Diffuse Axonal Injuries, Interruptions and Treatment Using Audio-Visual Entrainment

- Dave Siever, C.E.T.

Abstract

The concept of entrainment is a biological response in an organism to a stream of stimuli in which the organism in some way shows a resonant response in frequency to the stimuli. The concept Audio-Visual Entrainment (AVE) involves the presentation of a continuous stream of visual flashes of light and pulses of auditory tones. This is purported to induce a brainwave frequency response, matching that of the stimuli, also known as the frequency following response and more recently, brainwave entrainment. The frequency model of AVE is what most people think of when they consider AVE, however many other events occur simultaneously. The frequency following aspect of AVE is only a small portion of AVE and sometimes it doesn't occur at all. Other effects of AVE include dissociation and hypnotic induction; autonomic nervous system calming; increased cerebral blood flow; increased neurotransmitters and unexpected brain-enhancing effects.

Concussions are typically categorized as the diffuse axonal traumatic brain injury (DA-TBI), which is characterized by an interruption in the axonal pathways, either descending from the neurons in the cortex to the thalamus or from the thalamus to the cortex. Given that DA-TBIs rarely show on an MRI, it might be more precise to consider them as an interruption in signal transmission rather than an injury. DA-TBIs impair both cognitive function and emotional control, resulting in a spectrum of psychological disorders. EEG recordings of a person with a DA-TBI show loss of alpha activity (the main thalamocortical rhythm), agitated beta activity and aberrant delta and beta phase between the various cortical regions. Neurological deficits from DA-TBIs, caused by either injury or fever, can remain for decades.

Through its various mechanisms of action, AVE can quickly re-excite these neurons and in turn, reestablish the thalamocortical loops, thus restoring cognition, emotion control and affect.

Introduction

Audio-Visual Entrainment (AVE) is a technique using flashes of lights into the eyes and pulses of tones into the ears at specific frequencies. The frequency of the lights and tones used are in the brainwave frequency range from .5 to 40 Hz. *AVE is one of the most intriguing stimulation technologies as AVE devices have been shown to influence, in varying degrees, brain activity by a myriad of influences, not simply frequency driving.* Given these other effects, we could ponder if we should simply rename the technique as audio-visual stimulation (AVS). Our senses are constantly bombarded by AVS. Consider watching TV or sitting on a street corner watching the traffic. These activities consist of abundant quantities of AVS, yet they don't have much of an impact on the brain. For instance, when AVE is randomized at $\pm$ 1 Hz (for example, 10 Hz

would randomize from 9 to 11 Hz), entrainment is reported to provide a significant clinical impact. At $\pm$ 2 Hz, the clinical effect is poor and at $\pm$ 3 Hz, the clinical effect is all but lost. So it appears that the myriad of effects from AVS only occur when the stimulation is kept fairly rhythmic and therefore entraining, hence AVE. For this reasoning, we will use the term AVE throughout this article. Because AVE affects such a diverse range of neurological processes, it has applications spanning from simple meditation and boosting cognition to the treatment of complex issues such as depression, pain and diffuse axonal injuries (the most common type of brain injury). But first, let's understand the physiology of AVE.

Physiology of AVE

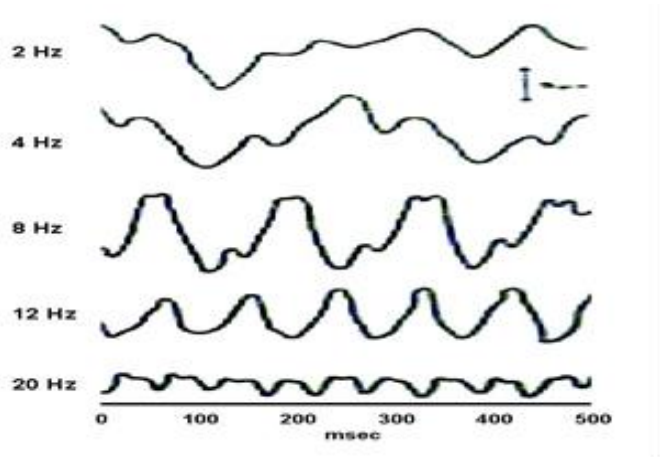
While clinicians often think of AVE as only entraining its frequency into the brain, a host of complementary effects are occurring simultaneously, as shown below:

1. Brainwave entrainment;
2. Dissociation/hypnotic induction;
3. Autonomic nervous system calming (including heart rate variability);
4. Increased cerebral blood flow;
5. Increased neurotransmitter activity;
6. Unexpected effects.

(1) Brainwave Entrainment

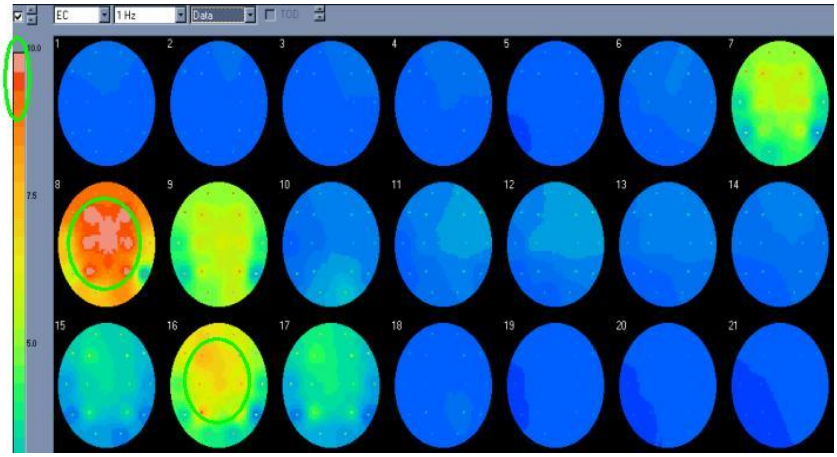
One mechanism of action attributed to AVE, and to some degree all stimulation technologies, is related to frequency driving of brainwave activity, known as the frequency following response and more recently as entrainment. Entrainment occurs when a bodily rhythm reflects the rhythm of the stimuli to which it is exposed. For example, brainwaves observed via EEG reflect the dominant brainwave frequency duplicating the frequency of auditory, visual or tactile stimuli. Photic driving of brainwaves was first discovered by Adrian and Matthews back in 1934, while auditory entrainment was first demonstrated by Chatrian and colleagues in 1959. Photic entrainment occurs best near one's own natural alpha frequency (Toman, 1941; Kinney et al., 1973). AVE utilizing square-wave photic stimulation should be avoided in those with photosensitive epilepsy, where flashing lights of certain frequencies may trigger a seizure (Ruuskanen-Uoti & Salmi, 1994; Erba, 2001; Trenité, et al., 2001) while sine-wave stimulation does not produce harmonics and may well be safer for epileptics (Donker, et al., 1978; Regan, 1966; Townsend, 1973; Van der Tweel & Lunel, 1965). Figure 1 shows the EEG effects of square-wave (xenon flash) photic entrainment at a variety of frequencies.

Figure 1. EEG Showing Photic Entrainment (Kinney, et al, 1973).



AVE effects are primarily associated with frontal, parietal, occipital brain regions and near the vertex (Frederick, et al., 1999). For example, Figure 2 shows a qEEG (quantitative electro-encephalograph), or "brain map" from the SKIL (Sterman-Kaiser Imaging Labs) database, in 1 Hz bins showing the frequency distribution of AVE at 8 Hz. The area within the circle at 8 Hz shows maximal effects of AVE in central, frontal, and parietal regions (at 10µv in this case) as referenced with the oval area on the legend. It is through associated influences on frontal brain regions that AVE has been shown effective in reducing depression, anxiety, and attentional disorders. A harmonic is also present at 16 Hz (the circled image), which is typical of semi-sine wave (part sine/part square wave) stimulation.

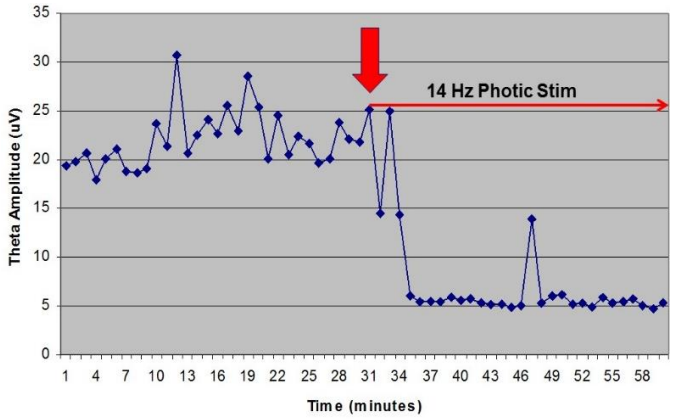
Figure 2. Brain Map in 1 Hz Bins - During 8 Hz AVE (SKIL-Eyes Closed).



In as much as entrainment can enhance brainwaves at a particular frequency, entrainment can also inhibit the half frequency of stimulation, which is useful for treating those with excess brainwave activity at slower frequencies such as with depression, ADHD, struggling college students and seniors with age-related cognitive decline (Siever 2003c). For instance, those with ADHD show excess frontal theta (5 to 7 Hz) activity and too little SMR (12 to 15 Hz). By stimulating in the frequency range of 12 to 15 Hz, the sensorimotor rhythm (SMR) activity over

the motor strip is enhanced while frontal theta activity ranging from 6 to 7.5 Hz is inhibited, thus accomplishing both outcomes at once (frontal lobe activation and motor-strip calming). Figure 3 shows excessive theta activity from an ADHD child. At the 30-minute mark (red arrow), SMR stimulation at twice his dominant theta (roughly 14 Hz) was given, which quickly suppressed his theta activity (Collura & Siever, 2009).

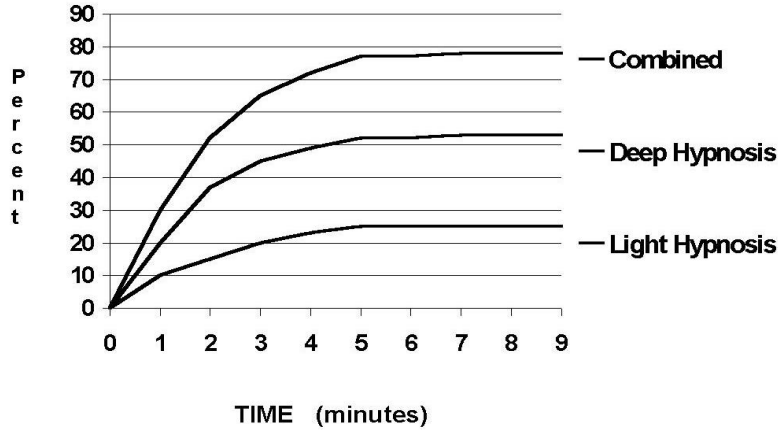
Figure 3. Theta Suppression (site FZ) Using AVE at Twice the Dominant Theta.



(2) Dissociation/Hypnotic Induction

Several studies have been completed since the 1950s on hypnotic induction and dissociation (Walter, 1956; Lewerenz, 1963; Sadove, 1963; Margolis, 1966; Leonard, et al., 1999 & 2000) and altered states of consciousness (Hear, 1971; Lipowsky, 1975; Glicksohn, 1987). The first study on dissociation induced via entrainment involved hypnotic induction, which found that photic stimulation at alpha frequencies could easily put subjects into hypnotic trances (Kroger & Schneider, 1959) as shown in Figure 4. Notice that nearly 80% of the participants in the study were in a hypnotic trance within six minutes of photic entrainment.

Figure 4. Photic Stimulation Induction of Hypnotic Trance (Kroger & Schneider, 1959).



(3) Autonomic Nervous System Calming

When people dissociate in a healthy, meditative way, there is profoundly reduced autonomic activity (sympathetic and compensating parasympathetic) and the autonomic nervous system returns to homeostasis (also known as *dissociation and restabilization* - DAR) (Siever, 2003a). Assisting clients with a history of trauma to dissociate (in a constructive and meditative way), during the course of treatment is important. For example, Figure 5 shows a typical reduction in forearm sEMG (electromyography recorded with surface/skin electrodes) and Figure 6 shows a typical increase in finger temperature during AVE (Hawes, 2000). Notice that restabilization begins after roughly six minutes of AVE, when the user begins dissociating and the autonomic nervous system settles down.

Figure 5. Forearm sEMG Levels during AVE (Hawes, 2000).

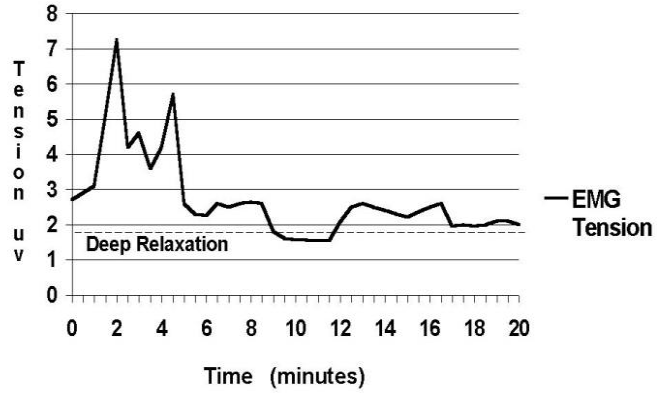
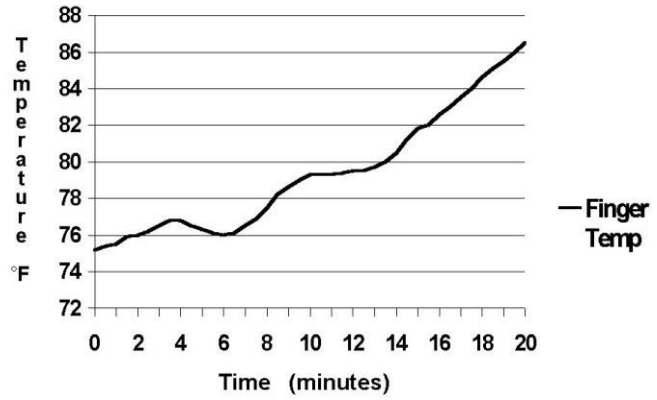


Figure 6. Peripheral Temperature Levels during AVE (Hawes, 2000).



Autonomic Calming and Heart Rate Variability

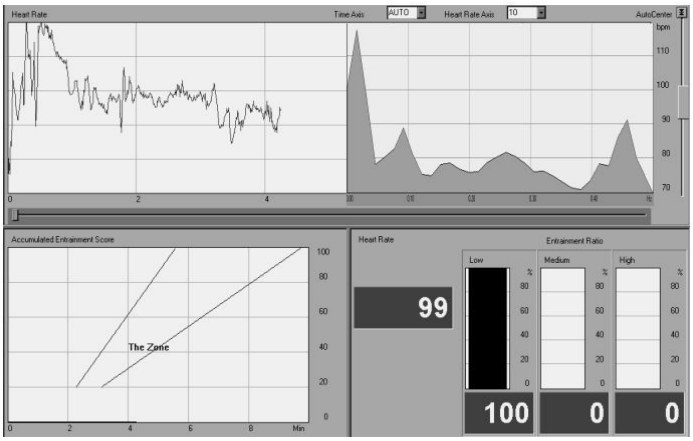
Heart rate variability (HRV) is a measure of variance of heart rate swing during inspiration and expiration. It also measures consistency in heart rhythm over time. It is an excellent measure of autonomic arousal and biofeedback method for learning meditative breathing (Gervitz, 2000).

Figure 7 shows a case study of a 30-year-old mother of two, struggling with post-traumatic stress disorder following a disturbing event with her recently separated husband. This study used a DAVID AVE device from Mind Alive Inc. The DAVID was used with a meditation protocol. The eyesets and the headphones are placed on the participant, but the lights and tones are not turned on. The DAVID devices have a heartbeat sound that is played through headphones at 24-beats per minute (bpm). The participant is instructed to breathe in for two heartbeats and exhale for two heartbeats. This makes a four-beat breathing cycle and six (24/4) breaths per minute. After about 10 minutes of breathing, the session is halted. There is a short break of a few minutes and the session is repeated, but now with the entraining lights and tones turned on.

The screen shot is taken from the emWave PC (www.heartmath.com). The upper-left window indicates the actual heartrate throughout the recording. The bottom-left window shows a score as determined by an algorithm which looks at the high versus low heart rate, roundedness of the waves and consistency over time. The lower-right window indicates the average heart rate and a score as to how well the heart followed a breathing rhythm of six breaths per minute (10-second breath cycles). The upper-right display shows the spectral analysis of the breathing. Given a 10-second breathing cycle, there should be a peak at 0.1 Hz and the spectral display would look much like a witch's hat. Sympathetic activity is indicated at low frequencies (below 0.1 Hz) while compensatory parasympathetic activity is indicated at frequencies above 0.1 Hz.

The woman's mind is constantly bombarded with distressing thoughts that she simply cannot stop. Although she is trying to breathe at six breaths per minute, she shows a spike and clamp (and unhealthy) heart rate frequency (as is typical with fear and anxiety). Her score is zero, her coherence ratio is 100% in the low category and her average heart rate is 99 bpm. Her spectral display looks like a mountain range with all of the autonomic activation.

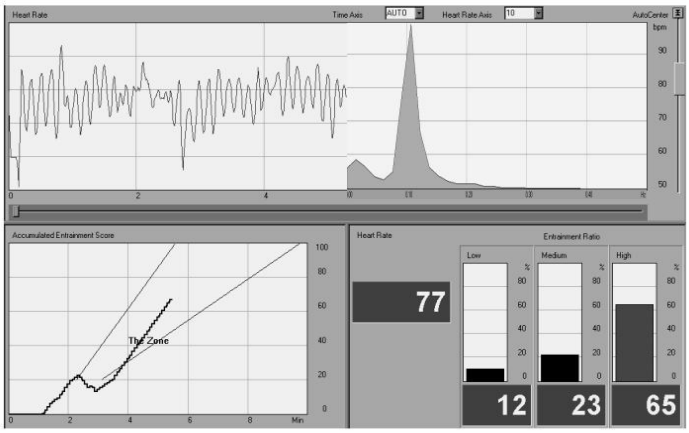
Figure 7. Heart Rate Variability Just Prior to 7.8 Hz AVE.



A few minutes later, she was put on AVE at 7.8 Hz and immediately began to calm down as seen in Figure 8. Recording began at after five minutes of AVE, at which time her heart rate had already fallen by 22 beats per minute. This recording shows the effects of DAR. Her breathing and heart rate quickly became stable. Aside from a few thoughts that crept in just past the two-minute mark, she was completely relaxed. Imagine what could be done in terms of preventing

the development of post-traumatic stress disorder if AVE devices were dispensed to people experiencing natural disasters or acts of terrorism.

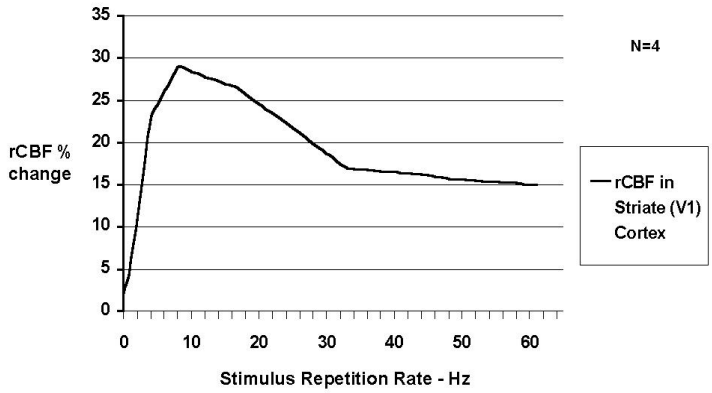
Figure 8. Heart Rate Variability during 7.8 Hz AVE.



(4) Increased Cerebral Blood Flow

Cerebral blood flow (CBF) is essential for good mental health and function. Measures of CBF show that hypoperfusion of CBF is associated with many forms of mental disorders including anxiety, depression, attentional and behavior disorders (Teicher, et. al., 2000), and impaired cognitive function (Amen, 1998; Meyer, et al, 1994; Meier, et al, 2015). Some of the purported beneficial effects of AVE have been attributed to increases in frontal region CBF (Fox & Raichle, 1985; Fox, et al, 1988; Sappy-Marinier, et al., 1992). For example, Figure 9 shows an increase of 28% in CBF within the striate cortex, a primary visual processing area within the occiput. As an interesting note, maximal increases in CBF have been shown to occur when stimulation techniques occur around 7.8 Hz, the "Schumann Resonance" of the earth (Balser & Wagner, 1960).

Figure 9. Cerebral Blood Flow at Various Photic Entrainment Repetition Rates (Fox & Raichle, 1985).

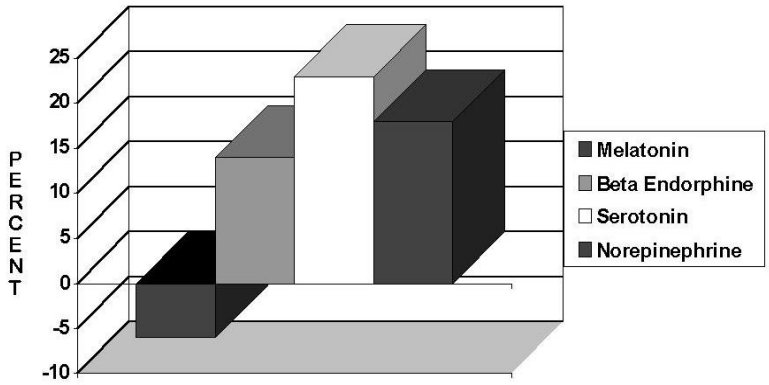


(5) Increased Neurotransmitter Activity

Neurotransmitters affect all of brain functioning. Some neurotransmitters such as glutamate and norepinephrine excite brain arousal while other neurotransmitters such as serotonin and GABA calm the brain down. With long-term stress and depression, the "feel-good" neurotransmitters such as dopamine, serotonin and norepinephrine shut down, leaving us feeling lethargic and sad.

For instance, Seasonal Affective Disorder (SAD) involves reduced levels of melatonin, a neurotransmitter that regulates circadian rhythms and hibernation in animals. Melatonin slows brainwaves and reduces CBF as it prepares us for sleep (Murphy, 1993). Endorphins are essential for blocking pain and are the basis of analgesic medication. Low levels of serotonin are part of most every psychiatric disorder. Moderate levels of norepinephrine (brain's adrenaline) are involved in mental vigilance. Norepinephrine is increased by caffeine, which is why we enjoy a cup of coffee so much in the morning. A direct correlation between psychiatric disorders including anxiety, depression, obsessive-compulsive disorder, schizophrenia, memory and cognitive disorders has been linked to brain neurotransmitter action (Emmons, 2010; Arco & Mora, 2009). There is evidence that cerebral-spinal fluid levels of melatonin fall while serotonin, endorphin, and norepinephrine levels rise considerably following 10 Hz, white-light AVE (Shealy, et al. 1989). Increases in endorphins reflect increased relaxation while increased norepinephrine along with a reduction in winter daytime levels of melatonin typically increase alertness (Figure 10).

Figure 10. Neurotransmitter Levels Following AVE (Shealy, et al., 1989).



(6) Unexpected Effects

Both non-entraining aspects of audio-visual stimulation and unexpected side-effects of entrainment are emerging within the literature. For instance, for treating Auditory Processing Disorder (APD), often comorbid with Autism Spectrum Disorder, the Tomatis Method^{TM}, a three-part technique in which a variety of filtered prenatal sounds, then filtered sounds of Mozart music and Gregorian Chants, followed by an active phase, are presented via headphones to the patient. Marked improvements in auditory processing, various forms of memory and cognitive improvements have been observed (Ross-Swain, 2007).

Intranasal Light Therapy (ILT) is a technique involving the shining of near infrared light into the nasal cavity from a light-emitting diode (LED) mounted on a small clip. Ample evidence of ILT has shown effectiveness in treating a multitude of neurological disorders including insomnia, mild cognitive impairment, Alzheimer's Disease, Parkinson's Disease, schizophrenia, migraine and stroke. The current theory of function has been based on optimizing the results for blood irradiation, which may be holding back the brain's potential for better neurological outcomes (Lim, 2013). Although the main premise of entrained light is that the frequency of the light pulses is affecting the brain, this does not preclude the fact that entrained light shines through the skull and into the brain directly, as does light therapy.

Many studies on near infrared (NIR) light therapy for a variety of brain issues ranging from depression to concussion have been emerging in recent years (Doidge, 2015). NIR light has been shown to improve mitochondrial function in hypoxic cells, increase adenosine triphosphate (ATP), important for cellular metabolism because it releases local nitric oxide, which in turn increases regional CBF. A recent study utilizing an array of head mounted transcranial near-infrared LEDs on people with mild traumatic brain injury, showed significant improvements in cognitive performance, social, interpersonal and occupational functions (Naeser et al., 2014).

One of the most intriguing effects of entrained light involves the application of 40 Hz (gamma) photic entrainment for one hour into the eyes of mice with the mouse equivalent of Alzheimer's Disease. This study showed that, within a few hours, beta-amyloid plaques were reduced in the range of 50 to 70% and microglia cell body diameter increased by 166% from consuming the beta-amyloid deposits (Iaccarino et al, 2016).

Clinical Research

Over the past several decades, AVE has been associated with several types of beneficial outcomes spanning a wide variety of clinical applications: for SAD (Berg & Siever, 2009), for improving concentration and memory in college students (Budzynski & Tang, 1998; Budzynski, et al, 1999; Budzynski et al., 2007; Wuchrer, 2009), for reducing worry in college students (Wolitzky-Taylor & Telch, 2010), for the treatment of ADD, ADHD and behavior disorders (Carter & Russell, 1993; Joyce & Siever, 2000; Micheletti, 1999), for regaining motor control in post aneurysm (Russell, 1996), for treating depression and risk of falling in seniors (Berg & Siever, 2004), for treating Alzheimer's (Budzynski, et al., 2002), for improving brain function and memory in seniors (Williams, et al., 2006; Palmquist, 2014), for reducing chronic pain from occupational injury (Gagnon & Boersma, 1992), for reducing symptoms of fibromyalgia (Berg et al., 1999), for reducing symptoms of temporo-mandibular dysfunction (Manns, et al. 1981; Morse & Chow, 1993; Thomas & Siever, 1989), for reducing anxiety during dental procedures (Morse & Chow, 1993; Siever, 2003b), for treating PTSD in war vets (Trudeau, 1999), and for improving sleep (Tang, et al., 2016). Whereas there is not space in this brief review article to describe all the studies cited, Table 1 lists a variety of clinical conditions, along with the number of studies (and the size of the population), as well as the type of demographic.

Table 1. Clinical Studies Involving AVE.

Condition	Studies (N)	Demographic
Attention Deficit Disorder (ADD)	4 (359)	School children
Academic performance in college students	3 (134)	College students
Reduced worry/anxiety in college students	3 (163)	College students
Drug rehabilitation	1 (44)	General population
Depression and anxiety	3 (93)	General population
Improved cognitive performance in seniors	1 (40)	From seniors' homes
Reduced falling and depression in seniors	1 (80)	From seniors' homes
Memory in seniors	1 (40)	Seniors - drop in
Dental (during dental procedures)	3 (>50)	Patients
Temporo Mandibular Dysfunction (TMD)	3 (76)	General population
Seasonal Affective Disorder (SAD)	1 (74)	General population
Headache (migraine and tension)	2 (35)	General population
Neurophysiology	>100 (>3000)	General population
Heart rate variability and hypertension	5 (148)	General Population
Hypnosis/dissociation/meditation	10 (>2000)	General population
Pain and fibromyalgia	5 (178)	General population
Insomnia	1 (10)	General population
Post Traumatic Stress Disorder (PTSD)	1(15) ~600 cases	Public, military, police
Literature reviews	5	Diverse population
Pre-menstrual Syndrome (PMS)	2 (23)	General population
Diffuse Axonal Injuries (interruptions)	1 (5)	Closed-head injuries

The Enigma of AVE

In this section, we turn our attention away from the traditional frequency driving effects of AVE to mechanisms and principles that underlie the brain's particular responses to entraining stimuli. There are several potential mechanisms that relate to nonlinear frequency-specific responses.

In particular, network properties and nonlinear effects of AVE may produce results beyond simple changes in arousal such as activation or relaxation. On the surface, a repetitive stimulus is nothing more than the combination of many single stimuli. However, one nonlinear effect of entrainment is the presence of a neuronal refractory period after each stimulus, which includes the time that a neuron requires to repolarize completely. If a stimulus is presented within the refractory period, the previous response to the next stimulus will be affected by that stimulus and may extend beyond the simple vector-addition of the stimuli, so that the response is now different.

But the function of the brain is acutely conditioned on factors such as synchrony, network connectivity and self-organization so complex neural processes can, and do, occur. Whereas physical processes such as blood flow, neurotransmitters, metabolic activity, and related physiology might be expected to reorganize themselves in a predictable fashion, neuronal network reactions to repetitive stimuli at various frequencies and via different modalities should be expected to be much more complex.

Neuroplasticity

Neuroplasticity is the ability for the brain to modify the ways it responds to stimuli. Stimuli may come from the environment or simply be thoughts one is thinking, which in turn can form dendritic connections. Neurons and their synapses may be excitatory or inhibitory and linearity of the response would be impossible to predict.

Neuroplastic effects of repetitive stimulation include those that involve changes in neuronal responsivity and tendency to activate, even after the cessation of the stimulation. Hebb's law (1961) states that "cells that fire together, wire together" which further indicates that neural networks change because of stimulated neuronal firing. But there is more to this story in that the "neurons that fire out of sync, wire out of sync." Often during learning, some neurons are firing and wiring together while others are firing and wiring apart simultaneously. For instance, cognitive training involves both learning a desired action and suppressing an undesired action.

Underlying mechanisms can include changes in receptor distribution and response, increases or decreases in the number of synaptic connections, cellular protein synthesis and metabolic or even anatomic changes. Once the neurons are off line, as with diffuse axonal interruptions, could active neuroplastic changes take these neurons "permanently" off line such that a hard push is required to reactivate them? A recent study in mice with intentionally damaged retinas and optic nerves found substantial regeneration in the group of mice that received intense visual stimulation (Lim et al, 2016), whereas the controls in a normal visual environment showed insignificant regeneration.

Repetitive stimulation, particularly when it produces metabolically significant activity such as action potentials, can have both immediate and lasting effects on the brain. These effects would be primarily due to the physical consequences of the increased activity of neurotransmitters, transmembrane transport mechanisms, and the associated vascular, respiratory, and recovery mechanisms. Vascular changes would include alterations in capillary structure and physiology and changes in blood flow. The associated recovery mechanisms take care of the removal of metabolic by-products, as well as the reuptake and transport of neurotransmitters. It is therefore reasonable to expect that repetitive stimulation can have effects on, not only neurons, but also associated structures including the glia, vascular structures, and other supportive tissues.

Clinical Examples of Enigmatic Effects of AVE

The five examples below indicate that the dissociative and CBF aspects of AVE are just as or likely even more important than the frequency following effects. As mentioned above, AVE affects CBF, neurotransmitters, dissociated states, and autonomic activity, all at the neuronal level.

For many years, we have observed that ADHD children fall asleep during beta frequency AVE and it is common for clients to fall into deep sleep with dual-frequency AVE of alpha/beta or SMR/beta frequencies. This is counter-intuitive, as higher frequencies such as beta (14 to 35 Hz) are typically associated with alertness, and yet AVE in the beta range, often puts the neuronally hypo-aroused user into a deep sleep, which also makes AVE a valuable tool for assessing sleep issues such as poor sleep-onset and sleep apnea.

Some Electro-Encephalography (EEG) and Quantitative EEG (qEEG) Terms

Before we start looking at the following case studies, we need to look at some definitions and to note that we are only going to address EEG and qEEG techniques and we will only review qEEG measures of greatest concern. There are a few methods for placing electrodes on the head. These EEG recordings were made using electrodes placed according to the international 10-20 EEG electrode placement system. This type of montage has 19 recording sites with two noise-cancelling electrodes on the earlobes. The term 10-20 means that the electrodes are placed at 10% and 20% intervals between the nose and inion from front to back and between the ear canals from side to side. The brainwaves being recorded are referenced against the ear inputs - this is termed *linked-ears* or a *referential* recording (referenced against the ears as opposed to other recording electrodes, known as a *differential* recording).

First, several minutes of EEG are recorded off the scalp. After these data have been saved as an EEG file, that data is downloaded into qEEG analysis software. Artifacts relating to eye-blinks and movements, plus muscle activity from jaw, neck and body movements are then removed with the intention of leaving only true EEG activity. The *quantity* (q) of true EEG activity remaining is processed with Fourier Frequency Analysis and other analysis such as coherence, comodulation, phase, etc. These data may also be compared to a database which was created using the exact same montage and with participants who were deemed psychologically normal according to psychometric testing. The analyses in the following examples have been processed

using the Sterman-Kaiser Imaging Labs (SKIL) qEEG software (Sterman & Kaiser, 1999; Lorensen & Dickson, 2004; Johnstone, Gunkelman & Lunt, 2005; Kaiser, 2007; Kaiser, 2008; Kaiser & Meckley, 2012).

The following qEEG analysis Figure 11 shows a picture of the electrode setup and Figure 12 shows the electrode cap being worn while experiencing AVE.

Figure 11. 10-20 Montage with Linked-Ears References (A1 and A2).

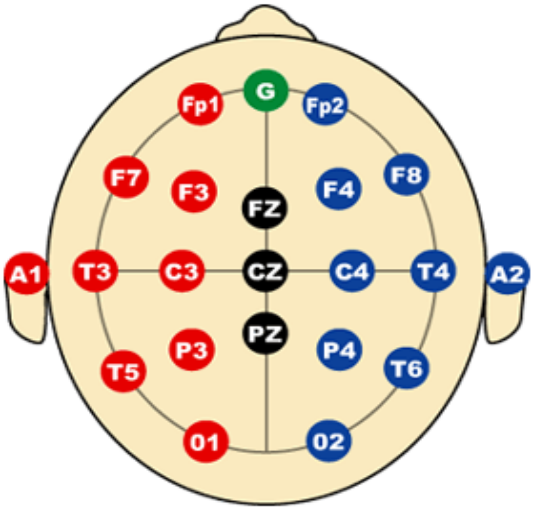
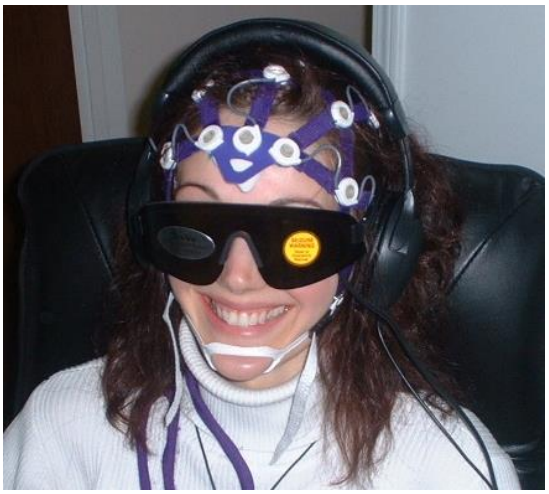


Figure 12. Person Wearing a 10-20 Electrode Cap While Using AVE.



Absolute Magnitude: The average voltage amplitudes averaged over time. Shown in microvolts (μv) for each frequency band.

Database Magnitude: The absolute magnitude in comparison to a database with the average healthy population.

Comodulation: Spindles and wavelets generally appear with temporal synchrony across brain regions. This measures the degree to which the spindles and wavelets are in alignment with respect to time. For instance, alpha is an idling rhythm, so when alpha spindles throughout the brain are in alignment, it means that the brain is taking small breaks together and processing information together. When the spindles are out of alignment, a part of the brain may be sending information to another part that is taking a break so the information doesn't get there. Conversely, a part of the brain may be requesting information and the part with the information is taking a break and still it doesn't get there. You see this a lot with people who have to be told something twice before they understand it or having to always check twice when driving in order to see traffic and road signs, or those who require a fair bit of time to recall someone's name or other information. Comodulation is compared against a normative database.

Delta Phase: This is a measure of the integrity of the white-matter extending from neurons to other regions of the brain, particularly the thalamus and associated structures. This entire *thalamocortical* synchronization loop normally takes 100 msec to complete. White-matter damage slows and may even stop propagation of signals to and from the neurons and the thalamus. When neurons lose this synchronization pulse from the thalamus, they begin to fire randomly and completely out of sync with both damaged and intact regions of the brain. Imagine you are at a concert and the band has encouraged the audience to clap to the beat of the song and everyone is doing so at two claps per second (delta). However, there are a few people who can't seem to get the rhythm right and are clapping on the off-beat. They are still clapping at two claps per second like everyone else, just 180° out of phase with the remaining audience. This would show up as a delta phase issue.

Beta Phase: This is a measure of synchrony between cortical areas. It may also reflect a general thalamocortical desynchrony of various Rich Club neuronal networks. These are densely packed neurons segregated into 66 clusters or hubs (Hagmann, et al., 2008).

Case Report 1 – Boosting Cerebral Blood Flow Effect

Giving a person with a slow dominant alpha brainwave the exact frequency of AVE can inhibit the same frequency. This is believed by the author to possibly be due to the increase in CBF. An inverse correlation between CBF and slow-wave EEG activity has been well established (Teicher, et. al., 2000).

CLIENT INFORMATION:

Condition: ADD and Fibromyalgia YOB: 1987

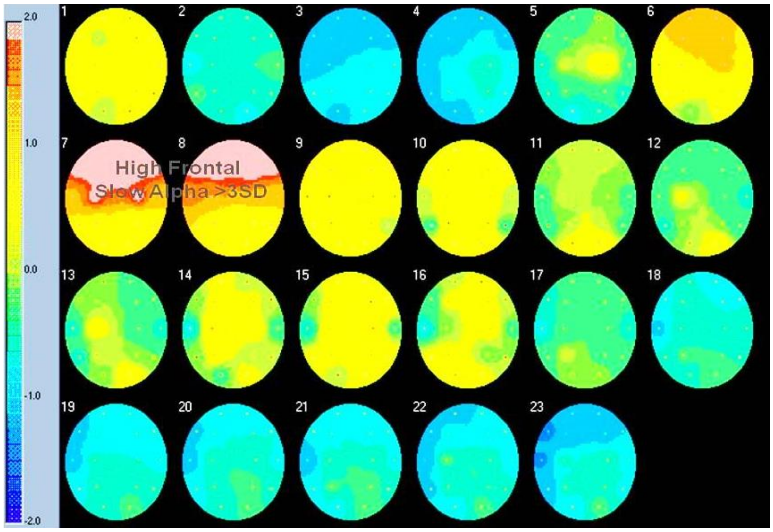
Sex: Female Handedness: Right

Medications: None

HISTORY:

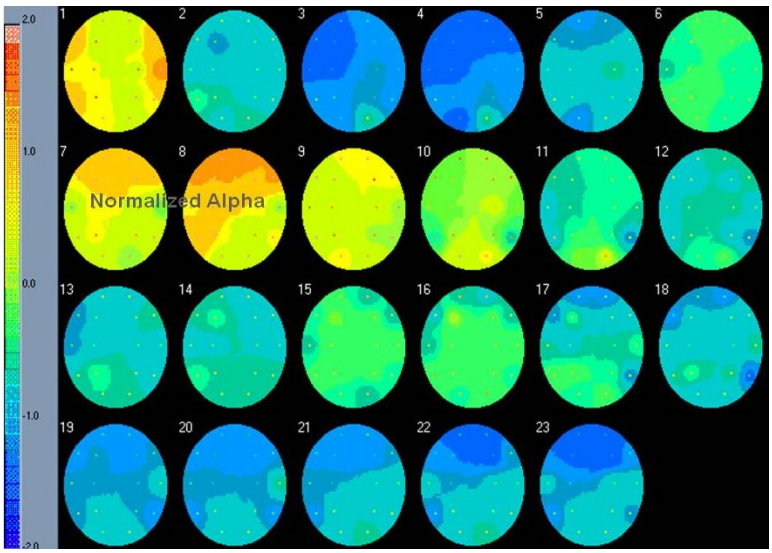
This college student has been struggling with low energy, poor focus, poor comprehension and mild pain throughout her body. She has been diagnosed by a medical doctor to have ADD and fibromyalgia. Because she struggled to read more than half a page of her text books at a time, she would read and reread in an attempt to learn her study material and often stayed up until 3 AM. Figure 13 shows her magnitude on the SKIL database in standard deviations (SD). Here we see that she is flooded with 7 to 8 Hz dominant and slowed alpha. She was only able to read two to three paragraphs before "fogging" out and quitting. The brainwave ranges are delta (1 to 4 Hz), theta (5 to 7 Hz), alpha (8 to 12 Hz), low beta and sensori-motor rhythm (12 to 15 Hz), faster beta (16 to 20 Hz) and even faster beta (21 Hz and above).

Figure 13. Eyes-Closed Z-Scored Alpha using Linked-Ears, Z-Scored Analysis on the SKIL.



During 30 minutes of AVE at 7.8 Hz, her 7 to 8 Hz activity did increase. However, 30 minutes following AVE, her abnormally high brainwaves at 7 to 8 Hz normalized. Given that Fox and Raichle (1985) and others have shown that photic entrainment increases CBF with the maximum increase at 7.8 Hz, it is plausible that the AVE recovered her brain function due to the CBF aspect of AVE. Figure 14 shows normalized brainwave activity 30 minutes following the cessation of AVE. This young woman was now able to read 10 pages before losing her attention.

Figure 14. QEEG Analysis 20 Minutes Post 7.8 Hz AVE.



Case Report 2 - Calming High Frequency Beta Type of Anxiety Using Beta AVE

It's been well known that beta stimulation effectively puts ADHD children into a deep sleep, whereas alpha, theta and delta entrainment will not. The mechanism behind this counterintuitive result may be that of dissociation, or perhaps beta AVE suppresses slow frequencies and thus puts on "the brakes," which in turn allows the child to relax.

CLIENT INFORMATION:

Condition: ADHD, Anxiety and Obsessive-Compulsive Disorder

YOB: 1958

Sex: Male

Handedness: Right

Medications: None

HISTORY:

This is a case of a 50-year-old male diagnosed with ADHD, but he also complained of intense generalized anxiety for "no good reason." Figure 15 shows his rough EEG, flooded with anxiety-producing beta EEG activity. Figure 16 shows his qEEG as compared with a normative database. Notice his excessive activation from beta activity as he was experiencing quite severe anxiety (pink areas from 13 to 15 Hz and 29 to 35 Hz).

To see if beta frequency AVE (17 Hz) would reduce his anxiety in the same manner that would put an ADHD child to sleep, he was placed on a DAVID Delight AVE unit using squared sine-wave photic stimulation and isochronic auditory tones. Within four minutes of AVE, his excessive beta activity quickly vanished along with his anxiety, as shown in Figure 17 (heartbeat artifacts are apparent in both Figure 15 and 17). Figure 18 shows his qEEG as compared with a normative database. Notice the difference between his pre-measures (Figures 15 and 16) and his post measures (Figures 17 and 18). Notice the calmer brainwaves in Figure 17 versus Figure 15. The calming effect lasted for about two days. He began a program using either beta or beta/SMR AVE every morning upon wakening so that he could go about his day with a calm demeanor.

Figure 15. Raw EEG of a 40-Year-Old Male with ADHD and Severe Anxiety.

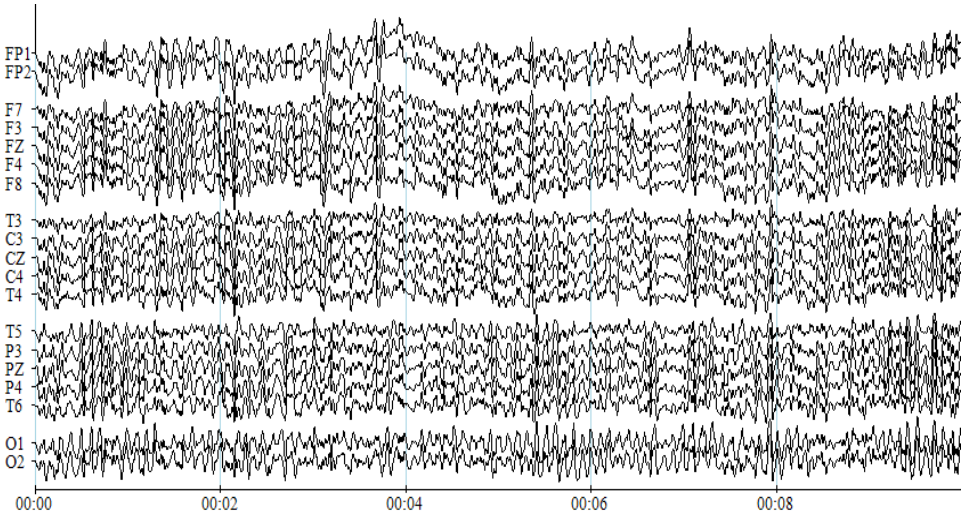


Figure 16. QEEG of a 40-Year-Old Male with ADHD and Severe Anxiety.

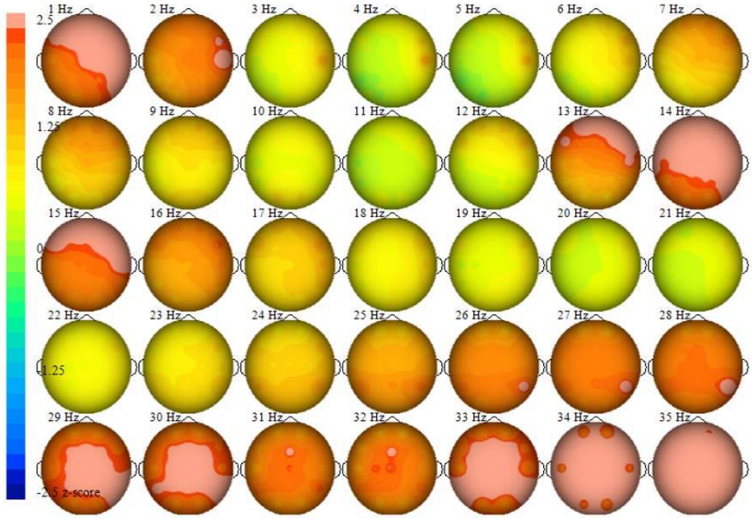


Figure 17. EEG of a 40-Year-Old Male with ADHD and Severe Anxiety.

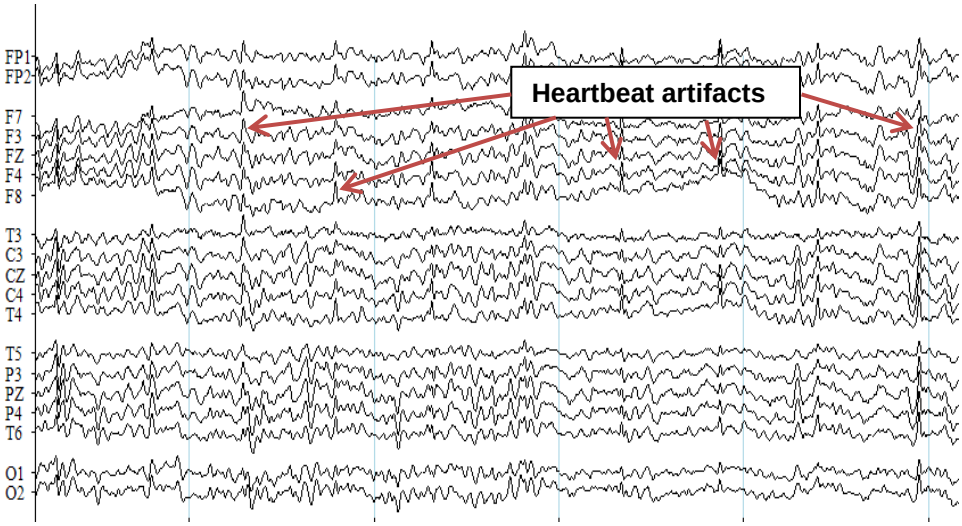
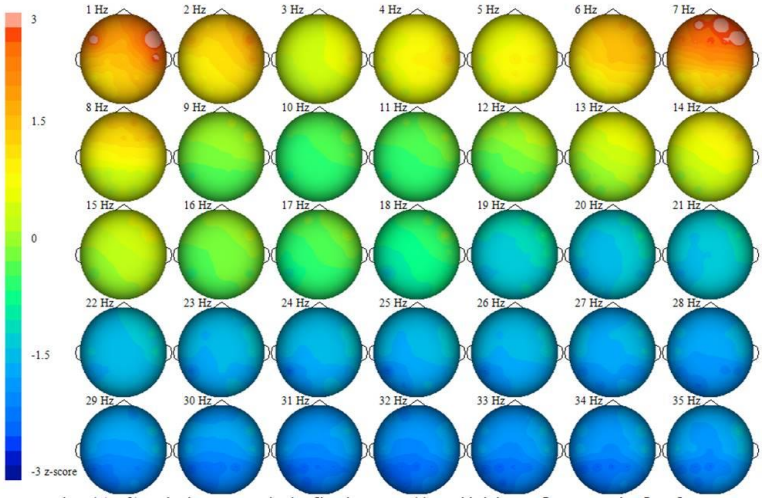


Figure 18. QEEG of a 40-Year-Old Male at Four Minutes of 17 Hz AVE.



Traumatic Brain Injury (TBI)

An excellent literature review by Ribbers in 2010, outlined the economic, emotional and socio-economic impact of TBIs of all types. In the USA 1.6 million people sustain a TBI annually, roughly 50,000 people die from a TBI and one year after injury, 125,000 continue to be disabled (Finkelstein et al., 2006). A broad range of psychological illnesses result from thalamic dysconnectivity. A study of 243 individuals at clinical high-risk for psychosis found that thalamocortical disconnects were particularly pronounced in the individuals who developed full-blown illness. A pattern of widespread hypoconnectivity between the thalamus, prefrontal and cerebellar areas (Anticevic et al., 2015; Dikmen et al., 2009) indicated clear evidence of an association between penetrating head injury and long-term cognitive impairments six months or longer post-injury. There were strong correlations between pre-post intelligence, volume of brain tissue lost and the brain region damaged. A systematic review on social functioning after TBI was performed by Temkin and colleagues in 2009 at least six months after the injury. They concluded there was a direct relationship between the severity of the injury and social outcomes. Depression and anxiety are highly correlated with TBI and persist for many months (Bombardier, 2010).

Thalamocortical Issues: Injuries or Interruptions of the Diffuse Axonal Type

The most common type of TBI is an injury or interruption of the diffuse axonal type (DA-TBI). A DA-TBI is a frequent result of traumatic acceleration/deceleration or rotational injuries of the head and sometimes even a fever. Depending on severity, symptoms range from mildly noticeable to persistent vegetative state in patients. DA-TBIs are most commonly the result from sports injuries and motor vehicle accidents and the most significant cause of disability in patients with traumatic brain injuries. It is called diffuse because unlike some other brain injuries that are localized in one area, a DA-TBI is widespread, usually affecting large brain areas. DA-TBIs are

described as the formation of several small tears or distortions at the gray-white matter junction and within the corpus callosum or other networks.

Fever has also been found to cause loss of brain function. Thompson, Pinto-Martin and Bullock, in 2003, found that patients with DA-TBI present on imaging studies were over nine times more likely to develop neurotrophic fever in patients with DA-TBI versus other forms of TBI (patients without diffuse axonal interruptions), all other factors being equal. Fever can also follow stroke and cardiac bypass surgery (Ginsberg & Busto, 1998). In a study of 110 patients admitted within 24 hours of stroke, more severe symptoms were associated with fever and *subfebrility* (temperatures between 37.5°C and 38.0°C) (Hindfelt, 1976).

Following a brain injury, the brain goes into a state of shock, called diaschisis. During diaschisis, inflammation is high and heavy energy demands are placed on the brain to facilitate repair. All of these events affect neurons, not only in the injured areas, but throughout the brain (Doidge, 2015, p 86). TBI-triggered thalamic inflammation has been linked to being caused by neuro-inflammation in the form of glial activation triggered by the TBI and has been shown to persist up to 17 years after traumatic brain injury (Ramlackhansingh et al., 2011). Chronic activation of microglia is implicated in many neurodegenerative disorders. In 2015, Scott and his colleagues further demonstrated using diffusion MRI to estimate axonal injury and show that thalamic inflammation is correlated with thalamocortical tract damage.

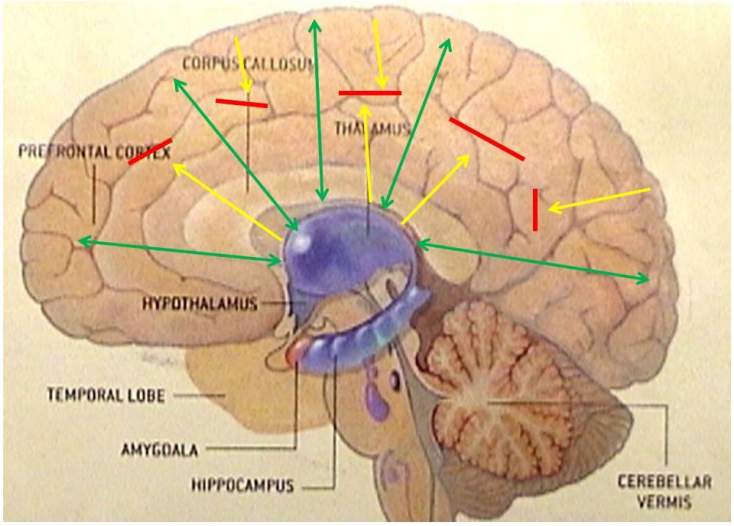
With hundreds of millions or even billions of neurons taken off-line, the famous phrase coined by Carla Shatz in 1992: "*neurons that fire together, wire together and neurons that fire out of sync, lose their link,*" principle comes into play and affected neurons continue to stay off-line from becoming disconnected in the first place.

A DA-TBI is best expressed as thalamocortical disconnectivity (TCD). The alpha rhythm is generated by billions of synchronized cortical-thalamic circuits and healthy alpha is close to 10 Hz (Schreckenberger, et al., 2004). In the case of a DA-TBI, there is an electrical blockage to either axons or dendrites in the loop between the cortex and the thalamus, as shown in Figure 19. This causes two things to occur:

1) the alpha rhythm becomes attenuated or eliminated entirely in certain brain regions. Typical eyes-closed alpha amplitude in healthy people shows an alpha/beta ratio (8-12Hz range / 20-30 Hz range) of approximately three or more (Kaiser, 2007). In those with DA-TBIs, the alpha/beta ratio is often less than one. This low alpha activity, coupled with excessive beta activity results in subjective feelings of agitation and anxiousness.

2) because the affected neurons are no longer receiving a synchronization pulse from the thalamus, they begin oscillating on their own in the 1 to 2 Hz range (low delta) (Thatcher, et al., 1989; Steriade, et al., 1993). Now that the neurons are oscillating without a synchronization pulse, delta phase is asynchronous and disconnected in relation to a database of a healthy brain and the delta phase measure will show abnormalities.

Figure 19. Diffuse Axonal Interruption or Injury Showing Thalamocortical Disconnectivity (Image: Scientific American).



Delta Phase Indicators of a DA-TBI: As mentioned, with the loss of the thalamic sync pulse, neurons no longer fire in rhythm and low-frequency endogenous random firing of neurons is revealed by poor delta phase. This random firing also makes the EEG appear "noisy," showing increased choppy-looking beta activity (Doidge, 2015, p 103). Individual neurons firing randomly would theoretically wash out in the KHz range and not be observable in the EEG. Of the 66 Rich Club neuronal hubs, consider that the thalamocortically disconnected hubs were firing randomly as complete units and in the 1 to 2 Hz range as shown by Thatcher, et al., 1989. Then these hubs would produce increasingly choppy EEG in correlation with an increasing number of disconnected hubs. With increasing severity of a DA-TBI, beta phase discontinuities would also increase in frequency. Low frequency beta phase abnormalities would appear in relation to small injuries while gamma phase discontinuities would appear in relation to large injuries.

The author has worked for several years with people struggling in the aftermath of a head injury and has observed alpha drop out in everything from forceps deliveries, fevers, physical head injuries and unknown causes. However, a lack of understanding of the underlying physiology within the psychological and psychiatric professions may result in many of those with DA-TBIs to be misdiagnosed as other disorders including the low alpha/high beta type of anxiety.

Beta Phase Indicators of a DA-TBI: The following scenario serves as an analogy to clarify synchronized delta versus asynchronous delta. Imagine you are at a concert listening to a band playing. The beat of the song is 120 beats/minute (2 Hz). The audience begins clapping their hands to the beat of the music, so everyone is doing so at two claps per second (delta). If a microphone was in the room processing the sound of the clap, spectral analysis would indicate it to be at 2 Hz. But once the song has ended, every person in the audience applauds the performers. Although each person continues to clap at roughly two claps per second or 2 Hz, but in their own time, everyone's clapping becomes desynchronized. Now thousands of

desynchronized claps are reverberating throughout the auditorium and a roar envelopes the room. And the larger the audience, the higher in pitch the "roar" appears (imagine the clatter from an audience of 10 people versus the roar from an audience of 1000). These disconnected claps would appear as high frequency noise, even though the sources are all at roughly 2 claps per second (2 Hz). In the brain, once the neurons (at 1 to 2 Hz) are desynchronized and firing in their own time, we would expect to see desynchronized beta and therefore a beta phase desynchrony. So far, few analyses have been done, but observations have shown beta phase desynchrony in the 15 to 40 Hz range, with the desynchronized beta frequency dependent on the size of the TCD. The larger the region of TCD the higher the frequency of beta phase discontinuity (n=10). These discoveries are in their infancy and require more exploration.

DA-TBI Case Examples

The following three examples demonstrate the diversity of presenting symptoms of DA-TBIs. We will review a young woman with anxiety and anorexia possibly resulting from a fever, a woman with lost cognition following a motor-vehicle accident and a young man dealing with binge alcoholism whose head injury as a child was a result of his skull being crushed by close to 2.5 cm (1") after a fall.

The remarkable aspects of this population are just how similar their brainwaves are, as they all show alpha dropout and poor delta phase. It's true that there are mild variances between them; the anorexic shows some signs of OCD and the young man with the childhood head injury shows severe alpha loss and delta phase problems. Despite these small variances, the underlying nature of a Da-TBI may manifest into a ***spectrum of disorders***. Despite the clinical diagnosis, these disorders should not be considered as entities in themselves until a DA-TBI has been ruled out.

AVE's Surprise!

To reestablish the lost alpha activity, it always seemed logical to provide entrainment at an alpha frequency. However, this technique has not been overly successful. What came as a surprise were two things: 1) beta stimulation at 20 Hz would reestablish an alpha rhythm at around 10 Hz, and; 2) dual frequency stimulation (where the left visual fields and left audio were presented at a different frequency than the right side) would reestablish the alpha rhythm. This technique provided left-side entrainment at SMR (12 to 15 Hz) and right-side entrainment at beta (20 Hz) frequencies. A solid physiological explanation as to why it works is yet to be revealed. All we know is that it produces fast and pronounced effects clinically.

Case Report DA-TBI 1 – Interpersonal Anxiety and Mild Anorexia (with OCD)

Research of Obsessive-Compulsive Disorder (OCD) showed similar brainwave signatures for hoarders, counters, cutters, anorexics and ritualists. Brainwave signatures were primarily slowed alpha with a locus over the anterior cingulate and delta phase issues similar to that of a DA-TBI (Siever, 2013).

CLIENT INFORMATION:

Condition: Generalized Anxiety and Obsessive-Compulsive Disorder (with Anorexia)

YOB: 1997

Sex: Female

Handedness: Right

Medications: 20 mg of Prozac

HISTORY:

This young woman struggled with social anxiety, mild obsessive-compulsiveness relating to her anorexia and delayed sleep-onset insomnia for about three years. To the observer, she appeared relaxed, peaceful and calm, but she was much more anxious than she appeared. She also had quite a tense left-side masseter muscle. She was a light sleeper most of her life. As nervousness grew about the challenges of entering college, her sleep-onset latency increased from approximately 20 minutes to two hours and her desire to eat was diminished.

Assessments and Work Done

1) While it's good to treat the qEEG, it's most important to treat the client. Therefore, we have the client track how they think things are going by completing a Symptoms Checklist-90-Revised (SCL-90-R, a 90-question self-reported questionnaire) during treatment. An overall score of 50 is the norm. Higher scores represent a poorer self-assessment than the normal population while lower scores indicate better than normal. A score of 60 is one standard deviation off the norm. Scores higher than 60 are considered clinical.

2) A 19-channel (10-20 montage), referential/linked-ears EEG and qEEG was completed for eyes closed, eyes open, AVE with eyes closed and post AVE with eyes closed.

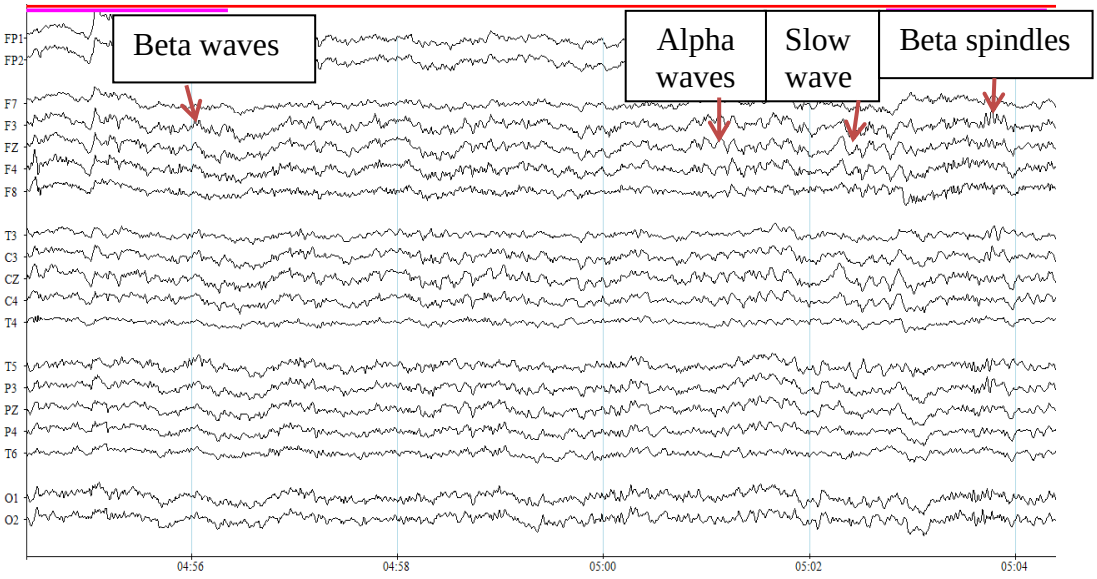
3) AVE at alpha/beta (left side at 10 Hz and right side at 19 to 21 Hz using headphones and split-field eyesets) frequencies to inhibit her slow brainwaves was used while her brainwaves were being recorded.

4) The client was given a DAVID Delight Pro, which uses a combination of AVE and Cranio-electro stimulation (CES). CES presents a small electrical pulse across the cranium. The resulting stimulus flows across the brain stem and primarily stimulates serotonin and endorphin production making it beneficial for calming the mind and reducing pain.

EEG and qEEG Analysis

In the snapshot of EEG activity shown in Figure 20, both slow wave and fast wave EEG are present. Here we see some spindled beta in the frontal, temporal, parietal and occipital regions. This generally represents an anxious and unsettled person. Fast brainwave activity may contribute to feelings of anxiety. FZ, CZ, and PZ (and to a lesser degree C3 and C4) are quite high in slowed alpha waves. Slowed activity over FZ and CZ (plus delta phase issues) are indicators of OCD.

Figure 20. Raw Waveforms Pre AVE – Eyes-Closed Condition.



What we see here in her eyes-closed analysis is slowed activity with the locus along the midline at FZ and CZ primarily (Figure 21), which is characteristic of OCD. When her data is compared against the SKIL database (Figure 22), it is apparent that she shows some degree of alpha/theta dropout across the frequency range of 4 to 16 Hz, with a significant loss at 10 Hz. To the best of her and her mother's recollection, she has never sustained a head injury, but has had fevers relating to viral infections. Therefore, this may not be a sign of concussion, but relating to a fever at one time in her life, leaving her with a diffuse axonal interruption, but not injury per se.

Figure 21. Absolute Magnitude Frequency Analysis in 1 Hz Bins as Raw Data in µVolts.

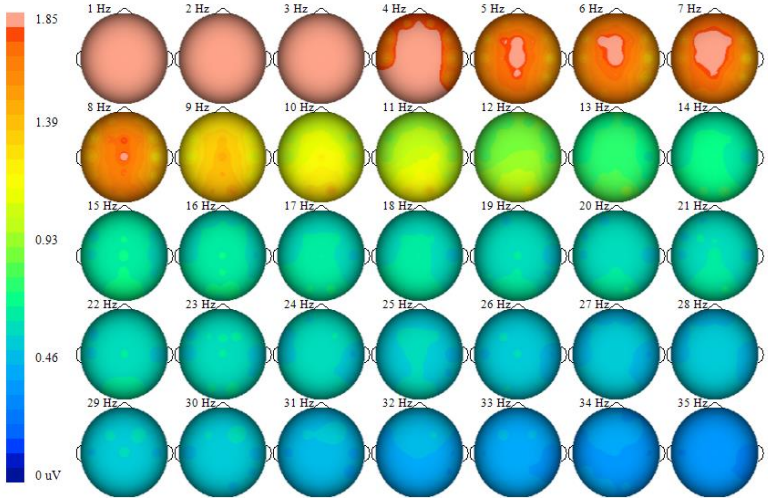
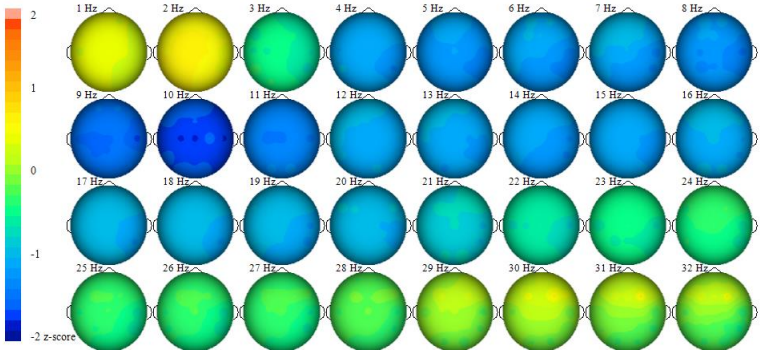
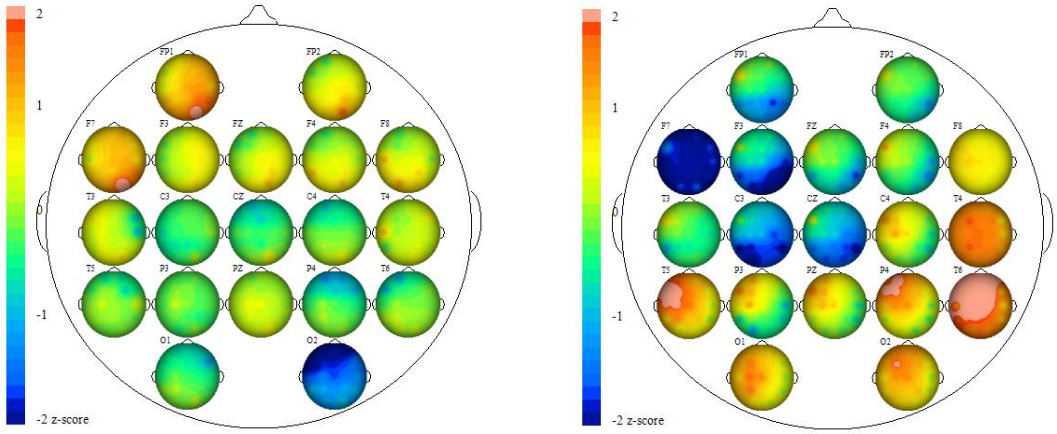


Figure 22. Absolute Magnitude Frequency Analysis as Compared Against a Database in 1 Hz Bins.



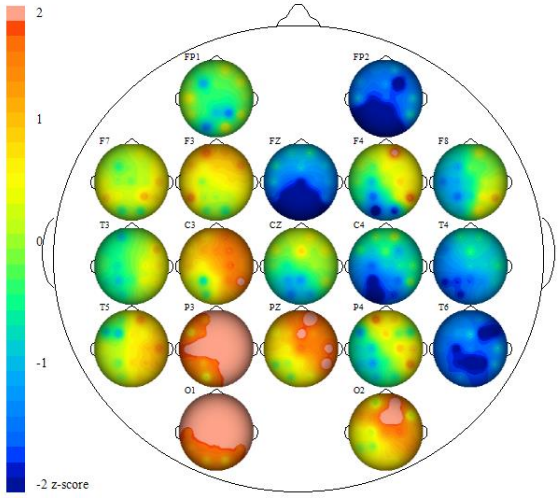
Data from several DA-TBI cases suggest that phase issues are generally not significant in the theta range. However, in this case, theta phase in both the eyes-closed and eyes-open conditions were more pronounced than with delta (Figures 23 and 24). In Figure 24, theta phase issues in the eyes-open condition (left-frontal to right posterior temporal) was particularly pronounced.

Figure 23 and 24. Theta Phase Eyes-closed and Eyes-open as Compared with a Database.



With this person, beta phase issues were most prevalent in the 24 to 30 Hz range (eyes-closed), but also strong in the low gamma range from 30 to 36 Hz, as seen in Figure 25. Beta was less significant in the remaining examples.

Figure 25. Beta Phase in the 30 - 35 Hz Range as Compared Against the SKIL Database.



EEG Assessment during Alpha/Beta AVE (Eyes-Closed Condition)

During AVE, this young woman (also with a sleep disorder), began generating spindled alpha at about six minutes. At nine minutes, she began producing large delta waves (Figure 26). Figure 27 shows her fast asleep, which is typical during an AVE session and yet counterintuitive, as it would seem difficult to fall asleep while being in a strange room with someone watching you and using higher frequency AVE. She has been using the alpha/beta entrainment at bedtime in her home since her initial visit and is sleeping well and no longer struggles with anxiety or interpersonal relations.

Figure 26. During Left-Side Alpha and Right-Side Beta AVE Using Headphones and Split-Field Eyesets.

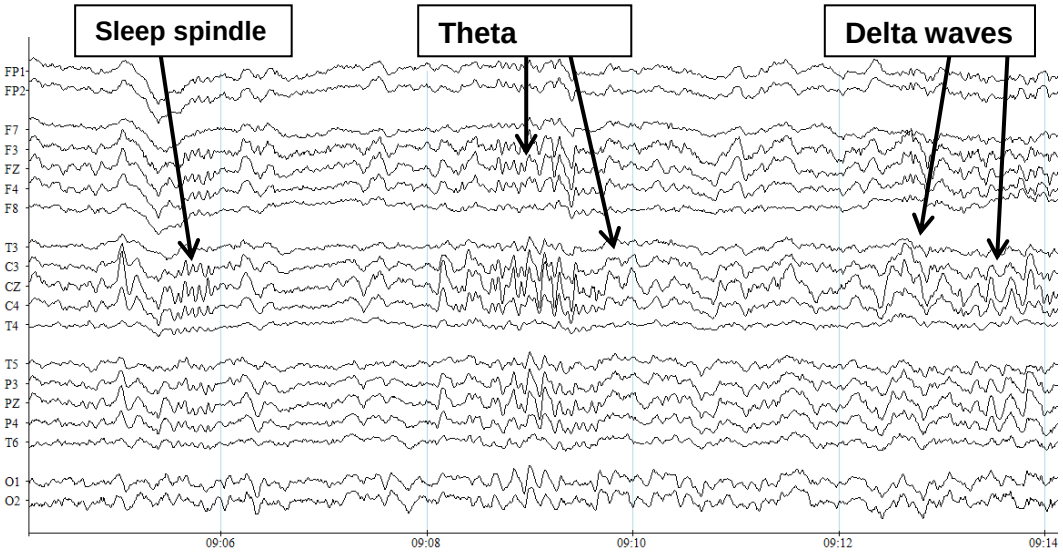
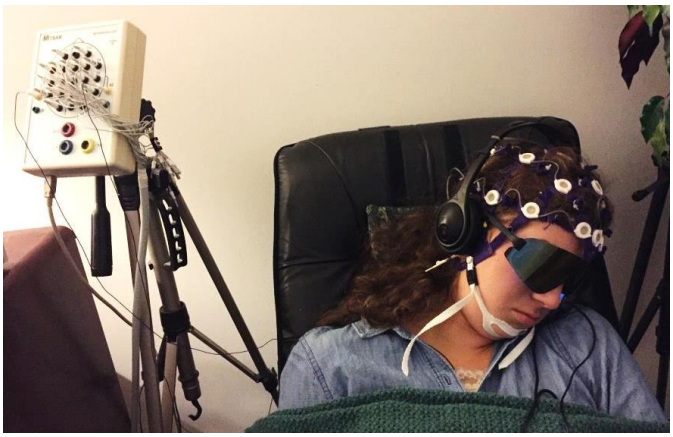


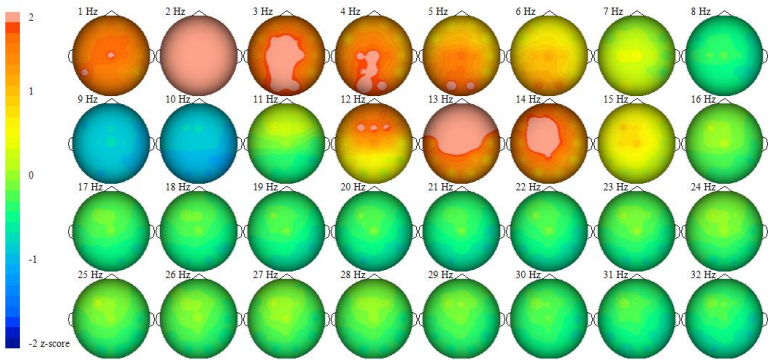
Figure 27. Subject is in Deep Sleep During Alpha/Beta AVE.



EEG was recorded during alpha/beta AVE. This stimulation provides 10 Hz on left visual-fields and ear and 20 Hz on her right visual-fields and ear. In Figure 28, we see her qEEG compared against the SKIL normative database. Her alpha has been clearly re-established, with a diffuse peak from 13 to 15 Hz. For reasons unknown to the author, this is a frequent occurrence during AVE. but settles over a few hours and appears to dissipate with ongoing AVE use.

Neither her theta or beta phase improved during AVE and unfortunately a post AVE recording was not taken with this subject.

Figure 28. Subject Shows Elevated Low Beta During Alpha/Beta AVE.



Cognitive/Emotional Self-Report

Her pre-treatment SCL-90-R score was 71 points. In Table 2, "1" is average and some items are higher than average or lower than average. Notice in Table 2, she believes to have major concerns with Interpersonal Sensitivity, Paranoid Ideation and Sleep (being of most concern). Other items are very low. When considering her low alpha activity and high beta activity, she is prone to anxiousness. Given that she is a socially active teenager, it makes sense that interpersonal sensitivity would score high. Following three weeks of AVE, her sleep was much improved, her daytime energy was good and her anxiety was all but gone.

Table 2. Pre & Three Weeks Post Alpha/Beta AVE Results as Indicated on the SCL-90-R.

	Pre AVE Scores	Post AVE Scores
Somatization	0.09	0.27
Obsessive-Compulsive	0.80	0.20
Interpersonal Sensitivity	1.67	0.00
Depression	1.00	0.00
Anxiety	0.70	0.00
Hostility	0.83	0.00
Phobic Anxiety	0.00	0.00
Paranoid Ideation	1.50	0.00
Psychoticism	0.20	0.00
Grand Total Raw Score:	71	5
Global Severity Index:	0.79	0.06
Vegetative Depression	0.7	0.0
Suicide Ideation	0.7	0.0
Dysfunctional Sleep	2.0	0.0
Chronic Fatigue	0.4	0.2
Decreased Cognitive Functioning	0.4	0.3

Findings and Conclusion

Her life has changed dramatically. As a result, she has put on 10 pounds of weight over a few months and has been feeling great. After one year of using AVE, she continued to do well and used her DAVID device on an as-needed basis. She was much more relaxed in school while getting good grades and her mother's own anxiety from concern for her had diminished greatly.

Case Report DA-TBI 2 - Jumpstarting the Brain with SMR/Beta AVE Following a Motor Vehicle Accident

CLIENT INFORMATION:

Condition: Closed-Head Injury from a Motor-Vehicle Accident

YOB: 1972

Sex: Female

Handedness: Right

Medications: None

HISTORY:

This wife and mother of two teenage boys was in a motor vehicle accident in 2011. She was in immediate pain and developed the following neurological issues as per her self-assessment.

1) Mental exhaustion and cognitive disconnects. Here are some quotes from client:

- "I would do an activity one day and it would not hurt while I was doing it but I would be very sore and exhausted a day or two later and it would often take days or weeks to recover."
- "I would have a conversation with someone and my mind would go blank talking to them. Then a day or two later, out of the blue, I would think of what I should have said to them."
- "I became unable to organize things in my life - my organizational skills disappeared."

2) Loss of spatial ability: She was bumping into everything and frequently dropping things.

3) Income stress: She has not been able to work since the accident.

4) Left temporal lobe issues: She reported having problems processing language and reading (Wernicke's Area). She also reported having problems expressing her thoughts and communicating effectively with people. This may reflect an issue with Broca's Area as well.

5) The client reported having trouble remembering, learning new things and fatigue. It's difficult to determine if this was an issue with the structure of her hippocampus (a key area involved with long-term memory), or if it was stress/anxiety related – as stress and anxiety impact this area significantly.

6) Given the client's brain showed desynchronization across most regions in coherence, comodulation and phase, she would struggle to focus on daily issues. Cognitive functioning involves the simultaneous integration of several brain areas. This explains why she struggled to organize her thoughts and actions.

Assessments and Work Done

1) SCL-90-R was recorded before AVE and three weeks and twelve weeks after AVE use.

2) A 19-channel (10-20 montage), referential/linked-ears EEG and qEEG was completed.

3) AVE at SMR/beta (left side at 14 Hz and right side at 20 Hz using headphones and split-field eyesets) frequencies to inhibit her slow brainwaves was used during EEG recording.

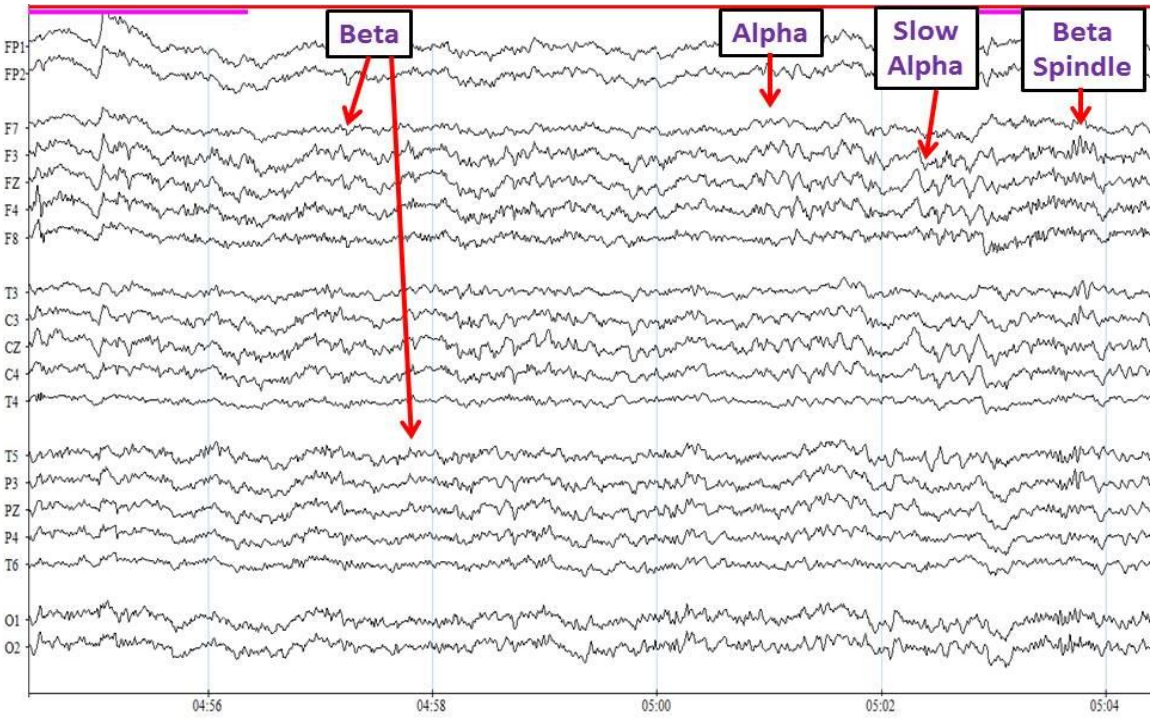
4) The client was given a Delight Pro, which uses a combination of AVE and CES, to clear her head and reduce her pain.

5) One month later, she was placed on several applications of transcranial DC Stimulation (tDCS) over T5 to help improve language and name recall. TDCS is the application of a direct current used to excite neuronal activity and performance. There are about 800 studies on tDCS to date with many of them showing clinical improvements such as motor and aphasias resulting from stroke.

EEG/qEEG Analysis (Eyes-Closed Condition)

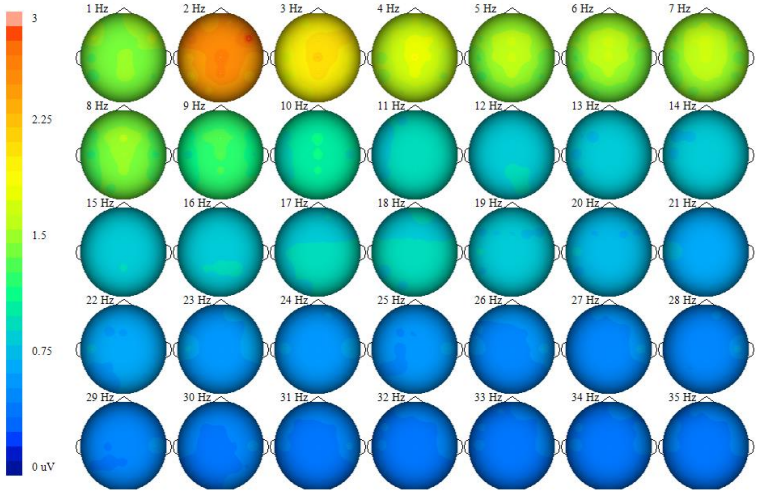
Referring to Figure 29, we see plenty of eye twitches. Note the ticks on FP1 and FP2, F7 and F8. This generally represents anxiety or sensitivity to anxiety. In this snapshot, small quantities of alpha waves are present only on certain channels and both slowed alpha waves and beta waves are present. Both of these abnormal brainwaves contribute to feelings of anxiety. Fast waves are often a side effect of too many slow waves and by speeding up the brain, the fast waves will diminish. FZ and CZ are high in slowed alpha. All channels are high in random beta and beta spindle activity.

Figure 29. EEG Prior to SMR/Beta AVE.



When looking at raw EEG, a spike in activity at 10 Hz (alpha) should be dramatically apparent, but only a slight amount of alpha is evident (Figure 30). This indicates an overall low-voltage EEG. Compare this against the alpha peak following AVE treatment as shown in Figure 35.

Figure 30. Raw Values of Absolute Magnitude Frequency Analysis in 1 Hz Bins at a 3 µVolt Scale.



The database indicates that the primary alpha frequency (9 to 11 Hz) is quite low (Figure 31). This beta frequency "noise" as shown above, is common in those with DA-TBI. Given the low alpha and excessive beta brainwaves, feelings of agitation and anxiousness plus difficulty in ability to settle down and focus are typical (this behavior is similar to that of an ADHD child).

Figure 31. Absolute Magnitude Frequency Analysis as Compared Against a Database in 1 Hz Bins.

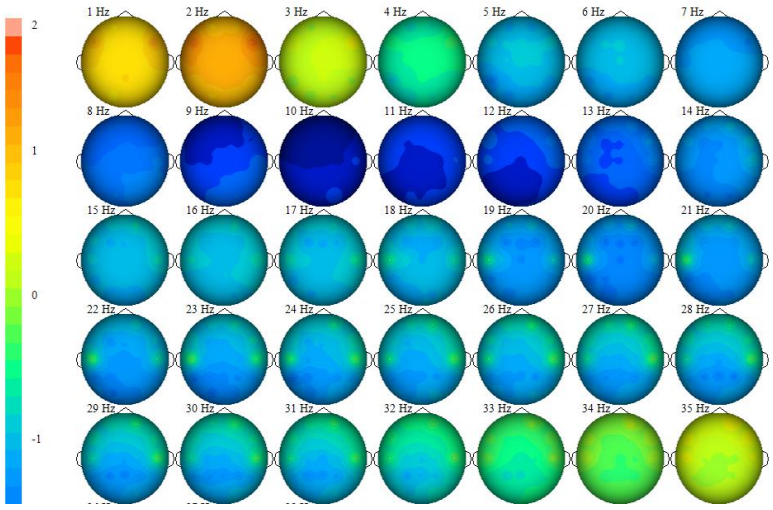
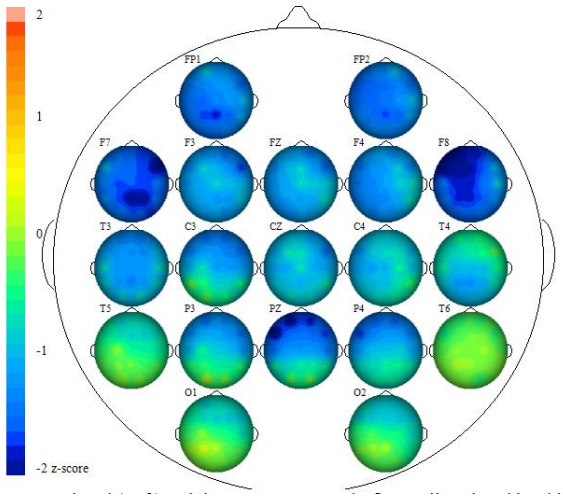


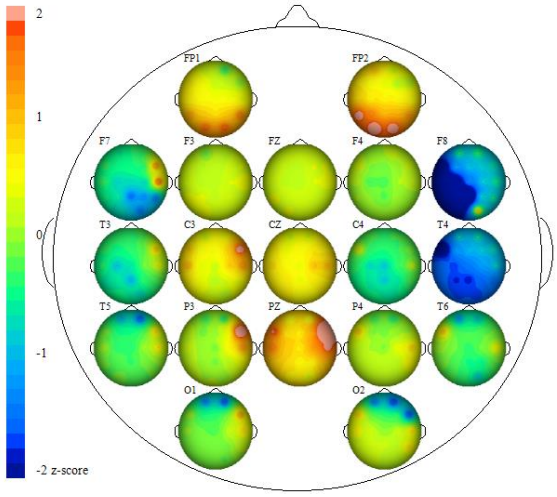
Figure 32 shows desynchronization in alpha (areas in blue) between several areas affecting reasoning, attention, verbal expression, emotional expression, cognitive processing and impulse control.

Figure 32. Alpha Comodulation as Compared with a Database - Eyes-Closed.



In Figure 33, we see several phase issues relating to a lack of connectivity between right temporal and frontal/occipital regions - another sign of a DA-TBI.

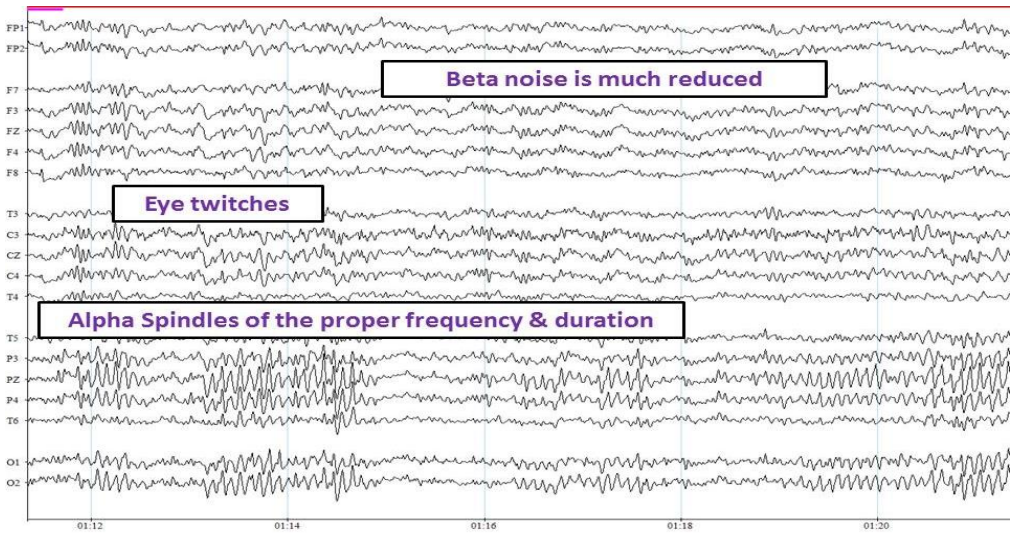
Figure 33. Delta Phase as Compared with a Database.



EEG Assessment during SMR/Beta AVE (Eyes-Closed Condition)

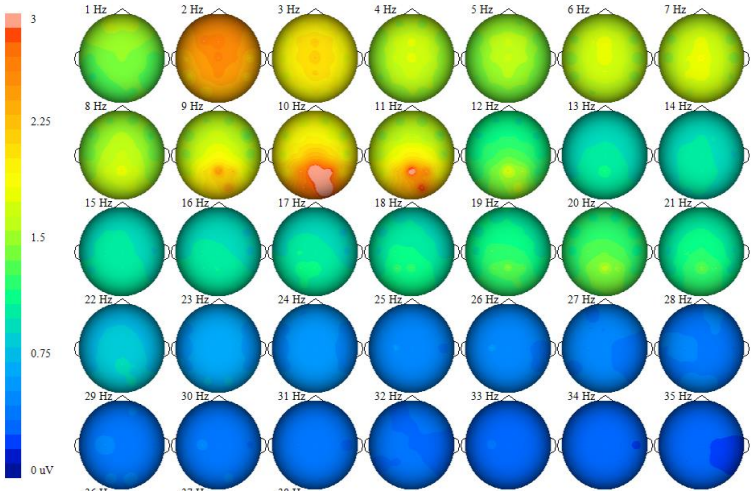
SMR/beta AVE involves stimulation in the 12 to 15 Hz range on the left side and 19 to 21 Hz stimulation on the right side. At roughly 20 minutes of SMR/beta AVE, alpha spindles began forming (Figure 34). When the session ended at 30 minutes, she exclaimed "Wow! My head is clear and I can think!"

Figure 34. Raw Waveforms Following 20 Minutes of SMR/Beta AVE.



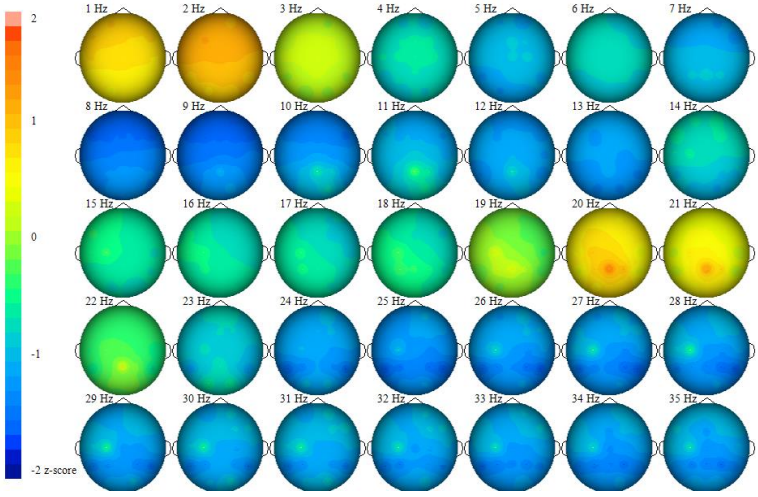
The stimulation recovered her lost alpha rhythm. In Figure 35, we see an alpha EEG from 10 to 11 Hz, which is where it should be (right parietal/occipital).

Figure 35. Absolute Magnitude Frequency Analysis in 1 Hz Bins at a 3 µVolt Scale.



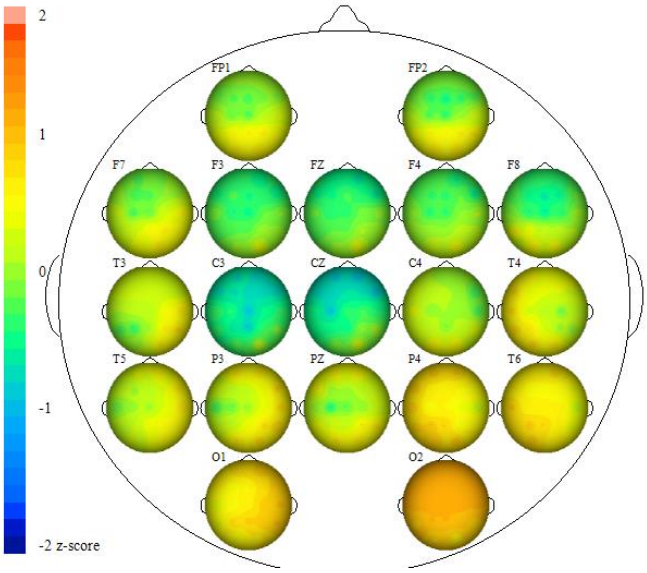
This is a fairly normal brain map as compared with the database (<1 SD). The main difference is the heightened 20 to 21 Hz activity - a result of the entrainment and not of concern (Figure 36).

Figure 36. Absolute Magnitude Frequency Analysis as Compared Against a Database.



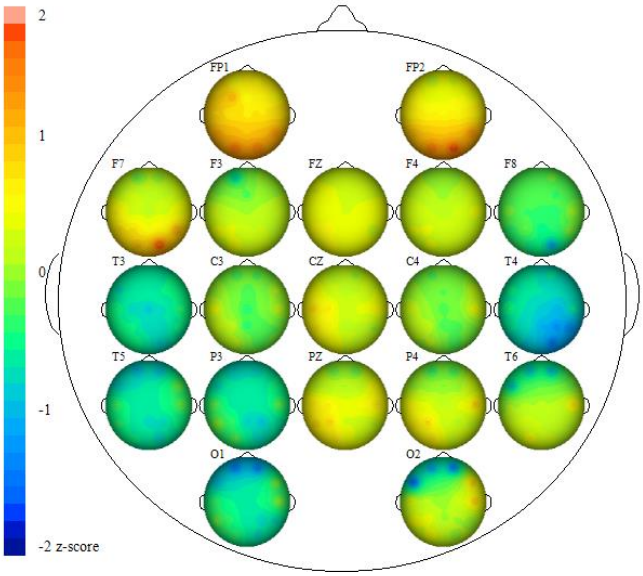
Comodulation is close to normal and not considered clinical (Figure 37).

Figure 37. Alpha Comodulation as Compared Against a Database.



Given that the neurons now have a thalamic synchronization pulse, the delta phase has been restored. There is mild frontal-occipital hypo-coupling, which is not considered clinical (Figure 38).

Figure 38. Delta Phase as Compared Against a Database.



In Table 3, we see her SCL-90-R results before and after using an SMR/beta AVE protocol for three weeks. The raw score dropped from 72 down to 10 in just three weeks. At three months, her score was further reduced to 5. This reflects a dramatic improvement in her state of mind.

We can see her most significant improvements are in somatization, obsessive-compulsiveness and depression, suicide ideation, fatigue, cognitive function and pain. Although never officially diagnosed with OCD, as a once independent, take-charge kind of person, she was constantly fixated on her issues and inability to resolve them. This increased focus also increased her perception of pain.

Table 3. SCL-90-R Results Pre and Post Three Weeks SMR/Beta AVE.

	Raw Scores Pre AVE	Raw Scores 3 Weeks Post AVE
Somatization	1.09	0.09
Obsessive-Compulsive	2.50	0.40
Interpersonal Sensitivity	0.67	0.11
Depression	0.77	0.08
Anxiety	0.70	0.10
Hostility	0.33	0.17
Phobic Anxiety	0.00	0.00
Paranoid Ideation	0.33	0.00
Psychoticism	0.20	0.00
Grand Total Raw Score:	72	10
Global Severity Index:	0.80	0.11
Positive Symptom Distress Index:	1.31	0.18
Vegetative Depression	1.2	0.0
Suicide Ideation	0.9	0.3
Dysfunctional Sleep	1.3	0.0
Chronic Fatigue	2.4	0.2
Decreased Cognitive Functioning	2.3	0.5
Physical Pain	0.8	0.3
Pain vs Somatization Index	0.7	0.0

Findings and Conclusion

1) She reported anxiety, loss of emotional control and impulsiveness, coupled with feelings of hopelessness and financial worries. Her magnitude qEEG analysis (not included) shows an OCD signature, but it's not known if her obsessiveness is inherent or developed as a result of her frustration with her condition and the medical profession.
2) Being that she was in litigation with the party whose car had collided with her, her lawyer had been reminding her for most of a year to write a victim-impact statement. Due to her DA-TBI, she had not yet managed to start writing her statement. Fortunately, after about three weeks of AVE, she wrote a full 15-page report, which was instrumental in her receiving compensation.
3) She is now doing well, is running her own business and was even able to coordinate a move into a larger new home.

Case Report DAI 3 - Jumpstarting the Brain of a Binge Alcoholic Using Alpha/Beta AVE

A low alpha/high beta EEG signature is typically associated with a tendency towards alcoholism. However, research has yet to determine if there is a phenotype of the low alpha/high beta type exclusive of DA-TBI causes of alcoholism. In this case, it is certain that DAI is a contributing or the sole cause of the alcoholism. It's possible that all low alpha/high beta signatures may lead to alcoholism simply to calm down an agitated and unsettled brain, whatever the cause.

CLIENT INFORMATION:

Condition: Alcoholism, Anxiety, Poor Sleep YOB: 1984

Sex: Male Handedness: Right

Medications: 75 mg of Effexor

HISTORY:

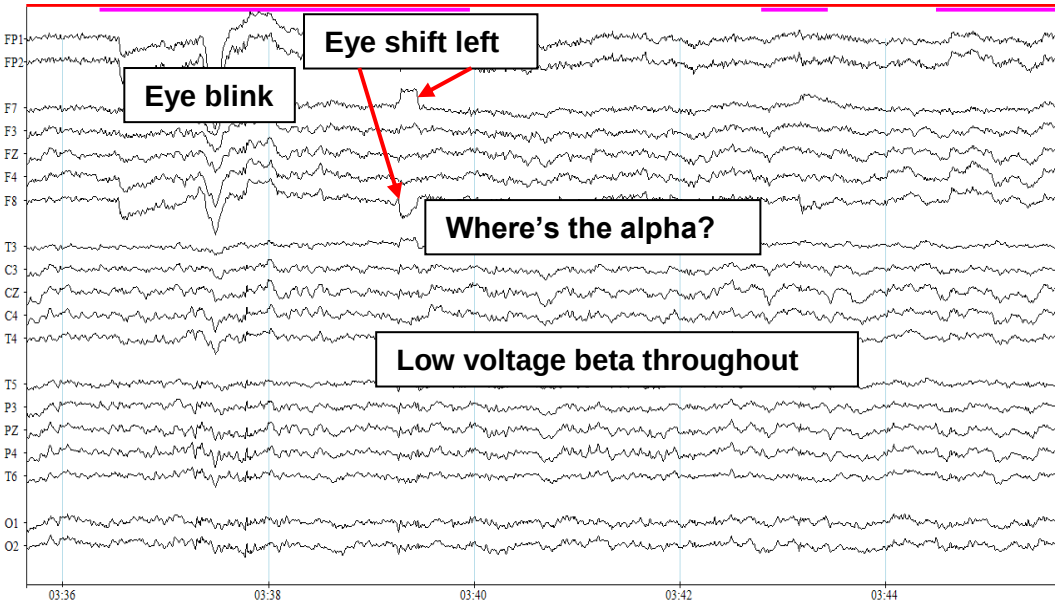
This 31-year-old man has been struggling with alcoholism for many years. He has poor grammar and difficulty maintaining employment due to his drinking. He was excessively aberrant in other measures such as comodulation and coherence, but for the purpose of this article, the focus of this investigation will remain with magnitude and phase as these are the main measures presumed to be vital in assessing DA-TBIs. The client's initial EEG-based gains were not as great as in the previous cases. However, his clinical improvements as indicated on the SCL-90-R combined with his reduced abusing of alcohol point to signs that the AVE was helping.

Following his EEG assessment, he was asked if he had ever sustained a head injury. Here is his history in his words:

- "Closed-head injury from a young childhood fall."
- "In Grade 1, I had a slide accident with major trauma to my left skull. Caved it in about two cm. I was in and out of consciousness. I don't feel like it has affected me and my life has been normal since. I've always lacked motivation."
- "We were in an inner tube being pulled by a skidoo. My leg got caught in the middle of the tube and I was sucked under. I was knocked unconscious."
- "Since high school, I had trouble socializing and was often a loner and binge drinking on weekends and then later on during the week."
- "In 2013, I lost my license in July from a DUI," (driving under the influence of alcohol).
- "In 2014, I spent six months at a treatment center."
- "In 2015, I spent seven weeks in rehab. I snuck in alcohol and was kicked out."
- "February 4, 2016. That was my last drink. Then I spent five days in detox."

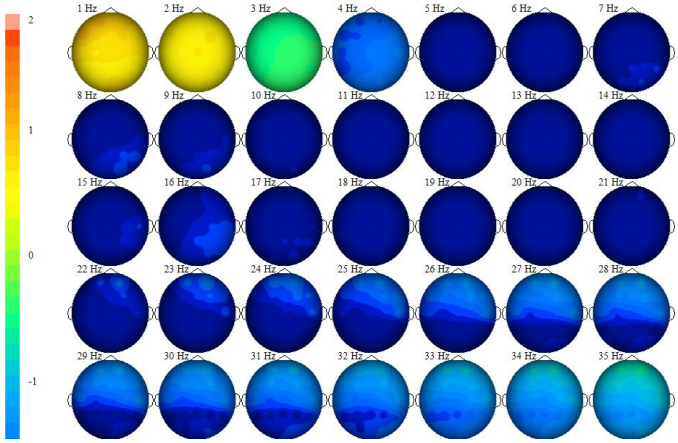
He had difficulty settling down during his EEG. What began as excessive facial tension, settled down to mostly eye blinks and twitches. In Figure 39, notice the low-voltage fast EEG activity and complete absence of alpha, theta and delta activity. The pink lines represent removed artifact such as eye-blinks, lateral eye movements and some posturing movement.

Figure 39. Raw Waveforms Pre AVE – Eyes-Closed Condition.



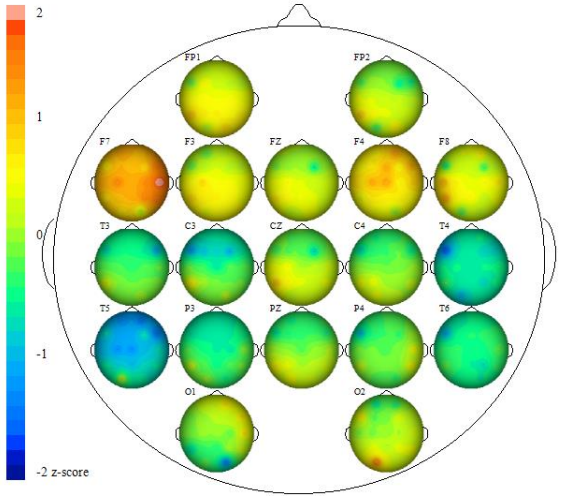
As seen in Figure 40, the brainwave activity is well below the norm, indicating a general brain shut down. This helps to explain his poor language, attention, emotional control and impaired cognition in general.

Figure 40. Absolute Frequency Magnitude as Compared Against a Database (2 SD).



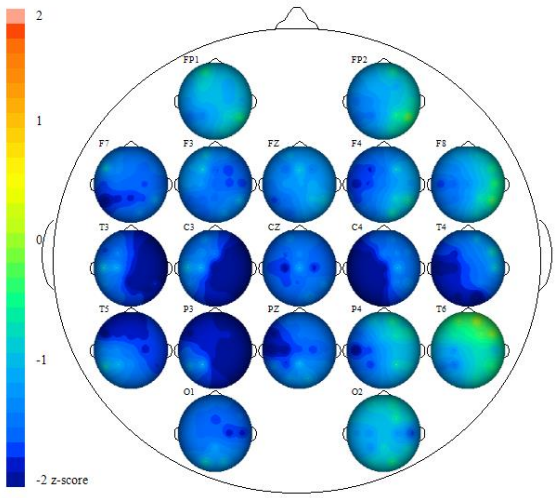
As mentioned previously, delta phase is an integral indicator of a DA-TBI. In Figure 41, however, it looks falsely better than it is. This is because his delta activity is so deficient, that the database has nothing to compare against, making it look better than it actually is.

Figure 41. Delta Phase as Compared Against a Database.



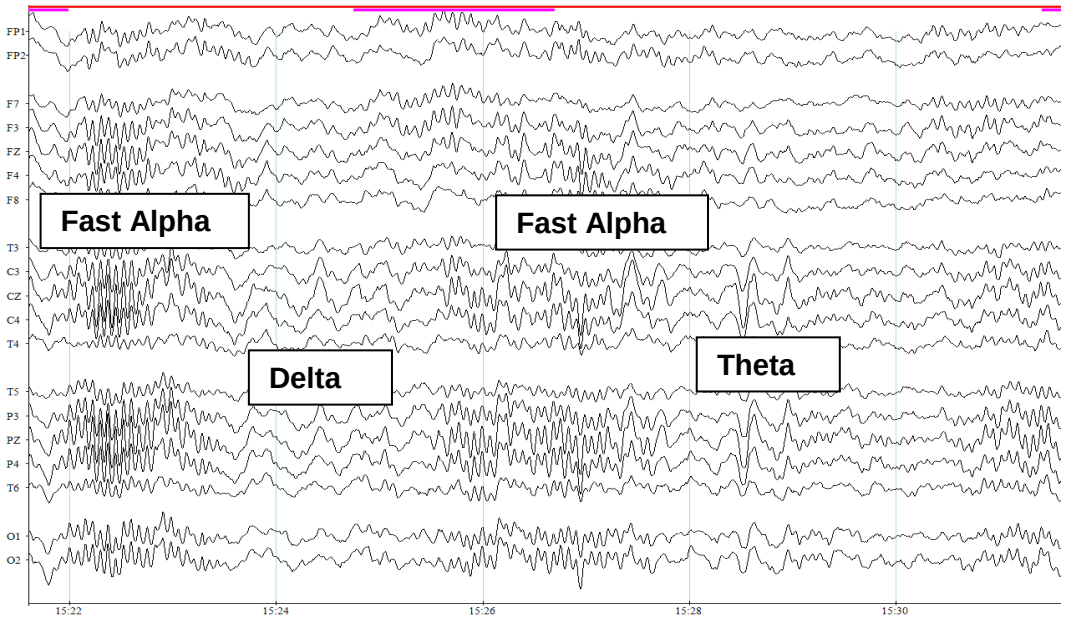
Comodulation is a measure comparing alignment of EEG generators throughout the brain. The larger generators of rhythmic activity are the hippocampal/septal and the thalamus (4 to 8 Hz and 6 to 9 Hz). The lateral geniculate bodies are the primary generators of the posterior alpha rhythm at O1 and O2 seen in EEG. The next generator in the thalamus is the pulvinar nucleus, which also oscillates at around 10 Hz in adults. It can be seen at PZ most prominently and also visible in the posterior cingulate or the cuneus and precuneus. Alpha comodulation, a measurement of alpha spindling, is linked to cognition and sensory integration. In Figure 42, we see the alpha generators have lost synchronization almost completely.

Figure 42. Alpha Comodulation as Compared Against a Database.



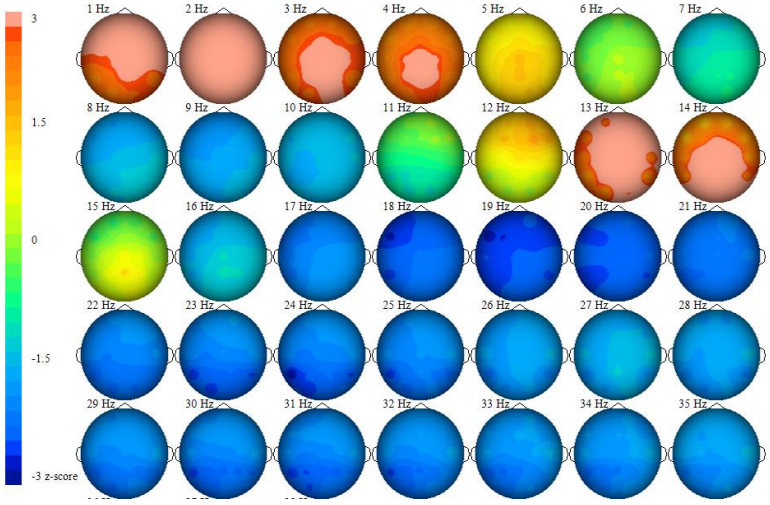
Given that alcoholics generally show a marker for depression, qEEG analysis shows that depression-related substance abuse often entails alpha asymmetry between the left and right dorsal-lateral pre-frontal cortexes (F3 alpha > F4 alpha in the 8 to 11 Hz range typically), so the Mood Booster protocol was administered even though this asymmetry was not observable in his qEEG. This protocol delivers stimulation to the left field using frequencies in the alpha band at 10 Hz and stimulation to the right field using frequencies in the beta range of 19 to 21 Hz. It has been shown that AVE quite effectively inhibits the half frequency of stimulation and therefore suppresses aberrant alpha. Given that the AVE was administered prior to the analysis being done, it turned out the client did not show any signs of depression and so the SMR/beta might have been a better choice. However, alpha/beta stimulation produced remarkable results, nonetheless. In Figure 43, we see recovered alpha spindling and some delta as he was falling in and out of sleep.

Figure 43. Raw Waveforms during 20 Minutes of Left-Side 10 Hz and Right-Side 19 to 21 Hz AVE at the 15-Minute Mark.



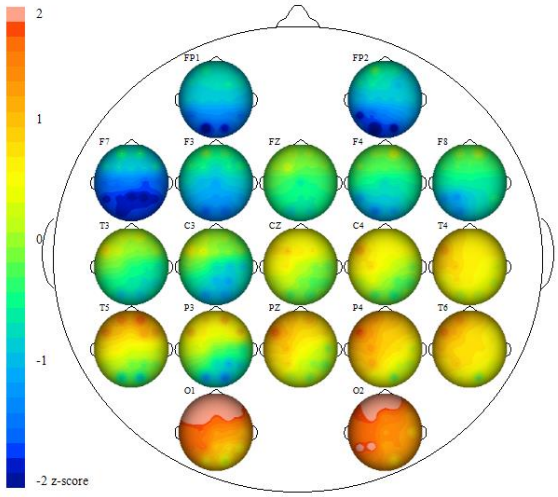
During alpha/beta AVE, we observe significant increases in the delta and the SMR/low beta band from 12 to 15 Hz (Figure 44). The delta activity (1 to 4 Hz) is present throughout the qEEG as the client fell into a deep sleep. We see that the alpha rhythm is fast. Given that slowed activity in any particular location is not evident, this indicates that he likely never sustained a significant grey-matter injury often seen in closed-head injuries and stroke – and this is good. We see the dark blue areas have faded indicating an increase to normalcy across the board of qEEG activity. Alpha activity is best near 10 Hz. The abundant activity seen at 13 to 14 Hz will hopefully settle down over time.

Figure 44. Absolute Frequency Magnitude as Compared Against a Database (2 SD).



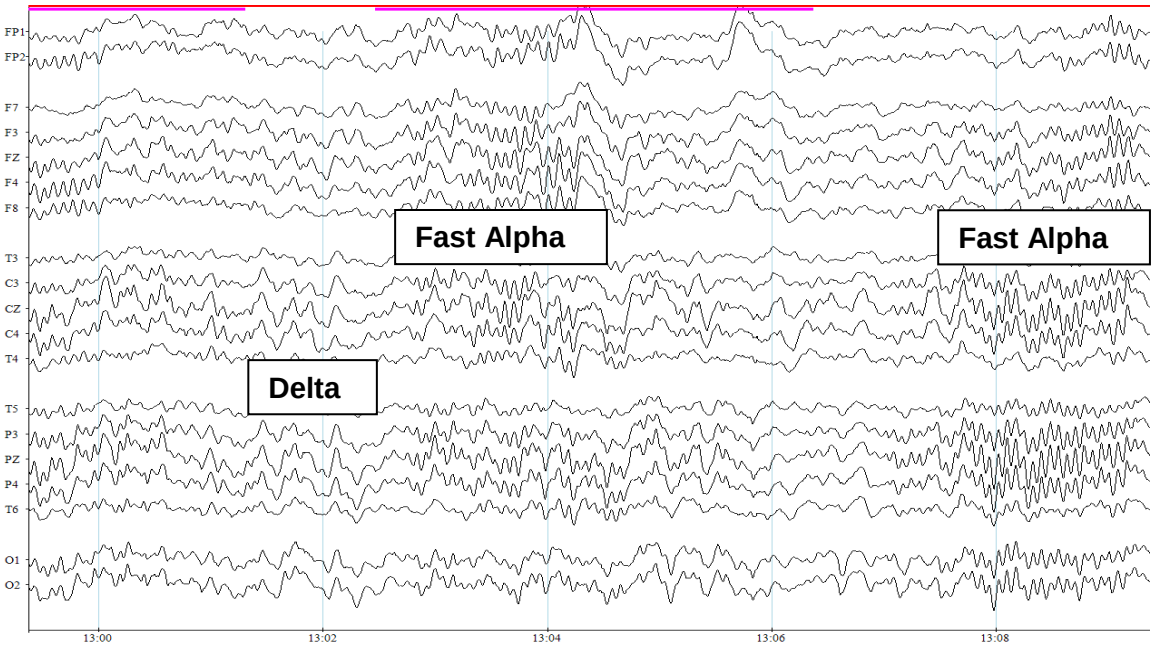
Given that the client was sleeping somewhat during the recording, much of the front to back phase issues are present as a result of sleep delta, as shown in Figure 45. However, the left to right anomalies have cleared up.

Figure 45. Delta Phase as Compared with a Database.



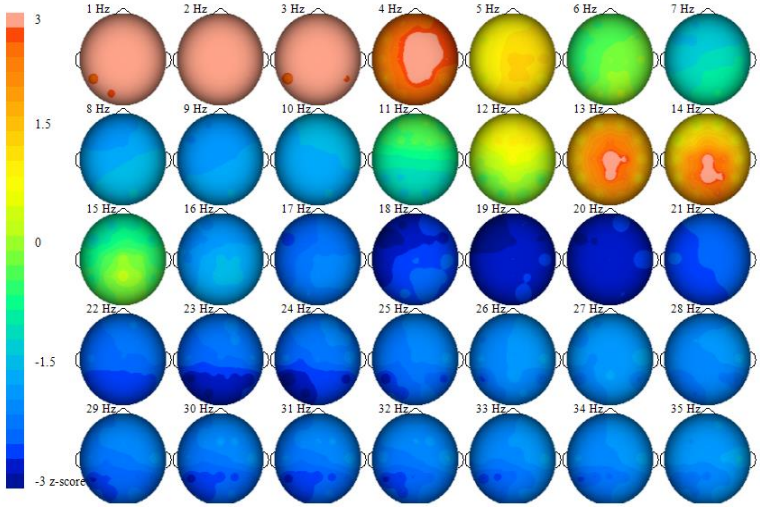
The client continued to sleep for 30 minutes past the cessation of AVE. In Figure 46, we continue to see nice alpha spindles, mixed with periods of delta, as he slept.

Figure 46. Raw Waveforms 15 Minutes Post Left-Side 10 Hz and Right-Side 19 to 21 Hz AVE.



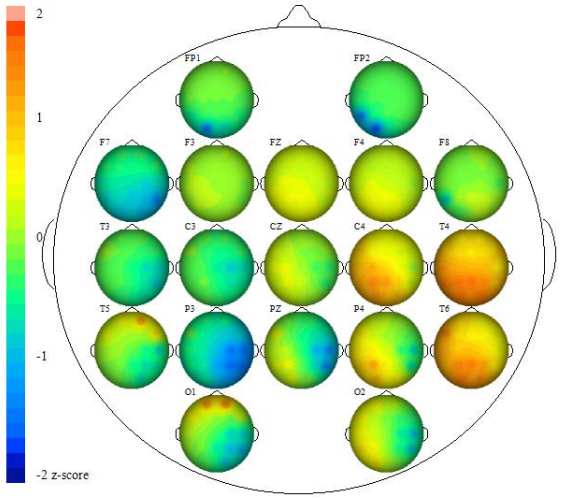
In Figure 47, we see that in making his own brainwaves post AVE, the fast alpha at 13 and 14 Hz has settled down a fair bit.

Figure 47. Absolute Frequency Magnitude as Compared Against a Database (2 SD).



We can see that while making his own brainwaves post AVE, his phase has normalized a great deal and shows quite optimal functioning (Figure 48).

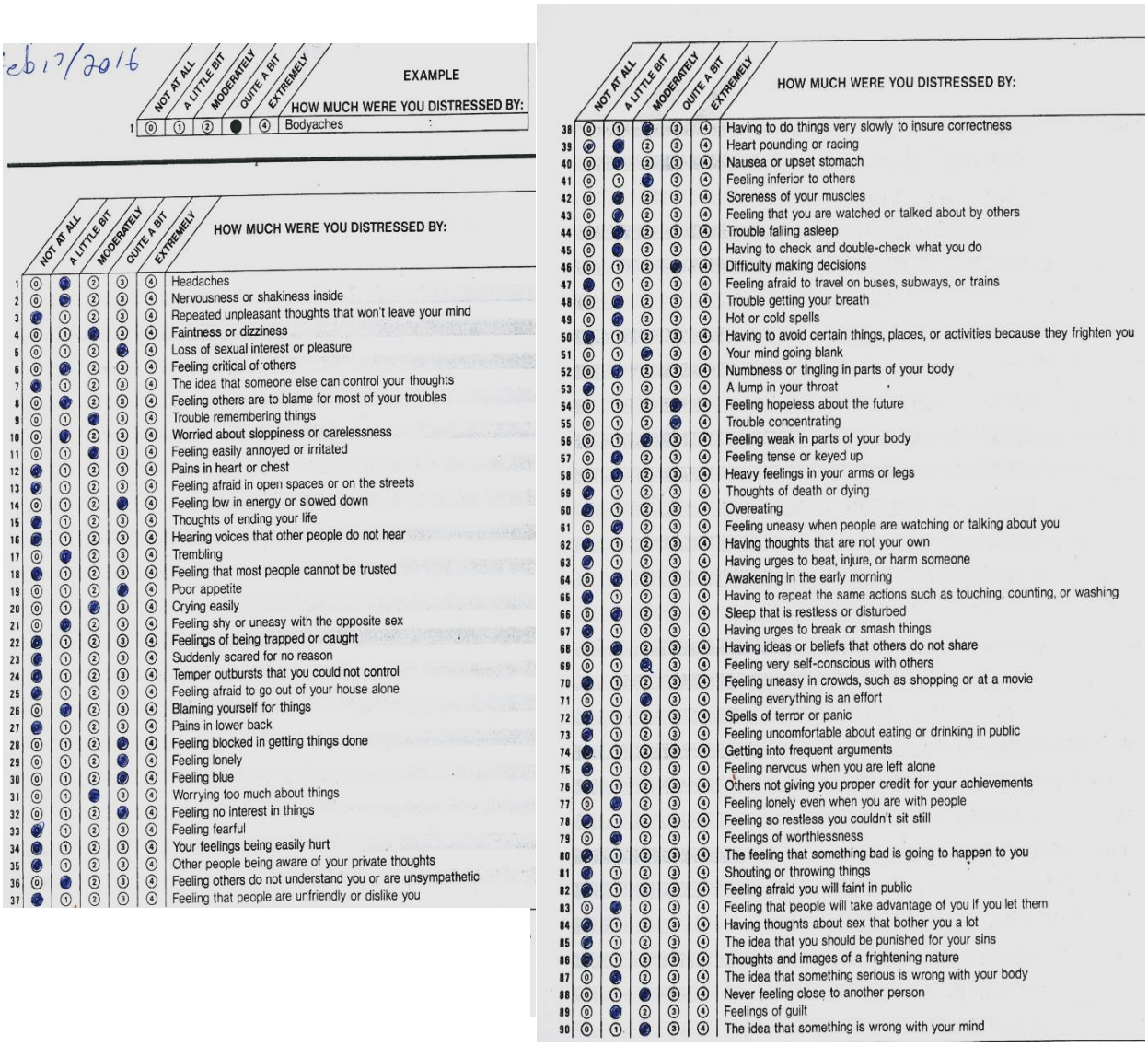
Figure 48. Delta Phase as Compared with a Database.



The client's raw SCL-90-R results (score = 85) are shown in the actual questionnaire (Figure 49a and b). Unlike the previous examples, his score was not processed, so his specific feelings can be observed. Notice his low energy, feeling blocked in getting things done, lonely, blue, lack of interest, difficulty making decisions, hopeless and trouble concentrating. These are typical behavioral and cognitive outcomes of a DA-TBI.

Following the Mood Booster session in the office, the client slept 11 hours that night. One week later, he was in for his followup. He indicated a score of 53 on the SCL-90-R. He was given a DAVID device to use at home. He was instructed to use the Mood Booster 2 (a 45-minute alpha/beta protocol) and given his trouble falling asleep, he was instructed to use the device and fall asleep with the session running. After nine days, he returned for followup and his SCL-90-R score indicated a score of 10 – a great improvement, as can be seen in Figures 50a and b.

Figure 49a and b. SCL-90-R Score on Intake (Score=85).

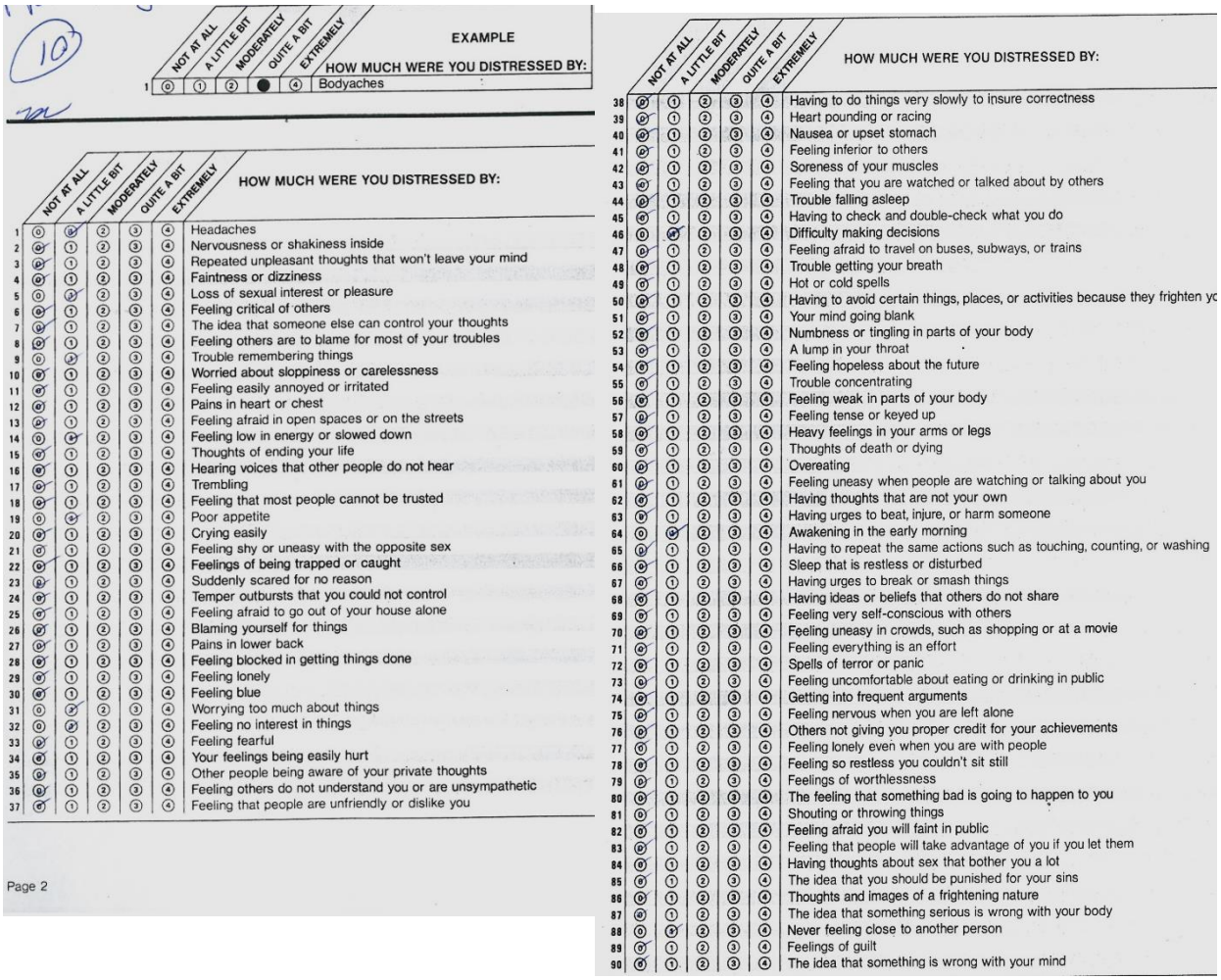


	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY	EXAMPLE HOW MUCH WERE YOU DISTRESSED BY:
1	0	1	2	●	4	Bodyaches

	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY	HOW MUCH WERE YOU DISTRESSED BY:
1	0	●	2	3	4	Headaches
2	0	●	2	3	4	Nervousness or shakiness inside
3	●	1	2	3	4	Repeated unpleasant thoughts that won't leave your mind
4	0	1	●	3	4	Faintness or dizziness
5	0	1	2	●	4	Loss of sexual interest or pleasure
6	0	●	2	3	4	Feeling critical of others
7	●	1	2	3	4	The idea that someone else can control your thoughts
8	0	●	2	3	4	Feeling others are to blame for most of your troubles
9	0	1	●	3	4	Trouble remembering things
10	0	●	2	3	4	Worried about sloppiness or carelessness
11	0	1	●	3	4	Feeling easily annoyed or irritated
12	●	1	2	3	4	Pains in heart or chest
13	●	1	2	3	4	Feeling afraid in open spaces or on the streets
14	0	1	2	●	4	Feeling low in energy or slowed down
15	●	1	2	3	4	Thoughts of ending your life
16	●	1	2	3	4	Hearing voices that other people do not hear
17	0	●	2	3	4	Trembling
18	●	1	2	3	4	Feeling that most people cannot be trusted
19	0	1	2	●	4	Poor appetite
20	0	1	●	3	4	Crying easily
21	0	●	2	3	4	Feeling shy or uneasy with the opposite sex
22	●	1	2	3	4	Feelings of being trapped or caught
23	●	1	2	3	4	Suddenly scared for no reason
24	●	1	2	3	4	Temper outbursts that you could not control
25	●	1	2	3	4	Feeling afraid to go out of your house alone
26	0	●	2	3	4	Blaming yourself for things
27	●	1	2	3	4	Pains in lower back
28	0	1	2	●	4	Feeling blocked in getting things done
29	0	1	2	●	4	Feeling lonely
30	0	1	2	●	4	Feeling blue
31	0	1	●	3	4	Worrying too much about things
32	0	1	2	●	4	Feeling no interest in things
33	●	1	2	3	4	Feeling fearful
34	●	1	2	3	4	Your feelings being easily hurt
35	●	1	2	3	4	Other people being aware of your private thoughts
36	0	●	2	3	4	Feeling others do not understand you or are unsympathetic
37	●	1	2	3	4	Feeling that people are unfriendly or dislike you

	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY	HOW MUCH WERE YOU DISTRESSED BY:
38	0	1	●	3	4	Having to do things very slowly to insure correctness
39	●	●	2	3	4	Heart pounding or racing
40	0	●	2	3	4	Nausea or upset stomach
41	0	1	●	3	4	Feeling inferior to others
42	0	●	2	3	4	Soreness of your muscles
43	0	●	2	3	4	Feeling that you are watched or talked about by others
44	0	●	2	3	4	Trouble falling asleep
45	0	●	2	3	4	Having to check and double-check what you do
46	0	1	2	●	4	Difficulty making decisions
47	●	1	2	3	4	Feeling afraid to travel on buses, subways, or trains
48	0	●	2	3	4	Trouble getting your breath
49	0	●	2	3	4	Hot or cold spells
50	●	1	2	3	4	Having to avoid certain things, places, or activities because they frighten you
51	0	1	●	3	4	Your mind going blank
52	0	●	2	3	4	Numbness or tingling in parts of your body
53	●	1	2	3	4	A lump in your throat
54	0	1	2	●	4	Feeling hopeless about the future
55	0	1	2	●	4	Trouble concentrating
56	0	1	●	3	4	Feeling weak in parts of your body
57	0	●	2	3	4	Feeling tense or keyed up
58	0	●	2	3	4	Heavy feelings in your arms or legs
59	●	1	2	3	4	Thoughts of death or dying
60	●	1	2	3	4	Overeating
61	0	●	2	3	4	Feeling uneasy when people are watching or talking about you
62	●	1	2	3	4	Having thoughts that are not your own
63	●	1	2	3	4	Having urges to beat, injure, or harm someone
64	0	●	2	3	4	Awakening in the early morning
65	●	1	2	3	4	Having to repeat the same actions such as touching, counting, or washing
66	0	●	2	3	4	Sleep that is restless or disturbed
67	●	1	2	3	4	Having urges to break or smash things
68	0	●	2	3	4	Having ideas or beliefs that others do not share
69	0	1	●	3	4	Feeling very self-conscious with others
70	●	1	2	3	4	Feeling uneasy in crowds, such as shopping or at a movie
71	0	1	●	3	4	Feeling everything is an effort
72	●	1	2	3	4	Spells of terror or panic
73	●	1	2	3	4	Feeling uncomfortable about eating or drinking in public
74	●	1	2	3	4	Getting into frequent arguments
75	●	1	2	3	4	Feeling nervous when you are left alone
76	●	1	2	3	4	Others not giving you proper credit for your achievements
77	0	●	2	3	4	Feeling lonely even when you are with people
78	●	1	2	3	4	Feeling so restless you couldn't sit still
79	0	●	2	3	4	Feelings of worthlessness
80	●	1	2	3	4	The feeling that something bad is going to happen to you
81	●	1	2	3	4	Shouting or throwing things
82	●	1	2	3	4	Feeling afraid you will faint in public
83	0	●	2	3	4	Feeling that people will take advantage of you if you let them
84	●	1	2	3	4	Having thoughts about sex that bother you a lot
85	●	1	2	3	4	The idea that you should be punished for your sins
86	●	1	2	3	4	Thoughts and images of a frightening nature
87	0	●	2	3	4	The idea that something serious is wrong with your body
88	0	1	●	3	4	Never feeling close to another person
89	0	●	2	3	4	Feelings of guilt
90	0	1	●	3	4	The idea that something is wrong with your mind

Figure 50a and b. SCL-90-R Score after Nine Days of Night Time Mood Booster Session (Alpha/Beta) AVE (Score=10).



EXAMPLE

	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY	HOW MUCH WERE YOU DISTRESSED BY:
1	0	1	2	●	4	Bodyaches

	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY	HOW MUCH WERE YOU DISTRESSED BY:
1	0	1	2	3	4	Headaches
2	0	1	2	3	4	Nervousness or shakiness inside
3	0	1	2	3	4	Repeated unpleasant thoughts that won't leave your mind
4	0	1	2	3	4	Faintness or dizziness
5	0	1	2	3	4	Loss of sexual interest or pleasure
6	0	1	2	3	4	Feeling critical of others
7	0	1	2	3	4	The idea that someone else can control your thoughts
8	0	1	2	3	4	Feeling others are to blame for most of your troubles
9	0	1	2	3	4	Trouble remembering things
10	0	1	2	3	4	Worried about sloppiness or carelessness
11	0	1	2	3	4	Feeling easily annoyed or irritated
12	0	1	2	3	4	Pains in heart or chest
13	0	1	2	3	4	Feeling afraid in open spaces or on the streets
14	0	1	2	3	4	Feeling low in energy or slowed down
15	0	1	2	3	4	Thoughts of ending your life
16	0	1	2	3	4	Hearing voices that other people do not hear
17	0	1	2	3	4	Trembling
18	0	1	2	3	4	Feeling that most people cannot be trusted
19	0	1	2	3	4	Poor appetite
20	0	1	2	3	4	Crying easily
21	0	1	2	3	4	Feeling shy or uneasy with the opposite sex
22	0	1	2	3	4	Feelings of being trapped or caught
23	0	1	2	3	4	Suddenly scared for no reason
24	0	1	2	3	4	Temper outbursts that you could not control
25	0	1	2	3	4	Feeling afraid to go out of your house alone
26	0	1	2	3	4	Blaming yourself for things
27	0	1	2	3	4	Pains in lower back
28	0	1	2	3	4	Feeling blocked in getting things done
29	0	1	2	3	4	Feeling lonely
30	0	1	2	3	4	Feeling blue
31	0	1	2	3	4	Worrying too much about things
32	0	1	2	3	4	Feeling no interest in things
33	0	1	2	3	4	Feeling fearful
34	0	1	2	3	4	Your feelings being easily hurt
35	0	1	2	3	4	Other people being aware of your private thoughts
36	0	1	2	3	4	Feeling others do not understand you or are unsympathetic
37	0	1	2	3	4	Feeling that people are unfriendly or dislike you

Page 2

	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY	HOW MUCH WERE YOU DISTRESSED BY:
38	0	1	2	3	4	Having to do things very slowly to insure correctness
39	0	1	2	3	4	Heart pounding or racing
40	0	1	2	3	4	Nausea or upset stomach
41	0	1	2	3	4	Feeling inferior to others
42	0	1	2	3	4	Soreness of your muscles
43	0	1	2	3	4	Feeling that you are watched or talked about by others
44	0	1	2	3	4	Trouble falling asleep
45	0	1	2	3	4	Having to check and double-check what you do
46	0	1	2	3	4	Difficulty making decisions
47	0	1	2	3	4	Feeling afraid to travel on buses, subways, or trains
48	0	1	2	3	4	Trouble getting your breath
49	0	1	2	3	4	Hot or cold spells
50	0	1	2	3	4	Having to avoid certain things, places, or activities because they frighten yo
51	0	1	2	3	4	Your mind going blank
52	0	1	2	3	4	Numbness or tingling in parts of your body
53	0	1	2	3	4	A lump in your throat
54	0	1	2	3	4	Feeling hopeless about the future
55	0	1	2	3	4	Trouble concentrating
56	0	1	2	3	4	Feeling weak in parts of your body
57	0	1	2	3	4	Feeling tense or keyed up
58	0	1	2	3	4	Heavy feelings in your arms or legs
59	0	1	2	3	4	Thoughts of death or dying
60	0	1	2	3	4	Overeating
61	0	1	2	3	4	Feeling uneasy when people are watching or talking about you
62	0	1	2	3	4	Having thoughts that are not your own
63	0	1	2	3	4	Having urges to beat, injure, or harm someone
64	0	1	2	3	4	Awakening in the early morning
65	0	1	2	3	4	Having to repeat the same actions such as touching, counting, or washing
66	0	1	2	3	4	Sleep that is restless or disturbed
67	0	1	2	3	4	Having urges to break or smash things
68	0	1	2	3	4	Having ideas or beliefs that others do not share
69	0	1	2	3	4	Feeling very self-conscious with others
70	0	1	2	3	4	Feeling uneasy in crowds, such as shopping or at a movie
71	0	1	2	3	4	Feeling everything is an effort
72	0	1	2	3	4	Spells of terror or panic
73	0	1	2	3	4	Feeling uncomfortable about eating or drinking in public
74	0	1	2	3	4	Getting into frequent arguments
75	0	1	2	3	4	Feeling nervous when you are left alone
76	0	1	2	3	4	Others not giving you proper credit for your achievements
77	0	1	2	3	4	Feeling lonely even when you are with people
78	0	1	2	3	4	Feeling so restless you couldn't sit still
79	0	1	2	3	4	Feelings of worthlessness
80	0	1	2	3	4	The feeling that something bad is going to happen to you
81	0	1	2	3	4	Shouting or throwing things
82	0	1	2	3	4	Feeling afraid you will faint in public
83	0	1	2	3	4	Feeling that people will take advantage of you if you let them
84	0	1	2	3	4	Having thoughts about sex that bother you a lot
85	0	1	2	3	4	The idea that you should be punished for your sins
86	0	1	2	3	4	Thoughts and images of a frightening nature
87	0	1	2	3	4	The idea that something serious is wrong with your body
88	0	1	2	3	4	Never feeling close to another person
89	0	1	2	3	4	Feelings of guilt
90	0	1	2	3	4	The idea that something is wrong with your mind

Findings and Conclusion

After one alpha/beta AVE session:

1) His alpha rhythm was reestablished, which brought on an instant calming effect.
2) He slept for 30 minutes after AVE had ended.
3) He slept 11 hours that night using the Mood Booster 2 protocol (alpha/beta).
4) He had no cravings for five days.
5) He continued sleeping well using AVE at bedtime.
6) His anxiety was reduced.

After nine days of alpha/beta AVE sessions at bedtime:

1) He had no more cravings at all.
2) He had no more anxiety.
3) He felt rested and grounded.
4) He felt sharper mentally.

He has relapsed with alcohol a couple of times since, but gradually gained control over his emotions, anxiety and cravings. As of August, 2016, he had been completely free of any alcohol consumption for two months! (Notice his markings on his SCL-90-R was much lighter after nine days of AVE treatment).

Conclusion

In summary, we do not yet have a complete understanding of the underlying mechanisms of repetitive stimuli and their effects on the brain. Many events are occurring simultaneously. With regards to deep brain structures, these findings are both new and exciting. Various Veterans Affairs' centers in the USA have noted that troops using AVE not only recover from PTSD, but also improve cognitively from DA-TBIs relating to shock waves from detonated explosives. Unfortunately, work with veterans has not had EEG or qEEG backing as proof of concept. Had it done so, these discoveries may have been revealed a decade ago.

The ability of AVE to jump start the cortico-thalamic loop has been compared with using a set of electrified paddles to jump start a heart in fibrillation. It is clear that the effects go well beyond simply "vibrating" the system and involve complex information-rich processes. The fact that repetitive stimuli can have effects on mental and emotional states speaks to the fact that the involvement of functional networks is of paramount importance. It's possible that AVE re-excites the Rich Club Neuronal Networks in the brain (Hagmann, et al., 2008).

One of the clinical concerns with head injury has to do with treating pre-existing conditions prior to treating the brain injury. However, in the three DA-TBI cases shown here, the anorexia, anxiety and binge drinking were ALL side effects of their brain injuries. These are NOT separate issues. They MUST be considered to be a spectrum of disorders concurrent with a DA-TBI.

Results shown in this article lend credence to the idea that we should be able to create a bottom-up analysis that puts repetitive stimuli in an objective and physiologically-based framework which will illuminate why such stimulation has the observed effects on how we think, feel, and perform in a myriad of situations.

The advantages of using the DAVID devices is that they show effectiveness in restoring brain function that has been suspended by a DA-TBI. The devices are safe, portable and low cost and can be sent home with a patient with minimal instruction. Figure 51 shows a DAVID Delight Pro system, which also includes CES, an electrical stimulation technique which delivers a small current across the brainstem. CES has been shown to liberate neurotransmitters, particularly serotonin and endorphins. Over 200 studies (~50 double-blind) have been completed on CES, covering drug abuse, depression, anxiety and insomnia. It is often used as an adjunct to AVE in more severe clinical cases involving PTSD, depression and brain injury.

Figure 51. DAVID Delight Pro Device.

References

Adrian, E. & Matthews, B. (1934) The Berger rhythm: Potential changes from the occipital lobes in man. *Brain, 57*, 355-384.

Amen, D. (1998). *Change your brain, change your life*. New York: Three Rivers Press.

Anticevic, A., Haut, K., Murray, J., Repovs, G., Yang, G., Diehl, C., McEwen, S., Bearden, C., Addington, J., Goodyear, B., Cadenhead, K., Mirzakhanian, H., Cornblatt, B., Olvet, D., Mathalon, D., McGlashan, T., Perkins, D., Belger, A., Seidman, L., Tsuang, M., van Erp, T., Walker, E., Hamann, S., Woods, S., Qiu, M., & Cannon, T. (2015). Association of thalamic dysconnectivity and conversion to psychosis in youth and young adults at elevated clinical risk. *JAMA Psychiatry*, 72(9):882-891.

Arco, A. & Mora, F. (2009). Neurotransmitters and prefrontal cortex-limbic system interactions: implications for plasticity and psychiatric disorders. *Journal of Neural Transmission, 116*, 941-952.

Balser, M. & Wagner, C. (1960). "Observations of Earth–ionosphere cavity resonances". Nature 188 (4751): 638–641.

Berg, K., Mueller, H., Seibel, D., & Siever, D. (1999). Outcome of medical methods, audio-visual entrainment, and nutritional supplementation in the treatment of fibromyalgia syndrome. *Unpublished manuscript*. Edmonton, Alberta, Canada, Mind Alive Inc.

Berg, K. & Siever, D. (2004). The effect of audio-visual entrainment in depressed community-dwelling senior citizens who fall. *Unpublished manuscript*. Edmonton, Alberta, Canada, Mind Alive Inc.

Berg, K. & Siever, D. (2009) A controlled comparison of audio-visual entrainment for treating seasonal affective disorder (SAD). *Journal of Neurotherapy, 13* (3), 166-175.

Bombardier, C., Fann, J., Temkin, N., Esselman, P., Barber, J., & Dikmen, S. (2010). Rates of Major Depressive Disorder and Clinical Outcomes Following Traumatic Brain Injury. *JAMA, 303*, 19, 1938-1945.

Budzynski, T. & Tang, J. (1998). Bio-light effects on the electroencephalogram (EEG). *SynchroMed Report*. Seattle, WA.

Budzynski, T., Jordy, J., Budzynski, H., Tang, H., & Claypoole, K. (1999). Academic performance enhancement with photic stimulation and EDR feedback. *Journal of Neurotherapy, 3*, 11-21.

Budzynski, T., Budzynski, H., & Sherlin, L. (2002). Audio visual stimulation (AVS) in an Alzheimer's patient as documented by quantitative electroencephalography (QEEG) and low resolution electromagnetic brain tomography (LORETA) [Abstract]. *Journal of Neurotherapy, 6*(1), 54.

Budzynski, T., Budzynski, H., & Tang, H. (2007). Brain brightening. In J. R. Evans (Ed.), *Handbook of neurofeedback: Dynamics and clinical applications* (pp. 231-265). New York, NY: Haworth Press.

Carter, J. & Russell, H. (1993). A pilot investigation of auditory and visual entrainment of brain wave activity in learning disabled boys. *Texas Researcher*. 4, 65-72.

Chatrian, G., Petersen, M., & Lazarte, J. (1959). Response to clicks from the human brain: Some depth electrographic observations. *Electroencephalography and Clinical Neurophysiology, 12*, 479-489.

Collura, T. & Siever, D. (2009). Audio-visual entrainment in relation to mental health and EEG. In T. Budzynski, H. Budzynski, J. Evans, & A. Abarbanel (Eds.), Introduction to quantitative EEG and neurofeedback: Advanced theory and applications (2nd ed., pp. 193–223). Boston: Elsevier.

Dikmen, S., Corrigan, J., Levin, H., Machamer, J., Stiers, W., & Weisskopf, M. (2009). Cognitive outcome following traumatic brain injury. *Journal of Head Trauma Rehabilitation, 24*, 430-438.

Doidge, N. (2015). The Brain's Way of Healing. Viking Books. NY, NY.

Donker, D., Njio, L., Storm Van Leeuwen, W., & Wieneke, G. (1978). Interhemispheric relationships of responses to sine wave modulated light in normal subjects and patients. *Encephalography and Clinical Neurophysiology*, *44*, 479-489.

Emmons, H. (2010). The Chemistry of Calm. Simon & Schuster.

Erba, G. (2001). Preventing seizures from "Pocket Monsters" A way to control reflex epilepsy. *Neurology, 57 (*10), 1747-1748.

Finkelstein, E., Corso, P., & Miller, T. (2006). The incidence and economic burden of Injuries in the United States. New York: Oxford University Press.

Fox, P. & Raichle, M. (1985). Stimulus rate determines regional blood flow in striate cortex. *Annals of Neurology, 17*, (3), 303-305.

Fox, P., Raichle, M., Mintun, M., & Dence, C. (1988). Nonoxidative glucose consumption during focal physiologic neural activity. *Science*, *241*, 462-464.

Frederick, J., Lubar, J., Rasey, H., Brim, S., & Blackburn, J. (1999). Effects of 18.5 Hz audiovisual stimulation on EEG amplitude at the vertex. *Journal of Neurotherapy, 3* (3), 23-27.

Gagnon, C. & Boersma, F. (1992). The use of repetitive audio-visual entrainment in the management of chronic pain. *Medical Hypnoanalysis Journal, 7*, 462-468.

Gervitz, R. (2000). Resonant frequency training to restore homeostasis for treatment of psychophysiological disorders. *Biofeedback, 27*, 7-9.

Ginsberg, M. & Busto, R. (1998). Combating hyperthermia in acute stroke: A significant clinical concern. *Stroke*, 29, 529-534

Glicksohn, J. (1986). Photic driving and altered states of consciousness: An exploratory study. *Imagination, Cognition and Personality,* 6, 167-182.

Hagmann, P., Cammoun, L., Gigandet, X., Meuli, R., Honey, C., Wedeen, V., Sporns, O. (2008). Mapping the Structural Core of Human Cerebral Cortex. *PLoS Biology, 6*, (7), 1479-1493.

Hawes, T. (2000). Chapter 14: Using light and sound technology to access "The Zone" in sports and beyond. In: Siever, D., The Rediscovery of Audio-visual Entrainment Technology. Edmonton, Alberta, Canada, Mind Alive Inc.

Hear, J. (1971). Field dependency in relation to altered states of consciousness produced by sensory-overload. *Perception and Motor Skills, 33*, 192-194.

Hebb, D.O. (1961). "Distinctive features of learning in the higher animal". In J. F. Delafresnaye (Ed.). Brain Mechanisms and Learning. London: Oxford University Press.

Hindfelt, B. (1976). The prognostic significance of subfebrility and fever in ischemic cerebral infarction. *Acta Neurolica Scandanavica*, 53, 72–79.

Iaccarino, H., Singer, A., Martorel, A., Rudenko, A., Gao, F., Gillingham, T., Mathys, H., Seo, J., Kritskiy, O., Abdurrob, F., Adaikkan, C., Canter, R., Rueda, R., Brown, E., Boyden, E., Tsai, L. (2016). Gamma frequency entrainment attenuates amyloid load and modifies microglia. Nature, 540, 230-251.

Joyce, M. & Siever, D. (2000). Audio-visual entrainment program as a treatment for behavior disorders in a school setting. *Journal of Neurotherapy*, *4* (2), 9-25.

Johnstone, J., Gunkelman, J., & Lunt, J. (2005). Clinical database development: characterization of EEG phenotypes. Clin. EEG Neurosci. 36, 99-107.

Kaiser, D. & Meckley, A. (2012). An Introduction to neurotherapy. In L. L'Abate and D. A. Kaiser (eds), Handbook of Technology in Psychology, Psychiatry, and Neurology: Theory, Research, and Practice. Hauppauge, NY: Nova Science Publishers.

Kaiser, D. (2007). Proprietary C++ software application for Microsoft Windows, Sterman-Kaiser Imaging Laboratory, Inc., Churchville, NY

Kaiser, D. (2008). Functional connectivity and aging: Comodulation and coherence differences. J Neurotherapy, 12 (2/3).

Kinney, J., McKay, C., Mensch, A., & Luria, S. (1973). Visual evoked responses elicited by rapid stimulation. *Encephalography and Clinical Neurophysiology, 34,* 7-13.

Kroger, W. & Schneider, S. (1959). An electronic aid for hypnotic induction: A preliminary report. *International Journal of Clinical and Experimental Hypnosis, 7,* 93-98.

Leonard, K., Telch, M., & Harrington, P. (1999). Dissociation in the laboratory: A comparison of strategies. *Behaviour Research and Therapy, 37,* 49-61.

Leonard, K., Telch, M., & Harrington, P. (2000). Fear response to dissociation challenge. *Anxiety, Stress, and Coping, 13,* 355-369.

Lewerenz, C. (1963). A factual report on the brain wave synchronizer. *Hypnosis Quarterly, 6* (4), 23.

Lim, J., Stafford, B., Nguyen, P., Lien, B., Wang, C., Zukor, K., He, Z. & Huberman, A. (2016). Neural activity promotes long-distance, target-specific regeneration of adult retinal axons. *Nature Neuroscience*, 01 June 2016. Published online 11 July 2016.

Lim, L. (2013). The Potential of Intranasal Light Therapy for Brain Stimulation. Presented at the North American Association for Photobiomodulation Therapy (NAALT) Conference, Palm Beach Gardens, Florida, February 2, 2013.

Lipowsky, Z. (1975). Sensory and information inputs over-load: behavioral effects. *Comprehensive Psychiatry, 16*, 199-221.

Lorensen, T., & Dickson, P. (2004). Quantitative EEG Normative Databases: A Comparative Investigation. J. Neurotherapy 8, 53-68.

Manns, A., Miralles, R., & Adrian, H. (1981). The application of audiostimulation and electromyographic biofeedback to bruxism and myofascial pain-dysfunction syndrome**.** *Oral Surgery, 52* (3), 247-252.

Margolis, B. (1966, June). A technique for rapidly inducing hypnosis**.** *CAL (Certified Akers Laboratories),* 21-24.

Meier, T., Bellgowan, P., Singh, R., Kuplicki, R., Polanski, D., & Mayer, A. (2015). Recovery of Cerebral Blood Flow Following Sports-Related Concussion. *JAMA Neurol.72*, 5, 530-538.

Meyer, J., Takashima, S., Terayama, Y., Obara, K., Muramatsu, K., & Weathers, S. (1994). CT changes associated with normal aging of the human brain. *Journal of the Neurological Sciences, 123*, 1-2, 200-8.

Micheletti, L. (1999). The Use of Auditory and Visual Stimulation for the Treatment of Attention Deficit Hyperactivity Disorder in Children. University of Houston. Ph.D. dissertation, unpublished. Edmonton, Alberta, Canada, Mind Alive Inc

Morse, D. & Chow, E. (1993). The effect of the Relaxodont^{TM} brain wave synchronizer on endodontic anxiety: evaluation by galvanic skin resistance, pulse rate, physical reactions, and questionnaire responses. *International Journal of Psychosomatics, 40* (1-4), 68-76.

Naeser, M., Zafonte, R., Krengel, M., Martin, P., Frazier, J., Hamblin, M., Knight, J., Meehan, W. 3rd, & Baker, E. (2014). Significant improvements in cognitive performance post-transcranial, red/near-infrared light-emitting diode treatments in chronic, mild traumatic brain injury: open-protocol study. *Journal of Neurotrauma, 1*;31(11):1008-17.

Palmquist, C. (2014). Brain brightening with audio-visual entrainment for memory enhancement in the middle-aged and senior population. Ph.D. dissertation, Saybrook University, California.

Ramlackhansingh, A., Brooks, D., Greenwood, R., Bose, S., Turkheimer, F., Kinnunen, K., Gentleman, S., Heckemann, R., Gunanayagam, K., Gelosa, G., & Sharp, D. (2011). Inflammation after trauma: microglial activation and traumatic brain injury. *Annals of Neurology*, 70(3), 374–83. doi:10.1002/ana.22455.

Regan, D. (1966). Some characteristics of average steady-state and transient responses evoked by modulated light. *Electroencephalogy and Clinical Neurophysiology, 20*, 238-248.

Ribbers, G. (2010). Brain injury: Long term outcome after traumatic brain injury. In: J.H. Stone, M. Blouin (editors), International Encyclopedia of Rehabilitation. Buffalo, NY: Center for International Rehabilitation Research Information and Exchange (CIRRIE).

Ross-Swain, D. (2007). The Effects of Auditory Stimulation on Auditory Processing Disorder: A Summary of the Findings. *The International Journal of Listening, 27*, 2, 140-155.

Russell, H. (1996). Entrainment combined with multimodal rehabilitation of a 43-year-old severely impaired postaneurysm patient. *Biofeedback and Self Regulation, 21*, 4.

Ruuskanen-Uoti, H. & Salmi, T. (1994, January). Epileptic seizure induced by a product marketed as a "Brainwave Synchronizer." *Neurology*, *44*, 180.

Sadove, M.S. (1963, July). Hypnosis in anaesthesiology. *Illinois Medical Journal*, 39-42.

Sappey-Marinier, D., Calabrese, G., Fein, G., Hugg, J., Biggins, C., & Weiner, M. (1992). Effect of photic stimulation on human visual cortex lactate and phosphates using 1H and 31P magnetic resonance spectroscopy. *Journal of Cerebral Blood Flow and Metabolism, 12* (4), 584-592.

Schreckenberger, M., Lange-Asschenfeld, C., Lochmanna, M., Mann, K., Siessmeiera, T., Buchholza, H., Bartensteina, P., & Gründer, G. (2004). The thalamus as the generator and modulator of EEG alpha rhythm: a combined PET/EEG study with lorazepam challenge in humans. *NeuroImage, 22*, 2, 637–644.

Scott, G., Peter, J., Hellyer, P., Ramlackhansingh, A., Brooks, D., Matthews, P., & Sharp, D. (2015). Thalamic inflammation after brain trauma is associated with thalamo-cortical white matter damage. *Journal of Neuroinflammation*, 12, 224.

Shatz, Carla J. (1992). "The Developing Brain." Scientific American. United States. pp. 60–67.

Shealy, N., Cady, R., Cox, R., Liss, S., Clossen, W., & Veehoff, D. (1989). A comparison of depths of relaxation produced by various techniques and neurotransmitters produced by brainwave entrainment. *Shealy and Forest Institute of Professional Psychology.* Unpublished manuscript.

Siever, D. (2003a). Audio-visual entrainment: History and physiological mechanisms. *Biofeedback. 31*, 2, 21-27.

Siever, D. (2003b). Audio visual entrainment: Dental studies. *Biofeedback, 31*(3), 29-32.

Siever, D. (2003c). Applying audio-visual entrainment technology for attention and learning. *Biofeedback, 31*, 4, 24-29.

Siever, D. (2013). ISNR Proceedings 21st Conference, Dallas, TX.

Steriade, M., Nuñez, A., & Amzica, F. (1993). Intracellular analysis of relations between the slow (<1 Hz) neocortical oscillations and other sleep rhythms of electroencephalogram. J Neurosci 13:3266-3283.

Sterman, M. & Kaiser, D. (1999). Topographic analysis of spectral density co-variation: normative database & clinical assessment. Clinical Neurophysiology, 110 (S1), S80.

Tang, H., Riegel, B., McCurry, S., & Vitiello, M. (2016). Open-Loop Audio-Visual Stimulation (AVS): A Useful Tool for Management of Insomnia? *Applied Psychophysiology and Biofeedback, 41* (1): 39-46

Teicher, M., Anderson, C., Polcari, A., Glod, C., Maas, L., & Renshaw, P. (2000). Functional deficits in basal ganglia of children with attention-deficit/hyperactivity disorder shown with functional magnetic resonance imaging relaxometry. *Nature Medicine, 6* (4), 470-473.

Temkin, N., Corrigan, J., Dikmen, S., & Machamer, J. (2009). Social functioning after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 24,* 6, 460-467.

Thatcher, R., Walker, R., Gerson, I., & Geisler, F. (1989). EEG discriminant analyses of mild head trauma. Electroencephalography and Clinical Neurophysiology, 73 (2), 94-106.

Thomas, N. & Siever, D. (1989). The effect of repetitive audio/visual stimulation on skeletomotor and vasomotor activity. In D. Waxman, D. Pederson, I. Wilkie, & P. Meller (Eds.), *Hypnosis: 4th European Congress at Oxford* (pp. 238-245). London, UK: Whurr Publishers.

Thompson, H., Pinto-Martin, J. & Bullock, M. (2003). Neurogenic fever after traumatic brain injury: an epidemiological study. *Journal of Neurology Neurosurgery & Psychiatry, 74*, 614–619

Toman, J. (1941). Flicker potentials and the alpha rhythm in man. *Journal of Neurophysiology, 4*, 51-61.

Townsend, R. (1973). A device for generation and presentation of modulated light stimuli. *Electroencephalography and Clinical Neurophysiology, 34*, 97-99.

Trenité, D., Guerrini, R., Binnie, C., & Genton, P. (2001). Visual sensitivity and epilepsy: a proposed terminology and classification for clinical and EEG phenomenology. Epilepsia, 42(5), 692-701.

Trudeau, D. (1999). A Trial of 18 Hz Audio-Visual Stimulation (AVS) on Attention and Concentration In Chronic Fatigue Syndrome (CFS). Proceedings of the Annual Conference for the International Society for Neuronal Regulation.

Van Der Tweel, L. & Lunel, H. (1965). Human visual responses to sinusoidally modulated light. *Encephalography and Clinical Neurophysiology*, *18,* 587-598.

Walter**,** W. (1956). Color illusions and aberrations during stimulation by flickering light. *Nature, 177*, 710.

Williams, J., Ramaswamy, D., & Oulhaj, A. (2006). 10 Hz flicker improves recognition memory in older people. *BMC Neuroscience, 7*(21), 1-7.

Wolitzky-Taylor, K. & Telch, M. (2010). Efficacy of self-administered treatments for pathological academic worry: A randomized controlled trial. *Behaviour Research and Therapy, 48*, 840-850.

Wuchrer, V. (2009). Study on memory and concentration. *Conducted at the Psychological Institute of the Friedrich-Alexander University Erlangen-Nürnberg.* Unpublished manuscript. Edmonton, Alberta, Canada, Mind Alive Inc.