Application of Sonified EEG in Alpha Neurofeedback Training For Anxiety Reduction

by

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Abstract

Electroencephalography (EEG) is a non-invasive neuroimaging method that records fluctuations in bioelectrical activity from the scalp. Oscillations in EEG activity can be categorized by bands of activity that are prevalent during different states of consciousness. Neurofeedback is a subset of biofeedback that uses real-time quantitative EEG metrics to help individuals regulate their own brain activity. Alpha waves (8-12 Hz) are EEG oscillations that are especially prominent during periods of relaxation with closed eyes. It has been shown that up-regulation of alpha activity through neurofeedback can decrease anxiety symptoms (Kamiya, 1969; Hammond, 2005), usually following hours of neurofeedback training. In this study, participants underwent a single fifteen-minute session of alpha-enhancement neurofeedback training to assess changes in self-reported anxiety, as measured by the State-Trait Anxiety Inventory. An auditory neurofeedback stimulus was represented as sine tone whose pitch was inversely correlated to the ratio of alpha amplitude to the average amplitudes of all frequency bands. A control group received sham feedback that was recorded from another subject. Experimental subjects showed a reduction in self-reported anxiety, although this reduction was not significant and not significantly different from changes in control-group anxiety. In addition, differences in alpha ratio pre and post training were not significant in either group, and changes in alpha ratio were not significantly correlated to changes in anxiety. More subjects, lower stimulus latency, and longer neurofeedback sessions would benefit future experiments aiming to reduce anxiety through alpha-enhancement neurofeedback.
1. Introduction

1.1 Electroencephalography

It has been known since the mid-19th century that the nervous system produces electrical signals. In 1848, Emil du Bois-Reymond theorized that peripheral nerves exude electrical properties; these findings, initially controversial, eventually inspired Richard Caton’s discovery of electrical activity emanating from exposed animal cerebrums in 1875 (Cantor, 1999). Fifty-four years later, Hans Berger recorded the first electroencephalogram (literally, “electrical brain recording”) from the scalp of a human subject. Thus, the field of electroencephalography (EEG) was born, and it has since become a fundamental non-invasive neuroimaging tool both in the laboratory and the clinical setting. Physicians use EEG to diagnose seizure activity, sleep disorders, and tumors. EEG can also be used to monitor brain activity of patients under anesthesia, or patients who have sustained traumatic brain injury.

Following Berger’s discovery, researchers began to harness the high temporal resolution of EEG to study a variety of topics in psychology, psychiatry, and neurological sciences. In 1939, Pauline and Hallowell Davis introduced a radical new application of EEG now known as the event-related potential technique, which studies how the brain produces stereotyped electrical activity time-locked to a sensory or cognitive event (Davis & Davis, 1936). In the 1950s, William Grey Walter triangulated EEG signals to determine surface coordinates of various frequency bands that could reveal the location of tumors, thus inventing the field of EEG topography.
(Kropotov, 2010). New EEG applications are constantly being introduced, and EEG systems continue to become cheaper, more portable, and more accurate.

In order to understand EEG analysis and EEG manipulation, it is important to consider the origin of the brain’s electrical activity, and how that activity is received by an EEG recording device. The upper layers of the cerebral cortex are composed of pyramidal neurons, which differ from other types of neurons in that their unique orientation patterns encourage simultaneous neuronal firing. Most of the time, these cells are firing asynchronously, creating external potentials that effectively cancel each other (Collura, 2014). When a collection of cells fire action potentials synchronously, however, the electrical activity emanates to the scalp and can be recorded by placing metallic sensors (electrodes) that detect voltage changes on the scalp.

Electrodes record signals preferentially from action-potential generators that are closest to them. Nevertheless, electrical activity from other sources may reach the site of interest and therefore distort the EEG signal. Muscle, ocular, and cardiac artifacts can all be present in an EEG recording; EEG data analysis should only be done after removing these artifacts or deleting sections that have a low signal-to-noise ratio (Evans & Abarbanel, 1999).

1.2 QEEG and Brain Rhythms

It is known that thalamic projections to the cortex, also known as thalamocortical radiations, are pervasive throughout the brain. These fibers relay important sensory and motor information to the cortex. Cortical neurons and thalamic
neurons engage in coordinated oscillations in action potential firing. More specifically, thalamic relay cells send bursts of activity to an adjacent thalamic nucleus known as the *Nucleus Reticularis Thalami* (nRt). In turn, cells in this layer respond with bursts of *inhibitory* activity back to the relay cells, which temporarily hyperpolarizes those cells. The relay cells slowly depolarize again, releasing action potentials that are sent to the nRt and the process continues in a cyclic manner. This waxing and waning of activation influences cortical neurons by way of thalamocortical radiations, and oscillatory activity in these cells inevitably transpires (Sterman, 1996).

These coordinated oscillations are represented as rhythmic waves in EEG recordings, and are categorized based on location and physiological/psychological state of the subject. In reality, EEG waveforms are incredibly complex and comprise a variety of frequencies. Nevertheless, when a certain frequency is dominant in a given waveform, it can be clearly visible. Berger himself noted the presence of a prominent 10 Hz oscillation in his early recordings, which he called “alpha waves.” Today, different cognitive processes are associated with defined bands of activity: delta (1-4 Hz), which are high-amplitude waves found during slow-wave sleep and may be indicative of some neuropathies; theta (4-8 Hz), which is associated with drowsiness and inactivity; alpha (8-12 Hz), which is most prominent above occipital and parietal regions, and is enhanced during relaxed yet attentive states with eyes closed; low beta (12-15 Hz), which has psychological correlates that are similar to alpha and comprises the sensory-motor rhythm (12-15 Hz oscillations above the
somatosensory cortex during inactivity); beta (15-35 Hz), which is distributed over various regions and is associated with thinking and alertness; and gamma (>35 Hz) which is elicited during multi-sensory information processing (Collura 2014).

Although basic EEG recordings provide qualitative information on the dominant rhythms present at a given electrode site, mathematical processing is required to extract further quantitative information. This brings us to the realm of quantitative EEG (QEEG). QEEG uses mathematics to transform and/or extract relevant spectral information from digital EEG data (Nuwer, 1997). One of the most basic QEEG analyses – and one that is relevant to neurofeedback – is the Fourier transformation. This transformation is applied based on the principle that any waveform, no matter how complex, can be described as a combination of simple sinusoidal components. Fourier analysis involves separating a waveform into these components such that each component waveform can be further analyzed. In this way, the original EEG is brought from the time domain to the frequency domain. Although temporal data is lost, the frequency domain reveals important information regarding the power of the frequencies that comprise the original waveform. For example, Fourier analysis can be used to determine the amplitudes of delta, theta, alpha, beta, and gamma waves in a defined EEG epoch. Today, the most popular method of translating EEG into the frequency domain is by applying a fast Fourier transform (FFT), which, as its name suggests, computes a Fourier transform relatively quickly by using a highly-efficient algorithm. The FFT is superior to other frequency analysis methods because it can transform data as EEG is being recorded, which is
necessary for real-time applications such as neurofeedback (Akin 2002). FFT typically outputs data as a power spectrum, with frequency ranges on the x-axis and amplitude or energy on the y-axis. This allows for extraction of amplitude information for specific frequency bands. With the improvement of computer technology, QEEG has become faster, more reliable, and more powerful than Berger could have ever imagined.

In reference to the frequency bands associated with these oscillations, neuroscientist Gyorgy Buzsaki wrote that “the borders between the different bands were evenly and arbitrarily drawn, like the straight-line country borders between the African nations drawn by the colonials” (2006). These ranges are useful in defining standards for classification and spectral analysis, but they should not be treated as rigid boundaries. More important than the frequency bands are the underlying mechanisms that produce the rhythms, and the physiological state associated with them.

1.3 EEG Sonification

After voltage information from EEG electrodes is sent to the amplifier, there must be an interface through which the practitioner can assess the data. Typically, EEG data is represented visually, with time and voltage fluctuations on the x- and y-axes respectively, quite similar to electrocardiogram data. Each row of data is stacked vertically by electrode, such that the EEG practitioner can view all data simultaneously.
Nevertheless, a new way of “reading” the EEG data has been growing in popularity since the 1960s. Sonification is a process whereby sound is synthesized from a data set, with the intention of making specific characteristics of that data perceptible (Kramer 1994). Sonification is similar to the process of audification in that both involve conversion of data to sound; in the case of audification however, the data is converted directly into a sound waveform. Audification has been performed on everything from seismic activity (Quinn & Meeker, 2001) to ultrasonic bat calls (Hermann et al., 2011), with both utilitarian and artistic intentions. With sonification, there is more freedom in the conversion process. Rather than a direct conversion to sound, the data set can control any defined parameters of a sound that may or may not have been generated by that data. For example, data fluctuations could control the final amplitude, pitch, or phase of a sound, or even all three parameters at the same time. Sonification has been used for biological signal monitoring (Glen, 2010), auditory feedback of motion (Cheng et al., 2013), and musical composition (Arslan et al., 2005), among other applications.

One of the earliest and most famous sonification demonstrations was performed in 1965 by experimental musician Alvin Lucier on his own brainwaves, in a piece entitled “Music for a Solo Performer” (1972). Lucier attached two electrodes to his temples and converted voltage information from the alpha band (in this case, 8-13 Hz) into sound. The resulting sonifications were played out of loudspeakers connected to various percussive instruments, and as Lucier’s alpha activity increased the instruments would rattle and create sound. Since this seminal composition, EEG
sonification for musical composition has been explored by engineers and artists alike. In 1971, a patent was filed for an “electroencephalophone,” which serves as an all-in-one EEG sonification system (Backerich & Scully, 1973). These devices went on to inspire compositions such as David Rosenboom’s “Brainwave Music” (Rosenboom & Humbert, 1975) and “On Being Invisible (Rosenboom, 1977).

Many studies have shown that non-expert populations can detect seizure activity by listening to sonified EEG, either offline using time-compressed audified EEG (Khamis et al., 2012), or in real-time (Baier et al., 2007; Loui et al., 2014). One may ask, however, why sonified EEG should be used over visually-presented EEG for seizure detection. A variety of answers have been suggested. First, musical waveforms and EEG waveforms are both oscillatory in nature, offering a natural mapping system for conversion of EEG into sound (Kramer, 1994). Second, unlike our eyes, our ears are constantly open and do not require foveation for sensory analysis, making the auditory system superior in its ability to continuously monitor sensory information. In addition, humans are adept at focusing on relevant auditory information, even in a noisy setting (e.g. the cocktail party effect; Arons, 1992). Studies have shown that human subjects are faster and better at monitoring complex physiological data when the data are sonically as opposed to visually displayed (Fitch & Kramer, 1994; Barrass and Kramer, 1999; Watson and Sanderson, 2004). This may be explained by the fact that the auditory system processes various streams of information in parallel, whereas the visual system processes information serially (Loui, et al., 2014). Lastly, people may be more motivated listening to sonified rather
than visualized data if the sonification is aesthetically pleasing. Since most people find listening to music to be an enjoyable experience, a sonification that sounds like music has great potential clinical value.

1.4 Neurofeedback

Biofeedback refers to a process whereby an individual receives real-time information (i.e. feedback) regarding the state of a physiological system, in a manner that allows the individual to change the functioning of that system for the purposes of health or performance improvement (AAPB, 2011). Neurofeedback is a subset of biofeedback in which the brain is the physiological system being adapted, and the goal is to modify brain function, psychology, or behavior. Neurofeedback is typically used in treating ADHD (Arns et al., 2009; Lansbergen, 2011), epilepsy (Tan, 2009), mood disorders (Hammond, 2005; Peeters et al., 2014), and alcoholism (Saxby, 1995). It has also been used to enhance cognitive performance (Escolano at al., 2014) and musical performance (Egner et al., 2003).

Although theoretically any functional neuroimaging method could provide feedback data, the term “neurofeedback” typically refers to EEG biofeedback. When neurofeedback research began, EEG was the predominant neuroimaging method, and due to its high temporal resolution and low cost relative to other neuroimaging methods such as functional magnetic resonance imaging, EEG has remained the most popular choice for neurofeedback. EEG neurofeedback is applied based on the premise that some abnormal brain states are marked by abnormal EEG activity, and that changing that activity will normalize the brain state. A typical EEG
neurofeedback paradigm would proceed as follows: (1) A subject’s EEG activity is recorded; (2) signal processing (QEEG) is performed to isolate one or more metrics (amplitude, frequency, etc.) that will control the feedback stimulus; (3) visual, auditory, or tactile feedback is produced and presented to the subject; and (4) the subject learns to control or change the stimulus, and, in turn, change his or her brain activity (Collura, 2014).

History of Neurofeedback

The practice of neurofeedback is founded on the pioneering work of Dr. Joe Kamiya at the University of Chicago in the 1960s. In 1962, Kamiya conducted a seminal study in which participants were verbally informed whenever their EEG showed high alpha activity; over time, participants were able to reliably report whether or not they were producing alpha. As a follow up study, Kamiya built a sine-tone generator that played a sound whenever a subject produced alpha above a certain threshold. After a dozen trials, subjects were able to voluntarily elicit high alpha activity using this feedback device. Kamiya also collected interesting verbal reports from his subjects regarding their personal experiences undergoing neurofeedback. Most subjects found the high-alpha state to be generally pleasant and relaxing, reporting that they felt free of worry and self-criticism. Relatedly, Kamiya noticed that practiced meditators were far better at the alpha-enhancement task, along with participants who seemed more in touch with their feelings. The alpha state was certainly a desirable one; Kamiya’s early neurofeedback subjects were compensated, but when word spread about his research, he recalls that people were “almost ready to
pay me to serve as [subjects], especially if I say I will let them turn on alpha for an extended period of time!” (1969).

Around the same time, Dr. Barry Sterman was conducting EEG experiments on cats, focusing on oscillations in the 12-14 Hz range. This particular frequency band, termed the sensorimotor rhythm (SMR), occurs over the sensorimotor cortex and manifests as spindle activity during both sleep and waking states with motoric stillness (Othmer, 2009). Sterman found that cats could be trained to elicit this rhythm when the activity was associated with a food reward. Unrelated to this work, Sterman was contracted by NASA to research the effects of the toxic rocket fuel monomethylhydrazine, which is known to cause nausea, seizures, and possible death in astronauts that come in contact with it. Surprisingly, Sterman found that cats that happened to have undergone SMR training in his earlier study had a higher seizure threshold than other cats when injected with the substance (Sterman et al., 1969). This research spawned an entire subfield of neurofeedback that focuses on SMR enhancement for the treatment of seizure disorders and ADHD (Budzynski et al., 2009).

Following the influential discoveries of Kamiya and Sterman, the field of neurofeedback exploded with innovative applications of the technique. In the early 1970s, it was shown that alpha control through neurofeedback could be used to enhance hypnotic susceptibility (Engstrom et al., 1970), and even communicate in Morse code (Dewan 1971). Dr. Barbara Brown published the popular books New
Mind New Body (1974) and Stress and the Art of Biofeedback (1977), which introduced the technique to the general public.

Although neurofeedback enjoyed this initial burst of success, a variety of factors decreased scientific interest in the phenomenon. Neurofeedback treatment is relatively cheap compared to other pharmacological and psychotherapeutic interventions, which is attractive to potential patients but threatening to the health-care establishment. In fact, drug lobbies in the early 1970s attempted to restrict biofeedback administration to only those with medical degrees in order to curb the industry’s growth (Evans, 2007). Lack of well-designed, controlled scientific studies made it difficult to incorporate biofeedback into mainstream medical practice, especially in an age when the concept of neuroplasticity was less accepted (Coben & Evans, 2010). In addition, the idea of brain control for the purposes of deep relaxation resonated with the “new age” spiritual movement of the hippie era, which tainted the reputation of neurofeedback as a serious science. Thus, neurofeedback experienced only modest advances in the late 1970s and early 1980s.

Thanks to better research practices and new developments in QEEG in the 1990s, neurofeedback received a second wind. Researchers began compiling databases of EEG activity from a normal (non-pathological) population; these databases, along with advancements in computational ability, led to the conception of the Z-score neurofeedback paradigm. In this paradigm, differences between subject EEG activity and normal EEG activity are calculated in real-time as Z-scores. The subject is then given feedback based on this metric, and is trained to normalize his or
her EEG patterns (Collura et al., 2010). The practice of Z-score neurofeedback intersected with the invention of Low-Resolution Electromagnetic Tomographic Analysis (LORETA), which provides statistical predictions of the subcortical brain regions producing scalp potentials at any given time. Z-score and LORETA neurofeedback can be used in tandem to normalize brain activity on a subcortical level. These and other advances have given neurofeedback a legitimate place in cognitive research. Today, there are at least three major professional biofeedback organizations, dozens of books on the topic, and thousands of published research articles (Evans, 2007).

**Mechanisms of Neurofeedback**

Neurofeedback is directly based on the principles of operant conditioning. In operant conditioning, an organism is rewarded in response to a certain behavior, leading the organism to associate that behavior with the anticipated reward. In neurofeedback, a particular brain state is reinforced by being temporally paired to the “reward” of a desirable feedback stimulus. Over time, the brain will spontaneously elicit the brain activity that is associated with the reward feedback (Collura, 2014).

Evans (2007) explains the efficacy of neurofeedback by relating familiar concepts of neurochemistry to bioelectricity. EEG abnormalities are directly analogous to chemical abnormalities in the brain; in fact, Evans argues that neuropathologies are just as likely to arise from electrical imbalances as they are from chemical imbalances. Therefore, treating these electrical imbalances would naturally treat the underlying illness. Othmer (1999) takes this ideology one step further,
arguing that neurochemical imbalances are mere aftereffects of more important bioelectrical changes. In this disregulation model, proper timing of thalamo-cortical interactions is necessary for normal regulatory functions of the brain, and any deviations from the norm will lead to pathology. Using EEG biofeedback, “re-regulation” can occur and restore the proper function of regulatory systems. In this model, brain function is emphasized over structure, and considerations of neural networks, frequencies, and phase relationships replace traditional pharmacological considerations of chemicals and neurotransmitters.

On a cellular level, these functional changes, both short- and long-term, are mediated by neuroplasticity. There exist two basic subtypes of neurotransmission: simple synaptic transmission and neuromodulation. Synaptic transmission is relatively fast-acting and involves Na\(^+\) and Cl\(^-\) channels. On the other hand, neuromodulation involves the relatively slower K\(^+\) and Ca\(^{2+}\) channels and works by modulating the firing characteristics of post-synaptic neurons. The brain stem exerts neuromodulatory control over thalamic and limbic neurons, which in turn control several cortical centers. These circuits, which determine electrical rhythmicity as described earlier, also control states of consciousness. It is postulated that neuromodulation promotes changes in brain rhythms, which consequently modify brain states (Abarbanel, 1999).

When neurofeedback is administered over time (20-40 sessions), these synaptic changes are strengthened and long-term potentiation (LTP) occurs. LTP is marked by increased synaptic strength following repeated stimulation of a given
pathway. This phenomenon was first studied in the hippocampus, where researchers showed that post-synaptic cell response could be enhanced after pre-synaptic neurons underwent high-frequency electrical stimulation (Bliss & Collingridge, 1993). LTP has characteristic developmental and temporal parameters in various brain areas; nonetheless, the process occurs similarly across the brain (Abarbanel 1999).

Neurofeedback therapy typically boasts low relapse rates, especially following well-established therapy protocols designed for ADHD; this can be explained by the fact that LTP strengthens neuromodulatory changes in brain function (Evans, 2007).

**Neurofeedback and Mood Disorders**

Although neurofeedback has been shown to effectively treat a range of disorders including Alzheimer’s disease and traumatic brain injury, this section will focus on the application of neurofeedback therapy for mood disorder treatment.

Major depression is often called the “common cold” of psychiatry, which speaks to its prevalence far more than its devastating effects on the sufferer. In 2012, sixteen million American adults over eighteen experienced some sort of depressive episode, and the disorder accounts for 8.3 percent of total years lived with disability in the United States (NIMH, 2009). A depressed person may find it difficult to eat, sleep, work, or perform activities that he or she used to find enjoyable. Currently, the most common treatments for depression include talk therapy and psychopharmacology; however, psychotherapy cannot directly treat the biological root of depression, and antidepressants are often ineffective and can cause negative
side effects. In addition, it impossible to know which antidepressant will work for a given patient, often forcing the patient to undergo multiple medication trials.

Neurofeedback, on the other hand, provides a biologically-mediated treatment for depression without any negative side-effects. Furthermore, QEEG allows neurofeedback practitioners to quantitatively assess electrophysiological abnormalities before therapy is administered. This is analogous to the hypothetical situation of a psychiatrist assessing the amount of serotonin in a patient’s cerebral spinal fluid before deciding appropriate dosage for a selective serotonin reuptake inhibitor. Prichep, Lieber, and John (1986) were the first to identify QEEG variables directly correlated with depressive disorders. Using these variables, they were 84% accurate in diagnosing depression in a new population of patients. Depression is typically associated with abnormally high alpha activity in left frontal areas. Since alpha waves indicate decreased activation of associated brain regions, it follows that left frontal areas are underactive in depressed patients. This finding led Davidson and his colleagues to formulate the now-popular approach/withdrawal hypothesis: the left frontal cortex is associated with positive affect and approach behaviors, whereas the right prefrontal cortex is associated with negative affect and avoidance behaviors.

Based on this hypothesis, most neurofeedback protocols for depression therapy seek to increase left frontal activation (Davidson et al., 1990). Baeher’s ALAY (alpha asymmetry) protocol for depression, which seeks to re-balance alpha activity in the left and right hemispheres, was shown to bring about long-lasting, significant decreases in depression in the subjects involved (Baeher et al., 2001).
Unfortunately, the study was not well-controlled so its results are difficult to interpret. Hammond (2000), finding the ALAY protocol to be unsuccessful for his patients, designed his own protocol, which involved beta reinforcement and alpha and theta inhibition in the left frontal region. This technique was successful for alleviating severe depression in Hammond’s subjects, even after an eight-and-a-half month follow up.

Neurofeedback treatment can also be used to treat anxiety, another potentially debilitating mood disorder. Approximately eighteen in one hundred Americans live with an anxiety disorder, and twenty-three percent of these cases are classified as “severe” (NIMH, 2009). Similar to depression, the most common treatments include medication and psychotherapy. Most anxiety neurofeedback therapy protocols involve training for alpha enhancement, based on reports that high alpha levels are associated with relaxation and decreased worry. In classifying anxiety, state anxiety refers to anxiety related to an event or perceived threat, whereas trait anxiety measures anxiety as a personal characteristic (Spielberger, 2010). Hardt and Kamiya (1978) showed that alpha-enhancement neurofeedback can be successful, but only in high trait anxiety subjects. Their results could not be explained by feelings of success following the neurofeedback tasks, as training for alpha suppression actually led to increases in anxiety scores. This study helped explain why some were not able to reduce anxiety using alpha enhancement (Paskowitz & Orne, 1973): the baseline levels of trait anxiety may have been too low to notice a significant change.
In a similar study, Rice et al. (1993) compared groups of volunteers suffering from generalized anxiety disorder that were subjected to various therapies, including four alpha neurofeedback sessions. Although alpha-enhancement training failed to increase baseline alpha levels, it did decrease anxiety levels, whereas other groups (including control) did not experience decreases in anxiety.

Other studies have shown success in treating other subcategories of anxiety. In a controlled study, Garrett and Silver (1976) were able to increase alpha and decrease anxiety in a population of students suffering from test anxiety, using an alpha-enhancement protocol. To determine the effectiveness of neurofeedback in treating PTSD, Peniston et al. (1993) compared an alpha-theta enhancement neurofeedback protocol to conventional PTSD therapy. All fifteen subjects in the neurofeedback group showed improvements in all ten subcategories of an anxiety level assessment, whereas the control group only showed improvement in one subcategory. Hammond (2003) used personalized protocols based on QEEG assessment to treat two subjects with obsessive-compulsive disorder; both subjects reported remission of symptoms and improved dramatically on OCD batteries following therapy.

1.5 Goals of the Present Study

This experiment assesses the effects of a single alpha-enhancement neurofeedback session on alpha activity and anxiety levels. We hypothesized that up-regulation of alpha activity with five short neurofeedback sessions would lead to positive changes in alpha activity between baseline sessions, and that any alpha increases would be correlated with decreases in self-reported anxiety. Although a few
studies have explored the use of alpha-enhancement for anxiety reduction, this study is different in at least four ways. First, most neurofeedback experiments utilize at least five hours of neurofeedback, broken into many sessions over the course of multiple weeks. Nevertheless, it has been shown that a single alpha-enhancement neurofeedback session can cause an increase in alpha power (Escolano et al., 2014; Konareva, 2005), and focusing on one session is advantageous in some ways. In comparing alpha and anxiety data within 20 minutes, it is possible to identify subtle changes in scores that may not be noticed in longer-term studies.

Another difference between the present study and most traditional studies is that it includes a control group receiving sham feedback (most prior studies did not include a control group). Comparing performance between the neurofeedback and the sham feedback groups helps eliminate possible confounding variables such as the effects of sitting with eyes closed, placebo, etc.

Third, in our sonification paradigm, brain activity controls the pitch of a tone, whereas most previous studies used volume as the variable feedback parameter (Hardt & Kamiya, 1978; Éismont et al., 2011). We hypothesized that attempting to decrease pitch would be more relaxing (and thus rewarding) for subjects than attempting to decrease volume.

Lastly, the auditory feedback used in this experiment was created completely from scratch. In most neurofeedback research – especially recent research – ready-made neurofeedback software such as BrainAvatar™ by BrainMaster Technologies, Inc. is used for feedback and analysis. Because we had complete control over the
data-acquisition and sonification algorithms, the software pipeline used in this experiment is completely customizable and can be individualized for each subject.

It is important to note that all recordings were performed while subjects’ eyes were closed. Most alpha-enhancement neurofeedback studies that use auditory feedback have subjects keep their eyes closed. Alpha activity is suppressed with visual activity (Collura, 2014), and it has been suggested that eyes-closed recordings be used in experiments where visual stimuli are not necessary (Barry et al., 2007). It has also been experimentally shown that attempts at alpha-enhancement neurofeedback with eyes open failed to decrease participants’ levels of arousal during stress (Bazanova, 2014). In addition, eyes-closed recording eliminates almost all blink artifacts in the final recording. EEG data are far cleaner without ocular artifacts, and the processes used to remove blink artifacts can result in loss of data.

2. Methods

2.1 Data Collection and Sonification

Continuous 32-channel EEG data were acquired using the BrainVision actiCHamp active-channel EEG amplifier. Electrodes were positioned according to the International 10-20 system. Head circumference was measured and an appropriately-sized cap was used for each subject. Data used for sonification came from electrode Oz, which showed high alpha activity in pilot experiments. All impedances were kept below 11 kΩ. Electrodes were referenced online to channel Fpz and were later re-referenced to channel Cz. Data were sampled at 1000 Hz per
channel, and were filtered offline with 0.5 to 30 Hz bandpass filter and a 60 Hz notch filter to remove any electrical interference.

Data were collected and visualized in BrainVision’s Pycorder software, which is an open-source, Python-based EEG acquisition program that allows real-time access of EEG data. Data were sent from Pycorder to MATLAB as a one-dimensional array, with metadata in the header detailing the number of channels and sampling rate. Data were then transformed into an $m \times n$ matrix, where $m$ represented channel count and $n$ represented the channel size, computed by dividing the data length by the channel count. Next, a fast Fourier transform (FFT) was applied to the data, using a data interval length of 1 second, with 50 ms between each segment. FFT data were binned into five frequency bands (delta = 0-4 Hz, theta = 4-8 Hz, alpha = 8-12 Hz, beta = 12-20 Hz, gamma = 20-50 Hz) so that alpha ratio (AR) could be computed. AR was computed as average alpha amplitude divided by the sum of average amplitudes from all five frequency bands. The AR data were sent over a socket to Max/MSP in the form of Open Sound Control (OSC) data using the pnet library for MATLAB.

In Max/MSP, OSC AR data were multiplied by 100 to convert the ratio into a percentage. These data were then scaled to float values from 110 to 880. Next, the data were smoothed according to the following function,

$$Y(n) = y(n-1) + ((x(n) - y(n-1))/\text{slide value})$$

where the output is equal to the last value added to the difference between the last value and the initial input divided by the slide value. A slide value of 10 was used,
such that the output changed $1/10^{th}$ as quickly as the input. These smoothed values then dynamically determined the pitch of a sine tone. In this way, AR and pitch were inversely related, where higher AR created a low-pitch tone.

2.2 Participants

Participants were 21 Wesleyan University students who ranged in age from 18 to 26 (11 female). Subjects were compensated with either course credit in Introductory Psychology (PSYC 105) or an Amazon.com gift certificate. Subjects were randomly placed into experimental ($n = 12$) and control ($n = 10$) groups. Descriptive statistics are shown in Figure 1.

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th># Female</th>
<th>Avg. Age</th>
<th>Shipley</th>
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<td>21</td>
<td>1.7</td>
<td>16.42</td>
</tr>
<tr>
<td>Control</td>
<td>10</td>
<td>5</td>
<td>21.4</td>
<td>1.65</td>
<td>16.1</td>
</tr>
</tbody>
</table>

Figure 1: Descriptive statistics for subject pool. Mean and standard deviation values were calculated for age, Shipley Institute of Living Scale of intellectual functioning scores, and pitch discrimination (PD) thresholds for both the experimental and control groups.

Subjects completed a brief background survey and a battery of tests which included the Shipley Institute of Living Scale of intellectual functioning (Shipley 1940) and a pitch discrimination threshold test that calculated pitch perception threshold with a three-up one-down adaptive staircase procedure centered around 500 Hz. Subjects were matched for age, sex, Shipley score, and pitch discrimination threshold. All subjects provided written consent. The study was approved by the
Institutional Review Board and the Wesleyan University Psychology Ethics Committee.

2.3 Experimental Protocol

After providing written consent, all subjects first completed the Spielberger State-Trait Anxiety Inventory (STAI), which includes ten questions regarding state anxiety (SA; see Appendix 1) and ten regarding trait anxiety (TA; see Appendix 2) Next, subjects provided basic demographic information via a survey administered prior to testing. The survey included items asking about musical experience and training, as well as history of mental illness and/or cognitive impairment, and language skills. All subjects reported having normal hearing. Participants then completed a pitch-discrimination test (perception and production), the Melodic Contour task of the Montreal Battery for Evaluation of Amusia (MBEA; Peretz et al., 2003), the Harvard Beat Assessment Test (HBAT; Fujii & Schlaug, 2013), the Shipley Institute Living Scale for non-verbal IQ, and the Interpersonal Reactivity Index survey (Davis, 1996). These data were acquired to assess for possible correlations between specific attributes/abilities and performance on the task.

Following EEG capping, subjects were moved into an electrically shielded, sound-attenuated room for recording. All electrical equipment (cell phones, etc.) was removed from the testing room, aside from the EEG battery and amplifier. First, all subjects provided 3-minute baseline recordings of brain activity. Subjects were told to keep their eyes closed, stay still, not to think of anything in particular, and to not fall asleep for three minutes. During this baseline recording, upper and lower limits of AR
were determined for the experimental group. In Max/MSP, maximum and minimum alpha percentage values during baseline were captured and stored; upper and lower alpha percentage limits were calculated by (alpha percentage + 3) and (alpha percentage – 3), respectively. Widening the range between the upper and lower limit gave the participants an opportunity during the experiment to produce an AR value outside of the baseline-recorded range. Therefore, if a subject’s alpha percentage maximum was 22 and minimum was 4, the upper and lower limits would be 25 and 1, respectively. These limit values were then inversely scaled to values between 110 and 880 as described in section 3.1.

After the baseline recording, five three-minute blocks of either authentic or sham neurofeedback training followed. All subjects in both groups were told that they would hear a tone, and that the pitch of that tone was correlated with their brain activity. They were told that when the pitch decreased they were in the correct brain state, whereas when the pitch increased, they were not in the correct brain state. Participants were told to sit back, relax, stay still, not think of anything in particular, and try to lower the tone. They were not given any mental strategies on how to do so, and were unaware of what mental activity was being tested. Audio was played through Sennheiser CX 1.00 earbud headphones. Subjects in the experimental group heard real-time sonified neurofeedback, whereas control subjects simply heard the pre-recorded neurofeedback of another experimental subject.

Following each neurofeedback session, subjects were asked if they 1) fell asleep, 2) felt like they had control over the pitch of the tone, and 3) had employed
any mental strategies in an attempt to lower the tone. All responses were recorded electronically.

When five neurofeedback sessions had been completed, a second three-minute baseline recording was acquired in the same manner as the first. Before leaving, the same STAI from the beginning of the experiment was administered again, and subjects filled out a short questionnaire that asked about perceived control over the pitch of the tone, how much they enjoyed experiment, and prior experience with EEG and meditation (see Appendix 3). Subjects were debriefed at the end of the session.

3. Results

3.1 Behavioral Results

Difference scores were calculated as a measure of change in anxiety level over the course of the experiment. Controls showed a slight increase in state anxiety ($M = .04, SD = 0.449$), but this difference was not significant ($t(9) = -0.282, p = .785$). Although the experimental group experienced a slight decrease in state anxiety ($M = -0.104, SD = 0.367$), this change was not significant ($t(11) = 0.985, p = .346$). Changes in trait anxiety were similarly non-significant, both for control ($t(9) = 0.510, p = 0.622$) and experimental ($t(11) = 1.589, p = 0.14$). Group differences in anxiety levels were not significant for state ($t(20) = -0.830, p = 0.385$) or trait ($t(20) = -0.563, p = 0.467$) anxiety.
3.2 Electrophysiological Results

EEG data were imported into BrainVision Analyzer 2.1 for analysis. All channels were re-referenced to electrode Cz. Data were then filtered using an IIR (infinite impulse response) 0.5 Hz high-pass filter, 100 Hz low-pass filter, and a 60 Hz notch filter to remove any electrical noise. EEG data was inspected for artifacts, and segments displaying high amplitude (± 200 Hz) or irregularly-steep voltage steps (50 µV/ms) were rejected. As this was an eyes-closed experiment, no specific measures were taken to reduce ocular artifacts, although any ocular artifact would be removed in the general inspection. Electrodes were subsequently averaged in groups to determine localization of frequency band changes in central (FC5, FC1, FC2, FC6, C3, Cz, C4, CP5, CP1, CP2, CP6), frontal (Fp1, Fp2, F7, F3, Fz, F4, F8), temporal (FT9, FT10, T7, T8, TP9, TP10), parietal (P7, P3, Pz, P4, P8), and occipital (O1, Oz, O2) brain regions. Prior to applying the FFT, data from each trial were segmented into 2s segments with a 10% overlap between each segment. A moving window FFT with 2.048 data interval, Hamming window, and 10% overlap was applied to each segment, and the resulting frequency spectrum was averaged across each segment to yield the final FFT data. Alpha ratio was calculated as mean alpha amplitude divided by the sum of mean amplitudes from each frequency band. All FFT data was then exported to SPSS (IBM Corporation) for statistical analysis.

Mean AR changes in pre- and post-neurofeedback sessions were calculated for both groups in five general brain areas, as shown in Figure 2. Data were also averaged from all electrodes to determine EEG activity across the entire scalp. No
significant differences in means were found between control and experimental groups for any brain regions, nor for differences between the grand averages (p>0.05).

![Mean Δα Ratio](image)

**Figure 2**: Mean changes in AR for experimental and control groups based on brain area, along with mean AR change averaged across all EEG electrodes. Standard error bars included.

AR changes were also averaged for each baseline session and all neurofeedback sessions in both groups for each brain area, as shown in Figure 3. No significant differences (p>0.05) were found between experimental and control groups for any given trail.
Figure 3: Changes in alpha ratio over the course of the seven EEG recording sessions in central (A), frontal (B), parietal (C), occipital (D), and temporal (E) regions of the brain. The red lines correspond to mean ratio values from the experimental group, whereas the blue lines correspond to mean values from the control group. Graph (F) represents averaged ratio values across all electrodes.

Figure 4 plots values of changes in AR between baselines against changes in the state portion of the STAI. AR changes did not significantly predict changes in STAI-state scores, for both control (F = 0.933, \( p = 0.39 \)) and experimental (F = 0.023, \( p = 0.89 \)). The coefficient of determination \( (r^2) \) for each group was 0.0012 and 0.0025 for control and experimental groups, respectively.
4. Discussion

This experiment examined the effects of a single alpha-enhancement auditory neurofeedback session on changes in the ratio of alpha (8-12 Hz) amplitude to all other frequency band amplitudes, along with the relationship between alpha ratio changes and shifts in self-reported anxiety before and after neurofeedback. The study included a control group that received sham neurofeedback, allowing for the elimination of confounding variables shared by both groups. No dedicated neurofeedback software or hardware was used in creating the sonified neurofeedback; this study successfully applied a novel sonification pipeline that has a high degree of malleability, and can certainly be used in future neurofeedback experiments.

In both control and experimental groups, AR changes were assessed for each three-minute neurofeedback session and pre- and post-neurofeedback. Although information on raw alpha amplitude was available, only alpha ratio data was used in Figure 4: Scatterplot of mean difference in $\alpha$ ratio between baseline tests against change in STAI-State self-reported anxiety scores, for experimental and control groups.
statistical analysis, for two reasons. First, descriptive statistics on alpha amplitude data showed discrepancies in baseline alpha amplitude ($M = 0.85 \mu V, SD = 0.31$ for experimental, $M = 1.16, SD = 0.54$ for control). In fact, average amplitudes for all frequencies were slightly higher for the control group in every trial. The difference between these means was not statistically significant, implying that this discrepancy occurred randomly. Calculating AR effectively normalized the data for both groups, therefore eliminating the effect of any baseline differences in voltage for each subject.

Second, the actual neurofeedback tone was directly related to the ratio of alpha amplitude to other frequency-band amplitudes, as opposed to the absolute alpha band amplitude.

As shown in Figure 2, average AR amplitudes increased after neurofeedback in both groups, with the exception of frontal alpha ratio. No statistically significant differences in alpha ratio were found for either group pre- or post-training, in any of the pre-defined general brain areas (frontal, central, parietal, occipital, and temporal). Average alpha ratio was calculated for each neurofeedback session; however, no significant difference was found between groups in alpha ratio for any given neurofeedback session (Figure 3).

The ability to self-modulate amplitudes of specific frequency bands – the basic tenant of neurofeedback – has been established in hundreds of controlled experiments since Kamiya’s original experiments (Othmer et al., 1999). It is hence unreasonable to assume that the lack of significant findings related to AR changes in this experiment disproves neurofeedback theory. Various experimental limitations
may have contributed to these results. The most significant limitation in this experiment is the time spent receiving training. In a meta-study of neurofeedback experiments for anxiety and depression, Hammond (2005) found the range of training time to be between two and twenty hours, and suggested that at least five hours is necessary for any electrophysiological changes to occur. In two recent studies, neurofeedback training was shown to cause minor changes in certain frequency bands (including alpha) in as short as nine minutes, although neither study yielded significant results, and the authors concluded that longer training sessions were necessary (Konareva, 2005; Escolano et al., 2014). Time restrictions in this study were imposed by logistical factors; a future study would benefit from more neurofeedback sessions per subject.

Latency in the sonification system may have also hindered subjects’ abilities to match brain events with the desired decrease in pitch of the stimulus tone. Tight temporal contiguity is vital to the success of any operant conditioning experiment (Lattal, 2010); in this case, the brain event (e.g. high alpha activity) must be followed directly by the corresponding auditory stimulus (e.g. low-pitched tone). It is estimated that the latency in the current sonification pipeline is ~1000 ms. This includes transfer of data between software, real-time FFT calculation, and the smoothing function in Max/MSP. Determination of exact latency time is a current work in progress, along with decreasing the overall latency of the system. Although there exists no standard latency limit in neurofeedback research, it is possible that subjects were incapable of
connecting brain activity to the stimulus response, either consciously or subconsciously, and were thus unable to enhance AR amplitude.

Although using alpha ratio as the primary metric for both the neurofeedback stimulus and offline analysis provided many benefits, a different mathematical reflection of alpha activity may have yielded more significant electrophysiological and/or behavioral changes. We hypothesized that since the presence of high alpha activity is inversely proportional to activity in other frequency bands such as theta (Hughes, 2005), providing information on AR would augment the stimulus response in the presence of high alpha activity. Nevertheless, some subjects may have displayed high variability in frequency bands other than alpha, while their alpha activity stayed fairly constant. In this case, the denominator of the AR expression would fluctuate while the numerator would remain relatively stable, making the stimulus pitch depend predominantly on non-alpha activity. In future experiments, the tone of the sonified stimulus could represent raw alpha or the ratio of alpha to a fewer number of frequency bands. Other more complex QEEG calculations could also be used for sonification, including coherence, symmetry, z-scores, or LORETA data.

Subjects in the experimental group showed a slight decrease in state anxiety scores ($M = -0.104$) whereas control subjects experienced a minor increase in state anxiety scores ($M = 0.04$) although neither of these changes was significant. Given the small subject pool and the short time interval between the administration of the pre and post STAI, it is not surprising that these differences were minor and not
significant. Group differences would certainly have been amplified with a larger number of subjects and multiple neurofeedback sessions.

In addition, although the STAI is the most direct psychological test for assessing anxiety, it is limited in a number of ways. First, the STAI comes with the drawbacks of any questionnaire, namely the problems of introspective limits and response factors (Greenwald et al., 2002). Introspective limits refer to the inability to report on personal characteristics that are not consciously accessible. On the other hand, response factors assume that a subject is compliant and has access to the information, but does not report accurately on the topic due to apprehension or some other cause. In the case of anxiety, participants may not accurately report their own anxiety levels because they cannot completely comprehend their emotional state (introspective limits), or because they are embarrassed about their answers (response factors). Measures were taken to reduce response factors: all subjects completed the STAI in the same sound-attenuated chamber without observation. Still, response factors such as faking and evaluation apprehension may have compromised the data. Furthermore, the identical STAI was administered before and after the short fifteen-minute neurofeedback session, and subjects’ memory of the first administration of the test may have affected their answers on the second administration.

One way to circumvent many of these limitations would be to use an implicit test of anxiety. The Implicit Association Task (IAT)–Anxiety implicitly measures anxiety by having subjects associate self-related words with and anxiety- and relaxation-related words and assessing reaction time. Although this test does not
explicitly gauge anxiety, it has been shown to be a valid and reliable measure of anxiety (Egloff & Schmukle, 2002). Most importantly, a test like the IAT-Anxiety would eliminate many of the limitations that come with self-report.

State-anxiety score changes were not significantly correlated with changes in AR in both experimental and control groups ($p > 0.05$). This analysis was most likely affected by the behavioral and electrophysiological limitations enumerated above, along with other limitations specific to the interaction between brainwave changes and anxiety. First, anxiety has been associated with abnormalities aside from low alpha (e.g. excess beta), and it is possible that alpha changes have no bearing on anxiety levels for the subjects in this experiment (Collura, 2014). On a related note, Hardt and Kamiya (1978) found that alpha increases were inversely correlated with anxiety changes only in subjects who displayed high pre-neurofeedback trait anxiety. In this experiment, no subjects displayed state or trait anxiety scores above three on a four-point Linkert scale, with mean pre-test scores being approximately two for both the control and experimental groups. It is clear that this study did not include high-anxiety subjects, and with a small subject pool, it would be impractical to separate subjects into groups based on anxiety level. With a larger sample size and more diversity in anxiety scores, it would be interesting to see if the results corroborate the findings of Hardt and Kamiya.

Following each neurofeedback training session, each subject reported his or her strategy for lowering the tone. Subjects were not given any explicit information as to what brain activity was being sonified or what mental strategy would be most
effective, but they were told to relax and not think about anything in particular. Most subjects described “focusing on breath,” “breathing steadily,” or “zoning out” as their tone-lowering strategy, but strategies ranged from synesthetic visualization (“picture tone lowering”) to emotional processing (“think happy thoughts”) to cognitive tasks (“mentally compute multiples of three”). It is known that high alpha activity is associated with relaxation and meditation (Kamiya, 1969); however, it is difficult to make quantitative assessments on these reports, as neurofeedback strategy was not controlled for. It would be interesting to replicate this study and enforce specific strategies, rather than having subjects attempt to formulate strategies on their own.

This study demonstrates the application of a novel neurofeedback system capable of sonifying any QEEG metric in real-time. It serves as a launchpad for further experiments that investigate optimal neurofeedback parameters or the therapeutic benefits of neurofeedback. Future studies can benefit from a larger sample size, longer neurofeedback sessions, multiple sessions over the course of many days or weeks, and reduced latency between alpha generation and feedback. The sonified data itself could be derived from a variety of other metrics, including raw frequency-band amplitudes, or measures of symmetry and coherence. To measure behavioral data, implicit tests could be implemented to reduce any self-report biases.

Barry Sterman has said that successful neurofeedback must be “correct, timely, and meaningful” (2008). In the quest to fully satisfy these conditions, there is much work to be done.
5. References


Bakerich, F., & Scully, R. (1973). Electroencephalophone and feedback system:
Google Patents.


6. Appendix

6.1 STAI (State Anxiety)
### 6.2 STAI (Trait Anxiety)

#### SELF-EVALUATION QUESTIONNAIRE

**STAI Form Y-2**

| Name __________________________ | Date __________ |

**DIRECTIONS**

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

<table>
<thead>
<tr>
<th>Statement</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>21. I feel pleasant</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22. I feel nervous and restless</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. I feel satisfied with myself</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24. I wish I could be as happy as others seem to be</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>25. I feel like a failure</td>
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<td></td>
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<tr>
<td>26. I feel rested</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27. I am &quot;calm, cool, and collected&quot;</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>28. I feel that difficulties are piling up so that I cannot overcome them</td>
<td></td>
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<td></td>
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<tr>
<td>29. I worry too much over something that really doesn’t matter</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30. I am happy</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>31. I have disturbing thoughts</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>32. I lack self-confidence</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>33. I feel secure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>34. I make decisions easily</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35. I feel inadequate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>36. I am content</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>37. Some unimportant thought runs through my mind and bothers me</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>38. I take disappointments so keenly that I can’t put them out of my mind</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>39. I am a steady person</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40. I get in a state of tension or turmoil as I think over my recent concerns and interests</td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>
6.3 Ending Questionnaire

**Ending Questionnaire**

1. Have you ever participated in an EEG experiment?  Y  N

2. Did you know what we were testing for in this experiment?  Y  N

3. Do you feel like you were in control of the pitch of the sound?  Y  N

4. On a scale of 1-7, how enjoyable was this experience?
   
   1  2  3  4  5  6  7

5. Have you ever regularly meditated?  Y  N
   a. If yes, please explain (when did you start, how regularly):

6. How often do you meditate?
   a. Never
   b. A few times a year
   c. Once a month
   d. Once a week
   e. More than once a week
   f. Daily
   g. I don't meditate now, but I have in the past

7. How many hours ago was your last caffeinated beverage? (If you don't consume caffeine, say N/A) ________