Audio-Visual Entrainment and Diffuse Axonal Injuries/Interruptions

- Dave Siever, C.E.T.

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.** As a result of 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 ± 1 Hz (for example, 10 Hz would randomize from 9 to 11 Hz), entrainment is reported to provide a significant clinical impact, at ± 2 Hz, the clinical effect is poor and at ± 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;
4. Increased cerebral blood flow;
5. Increased neurotransmitter activity.

**(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. By definition, 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)**

![Figure 1. EEG Showing Photic Entrainment (Kinney, et al, 1973)](image)

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 "brainmap" 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)**

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

In as much as entrainment can enhance a particular, non-square wave, entrainment can also inhibit the half frequency of stimulation. This is useful for treating depression and ADHD.
instance, those with ADHD show excess frontal theta (5 to 7 Hz) activity and too little SMR (13 to 15 Hz). By stimulating from 12 to 15 Hz, theta activity ranging from 6 to 7.5 Hz is inhibited, thus accomplishing both things at once. This technique also increases concentration in college students and cognition in seniors (Siever 2003c). 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 EMG Levels during AVE (Hawes, 2000)

![Forearm EMG Levels](image)

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

![Peripheral Temperature Levels](image)

**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 eye sets 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 heart rate 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 7.8Hz 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**

![Heart Rate Variability during 7.8 Hz AVE](image)

**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)**

![Cerebral Blood Flow at Various Photic Entrainment Repetition Rates](image)
(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)](image)

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 &
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 of 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

<table>
<thead>
<tr>
<th>Condition</th>
<th>Studies (N)</th>
<th>Demographic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attention Deficit Disorder (ADD)</td>
<td>4 (359)</td>
<td>School children</td>
</tr>
<tr>
<td>Academic performance in college students</td>
<td>3 (134)</td>
<td>College students</td>
</tr>
<tr>
<td>Reduced worry/anxiety in college students</td>
<td>3 (163)</td>
<td>College students</td>
</tr>
<tr>
<td>Drug rehabilitation</td>
<td>1 (44)</td>
<td>General population</td>
</tr>
<tr>
<td>Depression and anxiety</td>
<td>3 (93)</td>
<td>General population</td>
</tr>
<tr>
<td>Improved cognitive performance in seniors</td>
<td>1 (40)</td>
<td>From seniors’ homes</td>
</tr>
<tr>
<td>Reduced falling and depression in seniors</td>
<td>1 (80)</td>
<td>From seniors’ homes</td>
</tr>
<tr>
<td>Memory in seniors</td>
<td>1 (40)</td>
<td>Seniors - drop in</td>
</tr>
<tr>
<td>Dental (during dental procedures)</td>
<td>3 (&gt;50)</td>
<td>Patients</td>
</tr>
<tr>
<td>Temporo Mandibular Dysfunction (TMD)</td>
<td>3 (76)</td>
<td>General population</td>
</tr>
<tr>
<td>Seasonal Affective Disorder (SAD)</td>
<td>1 (74)</td>
<td>General population</td>
</tr>
<tr>
<td>Headache (migraine and tension)</td>
<td>2 (35)</td>
<td>General population</td>
</tr>
<tr>
<td>Neurophysiology</td>
<td>&gt;100 (&gt;3000)</td>
<td>General population</td>
</tr>
<tr>
<td>Heart rate variability and hypertension</td>
<td>5 (148)</td>
<td>General Population</td>
</tr>
<tr>
<td>Hypnosis/dissociation/meditation</td>
<td>10 (&gt;2000)</td>
<td>General population</td>
</tr>
<tr>
<td>Pain and fibromyalgia</td>
<td>5 (178)</td>
<td>General population</td>
</tr>
</tbody>
</table>
### 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, we are interested in how network properties and nonlinear effects 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 as a result of the 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. 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, not only on neurons, but also on associated structures including the glia, vascular structures, and other supportive tissues.

Clinical Examples of Enigmatic Effects of AVE

I will present five examples below which indicate that the dissociative and CBF aspects of AVE are just as or likely even more important than the brainwave driving (frequency 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 EEG and qEEG Terms

Before we start looking at our examples, we need to look at some definitions and to note that we are only going to address EEG and qEEG techniques. We will only review qEEG measures of greatest concern in these case examples. The first thing to note is that these are EEG recordings using a 10-20 EEG electrode montage. 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. Given that 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, EEG is recorded from the brain for several minutes. After the data have been saved as an EEG file, that data is downloaded into qEEG analysis software. Then artifacts relating to eye-blinks and movements, plus muscle activity from jaw, neck and body movements are 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. The data may also be compared to a database which was created using the exact same montage and with participants who were deemed psychologically normal in psychometric questionnaires. The analysis 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 some synchrony across brain regions. This measures the degree to which the spindles and wavelets are in alignment. 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 cortico-thalamic 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.
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 struggles to read more than half a page of her text books at a time, she would read and rereads in an attempt to learn her study material and often stays 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 brain wave 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 & 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 vs Figure 15. The calming effect lasted for about two days. He began a program using either beta or beta/SMR AVE every morning upon waking 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

Heartbeat artifacts
Cortico-Thalamic Issues: Head Injuries or Interruptions of the Diffuse Axonal Type

Diffuse axonal injury or interruption (DAI) is a frequent result of traumatic acceleration/deceleration or rotational injuries and sometimes even a fever. Depending on severity, symptoms range from mildly noticeable to persistent vegetative state in patients. DAI is 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, DAI is widespread, usually affecting a large area. It is traditionally described as the formation of several small tears or distortions at the gray-white matter junction and within the corpus callosum or other networks. A DAI typically results in thalamocortical dysconnectivity (TCD), a condition in which electrical signals between the neurons of the cortex and the thalamus are interrupted, as shown in Figure 19.
Figure 19. Diffuse Axonal Interruption or Injury Showing Thalamocortical Dysconnectivity (Image: Scientific American).

An excellent literature review by Ribbers in 2010, outlines the economic, emotional and socio-economic impact of TBIs on those inflicted with them. 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, 2006). A broad range of psychological illnesses result from thalamic dysconnectivity. A study of 243 individuals at clinical high-risk for psychosis found that TCD was 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).

TCD directly affects the alpha rhythm. The alpha rhythm is mediated by billions of synchronized cortical-thalamic circuits and healthy alpha is close to 10 Hz (Schreckenberger, et al., 2004). In the case of a DAI, there is an electrical blockage to either axons or dendrites in the loop between the cortex and the thalamus causing two things to occur: 1) the alpha rhythm becomes attenuated or eliminated in certain regions or entirely; and 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 (Thatcher, et al, 1989) and the delta phase measure will show abnormalities.

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.
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 (Ramplakhansingh 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 thalamo-cortical tract damage.

Fever has also been found to cause loss of brain function. Thompson, Pinto-Martin and Bullock, in 2003, found that patients with DAI present on imaging studies were over nine times more likely to develop neurotrophic fever in patients with DAI vs TBI patients without DAI, 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).

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. 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). The low alpha activity coupled with excessive beta activity results in feelings of agitation and anxiousness. Typical eyes-closed alpha amplitude in normal people shows an alpha/beta ratio of three or more. In those with DAIs, the alpha/beta ratio is often less than one.

The following three examples demonstrate the diversity of presenting symptoms of DAIs. 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 variances, I propose a spectrum of disorders arises from DAIs and this should be considered in that light and NOT formally treated for their individual clinical diagnosis until a DAI 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 was not overly successful. What came as a surprise were two things: 1) that beta stimulation at 20 Hz would reestablish an alpha rhythm at around 10 Hz, and; 2) that 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 DAI 1 – Interpersonal Anxiety and Mild Anorexia (with OCD)**

When conducting research with case studies with a hoarder, counter, cutter, anorexic and ritualist, the author was surprised to observe they all had similar brainwave patterns consistent with obsessive-compulsive disorder (OCD). They all showed a low-voltage, alpha dropout condition and delta phase issues similar to a DAI, but with a locus of slowed alpha/heightened theta EEG activity along the cingulate.

**CLIENT INFORMATION:**

Condition: Generalized Anxiety, 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, referential/linkered-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) Client was given a DAVID Delight Pro, which uses a combination of AVE and Cranio-electro stimulation (CES).

**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 brain wave 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 21](image1)

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

![Figure 22](image2)

Data from several DAI cases suggests 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.
Beta Phase: Imagine the scenario as presented in the delta phase example described previously where 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). But once the song has ended, every person in the audience applauds the performers. Each person continues to clap at roughly two claps per second or 2 Hz, but in their own time. So now, thousands of desynchronized claps are reverberating throughout the auditorium in a continuous roar. These disconnected claps would appear as high frequency noise. 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 problem. In the few number of thorough analysis I have done since my theoretical realization that beta phase issues could be coincident with delta phase issues, I have found that beta phase issues typically show in the 25 to 35 Hz range. More interesting is that this occurs in only about half the DAI population that I have observed (n~10). Beta phase could appear to be fine for two reasons. Perhaps with some people, the beta brain waves are in phase, or perhaps the high frequency being generated is above the high frequency limits of the database at 35 Hz. Either way, beta phase analysis might hold clinical significance and calls for further exploration. 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 or non-existent in the remaining examples.
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 24 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. She fell quickly to sleep while the EEG was recording (Figure 27).
EEG was recorded during alpha/beta AVE. This stimulation provides 10Hz on left eye-fields and ear and 20Hz on her right eye-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 13-15 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.

**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, that 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 and Three Weeks Post Alpha/Beta AVE Results as Indicated on the SCL-90-R

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

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. At 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 DAI 2 - Jumpstarting the Brain with SMR/Beta AVE Following a Motor Vehicle Accident

The author has observed alpha dropout in everything from forceps deliveries, undetermined causes, fevers and obvious head injuries. We have worked with people struggling in the aftermath of a head injury for many years now. However, the emphasis was directed more to those with grey-matter injuries, where a slow-frequency hot spot would show up in the qEEG and so we were puzzled as to how to deal with DAI.
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: Client 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) 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 in 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 reported before AVE and three weeks and twelve weeks after AVE use.
2) A 19-channel, 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) Client was given a Delight Pro, which uses a combination of AVE and CES, to clear her head and reduce her pain. 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.

5) One month later, she was placed on several applications of transcranial DC Stimulation (tDCS) over T5 (10-20 montage) 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

Raw Waveforms   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.
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 29. EEG Prior to SMR/Beta AVE**

**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). Noisy brain: This beta frequency “noise” as shown above, is common in those with DAIs. 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 DAI.

**Figure 33. Delta Phase as Compared with a Database**

![Delta Phase](image)

**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**

![Raw Waveforms](image)
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**

![Image of Alpha Comodulation](image1)

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**

![Image of Delta Phase](image2)
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

<table>
<thead>
<tr>
<th></th>
<th>Raw Scores Pre AVE</th>
<th>Raw Scores 3 Weeks Post AVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatization</td>
<td>1.09</td>
<td>0.09</td>
</tr>
<tr>
<td>Obsessive-Compulsive</td>
<td>2.50</td>
<td>0.40</td>
</tr>
<tr>
<td>Interpersonal Sensitivity</td>
<td>0.67</td>
<td>0.11</td>
</tr>
<tr>
<td>Depression</td>
<td>0.77</td>
<td>0.08</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.70</td>
<td>0.10</td>
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<tr>
<td>Hostility</td>
<td>0.33</td>
<td>0.17</td>
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<tr>
<td>Phobic Anxiety</td>
<td>0.00</td>
<td>0.00</td>
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<td>Paranoid Ideation</td>
<td>0.33</td>
<td>0.00</td>
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<td>Psychoticism</td>
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<td>0.00</td>
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<td>Grand Total Raw Score:</td>
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<td>10</td>
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<td>Global Severity Index:</td>
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<td>Positive Symptom Distress Index:</td>
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<td>0.18</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Raw Scores Pre AVE</th>
<th>Raw Scores 3 Weeks Post AVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetative Depression</td>
<td>1.2</td>
<td>0.0</td>
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<tr>
<td>Suicide Ideation</td>
<td>0.9</td>
<td>0.3</td>
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<td>Dysfunctional Sleep</td>
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</tr>
<tr>
<td>Chronic Fatigue</td>
<td>2.4</td>
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<tr>
<td>Decreased Cognitive Functioning</td>
<td>2.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Physical Pain</td>
<td>0.8</td>
<td>0.3</td>
</tr>
<tr>
<td>Pain vs Somatization Index</td>
<td>0.7</td>
<td>0.0</td>
</tr>
</tbody>
</table>

Findings and Conclusion

1) Anxiety relating to how her brain was not producing alpha waves plus loss of inhibition control 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 DAI, 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 DAI 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 DAI's. 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 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.”

Dave Siever

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• “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.”
• “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.”

EEG & qEEG Recordings

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 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.
As mentioned previously, delta phase is an integral indicator of a DAI. 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.
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 cortices (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 right-field stimulation 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.
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.
Given that the client was sleeping somewhat during the recording, much of the front/back phase issues are present as a result of sleep delta, as shown in Figure 45. However, the left-right anomalies have cleared up.

**Figure 45. Delta Phase as Compared with a Database**

![Delta Phase as Compared with a Database](image)

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**

![Raw Waveforms 15 Minutes Post Left-Side 10 Hz and Right-Side 19 to 21 Hz AVE](image)

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.
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).

The client’s raw SCL-90-R results are presented below (actual questionnaire). His score was 85 (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 DAI.

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

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

Figure 48. Delta Phase as Compared with a Database
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)**
Figure 50a and b. SCL-90-R Score after Nine Days of Night Time Mood Booster (Alpha/Beta) AVE (Score=10)

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) No cravings for five days;
5) Continued sleeping well using AVE at bedtime;
6) Reduced anxiety.

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

1) No more cravings at all;
2) No more anxiety;
3) No more cravings at all;
4) No more anxiety;
5) No more cravings at all;
6) No more anxiety.

Dave Siever
3) Feeling rested and grounded;
4) Feeling 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 has been completely free of any alcohol consumption for two months! (Notice his markings on his SCL-90R 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 DAI’s 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-excite the networks in the brain, which have recently been discovered using diffuse tensor imaging, sometimes referred to as Hagmann hubs (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 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 DAI.

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 DAI. The devices are safe, portable and low cost and can be sent home with a patient with minimal instruction. Figure 46 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.
References


