Audio-Visual Entrainment: Finding a Treatment for Post-Traumatic Stress Disorder

Dave Siever, Edmonton, Alberta, Canada, 2014

Abstract: Post-traumatic stress disorder (PTSD) is the aftermath of trauma. Trauma spans a diverse spectrum of unfortunate life experiences such as sexual abuse, assault, car accidents, and natural disasters. PTSD occurs when the inflicted are unable to cope. Following trauma, permanent changes occur within the brain that increases "racy-headedness," guardedness, anxiety, depression, insomnia, plus memory and cognitive impairments. The behavioral aftermath of PTSD typically involves increased aggression and drug and alcohol abuse. Audio-visual entrainment (AVE) has been shown to reduce anxiety and insomnia and improve coping for police officers. AVE has also been shown to reduce depression and anxiety among vets with chronic fatigue syndrome and fibromyalgia.

Introduction

The American Psychiatric Association defines psychological trauma as *a threat to life, to yourself or someone close to you accompanied by intense fear, horror or helplessness*. The ensuing brain damage from severe and chronic stress further brings about a host of trauma-related psychiatric disorders or *trauma spectrum disorders*, which include impairments in learning, reasoning, rationalizing, impaired alertness and increased destructive behavior including smoking, alcoholism, drug abuse, family violence and reckless risk taking (Bremner, 2002). Psychological trauma affects about half of all Americans sometime in their lives. As described by Bremner, PTSD comes about from one acquiring the knowledge or "wisdom" that the world is not a safe place, as once believed.

Every year, in the USA, more than 1 million children are confirmed as victims of child abuse (Teicher, 2002) and close to 50 million American adults have suffered from childhood abuse. Somewhere between 25 and 50 % of all Americans are exposed to a psychological trauma related to a wide variety of incidents including child abuse, assault, rape, car accidents, natural disasters, etc., at some time in their lives (Acierno, et al, 1999). Of these, about 15% will develop post-traumatic stress disorder (PTSD), roughly comprising 5 to 8% of the American population, making it one of the most common illnesses in the USA. PTSD is twice as common in women as men (Kessler, et al, 1995). PTSD is 10 times more common than cancer, yet society dedicates only one tenth the funding in PTSD research as it does for cancer research (Bremner, 2002).

About one million young men experienced the stress of the Vietnam War between 1963 and 1971 and several hundred thousand were deployed in the Gulf War from 1990 to 1991 (Bremner, 2002). Currently, U.S. Service Members serving overseas in theaters of operation in Afghanistan and Iraq are continually subjected to direct and indirect traumatic effects of combat, which includes shelling, artillery, missile attacks, watching people die and dealing with burnt, charred bodies. Service members assigned to combat support and service support units that are not on the front lines are just as exposed to the effects of PTSD, since rear echelon units no longer have the traditional distinction of being non-combative. The individual service member's physical condition, training and experience for combat will certainly prepare him or her for these various traumatic experiences often encountered during military missions. However, no matter how much an individual is trained to deal with the tragedy of war, trauma is inevitable.

Other major contributing factors related to PTSD are combat casualties, such as those related to a permanent disability such as amputation. These soldiers not only experience the immediate trauma from the event and struggle to dissociate from its significance, but also must deal with a physical irreversible change in their life, where psychological therapy is required for adaptation. The effects of PTSD are not one-sided. It also affects the spousal relationship, and puts as much or more stress on the spouse with the burden and apprehension of deployment and feelings of abandonment. Upon returning home, the dysfunction of PTSD has an immediate effect on the relationship of the veteran, spouse and family. If not diagnosed and treated promptly, PTSD quickly manifests itself into a socio-economic burden on society. To exemplify the far-reaching aspects of PTSD, it has been reported that more veterans have died in motorcycle accidents at home in the USA from thrill seeking (350 deaths) than in Afghanistan (259 deaths) (Edmonton Journal).

With the exceeding numbers of civilian and military Americans that suffer from PTSD, research to develop a non-drug treatment/method of therapy for treating PTSD, and without adverse side effects, would be an asset to both the inflicted as well as society.

Physiology of the Fear Response

The survival response rapidly activates via the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis is a "triangle" in which the hypothalamus and pituitary glands in the brain communicate with the adrenals. In response to stress, the hypothalamus releases corticotrophin-releasing factor (CRF), which causes the anterior pituitary gland to make adrenocorticotrophic hormone (ACTH). This in turn causes the adrenals to produce glucocorticoids such as cortisol and adrenalin which stimulates the spleen to increase red blood cells to send more oxygen to the muscles, dilates the pupils of the eyes for better vision, and releases endorphins to dull the sense of pain (Bremner, 2002). Cortisol also raises blood sugar concentrations, increases energy to the periphery and inhibits the immune system.

The Role of Serotonin in Behavior

Serotonin acts as the brain's brakes, keeping basic emotions (such as sex, mood, appetite, sleep, arousal, pain, aggression, and suicide ideation) in check. Serotonin also influences dominance and has been shown to be high in dominant male vervet monkeys and also found in salesmen with high sales performance. These salesmen averaged 180 ng/ml levels of whole blood serotonin (WBS), whereas the poor performers had average WBS levels of 140 ng/ml of blood volume (Walton, et al., 1992). A study by Raleigh (Kotulak, 1997) found that when sub-ordinate monkeys were given a serotonin uptake inhibitor like Prozac, they became dominant through friendship and alliances with the females. Dominant monkeys deficient in serotonin ruled with aggression. Like the "Prozac" monkeys and salesmen, college students with the most friends had serotonin levels 20 to 40% above the norm. Females have 20 to 30% more serotonin than men, which contributes to their reduced aggression (Kotulak, 1997). Those with high levels of serotonin "connect" better socially with improved ability to read facial expressions (Harmer, et al., 2003).

In an experiment with normal young males, the task was to be the first person to push a button when a light flashed. The successful person could give their partner an electric shock in the range from 1 (mild) to 8 (strong). Normally, the shocks given were mild and relatively "tit-for-tat." However, when one of the pair was given a serotonin antagonist, that person would frequently deliver more severe shocks (above "4") even if they received shocks that were mild. On the other hand, if one of the pair was given tryptophan, a pre-cursor to serotonin, that person would deliver milder shocks to his partner even if he received strong shocks from his partner, (Young and Pihl, 1988).

Low levels of serotonin are tied to loss of control (helplessness), which manifest this deficiency in temper and rage (Sapolsky, 2003). Reports from my clients indicate anxiety from traffic congestion, tight scheduling, computer problems, corporate "right sizing," the oxymoron of "customer service" with many businesses, and other factors that have come with the "modern" age. All of these "highlighters of helplessness" contribute to highly increased frustration and aggression much like studies with mice that are given random electrical shocks beyond their control (Sapolsky, 2003). So it's no wonder that even though the population of the USA has only increased by 40% from 1960 through 1991, aggravated assaults have increased by 600%, violent crime by 560%, rapes increased by 520%, and murders by 170%, according to FBI stats (Kotulak, 1997).

People low in serotonin (and 5HTP) have an increased risk of sexual deviance, alcoholism, fire-setting, obesity, and other impulse-control disorders (Kotulak, 1997). Conditions such as anxiety, depression and tendencies toward alcohol and drug abuse have been shown to run in families (Virkkunen, 1989), where a study of 114 male alcoholic and violent offenders and fire-setters showed that low levels of cerebral spinal fluid (CSF) 5-hydroxyindoleacetic acid (5-HIAA) and homovanillic acid (HVA) were strongly associated with a family history of paternal violence and alcoholism. A study by Linnoila (1983) of prisoners who were in jail for manslaughter, used serotonin levels as a basis in predicting with 84% accuracy, those who would recommit manslaughter after their release.

The Role of Norepinephrine in Behavior

Noradrenalin or norepinephrine (NE), a close relative of adrenaline is also a player in vigilance (Bremner, 2002). Low levels of noradrenalin are associated with under-arousal including lethargy and mental fuzziness (Amen, 1998), while higher levels are related to peak performance, and high levels correlate to impulsive "hot-headed" violence (Kotulak, 1997).

Norepinephrine is manufactured in the locus coeruleus (dorsal pons), a site in the brainstem. Long neurons project to multiple sites throughout the brain for direct and immediate release. NE activates in response to both internal stressors such as a drop in blood pressure due to a lack of blood and external stressors such as threats (Aston-Jones, Chiang & Alexinsky, 1991). During rest, feeding and grooming in primates, the NE system is quiet, but activates rapidly with the perception of a threat, increasing heart rate and blood pressure and behaviors of aggression (Aston-Jones, Chiang & Alexinsky, 1991). NE increases on an as-needed basis. Monkeys taught to play a video game showed increased NE activation along with alertness and vigilance as the game increased in challenge. However, a threshold exists where past a certain point of challenge,

the monkeys became more anxious and distracted and performance began to decline (Aston-Jones, Chiang & Alexinsky, 1991). Animal studies have shown that animals exposed to repeated stress and cannot escape leads to the emotion of *learned helplessness*, which correlates with the depletion of norepinephrine.

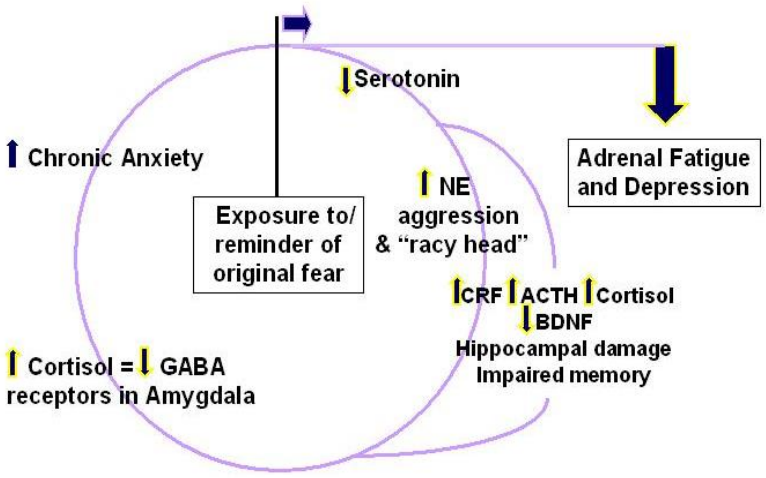
The Sympathetic Loop

The aftermath of trauma results in a double-edged sword. With the experience of trauma comes a fall in serotonin as the mind prepares to protect itself from further assault by keeping its guard up. However this drop in serotonin lowers the threshold where a person perceives threats which then increases the number of non-life threatening or social threats. Unfortunately, the limbic system never evolved beyond the "reptilian" stage and given that humans are primarily "pack" animals, very basic socio-emotional perceptions such as a mere wrong look or sense of rejection from anyone encountered in the acute/posttraumatic stress disordered person's travels (including family members or rejection from the individual him/herself) could constitute a threat and launch the PTSD survivor into aggression. Therefore, trauma poses a socio-economic toll, producing aggression and violence, family breakup, and lost productivity in the workplace, not to forget hundreds of millions spent annually on illicit and prescription drugs.

Survivors of trauma, once crossing the serotonin/norepinephrine threshold, get locked into a *sympathetic loop* as shown in Figure 1. Assuming that the original trauma has come and gone, and in the case of PTSD, the loop maintains itself from simple thoughts, reminders of, and imagination of past threats (and/or new threats with the lowered fear threshold). This continues a drop in serotonin and increased norepinephrine, and in turn maintains increased aggression and endless rumination or "racy head," which keeps the self-imagined fear going. (In part, this is caused by outgoing fibers feeding into the frontal cortex causing one to believe he/she is threatened, which in turn reinforces activation of the amygdala). As mentioned, this triggers the production of CRF, which, in turn, triggers ACTH, which then stimulates the adrenals as the body of the threatened individual prepares for battle. Cortisol antagonizes brain–derived neurotrophic factor (BDNF), ending in hippocampal neuronal necrosis (cell death) and impaired memory. The increased cortisol also causes GABA "strippage" within the amygdala, impairing the ability to relax, thus increasing tics, twitches and incidence of temporal-lobe seizures (Teicher, 2002).

And as the PTSD survivor alienates and loses his/her support network of family and friends, he/she feels more isolated and rejected. This perceived barrage of threats to the livelihood of the inflicted keeps the serotonin low, the cortisol flowing, norepinephrine (and anger) high and continuing amygdala GABA receptor strippage, resulting in the inflicted being no longer able to "idle," and in a constant "revving" state, constantly on guard and with this cycle feeding on itself. Eventually, the endocrine system exceeds its threshold, and "burns out," in a condition known as *hypoadrenia* or adrenal fatigue (Wilson, 2001). As the adrenals fatigue, so does the locus coeruleus resulting in reduced NE levels in the brain and increased suicide ideation.

Figure 1



Somatic Damage from Chronic Fear and Trauma

Continuous bouts of activation of the hypothalamic-pituitary-adrenal-axis (HPA) exact a personal toll on the body. This results in irritable bowel syndrome, tension and migraine headache, neck and spine problems, temporo-mandibular dysfunction, heart disease, skin rashes, slow recovery from viral and bacterial infections, insomnia, alcoholism and drug abuse (Everly, 2002) and ulcers, diabetes and osteoporosis (Bremner, 2002). The eventual adrenal fatigue leads to low blood pressure, chronic fatigue, and fibromyalgia, frequent respiratory infections and difficulty recovering from them (Wilson, 2001). Behavioral components are extreme fatigue in the morning – leading to consumption of caffeine, and an energy surge in the late evening – leading to consumption of alcohol and drugs for sleep (Wilson, 2001).

Cognitive Damage from Chronic Fear and Trauma

While acute (mild) stress seems to enhance mental function, chronic (severe) stress impairs hippocampal function, which in turn, may lead to multiple sclerosis, anxiety, depression, posttraumatic stress disorder, schizophrenia and Alzheimer's disease (Esch, et al., 2002). The most common structural changes from PTSD are reduced hippocampal volume (impaired experiential evaluation and memory), increased amygdalar activation (emotional activation) and decreased activity in Broca's area (impaired verbal expression), (Hull, 2002), and those going into combat with pre-existing smaller hippocampal volumes are predisposed to PTSD (Gilbertson, et al., 2002). Both Vietnam war-vets and women with abuse-related PTSD have reduced blood flow in the hippocampus and medial prefrontal cortex (Bremner, et al, 1999). The medial aspects of the pre-frontal cortex are instrumental in extinguishing fear responses to conditioned stimuli (Ledoux, 1996). People with PTSD do not have normal activation of the prefrontal medial cortex and are not able to extinguish their own fear responses while watching a movie involving violence (Bremner, et al., 1997), whereas people without PTSD are able to rationalize that they are only watching a movie and do not show a trauma response to the movie. This means that people with PTSD have crossed the threshold of being able to return to a relaxed

homeostasis and therefore live in an irrational and constant state of fear. The U.S. Army is researching the use of *Virtual Realty (VR) Exposure Therapy* with biofeedback for Iraqi veterans diagnosed with PTSD (Rizzo, 2005), which suggests traumatic association for adaptation. This is unlike dissociation for treatment of PTSD that is achieved by audio-visual entrainment (AVE).

This continued state of fear also inflicts damage to the frontal and temporal regions, known as *frontotemporal dementia* (Bremner, 2002). Frontotemporal damage further impairs a person's ability to control fear and the ability to reason and understand the significance of events in his/her life (Bremner, 2002), leaving the inflicted in a generalized state of anxiety, fear and confusion. Anxiety and fear increases cortisol in the brain. Cortisol counteracts a brain-nourishing hormone called *brain-derived neurotrophic factor* or BDNF (Bremner, 2002). Loss of BDNF leads to neuronal cell death within the hippocampus, which impairs declarative or explicit memory and the ability to recall details of events (Sapolsky, 2003). In fact, those with PTSD often cannot remember what they had for breakfast a few hours before and have extreme difficulty learning new things (Bremner, 2002). Unfortunately, PTSD inflicted dementia can affect persons as young as teenagers (Bremner, 2002). Dementia is a problem particularly for war veterans and is the reason why it is difficult to succeed in career retraining for civilian life (Bremner, 2002). The pre-frontal lobe damage and continued irrational fear continue to destroy the hippocampus. Hippocampal loss also plays a major role in the early onset of Dementia of the Alzheimer's Type, where the ability to form memories later in life is impaired. Those living in fear have further impairments in their memory and self-reliance in remembering, as they become seniors (Levy, 1996).

Affective Disorders Stemming from Trauma

Most people, in the aftermath of trauma, also succumb to affective disorders. Affective disorders pertain to disorders of emotion, including depression and anxiety and mania. Depression is the most common psychiatric disorder by far. About 14% of the American population will experience clinical depression in their lifetime. Of these, an alarming 15% will unfortunately commit suicide (Rosenfeld, 1997). The helplessness of depression is not a quiet, passive state; rather it is an active, all-consuming dreadfulness! The reality of this situation in the military is exemplified in The New England Journal of Medicine; Combat's Toll on a Soldier's Psyche by COL Charles Hoge's, MD , Chief of Psychiatry and Behavioral Science, US Army (Hoge, 2004).

Shealy, et al, (1992) studied blood-serum levels of five neurochemicals (melatonin, norepinephrine, B-endorphin, serotonin, cholinesterase) in depressives. He found that 92% of depressives had abnormal levels in at least one of the five neurochemicals tested and 60% showed three or more abnormalities. In over half of the depressives he found either elevated or low levels of norepinephrine/cholinesterase ratios. He also found magnesium deficiencies in 80% of depressed patients and 100% of those with depression were deficient in taurine, an amino acid found in meat and fish, which is used to help absorb fats and fat-soluble vitamins. His work supports the notion of dietary supplements for the treatment of depression.

The nucleus accumbens within the forebrain is a primary reward and pleasure center and is primarily sensitive to dopamine, serotonin and endorphins (Ratey, 2002). Recent research has shown that those with suicide ideation are also low in serotonin, dopamine and norepinephrine along with hippocampal shrinkage as the result of chronic sympathetic and adrenal (cortisol) activation (Ezzel, 2003). Stimulant drugs such as amphetamines and cocaine produce a sense of pleasure by changing the concentration of dopamine in the accumbens.

Arango and Mann (Oquendo, et al., 2003) observed with positron emission tomography (PET) scans, a direct correlation between ventral pre-frontal hypofunction levels of serotonin, also in the pre-frontal cortex and the severity of violence of the chosen suicide method. Slightly lower levels may produce death by an overdose of sleeping pills while extreme deficits will lead to the person jumping off of a cliff or blowing his/her brains out.

Serotonin has been well implicated as a driving mechanism for suicide, where both genetic factors and a string of upsetting life events combine to trigger suicide (Ezzel, 2003). In sectioned brains it is clear that suicide victims have fewer than average neurons in the orbital prefrontal cortex. A study by Chaouloff (2000) reinforced the hypothesis that the HPA axis, in reaction to stress, affects serotonin neurotransmission, partly through the actions of corticoids.

Violence and suicide are related. Aggression is aimed at others when there is a combination of *low serotonin* and *high norepinephrine*, whereas aggression is aimed inward (increased suicidal ideation) when there is a combination of *low serotonin* and *low norepinephrine* (Kotulak, 1997).

Antidepressants and Electroconvulsive Therapy in the Treatment of Depression

Several studies have examined cerebral blood flow (CBF) and metabolism using positron emission tomography (PET), single photon emission computerized tomography (SPECT) and functional magnetic resonance imaging (FMRI) analysis (Rubin, Sacheim, Nobler, & Moeller, 1994). Much controversy surrounds these studies. Functional imaging studies have shown confounding (both high and low) irregularities in metabolism, primarily in the basal ganglia, prefrontal and limbic areas that tend to normalize in those who respond to medication. In some cases, sleep deprivation reduces depression and is tied to reductions in abnormally high CBF within the anterior cingulate gyrus (Wu, et al., 1992). PET scans of those with seasonal affective disorder (SAD) showed both hypo and hyper perfusion of CBF in various regions of the frontal cortex, which normalized following treatment (Cohen, et al., (1992). Antidepressant medication has been shown to affect capillary permeability and the brain-blood barrier (Preskorn, Raichle, & Hartman, 1982). With electroconvulsive therapy (ECT), the electrodes are placed for whole-brain or right-side shocks. ECT has been widely used to treat depression. CBF reductions follow shortly after exposure to ECT, even with people who already have hypo-perfusion of CBF. For depression, ECT is generally administered to the right side (Rubin, et al., 1994). Right-side CFB reduction would help offset the "alpha" asymmetry, recognized in the QEEG field to be associated with depression and disturbed mood (Rosenfeld, 1997; Siever, 2003) by shutting down right frontal lobe function rather than boosting left frontal lobe function. The rational of using meds or ECT to impair the right hemisphere of the brain is akin to shooting a hole in the right tire of a car that is always pulling to the left because of a left flat tire. Even though this will

make the car drive straight, it will be very slow. This explains why those on anti-depressants have so much trouble with foggy-headedness and cognitive impairments. This is one of the reasons why pilots and special duty personnel (i.e., Nuclear Surety Program) are medically suspended from duty while on antidepressants. Rubin concluded that both antidepressants and ECT (even with clinical improvements) might further affect regions in the direction of further abnormality, not normalization.

Conclusion

The fear response involves the reduction of serotonin and activation of cerebral norepinephrine and the adrenals as the threatened prepare for battle. However, severe traumas can cause a dysfunctional never-ending activation of the fear response, which fatigues key neurotransmitters and the adrenals as it manifests into PTSD. The implications of PTSD include a combination of family and societal violence, alcohol and drug abuse, loss of wages and increased suicide ideation. Often, pharmaceutical agents and electroconvulsive therapy may alleviate the depression, but drive the brain further into dysregulation, leaving the patient feeling emotionally "numb" and struggling with cognitive impairments. A new non-drug, non-ECT approach needs to be considered.

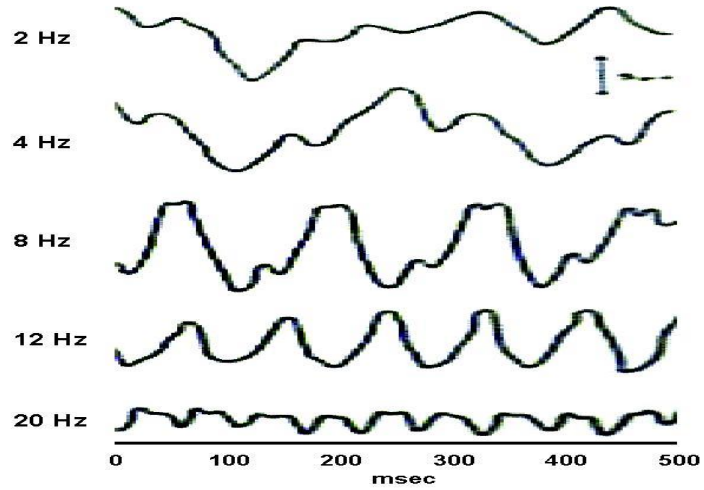
Audio-Visual Entrainment (AVE)

All sensory information, except for smell, must pass through the thalamus in order to gain access into other brain regions. Audio, visual and tactile stimulation all excite potentials within the thalamus and are loosely known as brain wave entrainment. AVE is the entrainment method using light and sound pulses only. Pulsed light is presented to the user via a pair of glasses (Tru-Vu Omniscreen™ Eyeset) with an array of flashing LEDs mounted within them. Pulsed tones are presented through a pair of headphones. In order for entrainment to occur, a repetitive stimulus of the proper frequency and sufficient strength to "excite" the thalamus must be present. The transmission of energy from AVE excites retinal cells in the eyes and pressure sensitive cilia in the cochlea of the ears. The nerve pathways from the eyes and ears carry the *evoked* potentials into the thalamus. From there, the entrained electrical activity within the thalamus is "amplified" and distributed throughout other limbic areas and the cerebral cortexes via the *cortical-thalamic loop*. This is a loop between the cerebral cortex and the thalamus that generates the *alpha rhythm* at approximately 10 Hz during neuronal rest (Demos, 2005). In essence, audio-visual entrainment (AVE) *is the continuous electrical response of the brain in relation to the frequency of the stimuli plus the mathematical representation (harmonics) of the stimulus wave shape.* The device, which delivers the entraining light and sound pulse-stream is called the *Digital Audio Visual Integration Device*, or DAVID. Because most maladies have an abnormal brain wave "signature", the DAVID device can help treat stress and anxiety, depression, insomnia, impact of trauma, racy mindedness, attention disorders, fibromyalgia and cognitive decline.

LEDs and xenon strobe lights contain much harmonic content due to the "squareness" or rapid turn-on and turn-off transitions of the stimuli and these harmonics are reflected within the EEG (Kinney et al., 1973). Entrainment occurs best near the natural alpha frequency from 9 to 11Hz (Toman, 1941). Kinney's study (Figure 2) shows strong and pure entrainment at 12 Hz. The

harmonics (small wavelets) seen in the EEG are a reflection of the harmonics produced in the EEG from the Xenon strobe-light stimuli.

Figure 2. EEG Showing Photic Entrainment at Various Frequencies

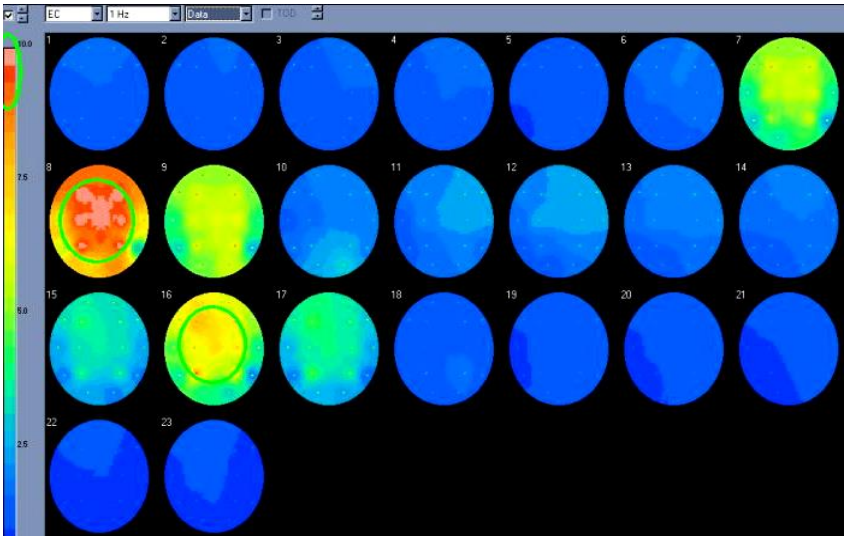


AVE at 18.5 Hz has also been shown to produce dramatic increases in EEG amplitude at the vertex (Frederick, Lubar, Rasey, Brim, & Blackburn, 1999). It was found that:

a) eyes-closed 18.5 Hz. photic entrainment increased 18.5 Hz EEG activity by 49%.
b) eyes-open auditory entrainment increased 18.5 Hz. EEG activity by 27%.
c) eyes-closed auditory entrainment increased 18.5 Hz EEG activity by 21%.
d) eyes-closed AVE increased 18.5 Hz. EEG activity by 38.3%.

Entrainment primarily shows itself in the frontal, central and parietal regions. (Siever, 2002). Figure 3 shows the results of a 19-channel quantitative EEG (QEEG), or "brainmap" (Demos, 2005) as processed through the Skil (Sterman-Kaiser Imaging Labs) database in 1 Hz bins (sorted into 1 Hz groupings) showing the frequency distribution of AVE at 7.8 Hz. The area within the green circle at 8Hz shows maximal effects of AVE in central, frontal and parietal regions (at 10 microvolts, in this case) as referenced with the area in the oval on the legend. It is through these effects that AVE has proven effective in treating depression, anxiety and attentional disorders. A second harmonic is also present at 16 Hz. (the circled image), which is typical of *semi-sine* wave (part sine/part square wave) stimulation.

Figure 3. Brain Map in 1Hz Bins -- During 7.8 Hz AVE (SKIL-Eyes Closed)



Body/Mind Effects of Audio-Visual Entrainment

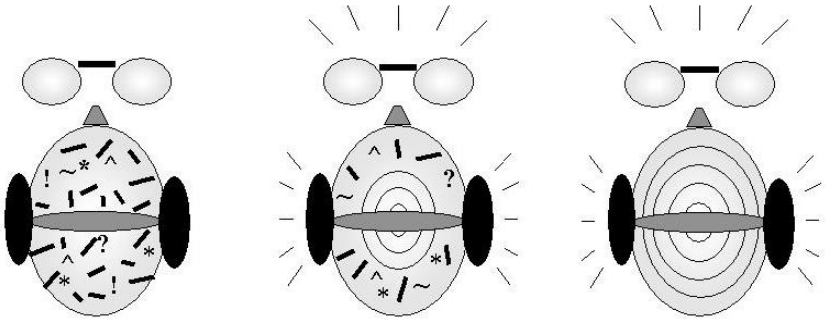
We conceptualize AVE as achieving its effects through several mechanisms at once (Siever, 2000). These include:

1) dissociation / hypnotic induction,
2) increased neurotransmitter,
3) altered cerebral blood flow,
4) normalized EEG activity.

Dissociation

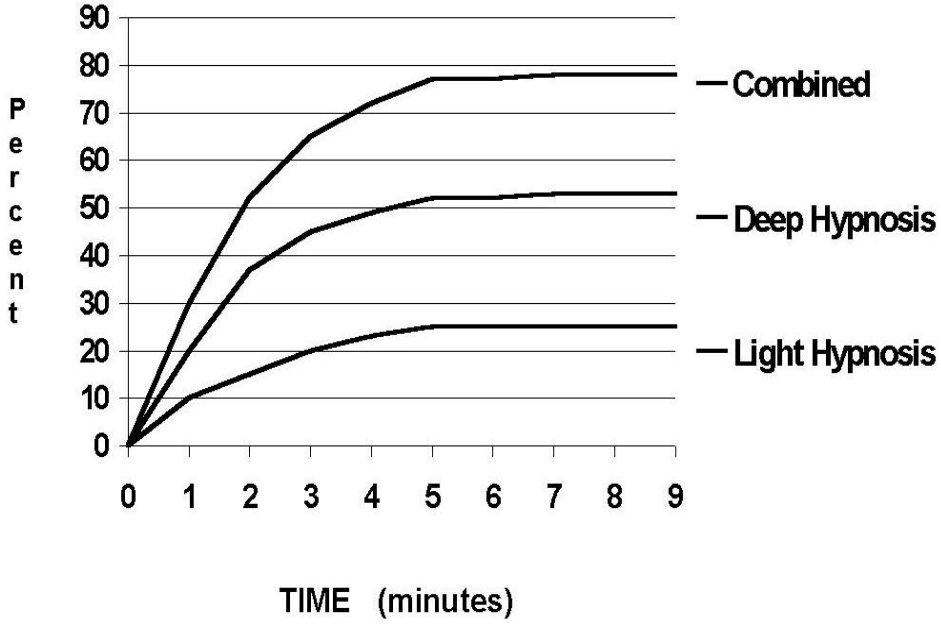
Dissociation, as a tool in psychotherapy, helps in diminishing the emotional component of disruptive memories. Dissociation, when referring to AVE, is a "disconnection" of self from thoughts and somatic awareness, as experienced during deep meditation (Figure 4). AVE induced dissociation is rapid, requires only 4 to 10 minutes in most cases and provides an excellent means for clearing the mind of destructive, fearful thoughts and allowing the person to relax and restabilize (Siever, 2000).

Figure 4. Dissociating from Negative Thoughts using AVE.



Visual entrainment alone, in the lower alpha frequency range (7-10 Hz), has been shown to easily induce hypnosis (a form of dissociation). It has been shown that nearly 80% of subjects enter into a hypnotic trance within six minutes during alpha photic entrainment (Kroger & Schneider, 1959), as shown in Figure 5.

Figure 5. Photic Stimulation Induction of Hypnotic Trance



Inducing dissociation using AVE delivered by the DAVID1 was found to be more effective than dot staring or stimulus deprivation (Leonard, et al., 1999). AVE using the DAVID Paradise demonstrated to be effective in clinically dissociating people with dissociative anxiety while simultaneously calming them down somatically and reducing their heart rate (Leonard, et al, 2000). As a result, AVE may be used as an effective desensitization tool for reducing dissociative anxiety that is sometimes seen in the PTSD population.

Limbic Stabilization

As mentioned, the amygdala initiates the activation of the fight-or-flight response, which activates the hypothalamus, which in turn controls all autonomic functioning and is responsible for the "tensed up" feeling in the body (chest breathing, shortness of breath, racing heart, cold, clammy hands, tense muscles, etc.) that is experienced during a fear response. Anyone who has consumed too much coffee will be familiar with these feelings.

AVE produces a calming effect on limbic structures, such as the amygdala and hypothalamus because properly applied AVE produces a *restabilization* effect where muscles relax (Thomas & Siever, 1989), electrodermal activity settles down, peripheral blood flow stabilizes (hand temperature normalizes to 86-90 F), breathing becomes diaphragmatic and slow, and heart rate slows and becomes uniform (Siever 2000).

Figures 6 and 7 show the calming effect of AVE on the somatic functions of forearm EMG, finger temperature (Hawes, 2000). Heart rate and heart-rate variability (HRV) are sensitive measures of stress (Stein, P., Kleiger, R. (1999). Figure 8 shows graphs of the "FreezeFramer" HRV analysis system manufactured by Heartmath. It shows dramatic improvements in both heart rate and HRV in a woman with adult onset PTSD. Within 10 minutes, heart rate dropped by 22 bpm, and she showed dramatic reductions in both sympathetic and parasympathetic activity ("blue mountains").

Figure 6. Forearm EMG Levels During AVE (Hawes, 2000)

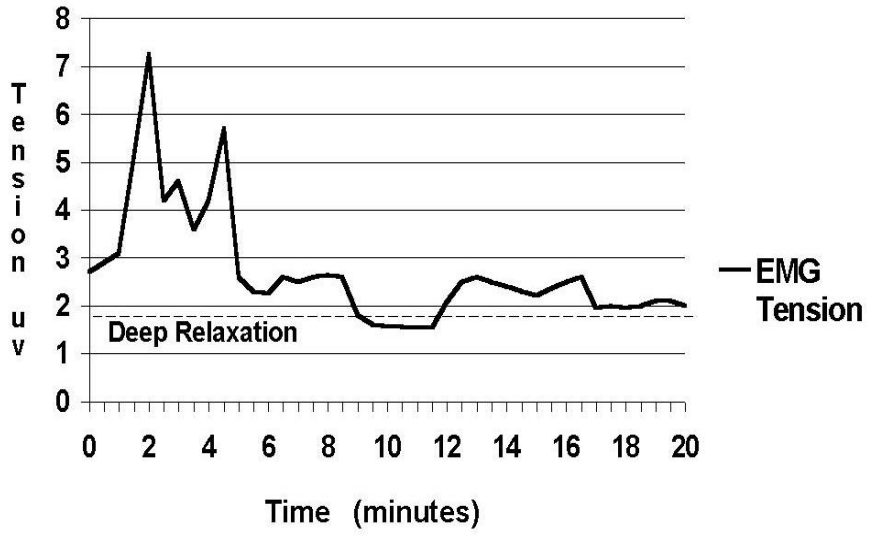


Figure 7. Peripheral Temperature Levels During AVE (Hawes, 2000)

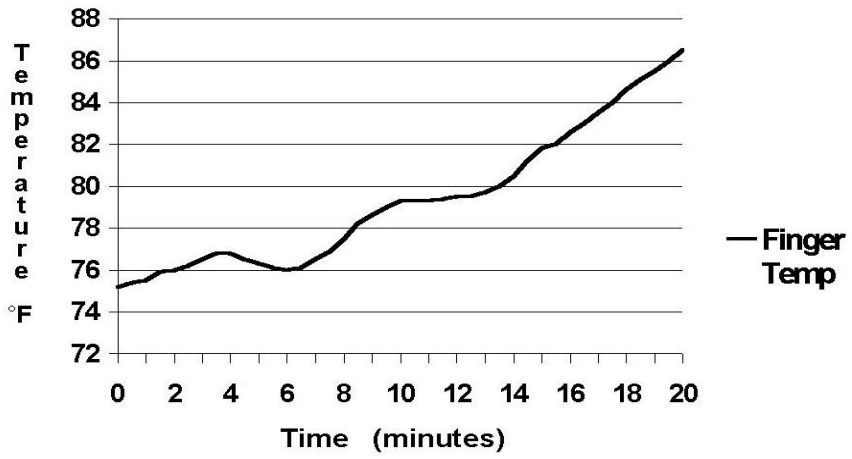
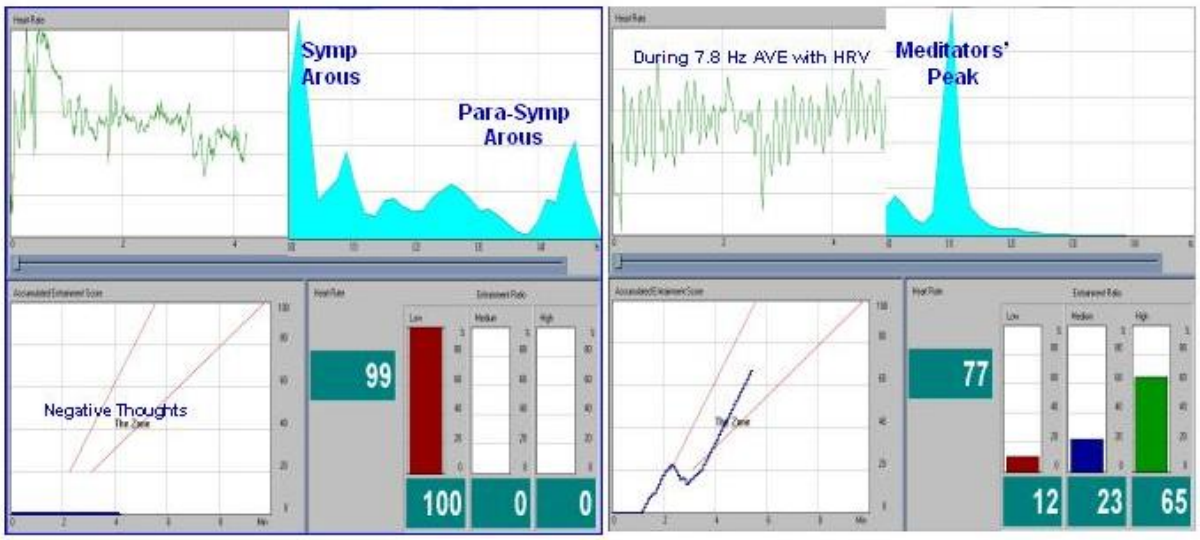


Figure 8. Pre and Post HRV in a Woman with Adult PTSD.

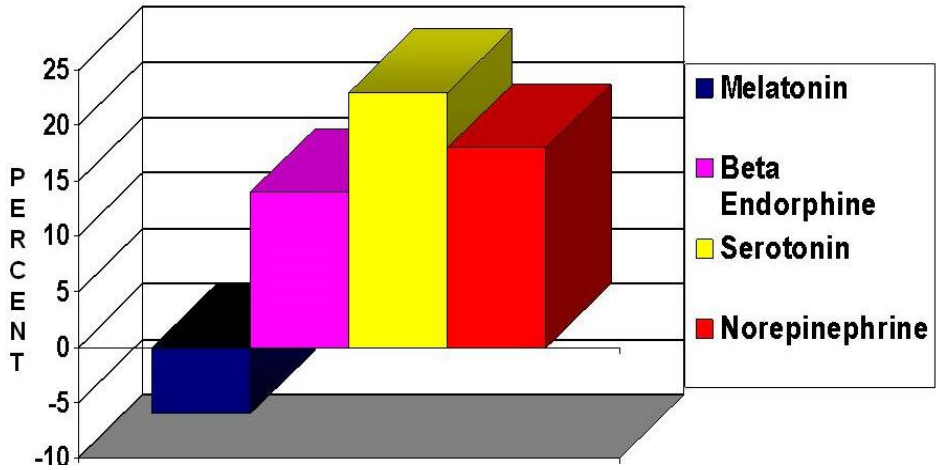


This lady's life was "turned upside down" when the police showed up at her door and charged her husband with molesting two young girls (ages 6 and 8) and possessing explicit child pornography. Between the moral trauma of realizing she shared her bed and children with such a sick aggressor, the loss of a mate, plus the rejection of her and her family from the local citizens of the small town where she lived, she moved to the city and filed for divorce. Her 'Ex' also moved to the city, continues to be aggressive and blames her for his "problem." He contends he has done nothing wrong, citing "the girls seemed OK with it." A judge awarded him alternate weekends of her young son (8 years old) and daughter (6 years old), which upsets her greatly.

Using AVE to Balance Neurotransmitters

As mentioned previously, people with clinical depression are low in serotonin, dopamine and norepinephrine. As shown in Figure 9, 30 minutes of white-light AVE at 10 Hz increased serotonin levels by approximately 23%, endorphin levels and norepinephrine by 18%, (Shealy, et al, 1989) leading to increased hopefulness, self-esteem, improved sleep, reduced pain and reduced anxiety.

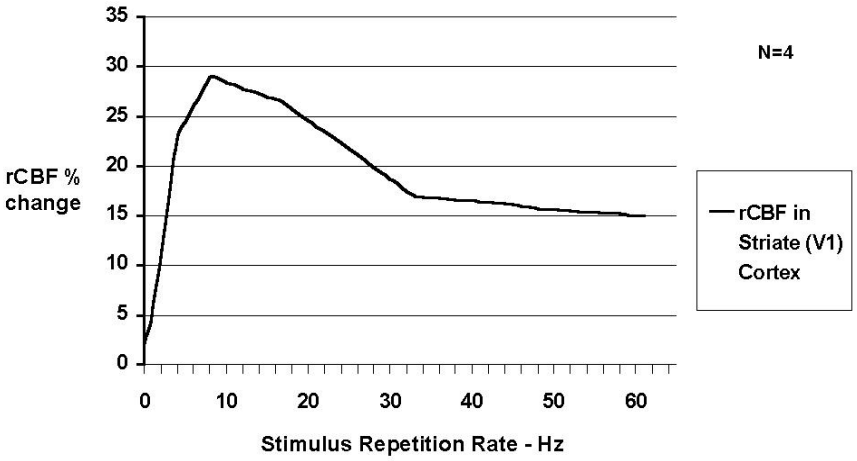
Figure 9. The Affect of White Light AVE on Neurotransmitter Production



Cerebral Blood Flow and Metabolism

SPECT and FMRI imaging of CBF show that hypoperfusion of CBF is associated with many forms of mental disorders. There is even greater concern regarding conditions involving hypoperfusion of CBF in frontal regions such as with trauma and PTSD. Frontal disorders include: anxiety, depression, attentional and behavior disorders, and impaired cognitive function (Amen, 1998). Adequate cerebral blood flow (CBF) is essential for good mental health and function. AVE increases brain glucose metabolism and cerebral blood flow (Sappy-Marinier et al., 1992). A study by Fox and Raichle (1985) showed overall increases in cerebral oxygen consumption by 5% and increased CBF in the striate cortex, (a primary visual processing area within the occiput), peaking at 28% increase at 7.8 Hz as shown in Figure 10. Accomplished Zen meditators also show a peak frequency of 7.8 Hz during meditation (Cade, 1987), and is the most common frequency used for reducing anxiety in AVE therapy.

Figure 10. Cerebral Blood Flow at Various AVE Repetition Rates



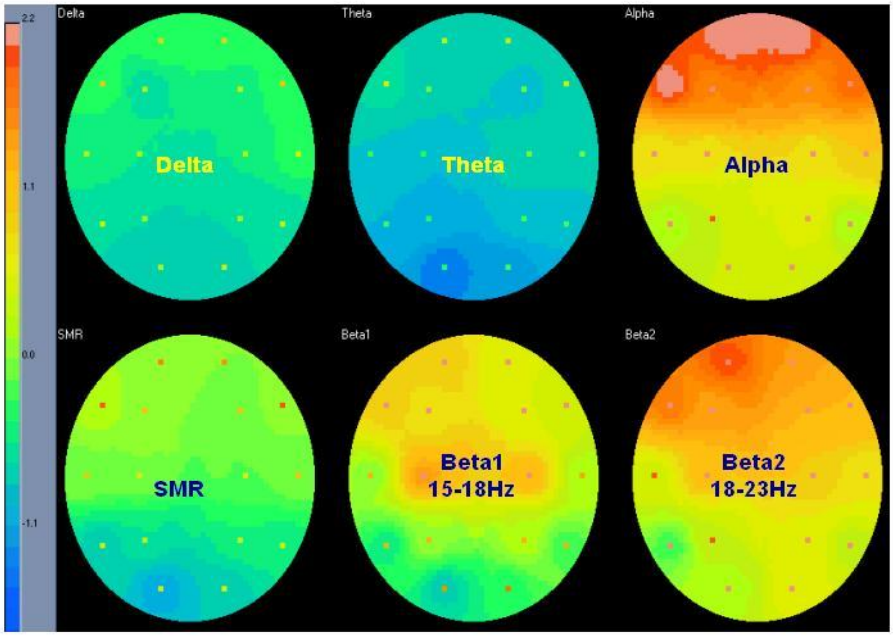
In addition, AVE has also been shown to increase CBF throughout various other brain regions including frontal areas (Mentis, et. al., 1997; Sappy-Marinier, et. al, 1992). A whole head PET analysis of visual entrainment at 0, 1, 2, 4, 7, and14 Hz on 19 healthy, elderly (mean age=64 years) subjects (Mentis, et. al., 1997) found that regional cerebral blood flow (rCBF) was activated differentially with the:

1) left anterior cingulate showing maximal increases in rCBF at 4 Hz.
2) right anterior cingulate showing decreases in rCBF with frequency.
3) left middle temporal gyrus showing increases in rCBF at 1 Hz.
4) striate cortex showing maximal rCBF at 7.8 Hz.
5) lateral and inferior visual association areas showing increases in rCBF with frequency.

Normalized EEG Activity

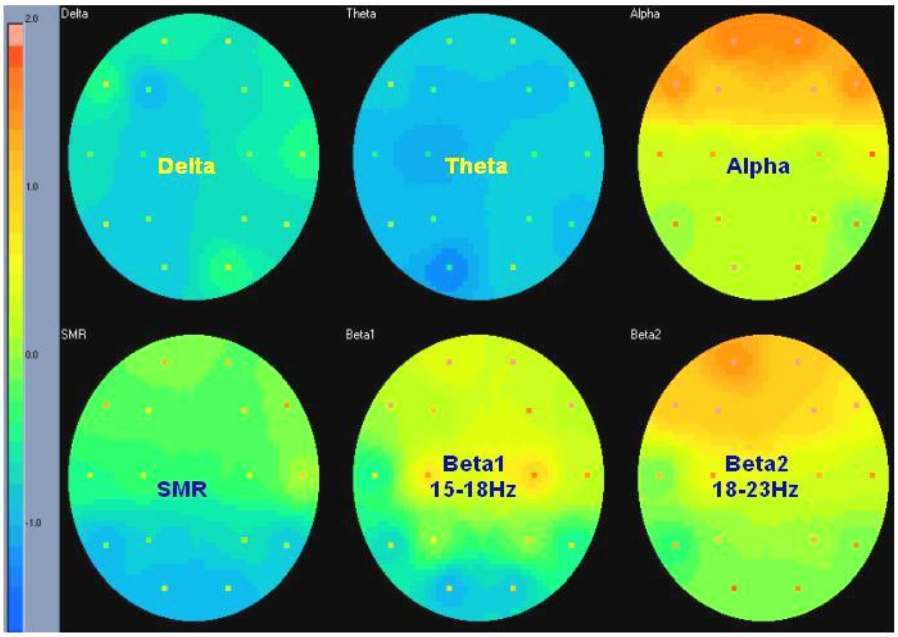
Figure 11 shows a fairly typical brain map in 1 Hz bins of a person with depression and anxiety from trauma as shown on the Skil database. The scale is 2.2 standard deviations (SD) and the pink area in the alpha view is actually 2.6 SD. Activity above 2 SD is considered a clinical abnormality. Notice that alpha activity is higher on the left side coincident with a personality based on withdrawal from negative stimuli. Also, the generalized red colored region is an indicator of generalized cognitive fatigue. The Beta2 activity is just approaching 2.2 SD (an indication of mild anxiety). Non-clinical persons have greater right frontal alpha associated with an attraction toward positive stimuli (Demos, 2005).

Figure 11. Brain Map of Individual with Depression and Anxiety (Skil-Eyes Closed)



Approximately 10 minutes after a 30-minute AVE session designed to reduce the symptoms of depression, both alpha and beta activity are normalized as shown below in Figure 12. Notice that the frontal alpha activity, as well as the Beta2 activity, is roughly 1.2 SD above average. The participant was also subjectively aware of his elevated mood and energy.

Figure 12. Brain Map Following a Mood-Booster AVE Session (Skil-Eyes Closed)



Conclusion

AVE is a powerful technique for treating disorders emanating from chronic fear, be it real or perceived. Chronic rumination, hypoperfusion of cerebral blood flow, loss of neurotransmitters, altered brain wave activity, and adrenal fatigue all contribute to PTSD and the continuation of PTSD. These effects also play a part in anxiety, bodily ailments of all kinds, aggression toward family and civilians at large, depression, substance abuse, and loss of work productivity.

The Digital audio-visual entrainment device (DAVID) has the ability to dissociate the inflicted away from destructive distressing rumination, increases blood flow, normalizes neurotransmitter production, calms the limbic system, restores the adrenals, and produces somatic relaxation. Therefore, the subjective benefits of AVE are reduced anxiety, improved sleep, increased energy, improved relationships with family and civilians, reduced physical problems, improved productivity and reduced dependence for self-medicating on alcohol and drugs. No study has been done to date on the use of AVE in the treatment of PTSD. However, there are hundreds of anecdotal cases of childhood and adult trauma, including abused women, police and emergency personnel confirming the benefits of AVE as a treatment methodology. AVE has been shown to reduce depression and impulsiveness while improving sleep in war vets with either chronic fatigue syndrome or fibromyalgia syndrome (Trudeau, 1999). AVE also has a proven history in treating posttraumatic stress related disorders for the Royal Canadian Mounted Police (RCMP) in Kamloops, British Columbia, where 80% of the officers respond with improved sleep onset, improved quality of sleep and reduced daytime anxiety (Carmichael, 2006).

About the Author

Dave Siever of Mind Alive, Inc. has lectured and assisted in workshops with leading psychological institutions including the Association of Applied Psychophysiology and Biofeedback, the International Society of Neuronal Regulation, the College of Syntonic Optometry, Walden University, the University of Alberta, A Chance to Grow Charter School, STENS Biofeedback Training Programs and other venues. He was also invited to lecture at the annual conference of the American College for the Advancement of Medicine in November 2006. Dave Siever has been designing and studying AVE since 1984 when he originally developed the DAVID1 to help performing arts students overcome stage fright.

Mind Alive, Inc.
David Siever, CET
President
6716 – 75 Street
Edmonton, Alberta, Canada, T6E 6T9
Office: (780) 465-6463

List of Relevant Publications

Does Stress Damage the Brain? – John Demos

Change Your Brain, Change Your Life – Daniel Amen

The Rediscovery of Audio-Visual Entrainment Technology – Dave Siever

New Technology for Attention and Learning – Dave Siever

Getting Started With Neurofeedback – John Demos

The AVE Session & Protocol Guide for Professionals – Dave Siever

The Brain's Way of Healing – Norman Doidge

References

Acierno, R., Kilpatrick, D., Resnick, H. (1999). Characterization of hippocampal norepinephrine release as measured by microdialysis perfusion: Pharmacological and behavioral studies. *Neuroscience, 27*, 897-904.

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

Aston-Jones, G., Chiang, C., Alenski, T. (1991). Discharge of noradrenergic locus coeruleus neurons in behaving rats and monkeys suggests a role in vigilance. *Progress in Brain Research*. Barnes & Pomeiano. New York: Elsevier Science Publishers.

Bremner, J., Innis, R., Ng, C., Staib, L., Duncan, J., Bronen, R., Zubal, G., Rich, D., Krystal, J., Dey, H., Soufer, R., Charnet, D. (1997). PET measurement of central metabolic correlates of yohimbine administration in posttraumatic stress disorder. *Journal of Traumatic Stress, 10*, 37-50.

Bremner, J., Narayan, M., Staib, L., Southwick, S., McGlashan, T., Charney, D. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *American Journal of Psychiatry, 156*, 1787-1795.

Bremner, D. (2002*). Does Stress Damage the Brain?* W.W. Norton & Company: New York, NY.

Butcher, J. N. (1994) The MMPI-2: A new standard for personality assessment and research in counseling settings. Measurement and Evaluation in Counseling and Development, 27, 131-150.

Cade, C. M., Coxhead, N. (1987). *The Awakened Mind*. Element Books: Longmead, Shaftesbury, Dorset.

Carmichael, J. (2006) Kamloops, B.C., Canada. Email to Dave Siever.

Chaouloff, F. (2000). Serotonin, stress and corticoids. *Journal of Psychopharmacology*, *14* (4), 419-421.

Cohen, R., Gross, M., Nordahl, T., Semple, W., Oren, D., Rosenthal, N. (1992) Preliminary data on the metabolic brain pattern of patients with winter seasonal affective disorder. *Archives of General Psychiatry*, *49* (7), 545-552.

Demos, J. (2005). *Getting started with neurofeedback*. W. W. Norton & Company: New York.

Edmonton Journal. Sunday, February 19, (2006). A6. *Reprinted from Daily Telegraph*, New York.

Esch, T., Stefano, G., Fricchione, G., Benson, H. (2002). The role of stress in neurodegenerative diseases and mental disorders. *Neuroendocrinology Letters, 23,* 199-208.

Everly, G. S., Lating, J. M. (2002). *A clinical guide to treatment of the human stress response.* New York: Kluwer Academic/Plenum.

Ezzell, C. (2003). New research addresses the wrenching question left when someone ends his or her own life. Why? The neuroscience of suicide. *Scientific American, 288, (2),* 45-51.

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

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.
Gilbertson, M., Shenton, M., Ciscewski, A., Kasai, K., Lasko, N., Orr, S., Pitman, R. (2002). Smaller hippocampal volume predicts vulnerability to psychological trauma. Nature Neuroscience. Published online 15 October 2002;doi10.1038/nn958.

Harmer, C., Bhagwagar, Z., Perrett, D., Vollm, B., Cowen, P., Goodwin, G. (2003). Acute SSRI administration affects the processing of social cues in healthy volunteers. *Neuropsychopharmacology, 28,* 148-152.

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

HeartMath LLC, 14700 West Park Avenue, Boulder Creek, California 95006 USA

Hoge, C. *The New England Journal of Medicine*, July 1, 2004; vol 351: pp 13-22. Charles W. Hoge, MD, colonel, U.S. Army; chief of psychiatry and behavior sciences, division of neurosciences, Walter Reed Army Institute of Research, Bethesda, Md.

Hoge, COL Charles Psy.D (January 2006) Article : *Hidden Combat Wounds* © 2000-2006 HealthyPlace

Hull, A. (2002). Neuroimaging findings in posttraumatic stress disorder. *British Journal of Psychiatry, 181,* 102-110.

Keane, T., Caddell, J., Taylor, K. (1988). Mississippi Scale for combat-related posttraumatic stress disorder: Three studies in reliability and validity. *Journal of Consulting and Clinical Psychology, 56,* 85-90.

Keane, T., Malloy, P., Fairbank, J. (1984). Empirical development of an MMPI subscale for the assessment of combat-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 52,* 888-891.

Kessler, R., Sonnega, A., Bromet, E., Hughes, M., Nelson, C. (1995). Posttraumatic stress disorder in the national comorbidity survey. *Archives of General Psychiatry, 52,* 1048-1060.

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

Kotulak, R. (1997). *Inside the brain: Revolutionary discoveries of how the mind works*. Kansas, Missouri: Andrews McMeel Publishing Co.

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

Ledoux, J. (1996). *The emotional brain*. 170. New York: Touchstone Books

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.

Levy, B. (1996). Improving memory in old age through implicit self-stereotyping. *Journal of Personality and Social Psychology, 71*, (6), 1092-1107.

Linnoila, M., et al. (1983). Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. *Life Sciences. 33*, 2609-2614.

McCormack, J.K., Patterson, T. W., Ohlde, C.D., Garfield, N.J., Schauer, A.H. (1990) MMPI configural interpretation as applied to posttraumatic stress disorder in Vietnam veterans. *Journal of Personal Assessments. 54* (3-4):628-38.

Mentis, M., Alexander, G., Grady, C., Krasuski, J., Pietrini, P., Strassburger, T., Hampel, H., Schapiro, M. Rapoport, S. (1997). Frequency variation of a pattern-flash visual stimulus during PET differentially activates brain from striate through frontal cortex. *Neuroimage, 5*, 116-128.

Oquendo, M., Placidi, G., Malone, K., Campbell, C., Keilp, j., Brodsky, B., Kegeles, L., Cooper, T., Parsey, R., van Heertman, R., Mann, J. (2003). Positron emission tomography of regional brain metabolic responses to a serotonergic challenge and lethality of suicide attempts in major depression. *Archives of General Psychiatry, 60* (1), 14-22.

Prescorn, S., Raichle, M., Hartman, B. (1982). Antidepressants alter cerebrovascular permeability and metabolic rate in primates. *Science, 217*, 270-272.

Ratey, J. (2002). *A user's guide to the brain*, 117. New York: Vintage Books.

Rizzo, A., Pair, J. (2005). Development of a VR Therapy Application for Iraq War Veterans with PTSD, USC Institute for Creative Technologies. White Paper, Contract No. DAAD 19-99-D-0046.

Rosenfeld, P. (1997). EEG biofeedback of frontal alpha asymmetry in affective disorders. *Biofeedback, 25* (1), 8-12.

Rubin, E., Sackeim, H., Nobler, M., Moeller, J. (1994). Brain imaging studies of antidepressant treatments. *Psychiatric Annals, 24* (12), 653-658.

Sapolsky, R. (2003) Taming stress. *Scientific American, 289* (3), 87-95.

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.

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.* A study done for Comprehensive Health Care, Unpublished.

Shealy, N., Cady, R., Veehoff, D., Houston, R., Burnetti, M., Cox, R., Closson, W. (1992). The neurochemistry of depression. *American Journal of Pain Management, 2* (1), 13-16.

Siever, D. (2000). The rediscovery of audio-visual entrainment technology. Unpublished manuscript.

Siever, D. (2002). New technology for attention and learning. Unpublished manuscript.

Siever, D. (2003). Audio-visual entrainment: 1. History and physiological mechanisms. *Biofeedback. 31* (2), 21-27.

Stein, P., Kleiger, R. (1999). Insights from the study of heart rate variability. *Annual Review of Medicine. 50*, 249-261.

Takahashi, T., Tsukahara, Y. (1972). Inhibitory Effect of Blue Color on Seizure Discharges (in Japanese). *Igaku No Ayumi, 83*, 81-82.

Teicher, M., (2002, March). Scars that won't heal. The neurobiology of child abuse. *Scientific American, 286* (3), 68-75.

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

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

Trudeau, D. (1999). Proceedings of the Annual Conference for the International Society for Neuronal Regulation.

Virkkunen, M., Eggert, M., Rawlings, R., Linnoia, M. (1996). A prospective followup study of alcoholic violent offenders and fire setters. *Archives of General Psychiatry, 53* (6), 523-529.

Walton, K., Gelderloos, N., Goddard, P., Gaudet, D. (1992, September). Signs of increased serotonin metabolism are associated with superior job performance and leadership ability. *Proceedings of the Second International Symposium on Serotonin*.

Wilson, J. (2001). *Adrenal fatigue (The 21st century stress syndrome)*. California: Smart Publications.

Wu, J., Gillin, J., Buchsbaum, M., Hershey, T., Johnson, J., Bunney, W. (1992). Effect of sleep deprivation on brain metabolism of depressed patients. *American Journal of Psychiatry, 149*, 538-543.

Young, S., Pihl, R., Ervin, F. (1988) Effect of altered tryptophan levels on mood and behavior in normal human males. *Clinical Pharmacology, II Supplement 1*, 207-215.