20 times the relaxed state, and eye blinks, up to 350 times (see below for more on eye movements). Also of interest, clapping the hands increases delta wave power by a factor of about 5 fold over that of the relaxed state.

Bringing the fear memory into attention without sensory input caused an increase in EEG delta wave power and z scores of 1.5 to 2 times or more that of the relaxed state (see supplementary material for an example). Diepold and Goldstein (2009) have earlier reported changes in the qEEG response when their patient focused on the fear memory state. Specifically, they report 25 statistically abnormal values of coherence and phase during the neutral baseline but only 8 during the fear memory state for this patient.

Examples of the average *absolute* power (as opposed to power as a multiple of that of the relaxed state reported above) during the clinical phase of the study are shown in Figure 5: relaxed state, 30 μV^2 ; fear memory only, 45 μV^2 ; and with sensory input from vibrating pads in the palms of the hands, 90 μV^2 . Examples of absolute power values obtained during the laboratory study are given in supplementary materials at the website <http://www.mindspace.org>.

As illustrated in Figures 6 to 8, wave power is not significantly changed when the sensory input procedure is changed from bilateral (e.g., tap right temple, then left temple alternately), to simultaneous (tap both temples simultaneously), or to unilateral tapping of the cheeks and temporal bones. Figure 9 shows the *decrease* of power sometimes associated with accommodation (adaptation) to repetitive sensory input.

The Special Case of Eye Movements

Most of the signal created by movement of the eyes laterally is the result of movement of an electrical dipole in the eyes, which generates a powerful electrical signal. This signal has a magnitude of about 150 μV^2 for a lateral eye movement of 30 degrees (Malmivuo, 1995, Figure 28.3) and constitutes noise (artifacts) in EEG records of eye movements. It largely obscures the electrical signal generated by the neurons themselves, which is the objective of most EEG analyses. The residual power found after taking into account this purely electrical component of eye movements suggests an approximate wave power from the neurons themselves of 30 to 40 μV^2 or about the same as that of tapping the neck or shoulders (Figure 10, see website <http://www.mindspace.org> for another estimate).

Based on results from all other upper-body somatosensory inputs, it seems likely that the effect at the neuronal level of

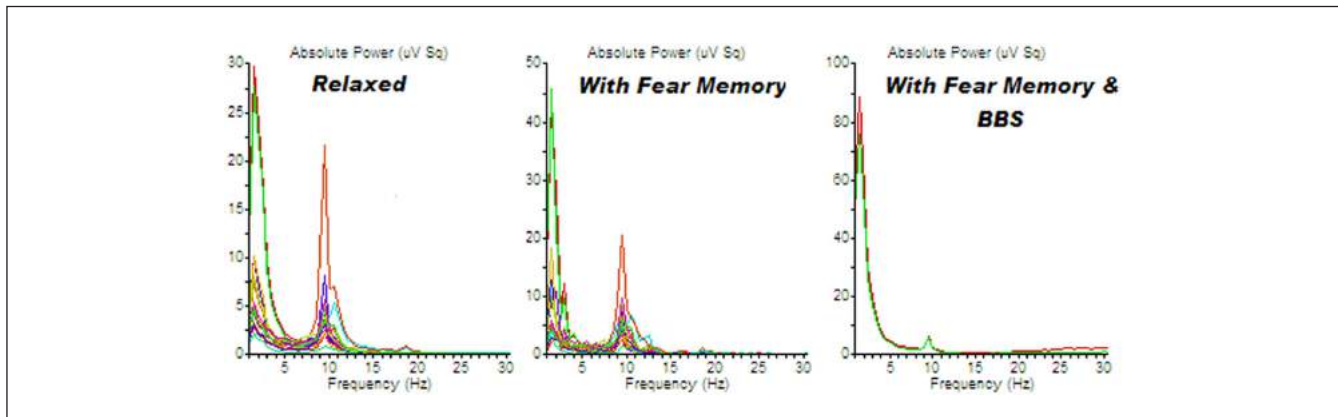


Figure 5. Examples of spectral power of EEG during three brain states recorded from Participant 2. Sensory input (BBS [bilateral brain stimulation]) was vibrating pads in palms of hands. Red and green lines are from the Fp1 and Fp2 electrodes, respectively. Note changes in scale between graphs

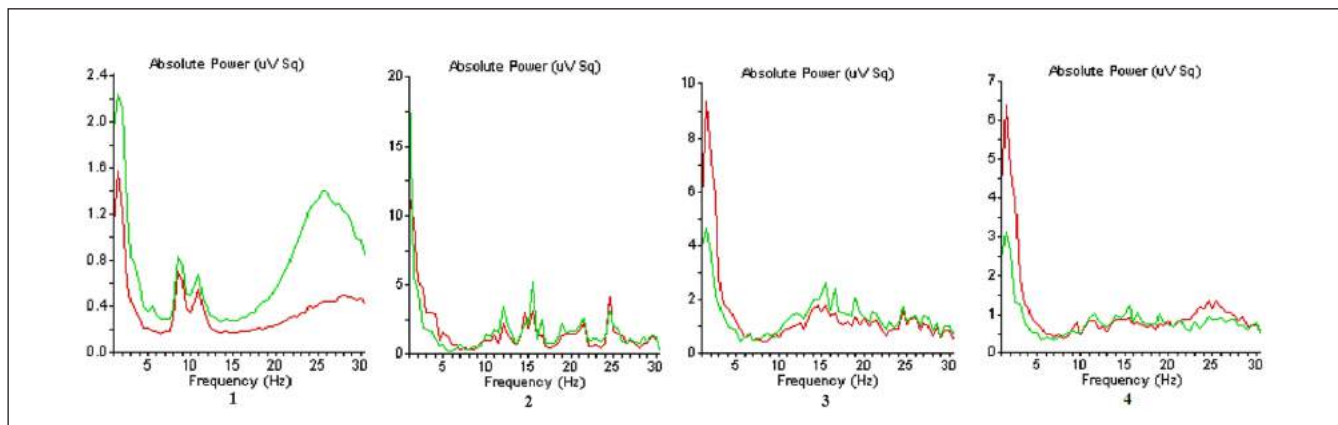


Figure 6. Power/frequency graphs, lab experiments (Participant 1). (1) relaxed state, (2) tap right temporal bone, (3) tap right and left temporal bone simultaneously, (4) tap temporal bone right and left in sequence (bilaterally). Maximum power occurs at about 1.5 Hz on all graphs and ranges from 2 to 17 μV^2 (note changes of scale between the graphs)

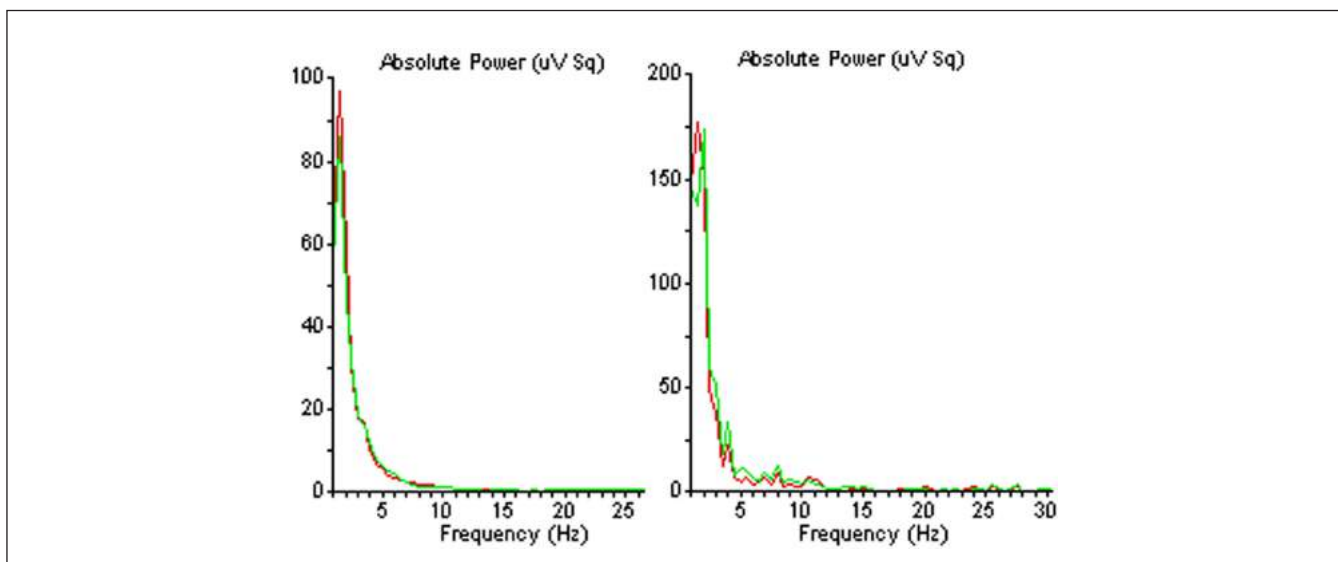


Figure 7. Power/frequency graphs from lab experiments recorded when tapping the cheeks bilaterally (left) and while tapping right cheek only (right)

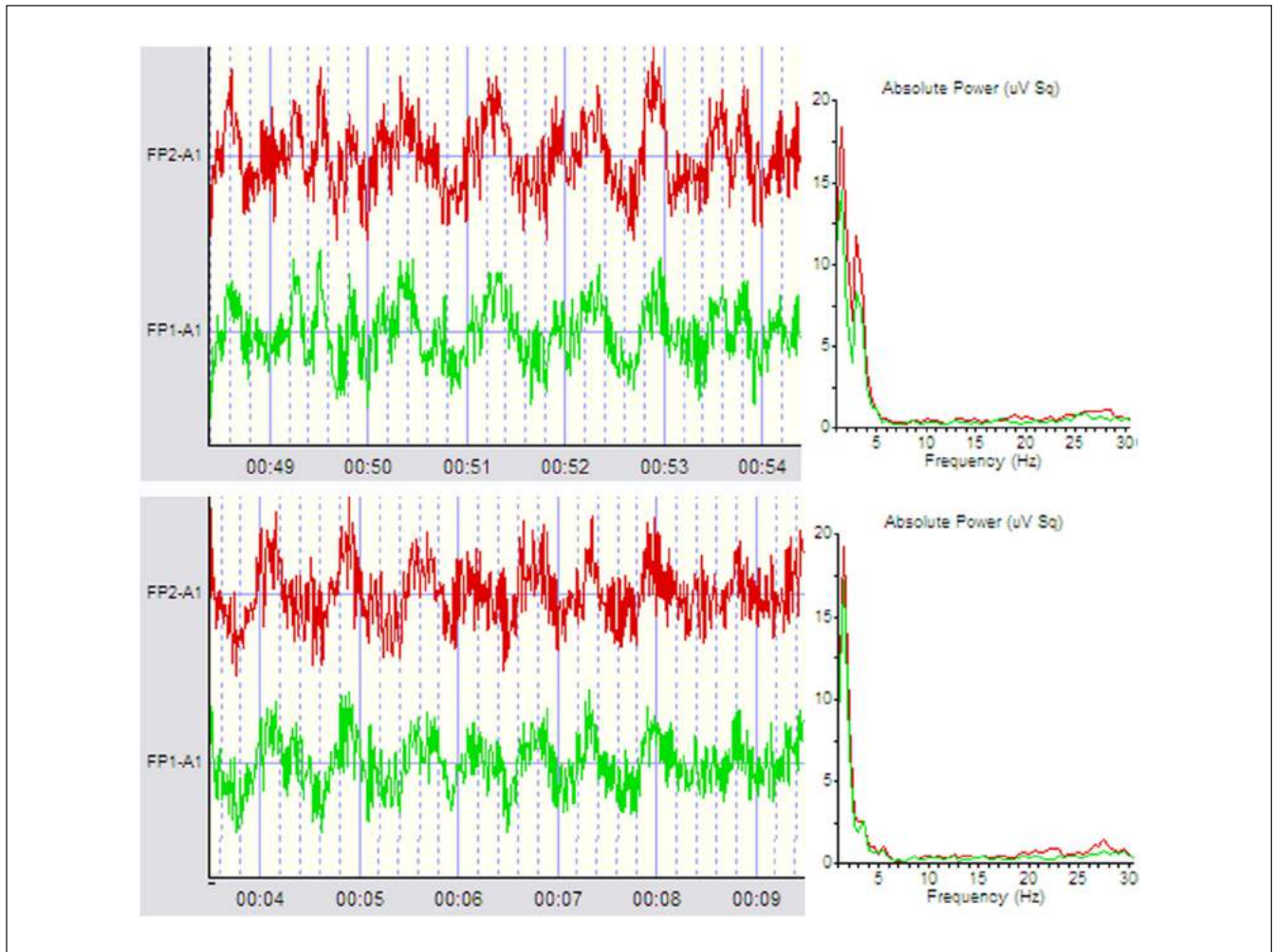


Figure 8. Lab experiment to illustrate similarity of waveforms created by bilaterally tapping shoulders (top), and simultaneously tapping shoulders (bottom). Power/frequency graphs on right of each panel show no significant differences

lateral eye movements is simply to cause bilaterally comparable and nearly simultaneous waves of neuronal depolarizations in both hemispheres.

Power at Energy Meridians and Gamut Points

As illustrated in Figure 11, tapping the “gamut point” (as defined by Callahan, 1996) on the back of the left hand, or any other point on the back of the hands, provides little more power than that of the relaxed state and much less than tapping the palm. Also, no significant differences in power were found when tapping meridian points such as the upper-inside corner of the eye as compared to tapping of nonmeridian points on the cheek. Other illustrations of power resulting from sensory input at meridians and gamut points are available at <http://www.mindspaces.org>

Number of Depotentiating Waves Required

Lin et al. (2003) found that depotentiation of most synapses mediating fear memories in animals subjected to tetanic stimulation occurred during the first few minutes of stimulation. They found that about 900 pulses were required for complete depotentiation (as also determined by Earnshaw & Bressloff, 2006). The principal neurons of memory areas of participants in the current study have a natural firing frequency averaging 1.78 Hz. If 900 pulses are required to depotentiate fear memory synapses in humans, these could be generated within about 500 s, or 8 min ($900/1.78 = 506$ s).

The least time required to eliminate a traumatic memory during Phase 4 of EMDR therapy in the Harper et al. (2009) study was 14 min for Participant 2. A count of delta waves on the EEG records of this participant suggests that about

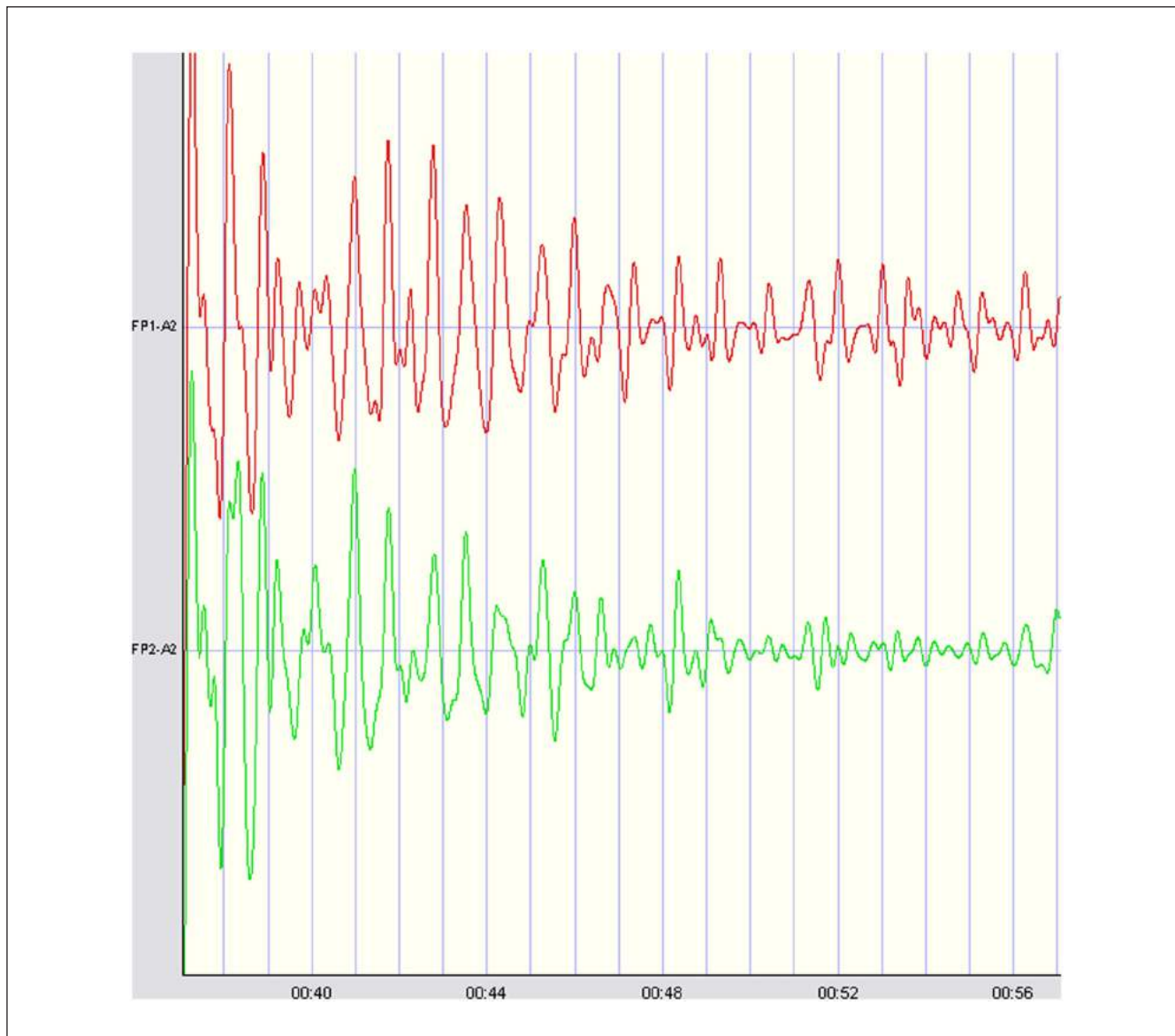


Figure 9. EEG showing adaptation of neurons over a 20-s interval when tapping the cheeks during the lab experiment 1.5Hz. There was no change in input over this interval. Eyes closed, delta filter applied

650 delta waves with power great enough to depotentiate AMPA receptors were produced during Phase 4 of the EMDR protocol by this participant, along with about 115 during exposure to the fear memory without brain stimulation. The participant reported being unable to feel the fear or even to remember the reason for fearing this memory after these approximately 765 pulses. A cutoff power value of 75 uV^2 was used in this calculation (detailed in backup data available at <http://www.mindspaces.org>).

Summary of Results

1. The average firing frequency of principal neurons in the memory areas of the brain for all participants in this study, taken over 20- to 30-s records, is 1.78 Hz, with a usual range of 1.5 to 2 Hz.
2. Bilaterally or unilaterally applied brain stimulation such as tapping produces essentially equal power and frequency in both hemispheres simultaneously.

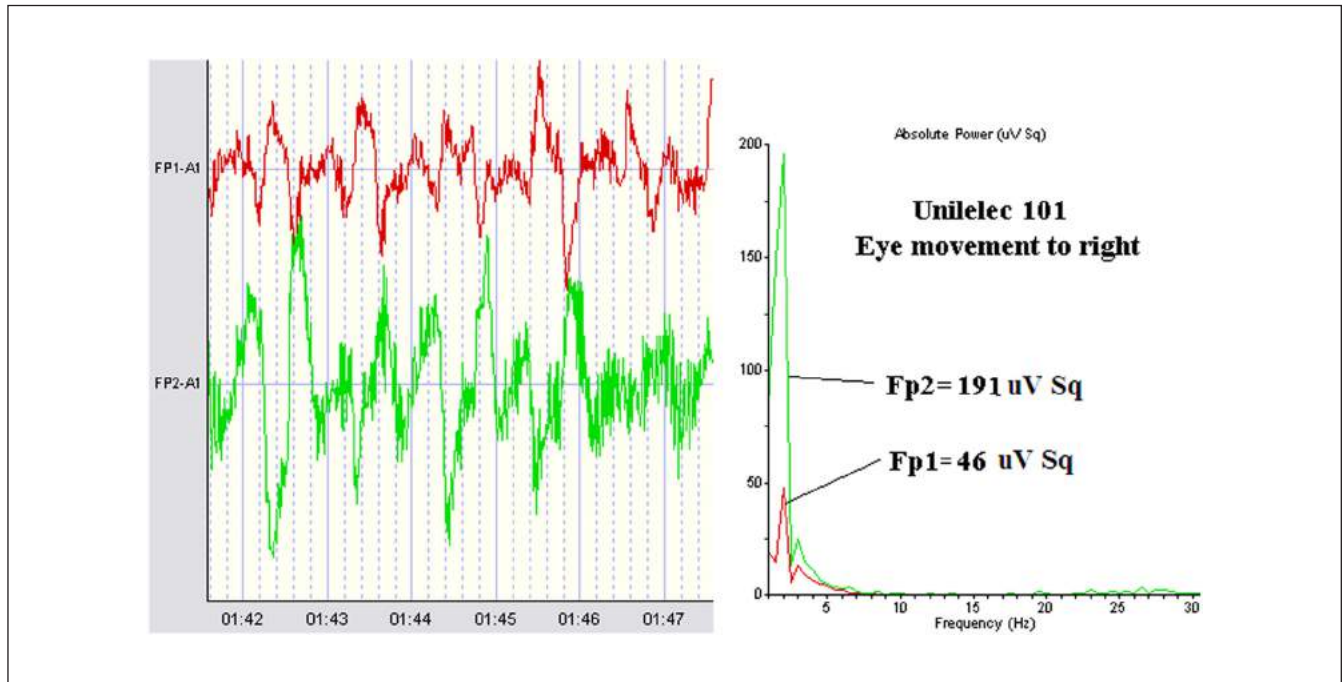


Figure 10. EEG produced by right lateral eye movements during lab experiment; difference of power between the two hemispheres (left, top curve; right, bottom curve) is principally due to the purely electrical component of eye movement in right hemisphere ($Fp1 = 46 \text{ uV}^2$, $Fp2 = 191 \text{ uV}^2$)

3. Stimulation at energy meridians and gamut points showed no power or frequency advantage over stimulation of any other points on the upper body.
4. An estimated 765 depotentiating neuronal pulses were necessary to remove the fear memory for Participant 2.

Discussion

The intensity of human emotions is governed largely by the amygdala, which has a design flaw: Unlike the hippocampus and neocortex, it has no adequate gain control mechanism (for comparison with the neocortex, see Hendler et al., 2003, and for discussion of comparative evolution, see LeDoux, 2003, p. 212). Gain control, as in the recording of a song, is used to control the volume of the recording. Similarly, gain control during memory recording can establish the relative intensity of the recorded emotions. Overdriving of the amygdalar mechanism during such recordings can result in pathological processing of sensory input during traumatic incidents. This often results in overlearning of some emotional memories, and in more extreme cases, it results in symptoms of PTSD.

It is thought that pathologically recorded emotional content mediated in the amygdala cannot be readily linked with the cognitive content of the memory from the hippocampus (Corrigan, 2002). For this reason, the brain cannot edit such

memories during slow-wave sleep, as normal memories are, to return them to a more nearly homeostatic (base) level. However, during dep. therapy, we can force the memory into attention and thus into a labile state where it can then be changed to a more reasonable response by the brain's natural processes.

It is to deal with psychological problems, particularly traumatic memories, arising from this flaw in the brain's design that dep. therapies were developed. Through use of our intellect, we have been able to compensate for poor evolutionary strategies by overriding the brain's usual programs and mechanisms; this is a clear case of a victory of mind over matter. Further possibilities for overcoming our evolutionarily derived amygdalar disadvantages are suggested below.

Implications of Results of Frequency Analysis

Relevance of powerful low-frequency waves to depotentiation therapies is that slow pulsation of neurons allows the calcium ion content within fear memory synapses in the amygdala to drop below the level at which AMPA receptors previously potentiated at higher frequencies are stable. The average frequency of the delta waves determined here, 1.78 Hz, is near the midpoint of the frequency range most conducive to synaptic depotentiation as determined primarily by animal studies of depotentiation by researchers such as Clem and Haganir (2010); Huang, Liang, and Hsu (2001); and Lin et al. (2003).

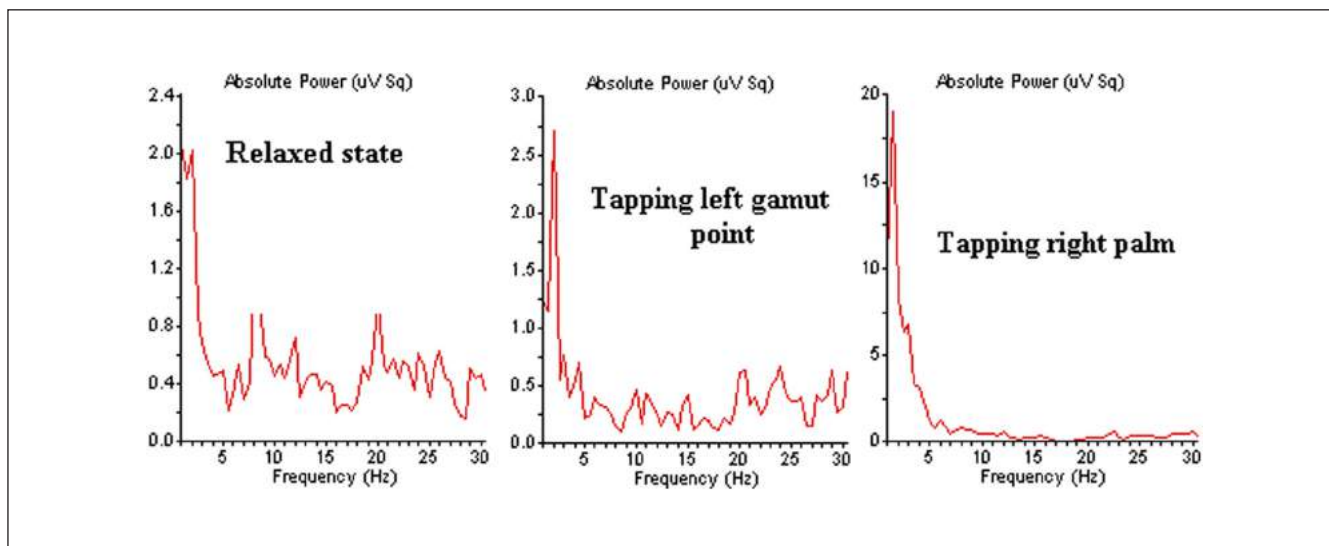


Figure 11. Power/frequency graphs of data from Fp1 electrode recorded during lab experiments: comparisons of relaxed state, tapping gamut point on left hand, and tapping of right palm. Note major scale change between palm taps and the other two states

It is thought that the principal neurons of the memory areas of the brain (mainly the hippocampus, amygdala, and areas of the prefrontal cortex) have this basic preferred frequency established by pacemaker neurons in corticothalamic regions and transmitted to the memory areas of the brain (as suggested by Steriade, Curró Dossi, & Nuñez, 1991). The average frequency of delta waves determined in the present study is close to the 1.7 Hz found in thalamic pacemaker neurons of the cat by Steriade et al. A similar value, about 1.86 Hz, was found in the auditory cortex of Rhesus monkeys by Lakatos et al. (2005, Figure 3). Both of these research groups calculated the frequencies by direct measurement of depolarization rates rather than by analysis of the EEG as done here.

It should be noted that there is a vast difference in reaction to sensory inputs in the memory areas of the brain as compared to that of the auditory cortex. Lakatos, Chen, O'Connell, Mills, and Schroeder (2007) found that the firing frequency of auditory cortex neurons changes to match the frequency of incoming sensory signals and that these signals also reset the phase of ongoing neuronal oscillations. This frequency and phase change enable greater accuracy of interpretation of incoming auditory signals. As noted by Harper et al. (2009), the neurons contributing to the EEG of the memory areas of the brain do not change from their basic firing mode when sensory input frequency changes. In the current study, evidence is also found that sensory input does not reset the phase of ongoing neuronal oscillations; the inputs, even when matching the preferred neuronal frequency, are often out of phase with the ongoing neuronal oscillations of the neurons creating the EEG.

The average firing rate of the neurons studied here, 1.78 times each second, is not clock like. The entire range from 1.5 to 2.0 Hz is seen in most recordings of more than a few seconds. The rapid variation in frequencies is likely the result of

rapidly changing intensity of attention or emotions. In addition to these short-term frequency changes, there often appear 3- to 5-s wave-like changes in power of the EEG (illustrated in supplementary information). Further analysis of these phenomena is beyond the scope of this study.

Activity in the memory areas of the brain during slow-wave sleep is time locked and synchronized by the high-amplitude slow waves (Luo, Honda, & Inoué, 2001; Wolansky, Clement, Peters, Palczak, & Dickson, 2006). As indicated by the behavior of the neurons monitored on the EEG in this study, the same is true during the waking state when sensory input radically increases delta wave power.

Significance of Results of Power Analyses

Repetitive sensory input applied to most locations on the upper body are shown in this study to cause remarkable increases in delta wave EEG power compared to the relaxed state, and it is concluded that all except tapping the back of the hand are sufficient to erase fear memories. This conclusion is based on the comparison of the spectral power of the relaxed state with that of fear memory only. The average power generated by fear memory alone is taken as the minimum for synaptic depotentiation because it is known from numerous case studies of exposure therapy without sensory input that several therapy sessions are required to erase a fear memory.

Evidence from this study indicates that the critical level for rapid depotentiation of fear memory synapses is about 2 times the power generated when thinking of the fear memory or about 3 times the power of the relaxed state. The great surge of power caused by almost any repetitive sensory input results in rapid depotentiation of fear memories. This accounts

for the great difference in contact time required for dep. therapy with and without sensory input (Harper et al., 2009).

The right amygdala is thought to be more reactive to negative emotional input than the left and may have a more powerful input to certain negative emotional states than the left (Iidaka et al., 2001, 2003). However, the therapist does not have to take this hemispheric dichotomy into account because the wave power produced by sensory input is sufficient to depotentiate all fear memory synapses in both amygdalae.

From this study and our clinical experience, we find that the client usually exhibits a delayed cognitive response to the change in the memory trace at the molecular level. Therefore, self-reports of fear memory status may lag the change at the molecular level in the fear memory circuits. Full realization of this vast change in the memory may come as a metacognition gradually over several days or even weeks. The memory seems to have to be “refitted” into the normal memory system since it has significantly decreased in relative subjective importance. This longer-term editing procedure set in motion by dep. therapy is likely to take place mostly during slow-wave sleep.

Low-frequency brain stimulation applied during dep. therapy quickly shifts the memory areas of the brain into a neuronal mode similar in power and frequency to that of slow-wave sleep (Harper et al., 2009). In both brain stimulation studies on animals and sleep studies in humans referenced above, this brain rhythm has been found to be ideal for the homeostatic adjustment of the power of synapses mediating memory. Homeostatic processes return synaptic potentiation toward baseline levels, and in the presence of normal waking levels of the neurotransmitter acetylcholine (ACh), they are conjectured to result in rapid and complete depotentiation of fear memory synapses (for review, see Harper et al., 2009). ACh levels are very low during slow-wave sleep (Gais & Born, 2004; Hasselmo, 1999; Power, 2004; Rasch, Born, & Gais, 2006), perhaps protecting the fear memory circuits in the lateral amygdala from complete depotentiation as seems to occur during dep. therapy. Details of naturally occurring homeostatic processes can be found in publications by Gilestro, Tononi, and Cirelli (2009); Liu, Faraguna, Cirelli, Tononi, and Gao (2010); Tononi and Cirelli (2003); and Yeung, Shouval, Blais, and Cooper (2004). Results of the EEG study reported here suggest that depotentiation therapies such as EMDR impose homeostasis on memory networks in the lateral tract of the amygdala by reproducing the wave pattern of slow-wave sleep during the waking state.

Gamut Points and Energy Meridians

During energy meridian therapy such as TFT (thought field therapy) and EFT (emotional freedom technique), the client is typically instructed to perform various activities such as moving the eyes in various directions while the gamut point on the left wrist is being tapped. The eye movements create powerful delta waves and are likely to be the sensory inputs

that result in depotentiation of fear memory synapses during TFT and EFT therapy, rather than the much less powerful waves created by tapping the gamut point. Also, evidence from this study of wave power suggests that tapping of specific points on energy meridians is no more effective than tapping other points on the upper body. At any rate, therapists decide for themselves what method is best for them and their clients on the basis of their own experience.

Information From Other Wave Frequencies

Although this study was primarily directed toward analysis of delta waves, which are necessary for synaptic depotentiation, important information can also be derived from analysis of other frequencies of the EEG wave spectrum. An example is the second most powerful waves observed in this study, 10-Hz alpha waves (10 neuronal firings per second). One of the significant aspects of alpha waves is that depressed patients may have higher alpha power than those with no depression (Lorenzen, Clarke, & Barry, 2006). Gregory et al. (2009) suggested that higher alpha power is required to decrease anxiety, a common goal for persons suffering from depression. This alpha wave behavior is in accordance with our findings: The participant suffering from major depression, in addition to PTSD, in our study had unusually high gamma wave power (shown in the supplementary information under alpha wave study at the <http://www.mindspace.org>).

Prospective Applications of Depotentiation of Amygdalar Synapses

The amygdala seems to have a more or less standard, built-in emotional repertoire, ready for elaboration whenever a relevant life event occurs. Depotentiation therapies deal with these activated emotions retroactively, for the most part, although Dr. Francine Shapiro (2001) has provided for prospective, future reactions in her EMDR protocol. Many people have reported that negative emotions in general seem to be less extreme after EMDR or other exposure therapies.

One simple way of incorporating this mechanism of taming the amygdala is to include sensory input while thinking or worrying about emotionally trying events we expect to happen during the following day. Mere consideration of expected emotional events while tapping the shoulder or cheek is beneficial; life encounters become less emotionally trying through use of this depotentiating procedure. Ultimately, through neurofeedback training, we might be able to power up the neurons that produce powerful delta waves at will. This could enable personal amygdalar control on a real-time basis.

The amygdala is under higher, cortical control by the VMPFC (Figure 1) in many situations, but this control may be overridden during traumatic events (Ruden, 2010, p. 27). A shorter route to the amygdala from sensory receptors is available for faster reactions to unexpected events through the sensory thalamus. Other methods of increasing control

of the amygdala can be learned, such as those taught during training of Special Forces military personnel. Undergoing “mock trauma shock,” perhaps through computer-generated virtual reality, can give us much greater resilience when exposed to real trauma. Including group dep. therapy for all military personnel as a part of basic training would also enhance resilience; targeting emotions likely to be experienced in battle would increase resilience on the battlefield. The author was an army medic and believes medics can be taught dep. therapy as an adjunct to their basic training. Other military personnel consider medics more approachable than mental health professionals, who are usually officers. Obviously, all these suggested extensions of dep. therapy must be used with great caution to avoid possible negative responses.

Cross-Species Use of Depotentiation Therapy

As LeDoux notes (2003, p. 212), the basis for emotional reactions is highly conserved across species. Other animals have the same evolutionarily challenged amygdala as ours. We can extend the benefits of depotentiation therapy to traumatized animals by finding ways to expose animals to emotions aroused by a past traumatic event while providing repetitive sensory input such as tapping the cheek or muzzle.

Conclusion

All depotentiation therapies have a high success ratio in eliminating the emotional aspect of fearful stress memories mediated in the amygdala. This study suggests that a wide variety of mild brain stimulation through sensory inputs is effective in erasing the physical basis for a fear memory in the lateral amygdala, including unilateral as well as bilaterally applied stimulation. Almost all inputs examined here produce powerful delta waves concurrently in both brain hemispheres regardless of the mode of input. The brain itself is bilaterally organized, and different reactions to such input occur within each hemisphere. In addition, this study suggests that tapping of energy meridians and gamut points creates no power advantage over tapping other points on the upper body. Nevertheless, sensory input in general dramatically speeds the process of fear memory erasure.

This laboratory and clinical study supports findings of many others such as Barrowcliff, Gray, Freeman, and MacCulloch (2004) concerning EMDR therapy, and the empirical results reported from the broad field of Energy Psychology by researchers such as Hartung and Galvin (2003).

Recommendations

Choose a method of sensory input that produces sufficiently powerful waves in the memory areas of the brain to quickly depotentiate fear memory synapses; do not rely solely on

tapping the knee or the back of the hand to provide sensory input. In refractory clients, repeating the therapy with higher power input such as tapping the shoulder or cheek may be necessary; if doubt exists for such clients, an EEG analysis will reveal the degree to which the fear memory is held in attention and whether the sensory input being applied is powerful enough to depotentiate fear memory synapses in both brain hemispheres.

Fast dep. therapy can be employed in mass emergencies following disasters such as earthquakes, floods, and wars; this might be best accomplished by developing and proving in relief camps a protocol for group therapy such as that proposed by Jarero and Artigas (2009) and used successfully in the field, for example, with children after the large earthquake near Istanbul in 1999 in which about 17,000 people were killed (Konuk et al., 2006). We can vastly extend the ways of targeting the amygdala to attain more direct and personal control of this ancient brain module.

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