Audio-Visual Entrainment as a Treatment Modality for Post-Traumatic Stress Disorder

***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, war, and natural disasters. PTSD occurs when the inflicted can no longer mentally cope with the situation. Following trauma, permanent changes occur within the brain that increases "racy-headedeness," guardedeness, anxiety, depression, insomnia, plus memory and cognitive impairments. The behavioral aftermath of PTSD also typically involves increased aggression and drug and alcohol abuse. Audio-visual entrainment (AVE) has been shown to reduce anxiety, insomnia and improve coping for police officers and military. 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 *"the development of characteristic symptoms of intense fear, helplessness, or horror following exposure to an extreme traumatic stress or involving direct personal experience of an event that involves actual or threatened death or serious injury, or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate*," (American Psychiatric Association, 1994).

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, Post Traumatic Stress Disorder (PTSD) comes about from 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, house fires, natural disasters, etc., at some time in their lives (Acierno, et al, 1999). Of these, about 15% will develop 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).

The Aftermath of War

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, improvised bombs, 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, but no matter how trained an individual is to deal with the tragedy of war, trauma is inevitable.

For all groups responding after deployment, there was a strong reported relation between combat experiences, such as being shot at, handling dead bodies, knowing someone who was killed, or killing enemy combatants, and the prevalence of PTSD. For example, among soldiers and Marines who had been deployed to Iraq, the prevalence of PTSD increased in a linear manner with the number of firefights during deployment: 4.5% for no firefights, 9.3% for one to two firefights, 12.7% for three to five firefights, and 19.3% for more than five firefights. Rates for those who had been deployed to Afghanistan were 4.5%, 8.2%, 8.3%, and 18.9%, respectively (Hoge, et al., 2004).

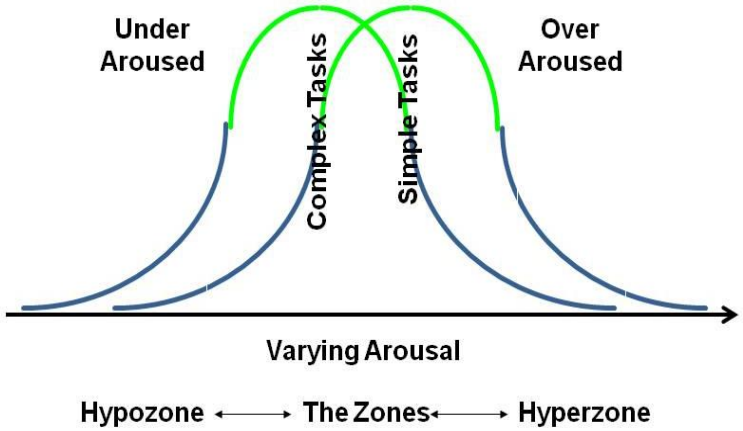
Other major contributing factors related to PTSD are combat casualties, such as those related to a permanent disability such as amputation. These soldiers not only experience the immediate trauma from the event and struggle to dissociate from its significance, but also must deal with a physical, irreversible change in their life, where psychological therapy is required for adaptation. The effects of PTSD are not one-sided. It also affects the spousal and family relationship, and puts as much or more stress on the spouse and children 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) as compared with 259 deaths Afghanistan (Edmonton Journal, 2006).

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.

About the Zone

Socialized mammals, and particularly humans, have two performance zones (Figure 1). There is one zone requiring higher arousal for simple tasks and the other requiring lower arousal for complex tasks. So what would be a simple task? Running fast, climbing a tree, spearing some food, punching an attacking animal or tribal enemy in the nose are examples of where we show peak-performance under high arousal, in other words, *fight-or-flight* activity. This is generally accompanied by excitement and often anxiety. A highly aroused state of body/mind involves the suppression of serotonin and increased production of norepinephrine, the brain's adrenalin (Bremner, 2002). This is why athletes who perform highly physical activities do better under the influence of caffeine, as caffeine has been shown to increase norepinephrine.

Figure 1. Arousal Curves for Different Types of Function.



Complex tasks, on the other hand, typically involve the cognitive mind on a much grander level than simple tasks. Complex tasks involve calculating a math formula, learning new concepts, and driving a car in busy traffic, but the most important aspect of calm-arousal is socialization.

Those with PTSD have lost their ability to learn new skills or occupations, reason and socialize (Bremner, 2002). The responsibility of making an income, pride in having a steady job and the toll on relationships with family and friends exacts a heavy price.

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 salesmen with outstanding sales performance. These salesmen averaged 180 ng/ml levels of whole blood serotonin (WBS), whereas 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 subordinate monkeys were given a serotonin reuptake inhibitor like Prozac, they became dominant through friendship and alliances with the females. Dominant monkeys deficient in serotonin ruled with aggression. Females have 20% to 30% more serotonin than men, which contributes to their reduced aggression (Kotulak, 1997). Like the "Prozac" monkeys and salesmen, college students with the most friends had serotonin levels 20% to 40%

above the norm. Those with high levels of serotonin, connect better socially with improved ability to read facial, verbal and body expressions (Harmer, et al., 2003).

In a serotonin experiment by Young and Pihl, (1988), pairs of normal, young males were given the task to be the first person to push a button when a light flashed. The winner had the option of also giving his opponent 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 mild shocks. On the other hand, if one of the pair was given tryptophan, a pre-cursor to serotonin, that person tended to deliver milder shocks to his partner, even if he previously received stronger shocks.

Low levels of serotonin are tied to loss of control and helplessness, which manifests as temper and rage (Sapolsky, 2003). Many of my clients report increased anxiety from traffic congestion, tight scheduling, computer problems, terrible customer service with many businesses, and other frustrations 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 statistics (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). A study of 114 male alcoholic and violent offenders and fire-setters showed that low levels of cerebral spinal fluid, 5-hydroxyindoleacetic acid (5-HIAA) and homovanillic acid 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 following their release from prison.

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). Above average levels are related to peak performance, while abnormally 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. However, NE activates rapidly with the perception of a threat; increasing heart rate and blood pressure and behaviors of

aggression (Aston-Jones, Chiang & Alexinsky, 1991). NE activates 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.

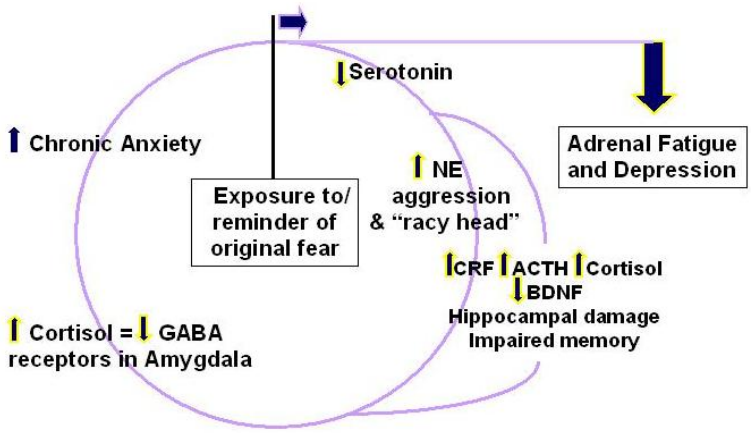
The Sympathetic Loop

The aftermath of trauma results in a double-edged sword. First, with the experience of trauma comes a fall in serotonin and increased norepinephrine, as the mind prepares to protect itself from further assault by "keeping its guard up." However, these neurotransmitter changes also lowers the threshold whereby a person perceives events in his environment as threats; a mere wrong look or sense of rejection from anyone encountered in the acute/posttraumatic stress disordered person's travels (including family members,) could constitute a perceived threat and launch the PTSD survivor into aggression against "threats" that were never previously perceived to be as such, thus maintaining a trauma-generating mindset to typical daily events. The PTSD survivor could blow up into rage from a baby crying, at the supermarket or into road-rage while driving. Therefore, trauma poses a socio-economic toll, producing aggression and violence, family breakup, and lost productivity in the workplace, as well as hundreds of millions spent annually on recreational and prescription drugs.

Unfortunately, the limbic system never evolved beyond the reptilian stage, and given that humans are primarily pack (social) 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.

Survivors of trauma, once crossing the serotonin/norepinephrine threshold, get locked into the *sympathetic loop* as shown in Figure 2. Assuming that the original traumatic event has come and gone, the loop is maintained from persistent thoughts, reminders of, and imagination of past threats (and/or new threats with the now reduced fear threshold). This continues to suppress 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). 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.

Figure 2



Somatic (Body) 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 enhances mental function, chronic (severe) stress impairs hippocampal function (Gurvits, 1996), and correlated with impaired memory, (Bremner, Randall, Scott, et al., 1995). PTSD may also lead to increased risk of multiple sclerosis, anxiety, depression, schizophrenia and Alzheimer's disease (Esch, et al., 2002).

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 in various regions of the brain. The most common structural changes from PTSD are reduced hippocampal volume, amygdala (emotional) activation, decreased Broca's area activity, and decreased pre-frontal lobe activity.

Brain Structural Damage from Trauma

People who were physically or sexually abused as children had smaller hippocampal volume and the same pattern of memory deficits as veterans (Bremner, Randall, Capelli, et al., 1995). Of particular interest is that depression was associated with an average 19% smaller left

hippocampal volume (Bremner, Narayan, et al., 2000).

The effects of increased cortisol also strips GABA within the amygdala, impairing the ability to relax, thus increasing tics, twitches and incidence of temporal-lobe seizures (Teicher, 2002). Decreased activity in Broca's area causes impaired verbal expression, (Hull, 2002).

Those living in fear have further impairments in their memory and self-reliance in remembering, as they become seniors (Levy, 1996). The military would benefit from assessing new recruits with brain Magnetic Resonance Imaging (MRI), as those going into combat with pre-existing smaller hippocampal volumes are predisposed to developing PTSD (Gilbertson, et al., 2002).

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.

Impact On Socialization

Socialization has brought about a rich wonderment of expressiveness. We have roughly 4000 facial expressions (Ekman, 2007 & 2009), over a thousand body-language positions and movements, and all kinds of verbal expressions and intonations (Navarro, 2008). Those with PTSD cannot perceive typical facial, verbal and body expressions - only the extreme ones. In a sense, they become perceptually Attention Deficit Disorder (ADD). They struggle to read social and emotional expression; they generally overreact in an impulsive and aggressive way; and often don't realize the hurt they inflict on their victims (Bremner, 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 those with PTSD keeps serotonin low, cortisol flowing, norepinephrine (and anger) high and the continual stripping of amygdala GABA receptors, resulting in the inflicted being no longer able to idle, and keeps them in a revving state, constantly on guard. Eventually, the endocrine system 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.

Both Vietnam War veterans 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).

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.

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.

Electroencephalographic (EEG) Changes from Chronic Fear and Trauma

The increased norepinephrine and cortisol levels in those with PTSD have an effect on EEG activity. The bulk of Quantitative EEG (QEEG) studies involving PTSD, suggest that most often there is reduced alpha activity and increased beta activity, coincident with high arousal (Jokic'-Begic' & Begic', 2003), and alpha asymmetry with heightened right-frontal activity in those who have developed depression (Gordon, et al, 2010; Rabe, et al., 2008) and elevated beta and theta activity (Begic, et al., (2001).

Affective Disorders Stemming from Trauma

Many people, in the aftermath of trauma, also succumb to affective disorders. Affective disorders pertain to disorders of emotion, including depression, 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 the main reward and pleasure center and is particularly 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 nucleus 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 depletion 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 of deceased suicide victims, it is clear that they 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). Functional imaging studies are controversial as they 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).

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 (quantitative electroencephalography) 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. This may explain why those on anti-depressants are troubled so much with foggy-headedness and cognitive impairments and 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 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 and never-ending activation of the fear response, which fatigues key neurotransmitters and the adrenals as it manifests into PTSD. The implications of PTSD include brain damage, a combination of family and societal violence, alcohol and drug abuse, loss of wages and increased suicide ideation. No adequate drug or medical treatment of PTSD exists today. Often, pharmaceutical agents and electroconvulsive therapy (ECT) may alleviate the depression, but usually drive the brain further into dysregulation, leaving the patient feeling emotionally numb and struggling with increased cognitive and social impairments. A new non-drug and non-ECT approach needs to be considered.

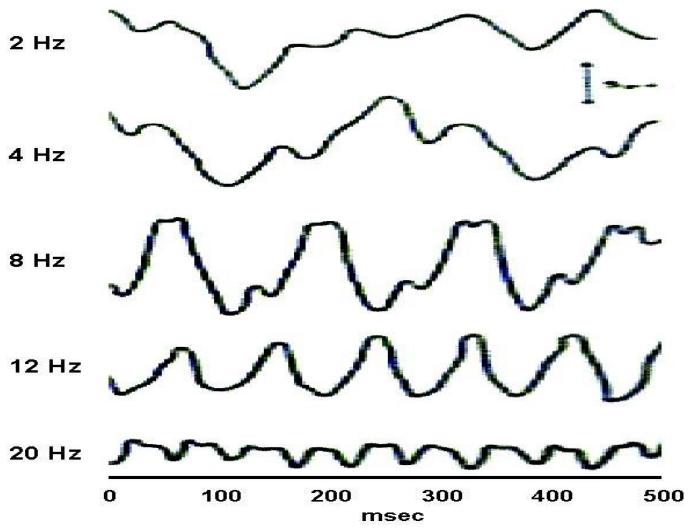
Audio-Visual Entrainment as a Treatment Modality for PTSD

All sensory information, except for smell, must pass through the thalamus in order to gain access into other brain regions. When lights are pulsed into the eyes or tones pulsed into the ears, the nerve pathways from the eyes and ears carry the evoked potentials into the thalamus. When a repetitive stimulus of the proper frequency and sufficient strength to excite the thalamus is present, their frequency signature is shown in the EEG. 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 signaling loop between the cerebral cortex and the thalamus that generates the alpha rhythm at roughly 10 Hz during neuronal rest (Demos, 2005). This effect of modulating the cortical-thalamic loop with light and sound is known as audio-visual entrainment (AVE). In essence, 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 Digital Audio Visual Integration Device (DAVID) AVE devices present pulsed light to the user via a pair of glasses (Tru-Vu Omniscreen™ Eyeset) with an array of flashing LEDs and pulsed tones through a pair of headphones. Because most maladies have an abnormal brain wave signature, the DAVID AVE device can help treat a host of maladies including anxiety, depression, insomnia, impact of trauma, a racy mind, attentional disorders, fibromyalgia and cognitive decline and risk of falling in seniors.

Entrainment occurs best near the natural alpha frequency from 9 to 11Hz (Toman, 1941). The results of a study by Kinney et al, (1973), shown in Figure 3, 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 square-wave, strobe-light stimuli.

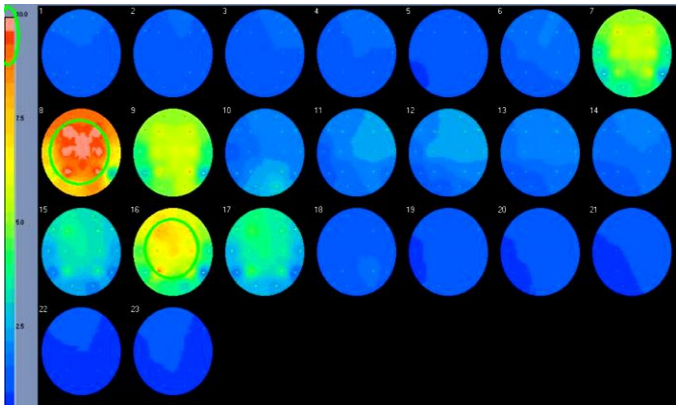
Figure 3. Occipital EEG showing photic entrainment at various frequencies



Entrainment primarily shows itself in the frontal, central and parietal regions. (Siever, 2002). AVE presents itself strongest at a person's natural alpha frequency, which is close to 10 Hz for the normal population (Toman, 1940). Given that PTSD most commonly causes enhanced beta and suppressed alpha activity, coincident with high arousal (Jokic'-Begic' & Begic', 2003), AVE can rapidly reverse the brain wave effects of PTSD as shown in Figure 4.

Figure 4 shows the results of a 19-channel QEEG (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 8 Hz 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 16 Hz., second harmonic is also present (the circled image), which is typical of *semi-sine* wave (part sine-wave / part square-wave) stimulation.

Figure 4. Brain map in 1 Hz bins - during 7.8 Hz AVE (SKIL-Eyes Closed)



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

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

Normalizing EEG Activity in Depression

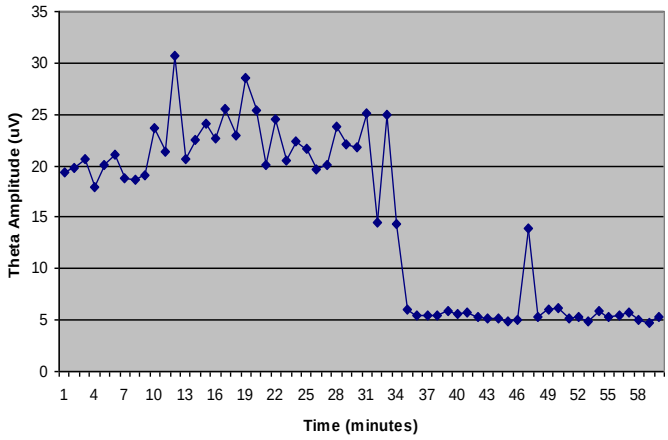
Studies suggest that a significant number of those with PTSD develop depression, characterized by frontal alpha asymmetry with more left frontal alpha activity as compared with right frontal alpha activity (Gordon, et al, 2010; Rabe, et al., 2008).

To help treat depression from an electro-neuro perspective and re-balance the frontal lobes we need to re-excite the left frontal lobe (the happy side) and suppress the right frontal lobe (the fear-based side). So two conditions must be met:

a) we must inhibit the excessive left-frontal alpha (thus "waking" it up).
b) we must simultaneously boost right frontal alpha (calming it down). Therefore, we need a means whereby we can affect both frontal lobes independently of each other.

It has been shown (above) that AVE clearly increases alpha and beta activity, but to treat depression, we also need a way that can suppress alpha in the left frontal lobe. Visual Entrainment has been found to *inhibit* brain wave activity at the ½ frequency of stimulation, thus satisfying condition a). Figure 5 shows an ADHD child with aberrantly high theta, in which 14 Hz visual entrainment was used to suppress it at the 30 minute mark. (Collura & Siever, 2009). Notice how rapidly the excessive theta disappears. In the case of depression, we can stimulate with 20 Hz and inhibit the 10 Hz alpha activity.

Figure 5. Suppression of Theta in an ADHD Child Using 14 Hz Photic Stimulation.



Independent hemispheric stimulation is accomplished by utilizing the optic chiasm (Siever, 1995), thus satisfying condition b). Stimulating the right fields of both eyes with a different frequency than the left-fields as shown in Figure 6, can accomplish this. Figure 6 depicts stimulus "A" at 12 Hz and stimulus "B" at 4 Hz. Notice the corresponding frequency evident in the opposite hemisphere of the brain. For treating depression, 18-21 Hz stimulation in the right fields would inhibit the left-frontal alpha from 9–10 Hz, thus boosting activity. Stimulating at 10 Hz in the left fields, will boost right-frontal alpha, thus calming activity.

Figure 6. Patented Technique for Independent Hemispheric Stimulation.

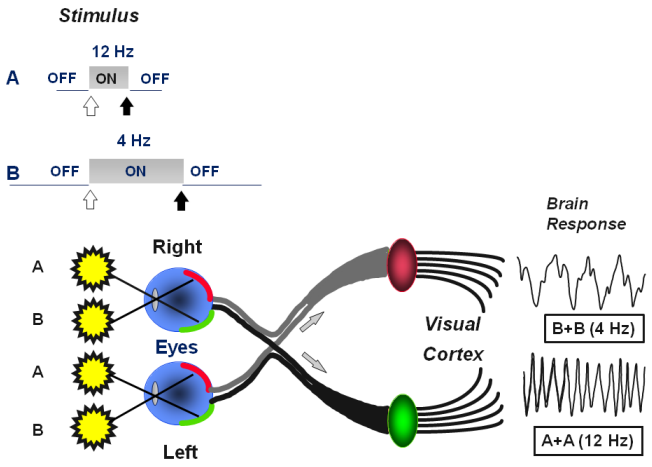


Figure 7 shows the QEEG (also referred to as a *brain map*) of a happy person as compared to the Sterman-Kaiser Imaging Labe (SKIL) normative database. This person constantly exhibits approach behavior towards socializing and what she considers to be fun activities. Notice that alpha activity is stronger in the right frontal lobe, the EEG signature typically associated with happiness.

Figure 7. Normative Brain Map of a Happy Person. (SKIL-Eyes-closed).

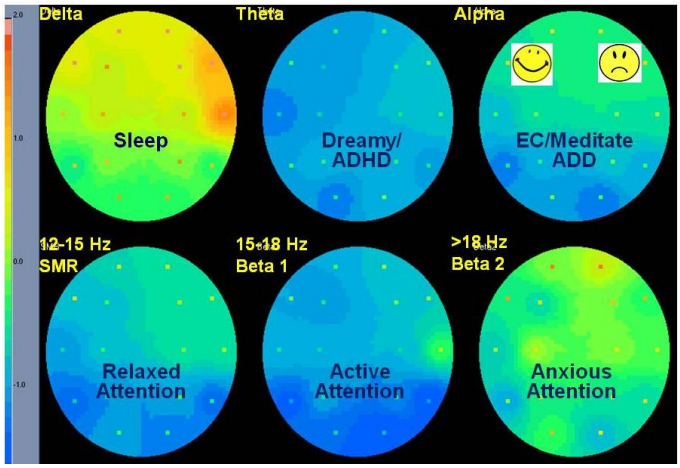
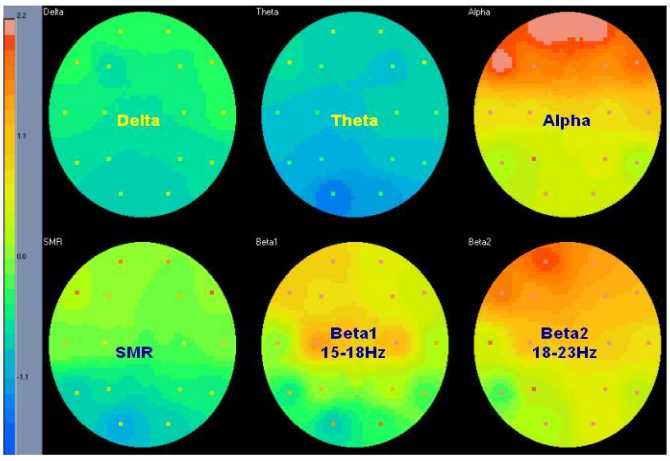


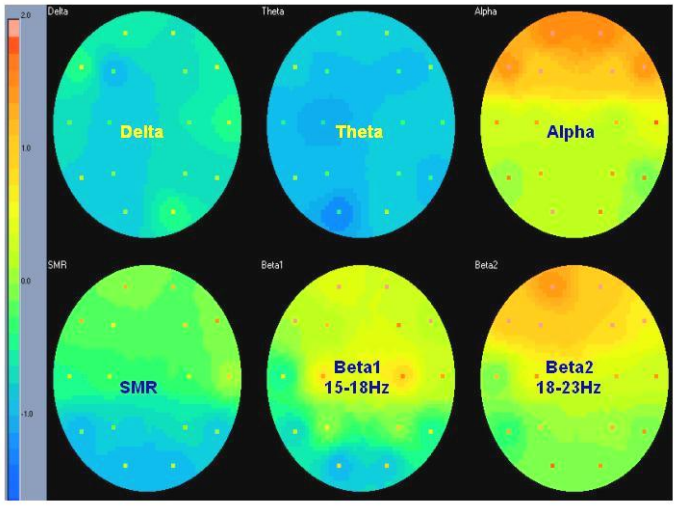
Figure 8 shows a fairly typical brain map of a person with depression and anxiety from trauma as shown on the SKIL database. The scale is 2.2 standard deviations (SD) and the pink area in the alpha view is actually 2.6 SD. Activity above 2 SD is considered a clinical abnormality. Notice that alpha activity is higher on the left side coincident with a personality trait based on a focus of withdrawal and avoidance from selfr-perceived negative stimuli. Also, the generalized red colored region is an indicator of generalized cognitive fatigue. The Beta 2 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 8. Brain Map of Individual with Depression and Anxiety (SKIL - eyes closed)



Approximately 10 minutes following a 30-minute AVE session designed to reduce the symptoms of depression, both alpha and beta activity is normalized as shown below in Figure 9. Notice that the frontal alpha activity and the Beta 2 activity, has been reduced to roughly 1.2 SD above the norm (non-clinical). The participant was also well aware of his elevated mood and energy.

Figure 9. Brain map following depression reduction AVE session (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 production
3. increased cerebral blood flow
4. normalized EEG activity

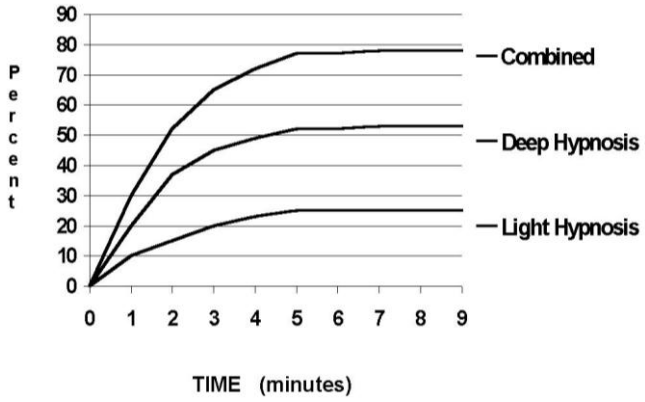
Dissociation

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 10). AVE induced dissociation is rapid, requires only 4 to 10 minutes in most cases and provides an excellent means for clearing a tormented, chattery mind of destructive, fearful thoughts and allowing the person to relax and restabilize (Siever, 2000).

Figure 10. Clearing the mind (dissociating) from negative thoughts using AVE.

Visual entrainment alone, in the lower alpha frequency range (7 to 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 11.

Figure 11. Photic stimulation induction of hypnotic trance



Inducing dissociation using AVE was found to be more effective than dot staring or stimulus deprivation (Leonard, et al., 1999). AVE also proved to be effective in dissociating people with dissociative anxiety. As expected, subjects experienced increased anxiety during dissociation, but simultaneous relaxation with slowed heart rate was also observed (Leonard, et al, 2000). The DAVID AVE proved to be effective in stopping distressing mental chatter and as an effective desensitization tool for reducing anxiety that is often 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.

Properly applied AVE produces a calming effect on limbic structures, such as the amygdala and hypothalamus, in which muscles relax (Thomas & Siever, 1989), electrodermal activity settles down, peripheral blood flow stabilizes (hand temperature normalizes to 86 to 90 F), breathing becomes diaphragmatic and slow, and heart rate slows and becomes uniform (Siever 2000). As a result, AVE can re-induce a relaxed state of mind and calm disposition, thus providing some badly needed time away from the distressing thoughts. Figures 12 and 13, show the calming effect of AVE on the somatic functions of forearm EMG (electromyography) and finger temperature (Hawes, 2000). Heart rate and heart-rate variability (HRV) are sensitive measures of stress (Stein, P., Kleiger, R. (1999). Figure 14 shows graphs of the emWave HRV analysis system by Heartmath. It shows dramatic improvements in both heart rate and HRV in a woman with PTSD after discovering that her husband molested two young girls. Within 10 minutes, her heart rate dropped by 22 bpm, and she showed dramatic reductions in both sympathetic and parasympathetic activity (notice the blue mountains).

Figure 12. Forearm EMG levels during AVE (Hawes, 2000).

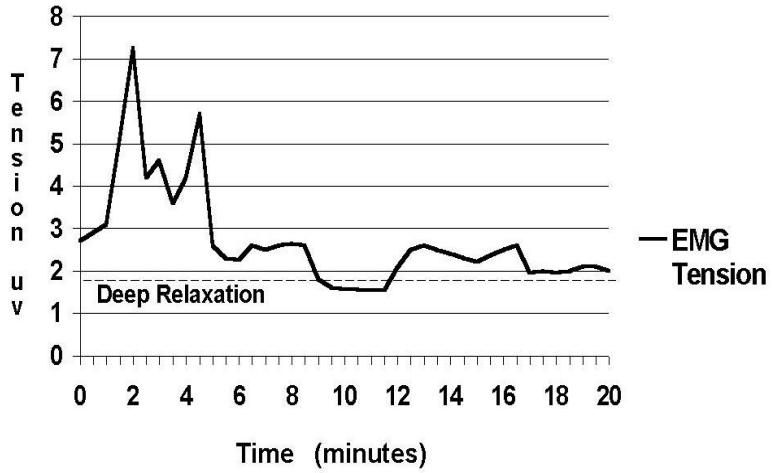


Figure 13. Peripheral temperature levels during AVE (Hawes, 2000).

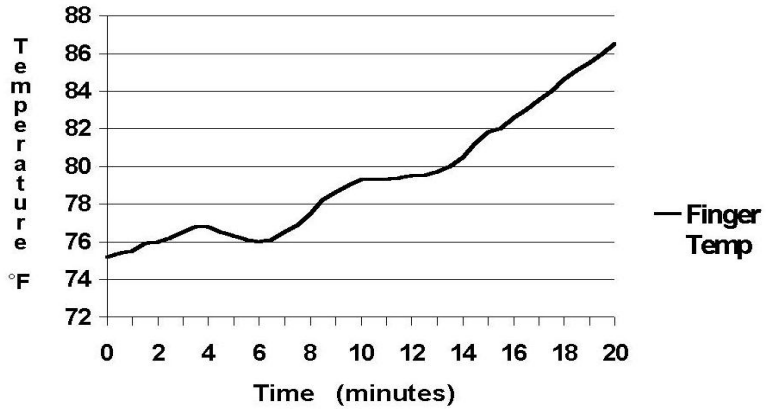
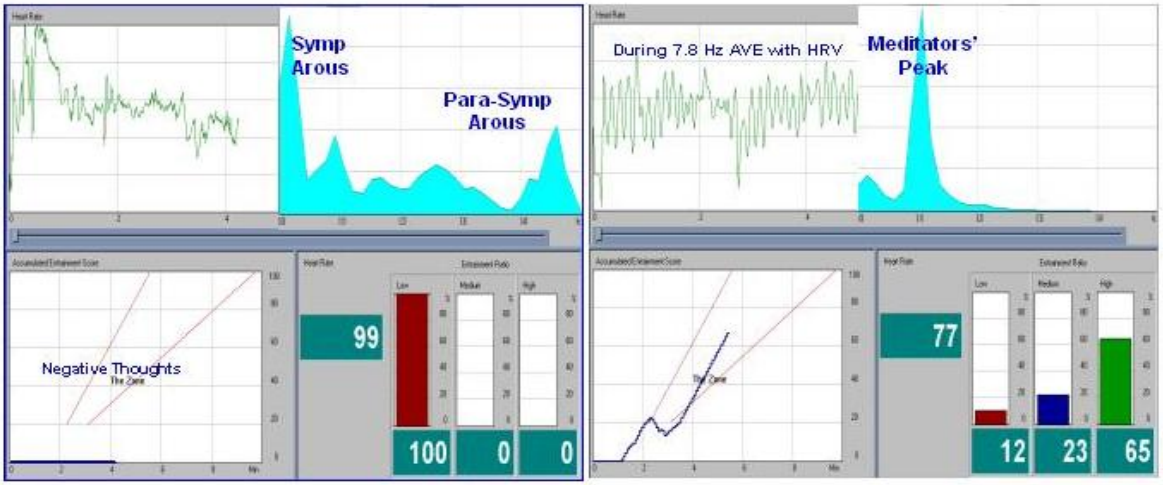


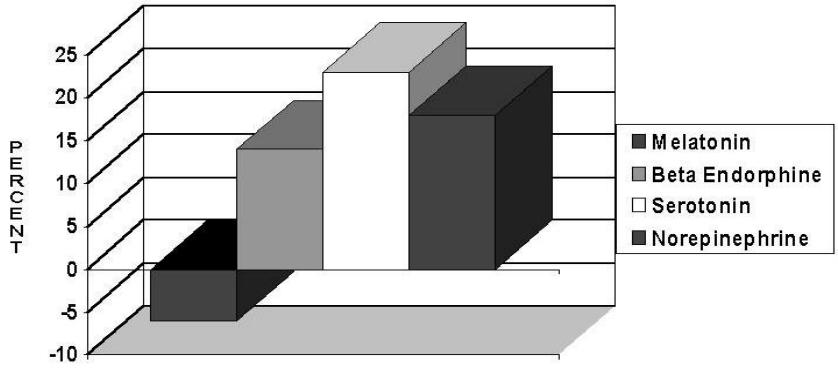
Figure 14. Pre and post HRV in a woman with adult PTSD



Balancing Neurotransmitters

As mentioned previously, people with lingering PTSD and clinical depression are low in serotonin, dopamine and norepinephrine. Figure 15 shows that 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, mental sharpness, improved sleep, reduced pain and reduced anxiety.

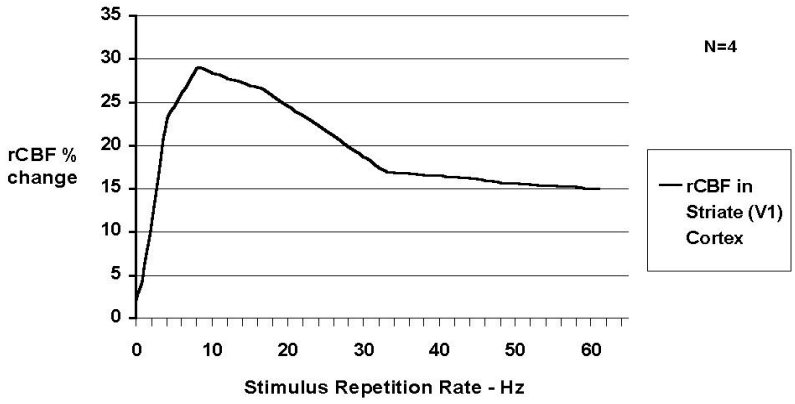
Figure 15. The Effect of White Light AVE on Neurotransmitter Production



Cerebral Blood Flow and Metabolism

SPECT and FMRI imaging of CBF show that hypoperfusion of CBF is associated with many forms of psychiatric disorders. Of particular concern are conditions involving hypoperfusion of CBF in frontal lobes. Frontal lobe issues include anxiety, depression, attentional and behavior disorders, and impaired cognitive function (Amen, 1998). Adequate CBF is essential for good mental health and function. AVE increases brain glucose metabolism and CBF (Sappy-Marinier et al., 1992). Figure 16 is a study by Fox and Raichle (1985) showing marked increases in CBF, with a 28% peak increase at 7.8 Hz in the striate cortex (a primary visual processing area). Overall whole brain oxygen consumption increased by 5%. Accomplished Zen meditators show a peak frequency of 7.8 Hz during meditation (Cade, 1987).

Figure 16. 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, and 14 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.

Studies

In 1995, David Trudeau, a physician with the VA Hospital in Minneapolis conducted a study on 15 war vets, all suffering from PTSD. The volunteer subjects received 60 daily sessions of AVE at 18 Hz. Pre and post intervention, QEEGs, Beck Depression Inventory (BDI), McGill Pain Questionnaire (MPQ), Test of Variables of Attention (T.O.V.A.), and DSM-IV Attention Deficit and Hyperactivity Disorders (ADHD) symptom checklist were done. None of the subjects had Wender Utah criteria for childhood ADHD.

As of summer 1999, ten subjects have completed the study. Following 60 daily sessions of 15 minutes of AVS at 18 Hz, there was a significant decrease in BDI scores from an average of 17 to 9 ($p<0.05$) and DSM-IV impulsivity-hyperactivity criteria from 3 out of 9 to 0 out of 9 ($p<0.01$). Consistent with the decrease in self-assessed impulsivity is a trend toward decreased impulsivity on the T.O.V.A. Anecdotally, subjects reported onset of dreaming and improved sleep and higher energy levels. Focus may be improved following AVS, and depression symptoms may be improved. Clearly more study is required, and further trials should include sleep assessment.

John Carmichael's PTSD Work with the Royal Canadian Mounted Police (2006).

Dr. Carmichael is the approved and designated clinical psychologist to the Royal Canadian Mounted Police in British Columbia, Canada. Currently, most of his private practice in clinical psychology is with police officers who most typically present with depression in which accumulated traumatic incidents have played a significant role, or with Post-Traumatic Stress Disorder (PTSD), or with both depression and PTSD. Given that the police have a tendency to wait until the last minute for treatment, their symptoms are very marked in both number and intensity and have been on-going for a considerable time.

However, since discovering our DAVID technology, Dr. Carmichael now includes audio-visual entrainment for all of his police clients with depression and/or PTSD once there is psychophysiological confirmation that they have mastered diaphragmatic breathing, that they can establish an RSA pattern, and that the entrainment creates desirable changes.

Most police officers continue to use the DAVID AVE devices on a daily basis. It is clear that well over 90% of his police clients find the DAVID helpful. Among the most common findings are:

- A rapid decrease in both autonomic nervous system hyper-arousal/hyper-reactivity and muscle tension (I show clients the changes during their first session with DAVID in my office);
- A longer and longer duration of these positive effects the more frequently they use the DAVID;
- A rapid increase in mental calmness and corresponding decrease in "monkey mind" (thoughts all over the place);
- Rapid improvements in sleep (reduced latency to sleep onset, decreased night waking, and increased sense of restfulness come morning) when they use it at regular bedtime and again if they wake during the night and are unable to fall back asleep within 15 minutes; and
- What appears to be self-initiated changes in both behaviour and cognitions even before any formal introduction of cognitive behavioural therapy.

Conclusion

Chronic rumination, hypoperfusion of CBF, 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. Interventions to help those with PTSD are poor at best and can have significant, unwanted side-effects.

The DAVID AVE dissociates those experiencing PTSD away from destructive distressing rumination, increases blood flow, normalizes brain wave and neurotransmitter production, calms the limbic system, restores the adrenals, and produces somatic relaxation. The subjective benefits of AVE are reduced anxiety, improved sleep, improved mood, increased energy, improved relationships with family and civilians, reduced physical problems, improved productivity and reduced dependence for medications or self-medicating on alcohol and recreational drugs.

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 90% of the officers respond with improved sleep onset and quality of sleep and with reduced daytime anxiety.

About the Author

Dave Siever of Mind Alive, Inc. has lectured and provided workshops with leading psychological institutions including the Association of Applied Psychophysiology and Biofeedback, the International Society of Neurofeedback and Research, the College of Syntonic Optometry, American College for the Advancement of Medicine, Walden University, the University of Alberta, Open University-England, A Chance to Grow Charter School, STENS Biofeedback Training Programs and other venues. Dave Siever has been designing and studying AVE since 1984 when he originally developed the DAVID1 to help performing-arts students overcome stage fright.

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