DEVELOPMENT AND PILOT TESTING OF A PROTOCOL FOR ASSESSING NEGATIVE TRANSFERENCE REACTIONS DURING OBSERVED AND EXPERIENTIAL INTEGRATION USING ELECTROENCEPHALOGRAPHY AND LOW RESOLUTION BRAIN ELECTROMAGNETIC TOMOGRAPHY

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ABSTRACT

Many investigations have provided evidence of neuronal and sensory symptoms associated with posttraumatic stress disorder (PTSD; Begić, Hotujac, & Jokić-Begić, 2001; Lanius et al., 2001, 2002, 2003, 2004; Woon & Hedges, 2009). One such effect of psychological trauma, known as "negative transference reactions", was explored in this study. Transference reactions refer to unconscious projections of clients' thoughts and feelings onto their therapists, among others. Over the last 16 years, a new trauma therapy known as *Observed and Experiential Integration* (OEI) has been discovered and developed (Bradshaw, Cook, & McDonald, 2011). OEI procedures were used in this study to assess and treat negative transference reactions. The purpose of this pilot study was to develop and test a protocol for assessment and amelioration of transference reactions using quantitative electroencephalographic (qEEG) and low resolution brain electromagnetic tomography (LORETA). The intent was to explore changes in cortical activity associated with transference reactions prior to, and following, OEI treatment targeting those reactions. Evaluations were employed in five phases: (a) baseline assessments, (b) stimulus source comparisons, (c) pretreatment transference assessments, (d) treatment assessments, and (e) posttreatment transference assessments. At each phase, qualitative interviews, psychometric measurements, and psychophysiological assessments were completed. Psychometric measurements included the Clinician-Administered Dissociative States Scale (CADSS) and the Transference Reaction Record (TRR). Psychophysiological assessments involved qEEG and LORETA. Findings indicated that at posttreatment there were significant decreases in the nature and severity of negative transference reactions and disturbing somatic symptoms. For one participant, LORETA

analyses revealed that from pre- to post-treatment (with left eye open --- the state associated with the most disturbing distortions), there was a shift in the origins of alpha activation from the hippocampus to the inferior temporal gyrus. Thus a shift, after OEI treatment, from activation of deeper brain structures to cortical areas, is consistent with a shift from visual memory activation to simple visual (facial) scanning and recognition. Implications for future research are provided.

Key-words: Observed and Experiential Intergration; negative transference reactions; qEEG; case study.

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CHAPTER 1: INTRODUCTION

Trauma refers to an extremely stressful life experience (Sar, 2008). According to Friedman (2003), trauma is a negative, unexpected, and overwhelming event, and/or an experience that leaves a person feeling powerless. "During the course of lifetime, approximately half of all men and women will be exposed to at least one traumatic event, such as assault, military combat, an industrial or vehicular accident, rape, domestic violence, or a natural disaster" (e.g., earthquakes; Friedman, 2003, p.1).

Trauma is regarded as a lifetime process that, over time, gets ingrained in an individual's brain and develops into what is known as Posttraumatic Stress Disorder (PTSD). PTSD is characterized by the presence of three clusters of symptoms: (a) reexperiencing traumatic events through flashbacks or hallucinations; (b) avoidance or *psychological amnesia*, or numbing or *emotional amnesia*; and (c) hyperarousal or hypervigilance (Friedman, 2003; Van Etten & Taylor, 1998). A broader definition of the criteria for PTSD is provided in the current version of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000).

1. For this diagnosis, the individual must have been exposed to a traumatic event by directly experiencing, witnessing, or being confronted with actual or threatened death or serious injury to self or to others, resulting in intense fear, helplessness, or horror.
2. The individual must persistently reexperience the traumatic event through recurrent and intrusive thoughts, images, or dreams. Additionally, the individual

manifests psychological distress in response to internal or external cues that resemble aspects of the traumatic event.

3. The individual avoids cues associated with the trauma, and experiences numbing including the following reactions: avoidance of cognitions, emotions, or dialogues related to trauma; avoidance of activities, settings, or individuals linked to the trauma; inability to remember vital aspects of the trauma; lack of interest in significant activities; feelings of disconnection or separation from others; restricted range of affect; and/or sense of foreshortened future.
4. The individual must experience ongoing hyperarousal, including the following indicators: sleep difficulties, problems controlling anger responses, problems with concentration, over-alertness, and exaggerated startle responses.
5. The symptoms in criteria 2, 3, and 4 must persist for more than one month.
6. The symptoms must cause significant dysfunction in the psychological, social, and/or occupational functioning of the individual.

The epidemiology of PTSD provides further understanding. PTSD prevalence in Vietnam veterans ranges from 20% to 30% (Novac, 2003). Lifetime prevalence of PTSD ranges from 1-14%, depending on the population and diagnostic methods (Van Etten & Taylor, 1998). In general community populations, women exhibit higher prevalence (10%) than men (5%; Connor & Butterfield, 2003).

Precipitating events such as wars, physical or sexual assaults, motor vehicle accidents, and natural disasters may produce a wide variety of psychological symptoms in addition to PTSD, including anger, shame, guilt, fear, and sadness. PTSD also results in negative beliefs (about self, others, and the world), seeing vivid and fragmented visual

images, and heightened physiological responses (e.g., heart rate and blood pressure; Brewin & Holmes, 2003; Orr & Roth, 2000). Exposure to trauma affects cortical pathways in the brain, specifically the limbic system. The amygdala and hippocampus are important structures in the limbic system that regulate emotion, fear, and memory. Neuroimaging studies reveal that PTSD results in reduced hippocampal volume and alterations in the other brain regions such as the amygdala, the anterior cingulate cortex, and the hypothalamus (Connor & Butterfield, 2003). It is evident, then, that PTSD affects both physiological and psychological reactions.

Traumatic events have cumulative effects on individuals in terms of symptoms (Novac, 2003). Only one such impact of psychological trauma, known as 'transference reaction' was explored in this study. The term *transference* was first used by Sigmund Freud in his psychodynamic theory (Rawn, 1987). In psychoanalysis, transference refers to "the strong sexual or aggressive feelings, positive or negative that patients develop toward their analyst during the course of treatment" (Feist & Feist, 2006, p. 49). Transference is reputed to be unconscious projection of one's thoughts and feelings onto another person. Given the right context, any kind of relational trauma cues can result in transference projections. In therapy, a client's transference is typically projected onto the therapist (Fischer, 2005; Gaensbauer & Jordan, 2009). As a result of this transference, individuals are often unable to separate themselves from their past traumatic experiences. It is vital, therefore, in treating PTSD, to minimize perceptual distortions that may negatively affect interpersonal relationships, as described in the following quote regarding transference and Observed and Experiential Integration (OEI) treatment (Bradshaw & Cook, 2008):

> This technique has benefited me, because I never knew I had problems! I had "unexplainable thoughts" and when I looked at people, they seemed to be looking at me funny. OEI helped me to see that I had distortions in my brain. I can now see the distortions. I can see the differences clearly–how my brain and my sight create different projections–far and near depending on which eye I cover. I've worked with pictures at home, and this technique has helped me to be more confident in my thinking and my self. My brain has been changed! (p. 58).

This is the personal testimony of a client who was sexually abused in foster care and struggled for years with a dissociative disorder. After receiving OEI treatment she reported significant improvements. The narrative of her struggle is one of many such stories told by people who face traumatic events in their lives.

Over the last several decades, a number of psychotherapies have been developed to treat PTSD, including: Cognitive Behavioural Therapy (CBT), Eye Movement Desensitization and Reprocessing (EMDR), and Stress Inoculation Training (SIT; Rothbaum, 1997; Seedat, Stein, & Carey, 2005). In the past 16 years, Bradshaw, Cook, and McDonald (2011) have discovered and developed a new psychotherapy for psychological trauma known as *Observed and Experiential Integration* (OEI). It is based on the assumption that multisensory representations of traumatic experiences can be reaccessed in the brain through the visual pathways, and underlying distortion, dissociation, and intensity can be dissipated in addition to correction of cerebral asymmetries (Bradshaw et al., 2011; Ndunda, 2006). As a result of relational traumas, some individuals experience perceptual distortions when they view the faces of people who remind them of someone who harmed or frightened them in the past. This can

extend to images associated via classical conditioning with harmful or frightening events or people, and can even include reactions to viewing one's own image in mirrors. These distortions are referred to in OEI as *transference reactions.* The OEI technique of *Switching* (alternate covering and uncovering of one eye at a time) appears to result in the integration of such emotional processes in the left and right hemispheres of the brain (Ndunda, 2006). As a result, clients experience shifts in emotions, perceptions, and sensations (Bradshaw et al., 2011). Between Audrey Cook (A.C) and Rick Bradshaw (R.B), they have applied OEI in their clinical practices during more than 45,000 hours of time, spread over 15 years (R. A. Bradshaw, personal communication, August 30, 2010). Additionally, there are numerous unpublished masters' theses (e.g., Austin, 2003; Grace, 2003; Lefebvre, 2004; McInnes, 2007; Ndunda, 2006; Williams, 2006), conference presentations (e.g., Bradshaw, 2008; Bradshaw, Grace, & Swingle, 2004; Bradshaw et al., 2009; Stewart, Dadson, & Bradshaw, 2009), symposia (e.g., Bradshaw et al., 2007; McDonald, Bradshaw, & Stewart, 2009; Williams et al., 2007), and the testimonies of clients (Bradshaw & Cook, 2008) that support the efficacy of OEI.

Bradshaw and his colleagues described the importance of the discovery of OEI transference checking and clearing procedures as follows:

> A large body of research supports the centrality of the therapeutic alliance in determining psychotherapy effectiveness (Horvath, 2006; Norcross, 2002; Obegi, 2008). Any barriers or distortions that impair this bond may substantiallyinterfere with treatment outcome. While working with clients using Switching to resolve core trauma symptoms and dissociative artifacts, we (A.C. and R.B.) began to notice that clients were exhibiting different responses to us as therapists,

> depending on which of their eyes was covered. When we made inquiries, it became apparent that many clients actually *perceived our faces* differently as they Switched. These were not just metaphorical or emotional shifts but, rather, involved major changes in the *appearance* of the therapist's face. Some of these involved apparent distances from the therapist (six inches away with one eye covered vs. six feet away with the other eye covered). Others involved changes in physical appearance of the therapist (looks like a mean, white-haired old man on one side, generating fear in the client; and looks like a kind, middle-aged, dark-haired man with the other eye covered). Some of these shifts are extreme, to the point where the therapist appears to have no face, or has a huge head and small body with one eye covered, but looks normal with the other eye covered. The encouraging discovery was that if a client repeatedly Switched (alternated the eye that was covered), while looking at the therapist (or partner, or child, or even his or her image in a mirror), the perceptual distortions dissolved. This has been extremely helpful for us as therapists and can be easily and quickly applied by psychotherapists practicing according to any therapy model (Bradshaw et al., 2011, pp. 130-131).

It is evident, then, that PTSD affects different parts of the brain, and that some of these brain changes affect visual perception of faces. According to Damsa, Kosel, and Moussally (2008), there are various brain imaging techniques such as positron emission tomography (PET), functional magnetic resonance imaging (fMRI), and single photon emission computed tomography (SPECT). For the purpose of this study, however, only descriptions of magnetoencephalography (MEG), quantitative electroencephalography

(qEEG), and low resolution brain electromagnetic tomography (LORETA) are provided. The temporal resolution of MEG and EEG are very similar (Parra, Kalitzin, & Lopes da Silva, 2004; Posner & Raichle, 1999). Additionally, LORETA is based on scalp-recorded EEG (Anderer et al., 2004) and the corresponding rapid temporal resolution will be most suitable for assessing rapid phenomena such as transference reactions.

In this study, the intent was to examine changes in brain electrical activity that occurred while a participant gazed at different faces. Changes in cortical activity were measured using qEEG with LORETA. Cortico-electrical activity associated with transference reactions was assessed pre- and post-OEI treatment. The participant received 90 minutes of OEI treatment targeting her transference reactions to one particular (most-triggering male) face. The objective in this pilot study was to develop and test a research protocol to *assess* transference reactions pre- and post-treatment. It had not been determined, for example, whether transference reactions occurred with equal intensity in response to, still photographs, video recordings, or live individuals. Both the subjective and objective experiences of an individual who experienced transference reactions needed to be more formally assessed, to more clearly define and understand this phenomenon. Such objectives led to the following research questions:

1. What changes in cortical activity occur during transference reactions before OEI treatment?
2. What qualitative responses occur during OEI treatment for transference reactions?
3. What changes in cortical activity occur during transference reactions after OEI treatment?

An overview of current literature on PTSD treatment, OEI interventions, and brain imaging techniques is provided in Chapter 2. In Chapter 3, the research protocol for the study is described. Chapter 4 is a summary of results, and Chapter 5 includes discussion of the findings relative to the literature, implications for clinical practice and future research, limitations of the study, and conclusions based on the findings.

CHAPTER 2: LITERATURE REVIEW

This literature review is divided into three sections. In the first section psychological treatments for PTSD are discussed, with detailed information on OEI. In the second section, different ways of measuring brain activity are described**.** Each assessment instrument and procedure is explained, with limitations, advantages, and empirical evaluations. In the third section, the clinical phenomenon of transference reactions is discussed. The literature review concludes with a summary of the purposes for this pilot study, and with research questions.

Psychological Treatments for PTSD

According to the current version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), the symptoms of PTSD are both physiological and psychological. The goals of PTSD treatment are: (a) reduction of intrusive and avoidance symptoms, (b) reduction of numbing, withdrawal, hyperarousal, and psychotic symptoms, if any exist, and (c) improvement in impulse control (McInnes, 2007, p. 20).

Clinicians use various empirically supported psychotherapies to treat PTSD (Silver, Rogers, Knipe, & Colelli, 2005). This section is not intended to provide an exhaustive review of psychotherapies to treat PTSD; however, it is a brief synopsis of major treatments (SIT, CBT, and EMDR). A detailed description of OEI is also presented.

Stress inoculation training (SIT). SIT was developed by Donald Meichenbaum to treat clients with phobias of many objects (Meichenbaum, 1977). His anxiety management training includes two distinct levels. Individuals who receive the first level of training participate in a discussion of the symptoms of trauma, and rationales for SIT.

Individuals who attend the second level learn coping strategies (e.g., deep breathing, muscle relaxation, cognitive restructuring, and assertiveness training), and applications in daily living (Livanou, 2001). SIT reduces physical, cognitive, and behavioural components of fear (Seedat et al., 2005). Kiselica, Baker, Thomas, and Reedy (1994) studied the effectiveness of SIT for anxiety, stress, and academic performance difficulties. Results revealed that, in comparison to the control group, the participating ninth graders (N = 48) who received eight sessions of SIT experienced significant improvement in their abilities to cope with anxiety and stress, but experienced no change in their academic performance. These treatment gains were maintained at 4-week follow up. Other research results indicate that SIT has been used successfully for anger management in juvenile delinquents and in treatment of individuals who were raped (Foa, Rothbaum, Riggs, & Murdock, 1991; Schlichter & Horan, 1981). Meichenbaum (1996) summarized various studies conducted with individuals who faced stressors (e.g., psychiatric illnesses, combat, or medical illnesses). Results support the efficacy of SIT.

Cognitive behavioural therapy (CBT). CBT was developed based on the assumption that emotional and behavioural responses are determined by the ways individuals structure, interpret, and evaluate their worlds. CBT therapists incorporate theories of learning, conditioning, and cognitive restructuring. This approach helps clients identify, evaluate, and alter dysfunctional thoughts and belief systems (Graham, 1998, p.2).

CBT is one of the most extensively researched psychotherapies (Butler, Chapman, Forman, & Beck, 2006; Livanou, 2001). Butler and his colleagues (2006) conducted a metaanalysis of 16 studies to review the efficacy of CBT for different types of disorders,

including PTSD. Studies of trauma-based CBT groups included participants who were survivors of various traumatic events such as accidents, assaults, domestic violence, and military combat. Results of the studies indicated large effect sizes for trauma-focused CBT compared to a wait-list group, implying a strong treatment effect. Results of other studies confirm the effectiveness of CBT for treating trauma survivors (Jaycox, Zoellner, & Foa, 2002; Klein et al., 2009; McDonagh et al., 2005).

Eye movement desensitization and reprocessing (EMDR). EMDR was developed by Francine Shapiro in 1989 for treating trauma (Edmond & Rubin, 2004). It involves rapid *saccadic eye movements* (induced by tracking a therapist's index finger as it moves laterally rapidly, across the visual fields of both eyes). Alternative forms of bilateral stimulations (e.g., audio or tactile), are also theorized to facilitate information processing, resulting in reductions of emotional intensity due to posttraumatic states (Edmond & Rubin, 2004; Friedman, 2003; Van Etten & Taylor, 1998). In EMDR treatment, clients are asked to identify traumatic events, focus on images of those events, and identify associated negative self-referencing beliefs. Clients are also asked to identify current emotions and accompanying body sensations. Next, they are asked to articulate desired positive self-cognitions (Silver et al., 2005). Clinicians then ask their clients to focus on traumatic memories while visually focusing on their clinician's fingers or other bilateral stimulations. After a set of 10-12 eye movements, the client is asked to rate both the traumatic memories and the positive cognitions (Friedman, 2003; Silver et al., 2005). This is continued until the client reports little or no distress regarding recollections of each traumatic experience, and achieves maximum validity for positive cognitions (Livanou, 2001; Silver et al., 2005). In this way, eye movements or other

bilateral stimulations together with imaginal exposure and cognitive restructuring, result in the integration of traumatic experiences (Seedat et al., 2005). Many studies have shown the effectiveness of EMDR for reducing PTSD symptoms (Edmond & Rubin, 2004; Ironson, Freund, Strauss, & Williams, 2002; Rothbaum, 1997; Silver et al., 2005; Van Etten & Taylor, 1998).

Observed and experiential integration (OEI). In this section, the background, theory, techniques, and empirical evidence for OEI are presented. Prior to offering this information, it is important to briefly describe another approach that involves alternating visual fields, which was developed during approximately the same time period as OEI. Similarities to, and differences from, OEI are clarified.

Dual brain psychology. Dr. Fredric Schiffer (1998), a Harvard University psychiatrist, began conducting experiments on postcommissurotomy patients (patients who have had their corpus callossa cut to separate the hemispheres). He postulated that there was a separate mind associated with each hemisphere of the brain, resulting in a *dual brain* model. "In one constellation, one mind is more mature, reasonable, and living in the present. The second mind, is immature in its cognitive and emotional aspects, is stuck back in an old trauma" (Schiffer, 1998, p. 80). He argued, however, that various possible relationships exist between the two minds. For example, both minds can be healthy or unhealthy. According to Schiffer, the goal of dual brain therapy is to bring the two minds to the point where they can collaborate, enhancing creativity and maturity. The central feature of dual brain therapy is *lateralized glasses* or shaded contact lenses. With these devices, everything but the lateral visual field on one side is blocked (hence the need for two pairs - one to cover all but the left lateral visual field, and one to cover

all but the right lateral visual field). The client is asked to wear the glasses that activate the immature view that Schiffer believes is trapped in past traumatic memories. Next, the client is asked to wear the glasses or lenses that bring to mind their mature and healthy views. This process allows clients to focus on one hemisphere at a time, with the result that therapists knowing which hemisphere needs work.

OEI development. Schiffer's dual brain model is very similar to another set of procedures that were independently developed in 1994 and 1995 by a marriage and family therapist in Canada by the name of Audrey Cook (Cook & Bradshaw, 2002). In her own practice of EMDR, Audrey Cook noticed that some clients with lifetimes of severe neglect and abuse were unable to track stimuli with both eyes. She tried doing EMDR 'one eye at a time.' From educational-kinesiology theory, she emphasized the notion that tracking one eye at a time would be more useful for clients. From this broad concept emerged OEI techniques, to heal clients from traumatic memories (Bradshaw et al., 2011; Cook & Bradshaw, 2002). This treatment was first referred to as *one eye integration* (to differentiate it from EMDR, which involves tracking stimuli with *both* eyes). The therapy is now known as Observed and Experiential Integration, or OEI. The first three OEI techniques were developed by Audrey Cook and have been further refined by both Audrey Cook and Dr. Rick Bradshaw (R. A. Bradshaw, personal communication, August 30, 2010).

Brain structures and functions associated with PTSD. Before introducing the theory for OEI, it is essential to understand brain structures affected by traumatic events. In this brief overview, an understanding of how the brain processes traumatic imprints is provided. According to Reite, Teale, and Rojas (1999), brain function is hemispherically

lateralized. In most humans, the left hemisphere is specialized for language, problem solving, sequential analysis, verbal information processing, labeling of perceptions, and cognitive categorizations. The right hemisphere is associated with nonverbal and paraverbal perception and assessment of emotions through voice tone and facial expression in others, and through visual or spatial communication (Kalat, 2004; Ray et al., 2006; van der Kolk, Burbridge, & Suzuki, 1997). In individuals with PTSD, there is marked lateral dominance of the right hemisphere over the left (Metzger et al., 2004; Vasterling, Rogers, & Kaplan, 2000). As a result of lateral dominance, individuals exposed to trauma cues will experience intense emotions, aroused bodily sensations, and remember fragments of memories, but will be unable to cognitively categorize their traumatic experiences (Bremner, Staib, et al., 1999; Ray et al., 2006). Some of the important brain structures and functions involved in the pathogenic processes resulting in PTSD are discussed below.

Amygdala. Multisensory information passes from the thalamus to the amygdala, which then assigns the valence (positive or negative) and the level of emotional intensity for the event. This brain structure is thought to integrate internal symbols of the external world, in the form of memories and associated emotional experiences (van der Kolk, 2001). The amygdala is involved in behavioural regulation, fear conditioning, emotionally-associated memories, and evaluation of potentially threatening stimuli (Woon & Hedges, 2009).

Results of brain imaging studies have shown that in people with PTSD, any form of trigger causes increased activity in the amygdala (particularly in the right hemisphere), contributing significantly to hyperarousal (Hull, 2002; Rauch et al., 2000; van der Kolk,

2001). Shin, Rauch, and Pitman (2006) completed a PET study, in which the participants with chronic PTSD were exposed to traumatic script-driven imagery. The results indicated that an increase in regional cerebral blood flow in the amygdala caused increased intensity of emotional response. A metaanalysis by Woon and Hedges (2009) confirmed greater amygdala volume in the right hemisphere than the left in participants with PTSD, when compared to those without PTSD.

Anterior cingulate cortex (ACC). The anterior cingulate cortex is another important area for emotional responses. It helps to differentiate current dangers from traumatic reminders. Hence, it acts as both an amplifier and a filter (van der Kolk et al., 1997). In comparison to the number of studies in which researchers examined hippocampal volume in PTSD participants, there are fewer studies on abnormalities in the ACC (Woon & Hedges, 2009). Such studies are needed, however, because individuals with PTSD have reduced activation in the ACC, leading to hyperarousal, which is consistently identified as a prominent symptom of PTSD (Bremner, Staib, et al., 1999; Hull, 2002; Lanius et al., 2001; van der Kolk et al., 1997). Research results indicate that people with PTSD have diminished ACC volume (Damsa et al., 2008; Shin, Rauch, & Pitman, 2006; Woodward et al., 2006).

Broca's area. A small part of the frontal lobe in the left cerebral cortex, near the motor cortex, is known as Broca's area. This is one of the key areas responsible for speaking (Kalat, 2004). Activities associated with this area include communicating one's feelings and experiences to others (van der Kolk, 2002). When used to assess individuals with PTSD, PET scans indicate that increased emotional arousal is associated with decreases in regional cerebral blood flow in Broca's area. This decrease, in turn,

decreases access to oxygen, causing 'speechless terror.' A person may see, feel, or hear the sensory elements of a traumatic experience, but be unable to translate the experience into communicable language (Hull, 2002; Shin et al., 1997; van der Kolk et al., 1997). Hence, traumatic memories are relived in the form of intense emotions, visual images, and somatic sensations (van der Kolk, 2001).

Corpus callosum. Neurons in each hemisphere communicate with neurons in the corresponding part of the contralateral hemisphere, through two bundles of axons known as the corpus callosum (Kalat, 2004). The corpus callosum along with the ACC, aids in integrating the emotional and cognitive aspects of traumatic experiences (van der Kolk et al., 1997; van der Kolk, 2001). In comparison to men, women tend to have thicker corpus callosa, and therefore experience greater communication between the hemispheres (Kalat, 2004). People who have experienced childhood abuse have been shown to have smaller than average corpus callosa, which contributes to diminished communication between the cortical hemispheres (Teicher et al., 2003). Another researcher also reported that individuals with PTSD have smaller corpus callosa than individuals without PTSD. Such abnormality in the corpus callosum leads to fragmentation of cognitive functions, and likely contributes to the symptoms of emotional numbing and dissociation (Villarreal et al., 2004).

Hippocampus. The hippocampus is located between the thalamus and the cerebral cortex. Its main function is to process declarative memories, in the context of time and space (Kalat, 2004). The hippocampus creates a cognitive map to help categorize experiences with other autobiographical information (van der Kolk et al., 1997). According to van der Kolk (2001), proper functioning of the hippocampus is

crucial for short-term memory. There are increasing numbers of studies to support the hypothesis that people with PTSD have smaller than average hippocampi (Bremner, 2006; Shin et al., 2006; Stein, Koverola, Hanna, Torchia, & McClarty, 1997; van der Kolk, 2001; Wignall et al., 2004). Adult survivors of childhood abuse with the diagnosis of PTSD, for instance, have hippocampi that are 12% smaller than those of healthy controls (Bremner et al., 1997). Furthermore, smaller hippocampal volume has been associated with deficits in declarative memory (Bremner, Staib, et al., 1999; Shin et al., 2006).

Decreased hippocampal volume is also cortically associated with PTSD symptoms. When an individual experiences a traumatic event, it gets logged in the hippocampus in *present tense* at *full intensity*. In PTSD patients, stress releases increased amounts of corticotropin releasing factor and abnormal responses in regional cerebral blood flow, resulting in fragmented memories (Shin et al., 2004; van der Kolk, 2001). Stress clearly affects the neurons associated with memory and damages the hippocampus (Bremner, Narayan, et al., 1999).

Theoretical assumptions. The human brain continuously receives, interprets, filters, stores, and transforms the sensory information received both from within the body and from the environment. Millions of neurons are involved in this process. This neuronal activity results in a response which produces internal satisfaction, in harmony with the demands of the environment. According to van der Kolk, Burbridge, and Suzuki (1997), individuals who have experienced trauma and have PTSD, however, are unable to place sensory input into its proper context in time and space. The question that arises, then, is how the mind processes input that is emotionally overwhelming.

Emotional intensity is clearly linked to limbic and paralimbic structures (van der Kolk, 2001). An important part of the limbic system is the amygdala which acts as a "smoke detector" and interprets whether or not incoming sensory elements are threats. For this reason, when individuals with PTSD are confronted with sensory cues that match sensory imprints from their traumatic experiences, the likelihood of intense physiological and psychological reactions is high because of the marked dominance of the right hemisphere over the left. Results of recent research indicate that high levels of arousal, activated by external stimuli in individuals with PTSD, interfere with the functioning of the frontal lobes, Broca's area; and the hippocampi, thalami, cingulate cortices, and dorsolateral prefrontal cortices (van der Kolk, 2002). It appears that this dysfunction is responsible for organizing trauma imprints as fragmented, sensory, and emotional traces, which are so important to treat.

Survivors of trauma often describe their inability to verbally describe or express their experiences, resulting in difficulty with traditional psychological treatments, especially talk therapies (Hull, 2002). A primary characteristic of OEI is that it is not primarily "talk therapy" (Bradshaw & Cook, 2008, p.11). The brain areas associated with talking and listening are the frontal and temporal cortices; whereas, the severe symptoms of PTSD (e.g., panic attacks, flashbacks, and nightmares) emanate from subcortical structures such as the limbic and paralimbic systems (van der Kolk, 2002). In current OEI theory (R. A, Bradshaw, personal communication, September 1, 2010), when individuals have PTSD, their traumatic events are not processed through the hippocampal-dentate complex into the higher cortex where they are perceived as past events. In contrast, they are imbued with emotional and somatic intensity via the

amygdalae and the thalami, but remain blocked at the base of the hippocampi in 'present tense' form. These blockages likely affect the dorsolateral prefrontal cortices, the anterior cingulate gyri, and some parts of the parietal and occipital lobes.

Bradshaw and Cook (2008) further explain that only small portions of the surface of the brain are involved in speaking and understanding speech. Traumatic imprints get trapped in more primitive areas of the brain. According to Bradshaw and Cook (2008), both the eyes have connections to both the hemispheres, and the visual pathway runs directly over the limbic structures. When traumatic experiences are hemispherically integrated, there is less tendency to be triggered, or to negatively misinterpret incoming stimuli as reexperienced traumatic events. It appears, then, that OEI works at a neuropsychological level to reduce the intensity of, and integrate, posttraumatic states.

One associated phenomenon often observed in OEI sessions is that when one eye is covered, a client may perceive the therapist as "a kind person" (e.g., young dark-haired man) but when the client covers the other eye, he or she might perceive the therapist as a "mean old white-haired man" (similar to an abuser perhaps, from the client's childhood years; Ndunda, 2006). Individuals with PTSD have cerebral asymmetry: greater right-sided than the left-sided activations when triggered (Metzger et al., 2004; Rauch, Savage, Alpert, Fischman, & Jenike, 1997; Vasterling et al., 2000). As a result of series of guided movements, it seems that integration of emotional and somatic intensity occurs between the hemispheres across the corpus callosum and the anterior commissure (McInnes, 2007). In short, OEI seems to repair the fragments of traumatic events into less intense, more cortical representations.

From the above discussion, it appears that the underlying processes involved in OEI are: (a) neuropsychological integration of traumatic memories, and (b) desensitization, or resolution of stored multisensory representations of events in the brain *without* feeling overwhelmed (Bradshaw & Cook, 2008). This theoretical perspective is extended in great detail by Bradshaw et al. (2011), and was also explored more fully in the present study, as changes were observed in response to transference reactions before and after OEI treatment.

OEI techniques. There are five sets of techniques involved in OEI treatment. These are *Switching*, *Sweeping*, *Glitch Massaging*, *Glitch Holding with Bilateral Stimulation,* and *Release Points*. The definitions and implications of each of these techniques are discussed below.

Switching. Switching involves the simple procedure of covering and uncovering one eye at a time, while recalling a traumatic memory, focusing on disturbing physical or perceptual sensations, or while looking at a facial image (Cook & Bradshaw, 2002). According to Bradshaw et al. (2011), Switching helps reduce core trauma symptoms (somatic and affective). It also aids in diminishing and resolving dissociative symptoms experienced during the processing of core trauma symptoms. Finally, it facilitates detection and resolution of negative transference reactions.

Sweeping. Audrey Cook found that people with severe trauma often report physical pains such as headaches (Bradshaw et al., 2011). In this process, the client is asked to cover one eye, follow the therapist's finger, which is usually 9-12 inches from the other side of the client's head and at the client's eye level. The therapist starts with his or her finger by the client's ear and arcs around the client's face to the other side of

the client's nose. The same sequence is repeated from the client's other ear to the other side of the client's nose. This procedure continues until the somatic symptoms no longer interfere with the client's ability to process core trauma symptoms. In addition to treating headaches, Sweeping is used for resolving artifacts like dizziness, drowsiness, and visual distortions.

Glitch massaging. The term *glitch* refers to a hesitation, skip, or spontaneous redirection of the eye(s) in the process of tracking a visual stimulus (Bradshaw et al., 2011). The client's eye(s) is guided by a stimulus across the visual field while focusing on disturbing memories or perceptions, and any halts or pauses in eye movements are noted. This process of guiding the client's eyes into, over, or through one or more of these glitches is called *Glitch Massaging*. Depending on the needs of each client, glitch massages can be vertical, horizontal, circular, elliptical, or diagonal on any given plane parallel to the surfaces of the eyes. Additionally, proximal-distal Glitch Massages are toward, and away from, the surfaces of the client's eyes.

A related procedure used in glitch work, is known as *track-to-target.* In this method, the client's *subjective* experiences are used to guide location(s) for the massage, instead of (or in addition to) the therapist's *objective* observations. The client is simply asked to inform the therapist when the disturbing symptoms are most intense, as the therapist tracks through the client's visual field (Bradshaw et al., 2011).

Glitch holding with bilateral stimulation. According to Bradshaw et al. (2011), this technique is for clients who are experiencing double-vision. From the therapist's perspective, it appears that either the client is cross-eyed or one eye is fixed on the stimulus while the other eye is "wandering." Such *visual splitting* is resolved when the

therapist holds the visual stimulus in the location where this splitting occurs, and the client applies bilateral stimulation through the use of audio sounds, or by alternately touching his or her left and right shoulders. Dissipation of dissociative symptoms can be further facilitated, if clients stand on a balance board. When clients dissociate, they lose their balance which, in turn, requires them to reconnect their bodies, facilitating traumatic processing.

Release points. Bradshaw et al. (2011) found that trauma processing can be quite uncomfortable for clients as they experience intense core trauma symptoms (e.g., nausea, or throat constriction). As a result of these experiences, clients are less motivated to continue OEI treatment. For this reason, it is imperative to relieve these clients using *Release Points*, which brings rapid and significant cessation of symptoms. In this process, the client is asked to cover his or her dominant eye, and track the therapist's finger horizontally, parallel to the surface of the client's abdomen (at the level of the lowest rib), and away from centre of the client's chest. During this process, there is a point along this continuum where a respiratory release occurs, chest compression stops instantly, and clients' experience relief from this precursor to panic attacks. At this point, the therapist asks the client to place his or her finger at the same Release Point and encourages the use of this technique between sessions, to gain relief from trauma symptoms such as panic attacks. This technique is also used to relieve clients of nausea and throat constriction. The same procedure (described above) is used, except the therapist tracks across in the *opposite* direction.

Empirical evidence. Controlled research on OEI has been done and reported (13 theses and over 40 conference presentations), and journal articles are in preparation. For

the purpose of this study, most of the research findings pertaining to OEI are drawn from unpublished master's theses and conference papers.

The very first master's thesis in which the efficacy of OEI treatment was explored was Grace (2003). This study involved 10 participants with mixed traumas who met the diagnostic criteria for PTSD. The purpose of this study was to provide preliminary information about the effectiveness of the OEI technique of Switching for reduction of PTSD symptoms from pretreatment to posttreatment. Participants were randomly assigned to a treatment group (n = 5) or a delayed-treatment control group (n = 5). Those in the treatment group received three 60-minute sessions of OEI Switching over a period of 2-weeks. Despite the small sample size, results of the study showed significant decreases in PTSD symptoms for the treatment group, in comparison to the control group.

Austin (2003) conducted another study with five participants, an extension of the first study by Grace (2003). The purpose was to compare PTSD symptom intensity and frequency from a 2-week baseline assessment to a posttreatment evaluation, after three 1-hour OEI treatment sessions. Additionally, remembrance patterns were examined across the three 1-hour treatment sessions using script-driven symptom provocation to evoke and assess traumatic memories. The results showed that PTSD symptoms dropped significantly from the baseline to the posttreatment assessment, after three sessions of Switching. At posttreatment, four of the five individuals no longer met the criteria for PTSD.

Lefebvre (2004) study consisted of 16 participants who received two levels of treatments. The first level of treatment included self-administered Switching for their headache symptoms. In the second level, participants engaged in Switching for two

minutes until their Subjective Units of Distress (SUD) scores were equalized for each of their uncovered eyes. Preliminary evidence from this study indicates that Switching is an effective treatment for somatic symptoms such as headaches (Lefebvre, 2004).

Ndunda (2006) conducted a study to investigate PTSD symptoms, social avoidance, and distress at pretest, posttest, and 3-month follow up. Participants included 29 women who had been sexually assaulted and met the diagnostic criteria for PTSD. They were randomly divided into three groups: cognitive processing therapy (CPT-R, n = 9), OEI (n = 10), and control group (n = 10). Some participants were on antidepressants and, except for one, all of them had previously attended at least one counselling session at some time in their lives. Both the CPT-R and OEI treatment groups received one 2-hour psychoeducation session and three 60-minute individual treatment sessions. The control group received various forms of relaxation and grounding techniques. The findings indicated that OEI treatment reduced the frequency and intensity of PTSD symptoms, and the relief was lasting (Ndunda, 2006).

Williams (2006) conducted an 18-month randomized controlled trial building upon the findings of Grace (2003). The purpose of the study was to compare the effectiveness of three treatments: OEI and CPT-R (administered by a therapist), and a self-administered breathing, relaxation, autogenic, imagery, and grounding (BRAIN or Control) treatment protocol, at pretreatment, posttreatment, and 3-month follow up. A total of 27 women who had been sexually assaulted and met the criteria for PTSD were randomly divided into three groups: OEI (n = 9), CPT-R (n = 8), and BRAIN (n = 10). PTSD symptoms, depression, and trauma-related guilt were assessed, along with changes in brainwave patterns in frontal and parietal areas. The researcher reported that at 3-

month follow up, the OEI group showed significantly greater reduction in PTSD symptoms compared to the BRAIN group; whereas the CPT-R group showed no such significant difference when compared to the BRAIN group (Williams, 2006). This result was sustained (Bradshaw et al., 2007).

Considering results from the above studies of OEI treatment (Austin, 2003; Grace, 2003; Lefebvre, 2004; Ndunda, 2006; Willams, 2006), it is clear that OEI is effective in reducing PTSD symptoms. These results were documented on multiple scales, including the Impact of Event Scale-Revised (IES-R) and the Clinician-Administered PTSD Scale (CAPS), in addition to qualitative findings from interviews with participants at the 3-month follow up (Houghton, 2006). In addition, it appears that OEI treatment helps reduce somatic symptoms such as migraine headaches. Results of these small studies require future replication with larger, and more diverse, samples. Although qEEG measurements were taken at pretreatment, posttreatment, 6-month follow up, and final assessments in the last study; none of the results provide evidence of significant changes in brain functioning due to OEI treatment. Neither do these studies provide confirmation of the theory that OEI works by affecting different parts of the brain.

Brain Imaging Techniques

The human brain can be perceived and analyzed on a wide spectrum of spatial resolutions, ranging from a single molecule to the whole brain. Over the past few decades, scientists have invented various techniques to assess brain functioning, in temporal resolutions ranging from a few milliseconds to a lifetime. According to Damsa et al. (2008), there are many ways of measuring brain activity. Only three of these are relevant for this study: Magnetoencephalography (MEG), quantitative

Electroencephalography (qEEG), and Low Resolution Brain Electromagnetic Tomography (LORETA). The temporal resolution (precision of measurement with time) of MEG and EEG are quite similar (Parra et al., 2004; Posner & Raichle, 1999). However, the spatial resolution (area covered by an imaging technique) of EEG is inferior to MEG (Knowlton & Shih, 2004; Reite et al., 1999). The functional neuroimaging method of LORETA is based on scalp-recorded EEG (Anderer et al., 2004), so it is logical that the temporal and spatial resolutions of measurements, and respective visual presentations, of these approaches will be very similar to those reported by EEG.

Magnetoencephalography (MEG). The brain is regarded as the "computer" that executes and commands various activities within the body. According to Cacioppo (2004), the cerebral cortex is a covering of about 2.6 to 16 billion neurons, with each neuron receiving 10,000 to 100,000 postsynapses in its dendrites. Parra, Kalitzin, and Lopes da Silva (2004) explained that these neurons produce the electrical and magnetic fields in the brain.

This magnetic activity can be measured by a neurophysiological technique known as MEG, which provides a passive noninvasive way to measure the magnetic fields generated by the brain (Brier, Maher, Schmadeke, Hasan, & Papanicolaou, 2007; Hari, Leväne, & Raij, 2000; Mantini, Franciotti, Romani, & Pizzella, 2008; Rotman Research Institute, n.d.). It was first measured by David Cohen in 1968, using a one-sensor magnetometer (Mantini et al., 2008; Parra et al., 2004; Reite et al., 1999). The first study conducted with MEG involved individuals with epilepsy. In 1993, whole scalp MEG was introduced, which greatly increased the reliability of the results of studies due to much shorter required recording time (Mäkelä et al., 2006).

Principle of MEG. In order to know how neurons create magnetic activity, it is important to have an understanding of *Faraday's law*. In 1820, Hans Oersted discovered that a current-carrying wire is capable of producing magnetic fields (Bueche, 1988; Otsubo & Snead, 2001). This implied that the motion of electrical impulses produces both electric potential and magnetic fields (Knowlton & Shih, 2004). The strength of these magnetic fields is greatest close to the current-carrying wire. Oersted postulated the *right-hand rule*, to make it easy to remember the direction of these magnetic fields (Bueche, 1988; Otsubo & Snead, 2001). According to this rule, if the thumb represents the direction of the current, then the finger curls determines the direction of the magnetic fields (Bueche, 1988; Müller & Kassobek, 2007; Reite et al., 1999). Additionally, the current-carrying wire which produces a magnetic field experiences a force due to the field known as *electromotive force* (EMF). In Faraday's law, the electromotive force in a closed circuit is equal to the time rate of change of the magnetic flux and the number of loops in a coil (Bueche, 1988). MEG measures the electromotive force generated by numerous neurons.

Construct of MEG. From Faraday's law, it is evident that any electrical current will produce an orthogonally-oriented magnetic field. Most often it is the layer of pyramidal cells in the cortex, which are generally perpendicular to its surface that gives rise to measurable magnetic fields (Müller & Kassobek, 2007). These magnetic fields induce current in the detection coil (which covers different regions of the brain) on the surface of the brain (Stern & Silbersweig, 2001). To register and measure these small signals, the detection coils are coupled to a superconducting device in a magnetically shielded room. This is known as the superconducting quantum interference device

(SQUID; Parra et al., 2004). These super conductors are essential for MEG recordings, because the tiny electrical currents generated by these magnetic fields would be lost in the energy required to overcome the impedances of the recording coil wire. SQUID removes these impedances, amplifies, and records the conduction changes caused by these minuscule magnetic fields (Knowlton & Shih, 2004). To maintain the superconducting properties of the SQUIDs, they are immersed in a dewar (a large insulated, helmet-like vessel) which contains liquid helium. The dewar is maintained at a very low temperature (-269°C), to keep the MEG equipment cool (Otsubo & Snead, 2001; Parra et al., 2004).

Functioning of MEG. A brief description of MEG functioning is essential to understand how neuronal activity is measured. The MEG device is described as a "very large hair dryer" and the bottom of this device is shaped like the inside of a helmet. A person sits on an adjustable hydraulic chair and his or her head is positioned inside the helmet (Rotman Research Institute, n.d.).

To measure the magnetic fields, an individual is presented with various stimuli. Any kind of stimuli (e.g., visual, auditory, or somatosensory) received by the sensory receptors will evoke neuronal activity, resulting in the production of electric currents and magnetic fields. Repeated presentation of a given stimulus produces the same electric and magnetic fields (Leon-Carrion, McManis, Castillo, & Papanicolaou, 2006). This magnetic energy then reaches the brain surface, where MEG captures these magnetic signals through the SQUID and then reconstructs the distribution of these fields along the head surface (Simos et al., 2006). When these fields are recorded on the head surface and averaged, the result is a series of event-related potentials (ERPs) and event-related fields

(ERFs). Additionally, ERFs are more readily distributed over the brain surface than ERPs, resulting in a mathematical estimation of the location and degree of activation of brain cells. These estimates are then superimposed on participants' MRIs to identify the brain structures (Leon-Carrion et al., 2006).

Hence, in this manner MEG signals represent the electrophysiological activity primarily produced in the cerebral cortex, where pyramidal cells are oriented parallel to the skull surface (Otsubo & Snead, 2001). These weak magnetic fields are measured in picoteslas (10^{-12}). Brain changes that occur as a result of reactions to events are measured in femoteslas (1^{-15}; Andreassi, 2000; Knowlton & Shih, 2004).

Empirical evidence. The main focus of this section is to describe how MEG has been used with individuals who have PTSD, although literature on measurement of PTSD with MEG is very limited. Preliminary evidence indicates that MEG has been used for studying a variety of psychological disorders, such as major depressive disorder (Fernández et al., 2005), epilepsy (Parra et al., 2004), aphasia (Breier et al., 2007), reading and language tasks (Simos et al., 2006), schizophrenia/psychoses (Reite et al., 1999), and autism (Rippon, Brock, Brown, & Boucher, 2007). In spite of limitations of the literature in this area, some of the studies in which MEG has been used to measure the cortical activity associated with psychological disorders that resemble PTSD are discussed below.

Kähkönen et al. (2007) studied auditory processing in 13 participants with unipolar major depressive disorder (MDD) and compared this with the auditory processing of 12 healthy subjects. This study involved both MEG and EEG devices. At baseline, participants watched a silent movie and ignored the tones produced through

plastic tubes and earphones. Later on they were presented with pure tones monaurally. Results using a MEG device with 306-channels indicated that participants with MDD had dysfunctions in auditory processing, which may be mediated through frontal-temporal neural circuits. This dysfunction indicates that reduced inhibitions and increased excitations of cortical neurons are responsible for regulating involuntary attention.

Leon-Carrion et al. (2006) examined the time course of brain activation in response to emotionally-evocative pictures. It was hypothesized that MEG would be able to localize brain activity during perception and processing of affective stimuli, and that brain areas would vary across time as the stimuli were perceived and processed. Participants were 10 students from the University of Texas Medical School. They were randomly subjected to three stimulus conditions (negative, positive, and neutral) for a time period of one second, with an interstimulus interval of 35 seconds. Stimulus conditions consisted of 60 pleasant, unpleasant, and neutral images. The results demonstrated that irrespective of the type of stimuli there was activity in the occipital cortex associated with perceptions, and in the inferior temporal gyrus associated with recognition. Next, activity occurred in the mesial temporal lobes associated with emotions. The researchers found that all participants showed activation in the left hemisphere, in response to unpleasant emotions. Mesial temporal lobe activation occurred either prior to, or parallel to, frontal lobe activation for the interpretation of incoming stimuli. This study revealed that MEG was able to identify brain structures associated with emotions.

Ray et al. (2006) conducted a study with 23 participants to examine psychological dissociation, and map cortical areas associated with such states. These participants were

imprisoned and had fled from Turkey to Germany. They reported experiencing various types of physical and psychological torture, and therefore met the PTSD criterion for dissociative symptoms. The control group consisted of 16 healthy university students with the same ethnic backgrounds as the participants who were tortured. For the participants who had been tortured, the MEG device (148 channels) showed slow delta wave (-1.5Hz to 4Hz) activity in the left ventrolateral frontal cortex. This dysfunction is produced by both structural and functional neural networks that are cut off from input.

From the above studies (Kähkönen et al., 2007; Leon-Carrion et al., 2006; Ray et al., 2006), it appears that MEG was able to provide details of brain activity associated with auditory processing, emotions, and psychological dissociation. Such a device works well for this fast-paced world, as it enables clinicians to identify brain abnormality in processing and administer treatment accordingly. These MEG studies involved participants of different ages, which also suggest that this instrument can be used effectively across most age groups.

Advantages of MEG. There are striking differences between MEG and other brain imaging techniques. To clarify some of these differences, advantages of MEG over other techniques are discussed, specifically compared to EEG.

Electrodes. MEG does not require a reference electrode. It therefore provides better estimates of scalp activity in studies during which rhythmic synchronization is being investigated (Parra et al., 2004). In addition, the SQUIDs used in MEG are superior to the electrodes used in EEG (Andreassi, 2000). The highly sensitive SQUID sensors are essential for reducing the effects of other magnetic fields (Müller & Kassobek, 2007). SQUID technology enables MEG to identify dipole patterns of focally-generated

neural activity, and simultaneously measure parallel processes (e.g., auditory processing) in the brain (Andreassi, 2000; Kähkönen et al., 2007; Ray et al., 2006). In contrast to EEG, these fixed sensors or detectors are not attached to the scalp surface, which aids in conveniently scanning the magnetic field patterns and contributes to the accuracy of results (Andreassi, 2000; Müller & Kassobek, 2007).

Instrumentation. MEG is a noninvasive technique so repeated measurements can be performed with participants (Hari et al., 2000; Mäkelä et al., 2006). MEG has very low risks and has no known short-term or long-term side effects (Rotman Research Institute, n.d.).

Recording and analysis. Brain imaging techniques such as PET and fMRI are used to assess neurophysiological activity seconds after a task has been performed. MEG differs from these techniques, because it directly measures neuropsychological activity in the form of magnetic flux as the task is being performed (Hari et al., 2000; Reite et al., 1999; Simos et al., 2006).

MEG measurements reflect magnetic activity generated by intracellular currents, whereas EEG corresponds to activity generated by extracellular currents (Baumgartner & Pataraia, 2006; Knowlton & Shih, 2004; Otsubo & Snead, 2001; Reite et al., 1999). This is because the current density is highest intraneuronally, producing magnetic fields that exit the head. The currents produced by extraneuronal cells are equal and widely distributed. Therefore, the net magnetic fields produced by these cells are likely to be cancelled or close intracerebrally and thus not seen outside the head (Reite et al., 1999). This suggests that EEG and MEG have different levels of sensitivity to the geometry, orientation, and configuration of stimulation (Parra et al., 2004). Furthermore, MEG

measures only a subset of neuronal activity that is tangential to the scalp surface, which increases the accuracy of source localization (Baumgartner & Pataraia, 2006; Hari et al., 2000; Knowlton & Shih, 2004; Mäkelä et al., 2006; Otsubo & Snead, 2001).

Interpretation of MEG signals requires one model of the brain; however, EEG calculations require multicompartmental models with known conductivities and shapes for the brain, skull, cerebrospinal fluid, and scalp (Parra et al., 2004). Additionally, the signals of the magnetic fields are less affected by the tissues lying between the source and the sensing device, which increases the accuracy of source localization (Andreassi, 2000; Knowlton & Shih, 2004).

Spatial and temporal resolutions. MEG also provides high spatial density of recording points, which are difficult to obtain with EEG (Parra et al., 2004). MEG does an excellent job of localizing brain activity in cortical areas, by tracing wave activities that originate in one area to processing in other areas (Knowlton & Shih, 2004). Once these magnetic fields are generated, they are less distorted by the resistive properties of the skull and the scalp, resulting in better spatial resolution (Baumgartner & Pataraia, 2006; Hari et al., 2000; Parra et al., 2004; Reite et al., 1999). There is less distortion of these magnetic fields, which are less susceptible to error because they traverse media such as tissues and fluids (Kähkönen et al., 2007; Reite et al., 1999). In addition, the skull serves as a low-pass filter for electrical potentials, which provides better conditions for recording fast brain activities. Yet another advantage of MEG over EEG is that, MEG records cortical activation at higher and lower frequency ranges, making it more useful for clinical research (Parra et al., 2004).

MEG has excellent temporal resolution and is capable of tracing neuronal activity in the range of milliseconds (Hari et al., 2000; Parra et al., 2004; Simos et al., 2006; Stern & Silbersweig, 2001). For this reason, MEG provides adequate information on both *where* and *when* brain structures are activated (Simos et al., 2006).

Limitations of MEG. Like any other technique, MEG is not exempt from limitations. It is important to consider any limitations of MEG which might render this technology inappropriate for the present study (or for similar future research). Limitations of MEG are discussed below, as follows.

Instrumentation. The magnetic fields detected by MEG are weak in nature. For this reason, the likelihood of possible interferences from much stronger magnetic fields (e.g., earth's magnetic field, fluorescent lights, or nearby hospital equipment such as MRIs) within the surrounding area is greatly increased. A magnetically-shielded room helps eliminate these interfering magnetic fields (Hari et al., 2000; Knowlton & Shih, 2004; Reite et al., 1999).

Since MEG is an expensive technology, the likelihood of performing long recordings similar to those carried out using EEG is not feasible (Baumgartner & Pataraia, 2006; Mäkelä et al., 2006; Parra et al., 2004). The high cost of MEG and the complexities associated with operations are hindrances to its acceptance in clinical practice (Parra et al., 2004; Reite et al., 1999).

Recording and analysis. MEG recordings are more prone to interference caused by noise than EEG (Andreassi, 2000). These noises are controlled for by placing two coils at a distance, so that magnetic fields at one end are larger than those at the other, and differences can be magnified (Knowlton & Shih, 2004). Also, during MEG

recordings, participants are required to keep their heads immobile. This limits the studies that can be performed with children. If a participant has a seizure in the MEG device, there may be injuries due to collision with the dewar (Hari et al., 2000; Parra et al., 2004).

Additionally, MEG measures neuronal activity that is tangential to the scalp surface, which means that deep and radial sources are neglected. MEG analyses are based on source modeling, which is very demanding and difficult for researchers unfamiliar with this approach (Hari et al., 2000). MEG signals can only be interpreted in the context of specific models (Reite et al., 1999).

Source localization. MEG does not provide adequate information on three dimensional (3D) distribution of electrical activity (Pascual-Marqui, Esslen, Kochi, & Lehman, 2002). Thus, MEG's capability to localize an event is quite confined (Rippon et al., 2007; Stern & Silbersweig, 2001). Additionally, spatial localization is based on a mathematical model that provides information regarding location, orientation, and strength of neuronal currents that are conducted between the brain and the skull. This leads to the *inverse problem* in MEG (Stern & Silbersweig, 2001).

In the absence of constraints, a magnetic field pattern can be produced from potentially infinite numbers of sources, resulting in interference with brain scan results (Otsubo & Snead, 2001; Parra et al., 2004; Rippon et al., 2007). For this reason, an assumption is made that the magnetic fields are generated by an equivalent current dipole (ECD; Mäkelä et al., 2006; Otsubo & Snead, 2001; Parra et al., 2004). The ECD construct is central to the principle of source localization (Reite et al., 1999). High-powered computational methods and improved source localizations of multichanneled MEG have been helpful in solving this problem (Rippon et al., 2007).

Artifacts. Disturbances caused by cardiac signals, eye movements, and muscle movements affect MEG acquisitions (Mantini et al., 2008). These disturbances can be a serious problem, in light of OEI interventions that often involve eye movements. Even if OEI techniques introduces minor muscle movements, however, Mantini, Franciotti, Romani, and Pizzella (2008) have developed an algorithm known as *independent component analysis* (ICA) shown to be effective for removing muscle artifacts and noise effects in MEG recordings. MEG signals are also sensitive to artifacts produced by moving objects such as dental material (Mäkelä et al., 2006).

Finally, experience with MEG in the clinic is still limited, due to its shorter history than EEG. Knowledge acquired from EEG is shaping the way MEG is currently used (Parra et al., 2004); however, from the above discussions it appears that MEG is not yet a suitable technique for this study.

Electroencephalography (EEG). Another brain mapping technique that performs neurophysiolgical measurement of brain electrical activity is called Electroencephalography (EEG; Müller & Kassobek, 2007). In order to grasp how electrical activity is measured by EEG, it is important to understand the process by which the brain produces these electric fields. According to Kalat (2004), the brain is composed of billions of neurons. The major components of a neuron are dendrites or branching fibers, a soma or nucleus, axons, and presynaptic terminals. These neurons produce small bursts of electricity resulting in weak electric fields, through a mechanism known as *action potential*.

Action potentials occur in axons and are propagated due to changes in the permeability of neuron membranes. Selective permeability of the membrane allows

important ions (e.g., sodium, potassium, chloride) to pass through the gates. A mechanism known as the *sodium-potassium pump* continuously transports three sodium ions outside the cell and draws two potassium ions into the cell (Kalat, 2004). In the resting state, very few sodium ions cross the membrane of a neuron except by means of the sodium-potassium pump. Potassium tends to flow into the cell in response to the electrical gradient and flows out of the cell due to the concentration gradient; however, when a neuron is stimulated, the gates open, sodium ions begins to flow freely and the membrane becomes slightly depolarized. When the potential across the membrane reaches a threshold, sodium ions rush into the neuron explosively, until the electrical potential of the membrane passes beyond zero to a reversed polarity. This increases sodium ion concentration by less than 1%. After the process is well in progress, potassium ions flow out of the axon, due to their excessive concentration inside the neuron, resulting in hyperpolarization. This process is known as the action potential. Clearly, it is this flow of ions from one side to other that produces weak electric fields. At the end of this process, the membrane returns to its original state (Kalat, 2004).

Construct and functioning. As mentioned by Müller & Kassobek (2007), an electroencephalograph is a device that measures electrical activity of the brain over time. EEG recordings can be obtained from the scalp surface and from brain tissues (Stern, Ray, & Davis, 1980). Participants wear a plastic or spandex cap and an investigator attaches or positions electrodes at various locations on the cap with an adhesive gel (Kalat, 2004; Müller & Kassobek, 2007). Electrode placement is determined by measuring and marking the scalp using a reliable and reproducible system (Müller & Kassobek, 2007). One such system arrangement is known as the *International 10-20 system* (various scalp

locations are either 10% or 20% of the distance between standard points used for measurement; Andreassi, 2000). Participants are then presented repeatedly with stimuli. These electrodes record, millisecond-by-millisecond, ERPs or overall electrical activity of neurons that are responding to various stimuli. As a result of repeated stimulation of the same cells, recordings are averaged to represent exact temporal resolution of brain activity (Posner & Raichle, 1999). The outputs from these electrodes are then amplified and recorded. Artifacts (e.g., eye movements, eye blinks) are removed from the raw EEG data. This artifact free data then is subjected to computer assisted imaging and statistical analysis using various softwares programs (e.g., WinEEG, Neuroguide database, Human Brain Index, Novatech). This software provides users with the amplitudes and frequency ranges of different brain waves.

It is important to note that the electrodes do not record the activity of one neuron, but an average of the whole population of cells in the area under the electrode (Kalat, 2004). In reality, an EEG is not a measurement of the electric current, but of the voltage difference between different parts of the brain. Resulting traces represent electrical signals from a large number of neurons (Müller & Kassobek, 2007). In this manner, brain abnormalities can be detected by EEGs (Kalat, 2004).

Types of waveforms. The output obtained from EEG recordings is in waveforms. Various waveforms are produced by EEGs. A brief description of these and the behaviour patterns associated with them are outlined below.

Andreassi (2000) stated that alpha waveforms consist of regular rhythmic oscillations of 8-12 Hertz (Hz = cycles per second). These waves are related to relaxed wakefulness and exhibit maximum amplitude over the occipital regions (Jokić-Begić &

Begić, 2003). These are disrupted with any type of mental activity. Waves are found over a wide scalp area, but rhythmic oscillations vary across the scalp. The waves produced are larger and slower ones (Andreassi, 2000). Increased alpha activity over the occipital regions is found in people with PTSD (Jokić-Begić & Begić, 2003).

Beta rhythms are irregular waveforms at higher frequencies (18-30 Hz) and lower amplitudes, which are observed when individuals are in vigilant states. These are smaller and faster waveforms, associated with emotional and cognitive processes (Andreassi, 2000; Jokić-Begić & Begić, 2003). People with PTSD have increased beta activity and maximum amplitudes over the frontal and central areas (Jokić-Begić & Begić, 2003).

Gamma waves oscillate with a frequency range of 30-50 Hz and are produced in response to sensory stimuli such as auditory clicks, or flashes of light. Delta waves are very low in frequency (0.5-4 Hz), but often high in amplitude. They occur when individuals are in states of deep sleep. If they occur in people who are awake, it could indicate some kind of brain abnormality, depending on where on the scalp the measurements are taken (Andreassi, 2000).

Theta waves are less common and have frequency ranges from 5 to 7 Hz. These waves are produced more in children, (e.g., in the pleasurable moments of babies; Andreassi, 2000). They are associated with drowsiness, daydreaming, and subjective derealisation (Begić et al., 2001).

Empirical evidence. There are a limited number of studies in which EEG has been used to research PTSD (Begić et al., 2001; Jokić-Begić & Begić, 2003); however, research using EEG to assess brain asymmetries and emotions is one of the most promising applications (Cacioppo, 2004). Metzger et al. (2004) studied relationships

between measures of PTSD symptoms and EEG alpha symmetry in female Vietnam War nurse veterans (N = 50) with and without PTSD. EEG results indicated that PTSD arousal symptoms and depression were associated with relatively greater right-sided parietal asymmetry.

Jokić-Begić and Begić (2003) studied 116 participants. They examined EEG patterns in combat veterans with PTSD (n = 79) and without PTSD (n = 37). Results indicated that combat veterans with PTSD exhibited decreased alpha and increased beta activity in the frontal, central, and occipital regions. Alpha and beta rhythms in these areas are potential markers of changes in EEG activity due to (or associated with) PTSD.

Another study was conducted by Begić, Hotujac, and Jokić-Begić (2001) to investigate differences in EEG patterns between veterans with PTSD (n = 18) and healthy non-veterans (n = 20). Results indicated that there was a marked increase in EEG theta activity in the central region. There were also increases in beta activity over frontal, central, and left occipital regions. No differences were found between the two groups in delta and alpha frequency ranges. These researchers explained that increases in theta activity could be due to changes in anatomical structures (e.g., amygdala, hippocampus) associated with PTSD. They suggested that increases in beta activity might be due to cortical hyperexcitability, prolonged wakefulness, attention disturbances, emotional activation, or restlessness (Begić, et al., 2001; Jokić-Begić & Begić, 2003).

Through EEG analyses, other researchers have also found greater right frontal cortical activity associated with the experiencing and expression of negative, withdrawal-related emotions, behaviours, and motivations, which are key symptoms of PTSD (Cacioppo, 2004; Davidson, 1988; Rabe, Zoellner, Beauducel, Maercker, & Karl, 2008).

Davidson (1988) suggested that frontal asymmetries are associated with affective experiences, while parietal asymmetries are more affected by cognitive demands.

EEG findings from Williams (2006) showed no significant changes in frontal or parietal regions from pretreatment to posttreatment or 3-month follow up among women with PTSD who had been sexually assaulted. One rationale for this lack of change in qEEG findings in response to three different treatments is that only small treatment doses (three 1-hour sessions of active therapies and 2-hours of psychoeducation) were provided.

In most of these studies (Begić et al., 2001; Cacioppo, 2004; Davidson, 1988; Jokić-Begić & Begić, 2003; Metzger et al., 2004; Rabe et al, 2008; Williams, 2006) EEG research findings were associated with frontal asymmetries and emotions. From the above studies, it also appears that EEG asymmetries are markers of underlying neural processes (Cacioppo, 2004). Additionally, Begić et al. (2001) mentioned that such electrophysiological techniques are not frequently used in studies of PTSD, and results obtained from them are sometimes contradictory. In veterans with PTSD, for example, Begić et al. (2001) did not find any unusual levels of alpha activity, while Jokić-Begić and Begić (2003) found decreased alpha activity. This implies that continued efforts are needed to identify neural processes underlying EEG asymmetries in emotionally-evocative situations (Cacioppo, 2004).

Advantages of EEG. Over the past few decades, EEG has been widely used in experimental psychology, and therefore is a well established technique (Parra et al., 2004; Rösler, 2005). In the present study, EEG recordings of the participant were taken. To justify the use of EEG for this study, it is essential to review the advantages of this technology.

Instrumentation. EEG has been employed for detecting brain activity in various motor and mental activities such as sleep, attention, intelligence, and emotional expression (Andreassi, 2000; Davidson, 1988). In comparison to other brain imaging techniques, EEG is quite inexpensive (Davidson, 1988; Rabe et al., 2008; Rösler, 2005). EEG involves a noninvasive procedure, so it can be used without physician assistance. Long-term and repeated measurements of EEG have been found to be harmless to participants (Davidson, 1988; Rösler, 2005; Müller & Kassobek, 2007).

Recording. EEG recordings do not require special constraints on task presentations, therefore intense emotions can be assessed (Davidson, 1988). Radial cells (cells that have long axes and are radial to the brain surface) are best detected by EEG, because maximum electrical positivity or negativity is directed to the electrodes (Reite et al., 1999).

Temporal resolution. EEG has a high temporal resolution, and therefore is able to measure changes in milliseconds in amplitude, frequency, and latency of brain electrical activity (Davidson, 1988; Müller & Kassobek, 2007).

Limitations of EEG. Like other brain imaging techniques, EEG is also not exempt from limitations. Some of the important limitations of EEG are outlined below.

Electrodes. In EEG, the number of electrodes placed and their location depends on the purpose of recordings. To ensure consistent potential, large numbers of electrodes are used to attain a thorough sample of electrical activity; however, the use of numerous electrodes can cause uneasiness for participants. Instability of these electrodes against the scalp creates artifacts resulting in fluctuations in voltage and obscuring EEG wave forms. The process of attaching electrodes to the scalp requires skill, and mishandling

can interfere with waveforms and effect EEG results (Müller & Kassobek, 2007; Stern et al., 1980).

Recording and analysis. According to Stern, Ray, and Davis (1980), waveforms produced are minuscule, resulting in electrical hindrances and interferences at every stage of instrumentation with EEG. Electrical potentials measured by EEG are attenuated or distorted in strength, and spatially blurred, because the electrical activity being recorded has to pass through the cerebrospinal fluid, skull bone, and skin of the scalp, before it reaches the electrode's surface (Andreassi, 2000; Knowlton & Shih, 2004). Furthermore, the recorder which amplifies waveforms must be capable of amplifying frequencies from less than 1 Hz to 100 Hz, which is technically difficult. Some qEEG devices are unable to register frequencies higher than 50 Hz, so waveforms at higher frequencies are attenuated (Stern et al., 1980). Additionally, amplitudes of waves measured by EEG recordings are reduced. Cells with tangential orientations cannot be detected by EEG, because their planes of maximum potential lie at right angles to the electrodes (Reite et al., 1999).

Analysis of these waveforms is also a difficult task. EEG calculations require multicompartmental models with known conductivities and shapes for the brain, skull, cerebrospinal fluid, and scalp. Inaccuracy in estimating these conductivities affects interpretation of the electrical sources (Parra et al., 2004). EEG measurement results in enormous volumes of amplified waves. In the process of characterizing waves, some waves remain unquantified and unidentified, due to the complex nature of numerical analyses (Stern et al, 1980).

Artifacts. In EEG, readings from the frontal and prefrontal scalp areas are susceptible to physiological interferences (e.g., eye blinks, eye movements, head movements, jaw clenching, and frowns), which constitute muscle artifacts (Andreassi, 2000; Davidson, 1988; Stern et al., 1980). Some eye blink artifacts can be easily removed posthoc, but those associated with slower eye movements are difficult to trace, and they particularly affect delta and theta rhythms. These slower eye movements can be directly examined with concurrent electrooculographic (EOG) recordings (Davidson, 1988). EOG involves the use of bipolar electrodes placed above and below the left eye, to monitor eye movement artifacts (Jokić-Begić & Begić, 2003).

According to Davidson (1988), other drawbacks of EEG include muscle artifacts, which are found in the beta frequency band width. For this reason, it is vital to examine beta asymmetries with extreme caution. Energy associated with muscles is generally registered at frequencies above standard EEG waveforms. Muscle artifacts cannot be easily filtered, and therefore intrude into frequency band widths. These interferences are most likely to be encountered in studies that involve movements, such as facial expressions pertaining to emotions. Such facial muscle asymmetries increase the likelihood of asymmetries in muscle artifacts, contributing to biases in the beta frequency range of EEG data (Davidson, 1988). In the present study, Switching (alternate covering and uncovering of eyes) involved the use of hand muscles, which would normally raise the likelihood of muscle artifact in EEG findings; however, the use of an eye patch can reduce the likelihood of such contamination of data.

Spatial resolution. According to Pascal-Marqui, Esslen, Kochi, and Lehmann (2002), EEG measurements are generated by the cortical pyramidal neurons undergoing

postsynaptic potentials. EEG has limited spatial resolution, which means that this technology has limited ability to map brain electrical activity (Rabe et al., 2008; Rösler, 2005). EEG also does not provide sufficient information on the three-dimensional distribution of neuronal electrical activity (Pascual-Marqui et al., 2002).

Techniques employed. Davidson (1988) explained that EEG recordings involve measurement of potential differences between two electrodes. EEG recordings can be done using either monopolar or bipolar techniques. Monopolar technique involves the use of a reference electrode (placed in some inactive area, e.g., the earlobe or tip of the nose) and an active electrode (placed in a cortical area of interest). The rationale for linking these two sites together is to provide a common reference for the electrodes on both sides of the scalp. Some researchers argue that practically none of these areas can be called 'inactive' (Andreassi, 2000; Davidson, 1988). Bipolar technique involves placing two active electrodes over cortical areas of interest. The difference or algebraic sum of the electrical potentials beneath the two regions is recorded (Andreassi, 2000).

Low resolution brain electromagnetic tomography (LORETA). From the above discussions of MEG and qEEG approaches to neuroimaging, it can be inferred that neither of these techniques are sufficient in their current form to measure all possible brain changes. This insufficiency suggests that it can be valuable to extend existing approaches to EEG imaging with a validated source localization algorithm known as low resolution brain electromagnetic tomography (LORETA). LORETA makes it possible to localize the electrical sources in brain regions (Korb, Cook, Hunter, & Leuchter, 2008). In light of the discussion regarding the various frequency bands associated with EEG, it is evident that these bands reflect different functions and behave statistically

independently (Pizzagalli et al, 2002). For this reason, a tomographic source location method (LORETA) was used to determine the underlying neuronal sources of EEG frequency bands associated with PTSD symptoms.

LORETA was first presented by Roberto Pascual-Marqui in 1994 (Anderer et al., 2004). This neuroimaging strategy identifies brain areas that contribute to electrical fields recorded on the scalp (Cannon, Lubar, & Baldwin, 2008; Mientus et al., 2002; Pascual-Marqui et al., 2002). Electrical activity in the cortices is computed and mapped onto a dense grid array, resulting in a low error solution for source generators. Thus, LORETA provides sufficient 3D information regarding electrical neural activity that synchronizes the strength between neighboring neurons (Pascual-Marqui et al., 2002).

LORETA is based on two assumptions: (a) the smoothest of all possible source distribution is the most plausible one (Anderer et al., 2004; Frei et al., 2001), and (b) the neighboring neurons are activated synchronously and simultaneously in terms of orientation and strength (Clemens et al., 2008; Mientus et al., 2002; Pascual-Marqui, Michel, & Lehmann, 1994; Pizzagalli et al., 2002).

Analysis principle of LORETA. LORETA employs a three-shell spherical (or head) model which includes the scalp, skull, and brain, and is based on the digitized Talairach atlas (Talairach & Tournoux, 1988), available from the Brain Imaging Center at the Montreal Neurological Institute (Clemens et al., 2008; Grave de Peralta Menedez & Gonzalez Andino, 2000; Lubar, Congedo, & Askew, 2003). In LORETA, the brain component or solution space is restricted to cortical gray matter and the hippocampi using the digitized probablitly atlas of the Montreal Neurological Institute. The gray matter portion of the model is divided into an arbitrarily chosen frequency band on a

dense grid of 2394 voxels, which permits a spatial solution of 7mm (Clemens et al., 2008; Lubar et al., 2003; Pascual-Marqui et al., 2002). At each voxel, LORETA computes the current density (unit = amperes per square meter, Å/m²) as the linear weighted sum of the scalp electric potentials. In this manner, LORETA analyses allow a 3D tomography of the brain's electrical activity (Clemens et al., 2008; Pizzagalli et al., 2002).

In summary, LORETA computes the distributed electrical activity within the cererbral volume and produces a low error solution for source generators (Cannon et al., 2008; Clemens et al., 2008). "LORETA images represent either the electrical activity at each voxel as squared magnitude of the computed current density or values of voxel-by-voxel statistics of brain regional electrical activity" (Mientus et al., 2002, p. 99).

Empirical evidence. There is empirical evidence of LORETA use in assessment of various physiological conditions; for example, LORETA revealed activation in language areas, auditory cortices, motor cortices, and visual cortices (Pascual-Marqui et al., 2002), associated with epilepsy (Clemens et al., 2008), schizophrenia (Mientus et al., 2002), depression (Korb et al., 2008; Lubar et al., 2003) and in the determination of drug effects on different brain areas (Frei et al., 2001).

A study involved 28 participants who were recovering substance abusers (RSA) and 28 nonclinical controls. The purpose of the study was to demonstrate that during a Self-Perception and Experiential Schemata Assessment (SPESA), neurophysiological patterns demonstrated by the two groups (RSA and the control) would differ. The data showed that there were, indeed, significant differences between the two groups during the both the assessment condition and baseline. sLORETA analyses revealed that the RSA group exhibited a pattern of alpha activity in the right amygdala, hippocampus, Broca's

area, uncus, insular cortex, and orbitofrontal regions during SPESA condition. In other words, standardized LORETA was able to reflect possible neural pathways related to negative self-perceptions in participants who were reovering from addiction (Cannon et al., 2008).

Pizzagalli et al. (2002) studied brain electrical activity in 38 participants with Major Depressive Disorder (MDD) and compared them with 18 healthy controls. LORETA was employed to identify the sources of EEG frequency bandwidths. The researchers found that individuals in the MDD group demonstrated increased activation of beta rhythms in the right inferior and superior regions, and hypoactivation in the posterior cingulated and precuneus. Another study was conducted by Korb, Cook, Hunter, and Leuchter (2008) to examine abnormal brain functions in MDD. Through LORETA findings, these researchers found high current density in delta, theta, beta, and alpha bands in the anterior cingulate cortex, dorsolateral prefrontal cortices, medial prefrontal cortices, and orbitofrontal cortices in the MDD group.

In comparison to 20 healthy controls, LORETA findings in 19 unmedicated participants who had schizophrenia revealed an increase of delta activity in the anterior cingulate gyrus and temporal lobe. In addition, 19 participants who had schizotypal personality disorder and 30 unmedicated participants who had major depression, showed decreased beta, theta, and delta activity in the anterior cingulate and decreased alpha activity in the temporal lobe (Mientus et al., 2002). In another study, Cannon, Lubar, Thornton, Wilson, and Congedo (2004) used LORETA to demonstrate activation of the limbic system during emotional memory processing (e.g., anger). A total of 12 students from the University of Tennessee participated in this study. Participants were subjected

to two (baseline and anger) conditions. Eyes-open baseline condition was obtained using EEG. In the anger condition, participants were asked to access a memory that created intense anger and to retain that state as long as possible. The researchers found that the anger condition in these participants resulted in more beta frequency activity in the limbic structures of the right hemisphere. This finding indicates that changes in the limbic structures can be recorded and examined using LORETA.

From the above studies (Cannon et al., 2004; Cannon et al., 2008; Korb et al., 2008; Mientus et al., 2002; Pizzagalli et al., 2002), it is evident that LORETA analyses facilitate the identification of the cortical areas that contribute to the scalp's electrical fields. LORETA results also revealed brain changes in the limbic system. This capability makes LORETA an acceptable algorithm for the present study. It is noteworthy, however, that the present author has been unable to identify brain activity of PTSD subjects using LORETA. Consequently, the significance of this pilot study as an initial attempt to assess traumatized individuals is all the more evident.

Advantages of LORETA. Both EEG and LORETA were used in the present study. For that reason, it is important to describe some advantages of this algorithm.

Instrumentation. LORETA is a noninvasive method based on scalp-recorded EEG (Anderer et al., 2004). This neuroimaging technique offers a 3D, distributed, linear, and discrete solution to the inverse problem of EEG and MEG by computing the cortical localization of neuronal activity from the scalp distribution of the electrical fields (Clemens et al., 2008). This solution is based on the assumption that the smoothest of all possible activity distributions is the most plausible one. This assumption is supported by

electrophysiology, where neighbouring neuronal populations show highly correlated activity (Anderer et al., 2004; Frei et al., 2001).

Recording and analysis. LORETA correctly localizes deep sources of brain electromagnetic activity via functional mapping (Pascual-Marqui et al., 2002). This reconstruction is independent of the reference electrodes used in EEG recordings, resulting in greater uniformity of results across laboratories (Lubar et al., 2003). Additionally, LORETA takes into account fewer assumptions, which make its interpretation easier than other techniques (Connemann et al., 2005). Such functional mapping provides high temporal resolution for brain electrical data (Frei et al., 2001). As a result, LORETA has been used extensively in electrophysiological research (Lubar et al., 2003).

Limitations of LORETA. LORETA has both advantages and limitations. Some of the main limitations of LORETA are explained below.

Instrumentation. The human head model in LORETA is based on EEG (Anderer et al., 2004). It implies that, like EEG, LORETA is highly subjected to muscle artifacts. According to Kincses (as cited in Pascual-Marqui et al., 2002), the electrophysiological and neuroanatomical constraints of LORETA are arbitrary and have no physiological meaning.

Spatial resolution. Despite its correct localization, LORETA gives a blurred (or low resolution) image (Anderer et al., 2004; Frei et al., 2001; Pascual-Marqui et al., 2002). This blurriness is a direct consequence of the smoothness constraints, resulting in unique and optimal 3D distributions of the brain's electrical activity (Pascual-Marqui et al., 1994). The minimum Laplacian approach and the grid spacing of solution space

results in low spatial resolution for LORETA, in comparison to other imaging modalities. Another concern is that LORETA does not include deeper brain areas that might be contributing to surface electrical fields (Connemann et al., 2005).

In spite of these limitations of EEG and LORETA, these methods were used in the present study. In practical terms, a low resolution tomography of the electrical activity at every moment in time still provides the advantage of high temporal resolution for the resulting electrical recordings (Pascual-Marqui et al., 1994).

Transference in OEI

In this study, OEI treatment was provided to a participant who exhibited negative transference reactions. The following definitions are provided to assist in understanding the construct and nature of these transference reactions in therapeutic relationship.

Background: Transference in psychoanalysis. Transference was one of the greatest discoveries of psychoanalysis by Sigmund Freud. He showed that "transference is an unconscious displacement of early images - especially parental ones - onto the current figures" (Rawn, 1987, p. 108). These displacements include feelings, fears, wishes, unconscious attitudes, conflicts or fantasies. Transference involves replication or repetition of aspects of a past experience in a distorted form with a new object (Rawn, 1987; Shevrin, Bond, Brakel, Hertel, & Williams, 1996).

Transference and trauma. With respect to trauma, transference projections become even more complex (Fischer, 2005). According to Gaensbauer and Jordan (2009), trauma experienced in early childhood leaves indelible, conscious, and unconscious memory imprints. Usually, unconscious memories are triggered in the context of emotional themes or environmental stimuli associated with traumas. Further,

manifestation of these unconscious memories occurs in various forms, including somatic sensations, intrusive thoughts, fears, reexperiencing of emotions, behavioural enactments, and transferences. Due to the inconsistent nature of these memories, however, their expression reflects only certain aspects of a traumatic event rather than the whole trauma. This means that transference projections appear to be compartmentalized, partial, and transient (Gaensbauer & Jordan, 2009). Additionally, Bradley, Heim, and Westen (2005) have identified five dimensions of transference: angry/entitled, anxious/preoccupied, avoidant/counterdependent, secure/engaged, and sexualized.

Transference reactions in OEI. Rawn (1987) argued that, in general, all human interactions involve transference to a certain degree. It is impossible to relate to others in terms of who they "actually" are. In therapy, the client's attitudes, states of mind, orientations, and reactions influence the process. Hence, transference is reactivated in treatment and is an integral part of a therapeutic relationship (Fischer, 2005).

According to Bugental (cited in Fischer, 2005), "Transference is not just the patient's way of perceiving and responding to the therapist. It is an evocation of the subself of the patient that has been symbiotically related to the earlier figure" (p. 32). Transference reflects the clients' inner and unconscious experiences onto the therapist. It is the client's distortion of what would otherwise be a consensually validated perception, resulting from projection of aspects of him/herself onto the therapist (Rawn, 1987). Such projections can vary, from the client exhibiting minimal feelings toward the therapist, to the therapist being the centre of the client's concerns. Fischer (2005) posited that transference reveals the client's engagement in the process, with the therapist being seen, or confused with other significant individuals.

According to Gaensbauer and Jordan (2009), when trauma is triggered the client perceives two roles in the therapist: one as a "sideline figure" and the other as a therapist. The therapist witnesses the client's feelings, as victim and perpetrator, that is, it gives the therapist a sense of how the client is functioning in relation to significant others in his/her life. If the therapist is not self-aware, the therapeutic relationship can be clouded by transference, so it is crucially important that therapists distinguish themselves from transference projections and act accordingly (Fischer, 2005).

From the above discussion, it is clear that transference occurs when a client have experienced a negative, overwhelming, and distressing event with people. The more the OEI clinician reminds or resembles (in terms of facial characteristics, gender, age, race, height, hair or eye color) the client of the person who have hurt, clients were having perceptual distortions or reacting negatively (expressing fear, anger, or sadness in response) to the therapist face.(Bradshaw & Cook, 2008). These distortions are referred to in OEI as *transference reactions* (Bradshaw et al., 2011).

In summary, transference demonstrates a vital part of a therapeutic relationship. Transference serves as a bridge between past experiences and present states of clients. For maximum benefit to clients, it is crucial to bring together a current stimulus, past experiences, and transference reactions to the therapist. A majority of therapists agree that transference offers both insight regarding the client's consciousness, and a therapeutic object that can cure (Rawn, 1987).

The aim of a dynamic psychotherapeutic model is the explanation and interpretation of a reaction that has occurred due to displacement. This involves working through the client's transference projections using immediacy, which is not an easy task

(Rawn, 1987). Finally, the empathetic and supportive presence of a therapist can help clients remain grounded, and facilitate resolution of affectively difficult issues, resulting in perceptions with fewer distortions (Gaensbauer & Jordan 2005; Rawn, 1987).

Purpose of the Study

In this section, a summary of this literature review, rationales for this pilot study, and research questions are presented. Implications of this study for mental health and future research are discussed later in this document.

Grace (2003) documented the foundational finding that OEI treatment results in reductions in the frequency and intensity of PTSD symptoms. Williams (2006), in her study of women who had been sexually assaulted, found that OEI treatment resulted in significant reductions in PTSD symptoms; however, she did not find significant changes over time in hemispheric asymmetries or other qEEG markers associated with PTSD. This finding leads to the growing need to explore relationships between brainwave patterns and underlying emotions associated with PTSD.

To date there has been no study to assess simultaneous changes in multiple cortical regions in response to OEI treatment, using qEEG with LORETA. The implications of some of the studies reviewed (Leon-Carrion et al., 2006; Rabe et al., 2008; Williams, 2006) are that there is increasing need to document not only the empirical efficacy of a therapeutic intervention for reducing PTSD symptoms, but also to examine changes that occur in the brain. The author of another study stated the need to examine lateralization of brain activity in the limbic system, with exploration of concommitant emotional elements (Cannon et al., 2004). According to Shin et al. (2006), studies should also examine brain structures and functioning in participants *before* and

after treatment. There is a great need for psychoneuro-physiological assessment of individuals to provide detailed information about the effects of OEI treatment on different brain regions (Grace, 2003; Williams, 2006). Also according to Stern and Silbersweig (2001), future studies will involve increasing use of various combinations of functional imaging techniques such as EEG, MEG, and fMRI. It is hoped that such studies will enhance our understanding of human perceptions, cognitions, emotions, and behaviours. The results of such studies could shed light on the pathophysiological mechanisms underlying neuropsychiatric disorders. It would also aid in providing improved diagnostic and therapeutic strategies to help clients.

The main purpose of this exploratory study is the development and pilot testing of a research protocol for exploration of cortical activity associated with OEI transference reactions and resolutions using qEEG and LORETA. This purpose leads to the following research questions:

1. What changes in cortical activity occur during transference reactions before OEI treatment?
2. What qualitative responses occur during OEI treatment for transference reactions?
3. What changes occur in cortical activity during transference reactions after OEI treatment?

The tests of these research questions have yielded valuable information and contributed significantly toward improvement of mental health services. Such a neuropsychological assessment of negative transference reactions could pave the way to new understandings of how OEI treatment is effective, from a biopsychosocial

perspective, and could be measured accurately and reliably. Results of this study suggests how OEI treatment can quickly impact and alter brain functioning. These results also advance the field of Counselling Psychology by offering scientific evidence of rapid remediation of psychological symptoms that can affect treatment outcome. Such an investigation also lends support to the theoretical framework of OEI by providing scientific evidence of positive perceptual changes that can affect interpersonal functioning, following brief OEI treatment.

In this study, both qEEG and LORETA techniques were used. EEG is a measure of variation in brain activation across scalp sites, while LORETA is used to estimate current density resulting from divergence in electrical responses from the scalp (Cannon, Lubar, Congedo, & Thornton, 2007). Neither qEEG nor LORETA alone can provide a complete understanding of changes in brain activity, but the combination of these two approaches would greatly enhance our knowledge of underlying brain activity. The use of both these techniques would help clinicians collect adequate information for diagnosis, and for guiding and tailoring treatment plans.

Exploratory dimensions. There exists a large body of literature which demonstrates the efficiency of various treatments (e.g., SIT, CBT, and EMDR) for PTSD symptoms (Silver et al., 2005); however, research on the efficacy of OEI regarding PTSD symptoms is in its infancy. This study has added a new dimension to the evaluation of this emergent therapy. The aim of this exploratory study was to examine changes in different brain areas before, and after, OEI treatment associated with the the viewing photographs and/or video of psychologically triggering face. The exploratory nature of this study opens a wide range of possible outcomes in terms of brain changes. In this

section, anticipated outcomes based on the existing body of literature were discussed. A key point to remember is that the foundations provided in these literature reviews is based on use of brain imaging techniques *other* than EEG and LORETA (e.g., PET, MRI). Major brain areas associated with trauma that are examined at different phases of this study, and described in the following sections include:

Amygdala. Studies reveal that, in people exhibiting PTSD symptoms, triggering causes increased activation in the amygdale (Rauch et al., 2000; Shin et al., 2006; van der Kolk, 2001). It was highly likely that at the pretreatment assessment, brain analyses would show increased activation in the amygdale; whereas, after OEI the intensity of activity intervention would decrease. This change was expected to be sustained at the posttreatment assessment.

Anterior cingulate cortex (ACC). Studies have revealed that, in comparison to control groups, groups of participants with PTSD symptoms exhibited decreased activation in the ACC (Bremner, Staib, et al., 1999; Bremner et al., 2004; Lanius et al. 2003; Shin et al., 1999, 2001). Results of still other studies confirm that individuals with PTSD symptoms have diminished volumes in the ACC (Karl et al., 2006; Shin et al., 2006; Woodward et al., 2006). From these studies, it was anticipated that brain analyses would show decreased activation in the ACC at the pretreatment assessment. Since OEI works at a neuropsychological level, it was expected that there would be increased activation in the ACC at the posttreatment assessment(s).

Broca's area. There are studies which demonstrate that people who are exposed to trauma are unable to translate their experiences into communicable language (Hull, 2002; Shin et al., 1997; van der Kolk et al., 1997). On the basis of these findings, it was

predicted that at the pretreatment assessment, brain analyses would reveal lower activity in Broca's area, but that following OEI treatment there would be increases in activity that would remain stable at the posttreatment assessments.

***Corpus callosum*.** There is research evidence that people with PTSD have smaller than average corpus callosa (Teicher et al., 2003; Villarreal et al., 2004). For these reasons, it was expected that EEG and LORETA analyses would reveal more brain asymmetries at the pretreatment assessment, but after OEI treatment there would be positive changes, which would remain stable, at least until the posttreatment assessments.

***Hippocampus*.** MRI and PET scans reported in various studies reveal that people with chronic PTSD have smaller hippocampi than healthy subjects (Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Nazeer, et al., 2003; Karl et al., 2006; Shin et al., 2006; Stein et al., 1997). In light of these results, it was anticipated that brain analyses would reveal hippocampal changes during pretreatment assessments, but that a brief OEI intervention would cause increased activity in the hippocampal-dentate complex, which would be sustained in the posttreatment assessments.

CHAPTER 3: METHODOLOGY

This chapter is divided into four sections. In the first section, application of a mixed design (quantitative measures, qualitative interviews, and psychophysiological assessments) is discussed. The participant underwent various measurements in a case-based time series. A brief description of characteristics of the participant and recruitment strategies is provided in the second section. In the third section, procedures for data collection are described. It includes defining research variables. Additionally, psychometric properties, advantages, and limitations of each of the instruments and measurements are described. A detailed description of the research protocol is provided. In the fourth section, data analysis processes and ethical considerations are discussed.

Research Design

In this pilot study, the intent was to examine changes in the brain associated with OEI transference reactions. This was achieved in four phases, along with a baseline condition: Phase I involved development of the research protocol and testing of the relative effects of three forms of stimulation (photograph, video, and live person). In Phase II, the participant was engaged in pretreatment transference assessments in response to the photograph and videoclip of the nontriggering and most-triggering faces. Phase III involved application of OEI treatment, and Phase IV consisted of posttreatment assessments associated with OEI transference reactions in response to the most-triggering face.

A mixed design was applied in this study, including quantitative measures, psychophysiological assessments, and qualitative interviews. Quantitative measures included: the Clinician-Administered Dissociative States Scale (CADSS), and the

Transference Reaction Record (TRR). Psychophysiological assessements consisted of EEG readings with the application of LORETA algorithm. Qualitative interview questions addressed the participant's subjective experiences through each phase of the research project. Such a comprehensive approach was important, given the exploratory nature of the proposed study.

The participant underwent these phases in a case-based time series. Such an approach was warranted for the following reasons:

1. This study is idiographic in nature, which allows the researcher to attain a deeper understanding of how a participant behaves (Kennedy, 2005). This is particularly important, given the exploratory nature of the topic. No prior psychophysiological investigation of the OEI *process* has been completed to date, although psychophysiological *outcome* measures have been used in previous studies (e.g., Williams, 2006).
2. It allows measurement of changes that occur in a participant over time from pre-to post-treatment states, as well as changes that occur during the therapy process (Mertens, 2005). The findings from such an approach can reveal the unfolding nature of therapeutic change (Borckardt et al., 2008).
3. In this study, the input variable was the OEI treatment and the time of assessment. The outcome variables were changes in brain activity in various areas (e.g., amygdala, ACC, Broca's area, corpus callosum, and hippocampus), including CADSS, TRR, and qualitative responses. Through pre- and post-treatment transference assessments in response to the same visual stimuli in the time series, the researcher assessed whether OEI treatment had caused

observed changes in the above-mentioned (or other) brain areas. This case-based time series was helpful for assessing the efficiency and efficacy of OEI treatment (Mertens, 2005).

Participant Characteristics

The participant was an OEI clinician. Due to the exploratory nature of this study it was important to include only clinicians who had successfully completed OEI training and provided treatment of transference reactions in their own clinical practices. This way, the participant was fully aware of OEI procedures for detecting and clearing perceptual distortions such as negative transference reactions. Most importantly, the participant had to have experienced visual or perceptual distortions in his or her counselling sessions (i.e., in individual psychotherapy for her own issues). These requirements were intended to prevent the inadvertent recruitment of a participant who experienced overwhelming somatic and affective transference reactions. This was also important because the participant had to be able to sustain or tolerate her perceptual distortion(s) for approximately 30 seconds. If a general community population was recruited, it is quite likely that more intensity would be experienced than could be ethically tolerated during qEEG measurements (in extreme clinical cases, individuals can fall unconscious after 30 seconds of sustained focus on a triggering face).

Recruitment strategies. An email message was sent to all OEI clinicians and oral announcements were made in monthly OEI clinical training meetings (see Appendix A). These meetings are attended by certified clinicians who have completed OEI training and have experience assessing and treating transference reactions in their own clinical

practices. Prior to recruitment, this study was approved by the Research Ethics Board (REB) of Trinity Western University, Langley (see Appendix B).

Procedures for Data Collection

Data collection occurred in the offices of Dr. Paul Swingle in Vancouver, BC, where qEEG assessment equipment and LORETA software are housed. Interviews and psychometric assessments were administered at the same location. All assessments and treatment for the participant was completed in one day.

Input variables. The input variables were OEI treatment (one 90-minute session), and time of assessment. The five time points were: (a) baseline condition with both eyes-open, and both eyes-closed; (b) Phase I - evaluation of comparative triggering effects with a photograph of a person's face, a video, and the live person of same face; (c) Phase II - pretreatment transference assessments with a phtograph and video clip of a nontriggering face, and a most-triggering face, (d) Phase III - treatment assessments, and (e) Phase IV - posttreatment transference assessments with the photograph and video clip of a most-triggering face only. All five sets of OEI techniques (discussed previously) were incorporated in the treatment phase of this study (Bradshaw et al., 2011; Cook & Bradshaw, 2002).

Outcome variables. Since this was an exploratory study, the precise selection of outcome variables depended on qEEG and LORETA findings; however, the most likely regions for analyses were the hippocampus, the amygdala, Broca's area, the ACC, and the corpus callosum. Amplitudes of corticoelectrical signals in several frequency ranges (theta, alpha, beta, and high beta) were measured at 19 points on the scalp (in accordance to International 10-20 system of electrode placement; see Appendix C) at three different

time points (Phases I, II and IV as listed above under input variables). The Clinician-Administered Dissociative States Scale (CADSS) was administerd at baseline condition, Phase II (triggering condition) and Phase IV. In addition, the Transference Reaction Record (TRR) was administered at Phases II, III, and IV. Finally, except for Phase I, the qualitative interviews (see Appendix D).occurred at all the other time periods

Instruments and measurements. Tests and measurements used in this study included the CADSS, and the TRR. In this section, descriptions, psychometric properties, and limitations of these instruments are discussed. Brief rationales for selection of qEEG and LORETA technologies are also provided.

Clinician-Administered Dissociative States Scale (CADSS). According to Bremner et al. (1998), the CADSS is used for measuring present state dissociative symptoms. It consists of 27-items; of which 19 items are subject-rated and 8 items are observer-rated (see Appendix E). These 19 items were administered and read by the clinician and then the participant rated her response between 0 (*not at all*) and 4 (*extremely*). For the other 8 items, the clinician observed the behavior of the participant at the time of administration of 19 subjective items and made subjective evaluations of the degree to which the behaviour fit the Likert scale items. The three subscales of CADSS are depersonalization, amnesia, and derealization.

Bremner and other researchers (1998) conducted a study of participants with PTSD symptoms and dissociative disorders, as well as members of a healthy control group. No test-retest reliability was performed with the CADSS, because it measures changes in dissociation which vary with time. These researchers reported that internal consistency of the CADSS across all 27 items was .94.

The CADSS also has some limitations. According to Bremner et al. (1998), some items on the observer subscales are not correlated with total test scores. Trained raters are required, to improve the reliability of the observer rating subscale.

The rationales for using the CADSS are (a) it is a reliable and valid tool for measuring present-state dissociative symptoms, (b) it can be used as a repeated measure to assess dissociative states across times, and (c) and its observer-rated items provide in-depth information about the participant.

Transference Reaction Record (TRR). The four sections of this instrument parallel the four levels of OEI transference assessment. Left and right eye experiences at each level are recorded separately. The four levels are: (a) proximity, (b) appearance, (c) subjectively-experienced emotions and somatic sensations, and (d) projections of therapist thoughts and feelings (see Appendix F). Proximity involves perceived physical distance between a client and therapist. Appearance can include perceived emotional state (e.g., anger, sadness, fear, or shame), relative size, clarity of features, perceived colour and light associated with perceptions of the therapist's or other person's face. Emotions or somatic sensations include self-perceived client feelings of fear, sadness, anger, or shame; and bodily sensations of tension, pain, or numbness in eyes, hands, chest, head, throat, or abdomen, typically. Client projections of therapist thoughts or feelings (seeing the client as stupid, boring, angry, or irritated) comprised the last section or dimension on the TRR. Each of these four dimensions ranges on a scale from 1 (*lowest in terms of perceptual intensity*) to 5 (*highest in terms of perceptual intensity*). Since this is a pilot version of this instrument, no psychometric properties were available on this measure.

EEG and LORETA. The core purpose of this study is to get an in-depth understanding of changes that occur in the brain before, and after OEI treatment while focusing on a most-triggering face (i.e., involving negative transference reactions). Both qEEG and LORETA techniques have been used in a number of studies to date (Cannon et al., 2004). The main reasons qEEG and LORETA are a good fit for this study are summarized below.

EEG measures variation in electrical potential, while LORETA estimates current density, resulting in a quantifiable divergence on the scalp (Cannon et al., 2004). These techniques therefore complement each other, thereby enhancing our knowledge of underlying neuronal activity. The high temporal resolution of both EEG and LORETA, combined with the capability of LORETA to localize deep sources (Andreassi, 2000; Davidson, 1988; Pascual-Marqui et al., 2002) makes these technologies an ideal fit for the present study. In addition, the noninvasive nature of both the techniques (Anderer et al., 2004; Davidson, 1988; Rösler, 2005) makes it harmless for the participant, permitting repeated measurements over long time periods. Both qEEG and LORETA findings have provided information about distinctive brain patterns during the phases of this study.

Assessment and treatment procedures. The OEI treatment procedures for this study were nested within the larger assessment protocols (which were the four phases along with baseline condition). The research protocol included the following procedures.

Research protocol. The selected participant was led through the four phases (excluding the baseline condition) of the research protocol: (I) protocol development and testing for comparing transference stimulus formats, (II) pretreatment assesments with a nontriggering and a most-triggering face, (III) treatment application and analyses, and

(IV) posttreatment assessments with the most triggering face (see Appendix G). Step-by-step explanations are provided below:

1. *Baseline*: In the baseline condition, the participant was instructed to concentrate visually on the green screen of the computer. The participant was asked to relax, while qEEG recordings were done. These assessments were taken with (a) both eyes-open, and (b) both-eyes closed. This phase also included the qualitative interview and the CADSS.
2. *Phase I*: This phase involved exposure to various visual stimuli (e.g., photograph, video footage, and live person of the same face) for approximately 30 seconds each. This was done to evaluate the relative triggering effects of various visual stimuli, and to determine whether these visual stimuli could be tolerated for differing time periods, up to (and including) one minute. To minimize the impact of hand and arm movement, the covering of each eye was accomplished using an eye patch while q EEG recordings were done.
3. *Phase II*: This phase included the following stages
    a. *Nontriggering condition*: In this condition, the participant was instructed to concentrate visually on the faces of two female individuals (first on their photographs and then on video clips of their faces on a computer screen). It was found that these female images triggered the participant least in terms of transference reactions. As the participant concentrated on each image for one minute, qEEG recordings were taken. With the presentation of each face, assessments were taken first with both eyes-open, then with the right (dominant) eye open and left (nondominant) eye

covered, and finally with the left (nondominant) eye open and the right (dominant) eye covered. qEEG assessments were followed by the TRR and the qualitative interview.

b. *Triggering condition*: The participant was instructed to concentrate visually on the faces of two male individuals (first on their photographs and then on video clips of their faces on a computer screen). The faces of these men were found to trigger transference reactions (i.e., visual distortions) more than the faces of the women. One of the male faces triggered the most transference reactions, and so was selected for the qEEG measures transferred for analysis using LORETA, and for the posttreatment assessments. As in Phase I, qEEG recordings were taken first with both eyes-open, then with the right (dominant) eye open and left (nondominant) eye covered, and then the left (nondominant) eye open and the right (dominant) eye covered. These qEEG assessments were followed by completion of CADSS and TRR.

A qualitative interview was conducted with the participant at the end of this phase. The purpose of this interview was to help the participant process pretreatment transference reactions involving most-triggering and nontriggering faces, and to comprehend the nature and extent of her perceptual distortions.

4. *Phase III*: The participant was then escorted to another room in the clinic, where 90 minutes of OEI treatment were provided by an OEI clinician with master trainer certification. The stimulus, in this case was the photograph of the most-

triggering male face, which was set at the specified distance and constituted the target for the OEI treatment. This phase was followed by a qualitative interview and the TRR, to gain greater understanding of this process and experience from the participant's perspective.

5. *Phase IV*: This phase consisted of only one stage.
    a. *Triggering condition*: The participant was instructed to concentrate visually on the face (both the photograph and the video clip, on a computer screen) of the most-triggering male individual, and qEEG recordings were taken. The participant underwent assessments with both eyes-open, with the right (dominant) eye open and the left (nondominant) eye covered, and with the left (nondominant) eye open and the right (dominant) eye covered. These qEEG recordings were followed by completion of the CADSS and TRR.

        The participant's report of this triggering condition were incorporated in the final qualitative interview to gain a clearer understanding of the subjective differences between pre- and post-treatment assessments, involving the same triggering face. At the end of data collection, the participant was debriefed, and grounding and calming techniques were applied to ease the mild distress and dissociation she was still experiencing.

Data storage. All data and information received, including questionnaires, interviews, and video recordings of assessments and treatment sessions, were kept in a locked file cabinet in a locked office in the Department of Counselling Psychology at

Trinity Western University (TWU). The output of qEEG and LORETA were stored on a password protected computer, in a locked room at the offices of Dr. Paul Swingle.

Data Analyses

No formal or inferential statistics were performed, due to small sample size ($N = 1$) and lack of individual or group comparison. Standardized questionnaires were scored and the interviews were transcribed.

qEEG data were recorded, artifacted, processed, and then transfered for LORETA imaging. The LORETA-KEY software package was used to compute an average cross-spectral matrix for the participant in each of the EEG frequency bands: delta (1-3Hz), theta (3-7Hz), low alpha (8-9 Hz), high alpha (11-12 Hz), low beta (13-15 Hz), beta (16-25 Hz), high beta (26-28 Hz), and gamma (28-40 Hz; Swingles, 2008). This was done by an experienced neurotherapist and qEEG assessment specialist Michael Mariano; an employee of Dr. Paul Swingle & Associates in Vancouver, British Columbia, Canada.

The reported findings include a series of LORETA images that permit localization of the sources of scalp electrical activity (recorded qEEG measurements) in figures depicting three dimensions: front to back (coronal plane), side to side (sagittal plane), and top to bottom (horizontal plane; see, Appendix H). Additionally, tables of qEEG power spectra in selected bandranges (see Appendix I) and color topographic brain maps (see Appendix J) were also incorporated. A series of qEEG “Swingle signature” (see Appendix L) was selected for review from each power spectra table. These have been identified through more than 50 years of clinical experiences in psychophysiological measurements (Swingle, 2008, 2009), and include a series of ratios between mean amplitudes in selected bandranges at particular scalp locations. These data analyses,

which include graphical representations and descriptive statistics, provide indications of changes in brain areas that occurred from the assessment before to the assessment after OEI treatment.

Ethical Considerations

One of the recruitment strategies was to request participation from OEI clinicians and TWU Counselling Psychology faculty members who were aware of experiencing negative transference reactions in response to particular individuals. To minimize or avoid coercion with respect to recruitment of individuals with established professional relationships to the investigators, (a) it was explicitly stated orally and in the informed consent form that participation was completely voluntary, (b) it was emphasized that any individual's decision to participate in (or continue with) the study would not affect ongoing professional relationships, (c) it was recognized that the target population was fully aware of what OEI treatment involved, had been trained in OEI, and had observed transference reactions during OEI in their own clinical practices, and (d) it was stressed that the outcomes of the study (associated with the identities of any participant) would remain confidential.

The study required a certain degree of psychological discomfort, because it involved questions pertaining to trauma and dissociative symptoms. Additionally, the participant was asked to hold transference reactions for approximately 30 seconds. Such experiences elicited uncomfortable feelings, thoughts, and bodily sensations. As a result of her familiarity with OEI and related transference assessment and treatment procedures, however, the participant was much less disturbed by perceptual distortions and somatoform dissociative symptoms than individuals would likely be who had no such

experience or knowledge. In fact, the participant benefited by gaining greater understanding of underlying brain activity associated with transference projections and reduction of her own transference reactions.

CHAPTER 4: RESULTS

In this chapter, results are presented for protocol testing and analysis. For each phase, qualitative findings are followed by psychometric information and psychophysiological findings. Prior to presenting Phase I results, the outcomes of baseline condition are provided. In all cases, results pertain to one Caucasian female participant between the ages of 25 and 35 years. The participant had completed OEI training and had observed transference reactions in clients, so she was familiar with the assessment and treatment of such phenomena. Upon entry to the study she was asked to indicate an individual who she experienced a transference distortion with. A photograph and one-minute video clip of that individual was obtained prior to the study, and that individual was asked to come into the laboratory for a "live" assessment.

Since psychophysiological measurements were an important component of this study, it is important to provide a description of the brainwave profile of the participant, in terms of abnormalities observed in qEEG data. For the current study, the 'Quick-Q' scalp sites were C_z, O_1, F_z, F_3, and F_4 (see Swingle, 2009 for a detailed explanation of the entire Quick-Q protocol and interpretation of results). Alpha suppression at C_z and O_1 were not reported because data in the eyes closed condition were corrupted with eye-blink artifact. The amount of eyeball movement within the orbits with eyes closed contributed so much muscle artifact that the sample was non-usable. In general, the participant had a low theta/beta ratio in the occipital region (O_1) which indicated racing thoughts, inability to "shut the brain off", general anxiety, reduced distress tolerance, and often problems with sleep quality. Her hibeta to beta ratio at F_z was below the normative range, which suggests the possibility of excessive passivity (Swingle, 2009).

Her low alpha to high alpha ratio at F_z exceeded the normative range, which may be indicative of a cognitive deficit or sleep difficulty. Her beta comparisons between F_4 and F_3 (i.e., 15% or greater beta amplitudes in favour of the right) were above the normative range, which predisposes her to depressed mood states (Swingle, 2008, 2009). Studies also demonstrate that alpha frontal asymmetry (15% or greater elevation at F_3 relative to F_4) is seen more among both currently depressed individuals and nondepressed individuals who have histories of *past* depression. Depressed individuals have less left frontal activation than right (Davidson, 1988, 1992). Still other researchers have shown that left frontal *hypo*activation (i.e., higher alpha) reflects an individual's vulnerability to depression (Henriques & Davidson, 1990, 1991) and react negatively to emotional situations. From these studies it can be inferred that the participants' excessive alpha and beta value differences at F_3 and F_4 increase her susceptibility to depression in the presence of stressors.

Baseline Assessments

Qualitative interview findings. The participant was instructed to engage in a three-minute period with both eyes-open, followed by a three-minute period with both eyes-closed, during which qEEG information was collected. The eyes-open baseline condition posed no problem for the participant. She was able to remain relaxed, and did not engage in excessive blinking, tooth grinding, shoulder shrugging, or neck rolling (all of which interfere with accurate qEEG recordings, resulting in "artifact").

In contrast, the participant reported more difficulties during the both eyes-closed condition. She experienced anxiety, and was physically agitated at a moderate level of intensity (evidenced by excessive blinking, which rendered the qEEG recording invalid).

The major somatic symptoms reported were increased heart rate and tightness in her stomach. She reported that she typically manifested these symptoms when she "felt the need to know what was going on". She stated that she "didn't feel safe" with her eyes closed; however, these symptoms dissipated spontaneously within several minutes after the eyes-closed condition was complete.

Psychometric findings. The participant's baseline CADSS score of 12, was below the means for both PTSD patients (*M* = 18.9) and PTSD patients with comorbid disorders (*M* = 19.3), reported by Bremner et al. (1998; see Table 1). TRR assessment was not performed for this condition because the participant was not engaged in viewing a face.

Psychophysiological measurements. Unless otherwise noted, all baseline measurements reported were taken in the eyes-open condition. Five-point qEEG analyses revealed that low alpha/high alpha ratios at F_z, alpha comparisons at F_3/F_4, and beta comparisons at F_4/F_3 were within the normative range; however, her theta/beta ratio at O_1 and hibeta/beta ratio at F_z, were below the normative range (see Table 2).

Overall, the baseline findings were unremarkable, other than the somatic symptoms that were experienced by the participant, during both eyes-closed condition, which quickly dissipated spontaneously. During the both eyes-open condition, the abnormality observed in the occipital and frontal midline suggests that the participant was experiencing general anxiety and excessive passivity, respectively. These symptoms may have occurred due to unfamiliarity with the experimental protocol.

Table 1

Participant's Clinician Administered Dissociative State Scale (CADSS) Scores at Three Administration Times

Time of Administration	Subjective Items	Objective Items	Total
Baseline	12	1	12
Phase II	12.5	6	18.5
Phase IV	8.5	0	8.5

Note. The ranges of possible CADSS Scale Scores were: Subjective (0–76), Objective (0–32), and Total (0–108). The Phase II values are reported for the pretreatment triggering condition.

Table 2

Swingle Signature Scores during qEEG Recordings at Baseline

Swingle Signature		Control conditions	
Location(s)	Frequency(ies)	BO	BC
O_1	θ/β	†0.96	-
F_z	Hiβ/β	†0.30	-
F_z	Loα/Hiα	1.20	-
F_3/F_4	α	1.05	-
F_4/F_3	β	1.10	-

Note. Swingle signatures are ratios of amplitudes: one frequency at two locations or two frequencies at one location. O_1 = Electrode placed in the left side of the occipital lobe; F_z = Electrode placed in the frontal midline; F_3 = Electrode placed in the left side of the frontal lobe; F_4 = Electrode placed in the right side of the frontal lobe; α = Alpha; β = Beta; θ = Theta, Hiβ = High Beta; Loα = Low Alpha; Hiα = High Alpha; BO = Both eyes open; BC = Both eyes closed; - = No scores available; † = Below the normal range (Swingles, 2008).

Phase I: Stimulus Source Comparisons for Protocol Development and Testing

The main goal of this first phase of data collection following the baseline measures was to comparatively evaluate the triggering effects of a photograph, a video clip, and a live person, for the same face. The intent was to determine which stimulus source triggered the most transference reactions (perceptual distortions and emotional intensity). Only psychophysiological measurements were done.

Psychophysiological measurements. In response to the *photograph* of the triggering face, the participant's low alpha/high alpha ratio at $F_{z,}$ and her comparative beta values at F_4/F_3 exceeded the normative range; however, her theta/beta ratio at O_1 was below the normal range. In the 'right eye open' condition, the difference in beta values between F_4 and F_3 was to found to be greater than 15% (i.e., above the normal range); whereas, her theta/beta ratio at O_1 and the hibeta/beta ratios at F_z were below the normal range. When the 'left eye' was open, the low alpha/high alpha ratio at F_z and her difference in beta values between F_4 and F_3 were above the normative range, whereas her theta/beta ratio at O_1, and hibeta/beta ratio at F_z remained below the normal range. When the participant was exposed to the *video* of the most-triggering face, the same differences were observed in two conditions: (a) 'both-eyes open', and (b) 'right eye open.' In each case, her low alpha to high alpha ratios at F_z and her differences in beta values at F_4 and F_3 were above the normal range; however, her theta to beta ratio at O_1 and hibeta/beta ratio at F_z were below the normative range. In the 'left eye open' condition, her low alpha to high alpha ratio at F_z was also found to be above the normal range, and her theta to beta ratio at O_1 and hibeta/beta ratio at F_z were below the normal range. When a *live person* was the visual stimulus, analyses revealed higher than normal differences in beta

values between F_4 and F_3 in (a) 'both eyes open', and (b) 'right eye open' conditions. In each case, her theta/beta ratio at O_1 and hibeta/beta ratio at F_z were below the normative range. When the 'left eye' was open, the participant demonstrated a higher than normal low to high alpha ratio at F_z and greater than normal differences in beta values between F_4 and F_3; however, her theta to beta ratio at O_1 and hibeta/beta ratio at F_z were below the normative range (see Table 3).

In summary, it appears that the type of stimulus does not determine whether or not an individual will exhibit qEEG trauma markers in response to a triggering face. For this reason, future use of this protocol with additional participants can rely on photographs of the faces of most-triggering and nontriggering individuals. This is helpful for research purposes, since the consistency of photographic images is much greater than the consistency obtainable with either live or video stimuli. One interesting finding mentioned in the qualitative interview with this participant, was that the complex collection of characteristic evidenced in the live stimulus elicited her recall of *positive* aspects of the relationship (which to some extent offset the negative triggering properties of the facial stimulus). This is perhaps yet another reason to use photographs in future --- to avoid the additional variable of *recent* (as opposed to distant *past*) relationship history.

Table 3

Swingle Signature Scores during qEEG Recordings at Phase I with the Male Face Triggering the Strongest Transference Response

Swingle Signature		Visual Medium for presenting Target Face								
Location(s)	Frequency(ies)	Photograph			Video			Live person		
		BO	*R*	*L*	*BO*	*R*	*L*	*BO*	*R*	*L*
O_1	θ/β	†1.34	†0.97	†0.99	†1.26	†1.26	†1.39	†0.95	†0.93	†1.30
F_z	Hiβ/β	0.51	†0.32	†0.42	†0.33	†0.35	†0.36	†0.42	†0.43	†0.41
F_z	Loα/Hiα	*1.74	1.21	*1.68	*1.53	*2.37	*1.65	1.24	1.25	*1.97
F_3/F_4	α	0.86	1.02	0.88	0.89	1.06	0.94	0.90	1.04	0.74
F_4/F_3	β	*1.29	*1.26	*1.21	*1.39	*1.24	1.08	*1.74	*1.72	*1.48

Note. Swingle signatures are ratios of amplitudes: one frequency at two locations or two frequencies at one location O_1 = Electrode placed in the left side of the occipital lobe; F_z = Electrode placed in the frontal midline ;F_3 = Electrode placed in the left side of the frontal lobe; F_4 = Electrode placed in the right side of the frontal lobe; α =Alpha; β = Beta; θ = Theta, Hiβ = High Beta; Loα = Low Alpha; Hiα = High Alpha; BO = Both eyes open; R = Right eye open; L= Left eye open * = Above the normal range, † = Below the normal range (Swingles, 2008).

Phase II: Pretreatment Transference Assessments

Nontriggering condition.

Qualitative interview findings. Both emotionally and physiologically the participant experienced a state of *relative* calmness and relaxation when viewing photographs and video clips of individuals who did *not* trigger her anxiety (i.e., compared to her reactions to the most-triggering face). In this phase, however, the visual stimulus (face) of one person--- the younger female face --- bore some resemblance to a previous acquaintance who was angry, and therefore was reported as slightly anxiety-inducing.

Psychometric findings. CADSS assessment was not performed during the pretreatment nontriggering condition. Formal assessment using the TRR during exposure to nontriggering faces (two females, one younger and one older) indicated that there was no major transference reactions with either the left or right eye open, for any of the four dimensions (proximity, appearance, emotions/bodily sensations, or projected cognitions; see Table 4)

Psychophysiological measurements. According to the participant, image 2 was the most nontriggering face in terms of transference reactions. For that reason, unless otherwise indicated, the term "nontriggering" image refers to the face of the woman in image 2. During exposure to the *photograph* of image 2, in the 'both eyes open' condition, beta value differences between F_4 and F_3 were higher than normal; whereas, the participant's theta to beta ratio at O_1 and her hibeta to beta ratio at F_z were below the normative range. In both the other conditions ('right eye open', and 'left eye open') her low alpha/high alpha ratio at F_z was above the normal range; whereas, her theta/beta ratio at O_1 and her hibeta/beta ratio at F_z were below the normal range (see Table 5). For the

video of image 2, the same differences were observed in all three conditions: (a) 'both eyes open', (b) 'right eye open', and (c) 'left eye open' conditions. In each case, higher than normal beta amplitude differences were found between F_4 and F_3; but the theta/beta ratio at O_1 and hibeta to beta ratio at F_z were found to be lower than normal (see Table 6).

In summary, findings associated with the *least triggering faces* from the qualitative interview and psychometric assessments were unexceptional. Results displayed in Tables 5 and 6 shows, that between the photograph of the least triggering face (image 2) and the video clip of the least triggering face (image 2), a slight change in qEEG findings were observed in two conditions: (a) 'right eye open' and (b) 'left eye open.' In each case, there were changes in the low alpha to high alpha ratio at F_z and to beta comparisons between F_4 and F_3. These changes may be due to (a) decrease in anticipatory anxiety as a result of lack of negative properties associated with the observed face and/or (b) habituation as a result of familiarity with the face.

Table 4

Transference Reaction Record (TRR) Results for Triggering and Nontriggering Faces at different Administration Time

Administration Time	Dimensions of Perceptual Distortions	Right Eye Open (R)	Left Eye Open (L)	L-R Differences
Phase II: Pretreatment Triggering condition	Proximity	None		None
	Appearance	Eyes appear "slightly separated" and vary emotionally. Sad=1.5; Angry=3.5	Face appears larger; with "Eyes separated". They "stand out" from the rest of the face (especially the Right eye)	Far more visual distortion when looking through Left (nondominant) eye
	Emotions	"Blank" = 1.5, but <u>grounded</u> emotionally	"Blank" = 3, but with <u>fear</u> emotionally	More intense and negative emotions with Left (non-dominant) eye
	Bodily Sensations	Slight = 2 heartbeat increase	Major = 4.5 "pounding" heartbeat, heart rate increase, bad headache, and "body heaviness"	Much more intense and severe somatic symptoms with Left (nondominant) eye

	Projected Cognitions	None		None
Phase III: Treatment condition	Proximity	None		None
	Appearance	Sad appearance = 5; Fragmented = 1	Angry appearance = 5; Fragmented = 5; Left eye appeared to have a circle around it, rest of face was blurred.	Greater fragmentation and higher fear response with Left (non-dominant) eye
	Emotions	Sadness = 5	Weird" = 1	Sense of emotional sadness with Right (dominant) eye may be associated with physical feeling of being" cared for" in chest, with that eye
	Bodily Sensations	"Care Feeling" in chest	Laboured breathing inchest & nausea in stomach = 5	Much more severe somatic symptoms associated with Left (nondominant) eye

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	Proximity	None		No
Phase IV: Posttreatment Triggering condition	Appearance	Anger = 1; No distortions in colour	Anger = 1; Yellow tint = 1	No L-R differences for perceived emotion; Slight colour difference with Left (non-dominant) eye.
	Emotions	Agitation = 3	Numbness = 4	Evidence of Negative transference reaction in Left (non-dominant) eye
	Bodily Sensations	Chest congestion = 3; Headache = 3	Headache = 3	Slightly more somatic discomfort associated with right (dominant)eye
	Projected Cognitions	"Hard to think about"	"Hard to think about"	Evidence of dissociation

Note. None = No transference reactions registered. There were no transference reactions reported by the participant in Phase II nontriggering condition.

Table 5

Swingle Signature Scores during qEEG Recordings at Phase II with the Photographs of the Four Target Faces

Swingle Signature		Photograph as a medium for presenting Target Faces											
Loc(s)	Freq(s)	Image 1 (woman)			Image 2 (woman)			Image 3 (man)			Image 4 (man)		
		BO	*R*	*L*	*BO*	*R*	*L*	*BO*	*R*	*L*	*BO*	*R*	*L*
O_1	θ/β	†1.15	†1.06	†1.5	†1.23	†1.14	†1.06	†1.21	†1.05	†1.2	†0.1	†1.45	†1.32
F_z	Hiβ/β	†0.36	†0.29	†0.26	†0.32	†0.4	†0.38	†0.3	†0.29	†0.37	†0.36	†0.37	†0.32
F_z	Loα/Hiα	1.18	*4.06	*1.81	1.07	*1.83	*1.58	*1.97	*2.19	1.21	*1.71	1.18	*1.53
F_3/F_4	α	0.91	1.13	0.10	0.96	1.10	1.06	0.89	0.85	0.96	*1.26	1.04	1.00
F_4/F_3	β	*1.43	*1.93	*1.16	*1.22	0.9	1.05	*1.19	*1.32	*1.26	*1.27	*1.39	1.01

Note. Swingle signatures are ratios of amplitudes: one frequency at two locations or two frequencies at one location. Loc(s) = location/locations; Freq(s)=frequency/frequencies; O_1 = Electrode placed in the left side of the occipital lobe; F_z = Electrode placed in the frontal midline ;F_3 = Electrode placed in the left side of the frontal lobe; F_4 = Electrode placed in the right side of the frontal lobe; α = Alpha; β = Beta; θ = Theta, Hiβ = High Beta; Loα = Low Alpha; Hiα = High Alpha; BO = Both eyes open; R= Right eye open; L= Left eye open ;* = Above the normal range, † = Below the normal range (Swingles, 2008). The continuum of transference response ranged from weakest to strongest in the following sequence: Image 2, Image 1, Image 3, and Image 4.

Table 6

Swingle Signature Scores during qEEG Recordings at Phase II with the Video of the Four Targets Faces

Swingle Signature		Video as a medium for presenting Target Faces											
Loc(s)	Freq(s)	Image 1 (woman)			Image 2 (woman)			Image 3 (man)			Image 4 (man)		
		BO	*R*	*L*	*BO*	*R*	*L*	*BO*	*R*	*L*	*BO*	*R*	*L*
O_1	θ/β	†1.18	†1.45	†0.99	†0.77	†1.62	†1.31	†1.08	†0.94	†1.11	†0.98	†1.21	†1.11
F_z	Hiβ/β	†0.37	†0.3	†0.33	†0.3	†0.29	†0.27	†0.37	†0.30	†0.31	†0.32	†0.34	†0.32
F_z	Loα/Hiα	*1.87	*2.34	*2.07	1.31	1.32	1.45	1.26	0.54	1.33	0.93	*2.30	1.01
F_3/F_4	α	0.81	1.07	1.10	0.98	0.85	0.77	0.97	1.12	0.91	0.10	0.98	0.10
F_4/F_3	β	*1.71	*1.17	*1.33	*1.54	*1.33	*1.38	*1.26	*1.47	1.12	*1.27	1.13	*1.25

Note. Swingle signatures are ratios of amplitudes: one frequency at two locations or two frequencies at one location Loc (s) = location/locations, Freq(s)= frequency/frequencies, O_1 = Electrode placed in the left side of the occipital lobe; F_z = Electrode placed in the frontal midline ; F_3 = Electrode placed in the left side of the frontal lobe; F_4 = Electrode placed in the right side of the frontal lobe, α = Alpha; β = Beta; θ = Theta, Hiβ = High Beta; Lα = Low Alpha; Hiα = High Alpha; BO = Both eyes open; R= Right eye open; L= Left eye open; * = Above the normal range, † = Below the normal range (Swingles, 2008).

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Triggering condition.

***Qualitative interview findings*.** During exposure to the two male faces, (both of which were found to be more triggering than either of the female faces), the participant reported feeling agitated and "numbed out". She also reported congested breathing, increased heart rate, headaches, tightness in her stomach,"heaviness" in her legs ("…like…I'm walking in molasses or water"), and her body of feeling "weighed down" and "hard to move." Overall, her somatic symptoms were predominant (rather than her emotions or thoughts). She reported her somatic symptoms on the higher end of moderate (on a SUD scale from 0 *least* to 10 *highest*). This was consistent with her *post*treatment report, when she stated, "It's usually *afterwards* that I end up getting way more emotionally upset." Visual distortions were also experienced by the participant; more specifically, the most-triggering face appeared to be "masked" or "unreal", and the eyes seemed to "stand out" or be "isolated." The participant reported experiencing negative transference reactions immediately after seeing the triggering visual stimulus. She stated that she expereienced more intensity and more pronounced visual distortions with the left (nondominant) eye open.

Psychometric findings. CADSS scores reported in the present study were compared to means for PTSD patients (M = 18.9) and PTSD patients with comorbid disorders (M = 19.3), as stated in Bremner et al., (1998). The participant's score of 18.5 during the pretreatment triggered condition was approximately equal to both of these means (see Table 1), and higher than the CADSS score of 12 reported during the baseline condition.

On the TRR, in terms of *appearance,* the participant reported more visual distortions when looking through the left (nondominant) eye than through the right (dominant) eye. There were more intense and negative *emotions* present with the left (nondominant) eye open. She also experienced more intense *somatic* symptoms (e.g., heart pounding, headaches, and "heaviness" in her body) with her left (nondominant eye) open than with her right (dominant) eye open. No transference reactions were registered in terms of *proximal distance* or *projected cognitions* on the TRR in response to viewing the most triggering face (see Table 4).

Psychophysiological measurements. According to the participant, image 4 (male) was the most-triggering face in terms of transference reactions. Unless otherwise mentioned, the term "most-triggering face" refers to the face of the man in image 4. In response to the *photograph,* the low alpha/high alpha ratio at F_z, the differences in alpha value between F_3 and F_4, and the difference in beta values between F_4 and F_3 were above the normative range; during the 'both eyes-open' condition. When the 'right eye' of the participant was open, a higher than normal beta value difference was found at F_4 relative to F_3; whereas with her 'left eye' open, the low alpha/high alpha ratio at F_z exceeded the normal range. Her theta/beta ratio at O_1 and hibeta/beta ratio at F_z were below the normative range for all the three conditions ('both eyes open', 'right eye open', and 'left eye open'; see Table 5). In the case of exposure to the one-minute *video clip* of image 4, the two conditions (a) 'both eyes open', and (b) 'left eye open' conditions were accompanied with higher than normal beta values differences between at F_4 than F_3. In both these conditions, the participant had a lower than normal theta to beta ratio at O_1 and a lower than normal hibeta/beta ratio at F_z. When the participant's 'right eye' was open,

her low alpha/high alpha ratio at F_z was above the normal range; but her theta/beta ratio at O_1 and hibeta/beta ratio at F_z were below the normal range (see Table 6).

From the qualitative responses and TRR results, it was found that in the pretreatment triggering condition, the participant experienced severe somatic symptoms (e.g.; headaches, increased heart rate, and tension in her stomach), visual distortions, and intense negative emotions associated with the *most-triggering face* when her left (nondominant) eye was open. When comparing the qEEG findings from Table 5 (photograph of most-triggering face or image 4) and Table 6 (video of most-triggering face or image 4) a slight change in qEEG findings was observed. This change could be attributed to (a) the result of habituation with the face, and/or (b) greater stimulus variation in the video clip (than in the photograph), provided notably brief experiences of relief from observations of disturbing facial expressions in the video clip.

Phase III: Treatment Assessments

In this phase, only the photograph of the most-triggering face was presented as a visual stimulus. The participant underwent approximately 90 minutes of OEI treatment by a female clinician who was certified and experienced in OEI (an important distinction, since the most triggering face, in this case, was male).

Qualitative interview findings. The participant had a hard time focusing on the photograph of the most-triggering face. At the beginning of the treatment, she reported feeling physical agitation and moderate anxiety. During the treatment, the participant stated relief from her somatic symptoms. Her perceptual distortions (visual appearance of the most-triggering face) were more intense when she was simultaneously engaged in Switching while focusing on the visual stimulus. This was most emotionally upsetting

and for the participant, and was accompanied by physical symptoms of discomfort (e.g., "heaviness" in her legs, nausea, palpitations, and headaches) and distress. By the end of the treatment, the participant reported great relief from her somatic symptoms, except for a residual sense of 'fuzziness.' She also reported some "emotional disconnection". The participant affirmed that grounding techniques, and the therapist's presence ("being there" for her), were helpful and important aspects of this process.

Psychometric findings. On the *appearance* dimension of the TRR, the participant reported greater fragmentation and more fear wher her left (nondominant) eye was open. Severe *somatic* symptoms were also associated with the left (nondominant) eye open. There were also *emotions* associated with the right (dominant) eye, but they were actually positive (e.g., the physical feeling of "being cared for" in her chest). There were also more positive *projected cognitions and emotions* associated when her right (dominant) eye open than when her left (nondominant) eye was open. No differences were reported between the left and right eye open conditions in terms of perceived *proximity* (see Table 4). CADSS assessment was not performed during the Treatment phase.

In summary, it appears that during OEI treatment there were visual distortions associated when the left (nondominant) eye was open. These distortions were associated with intense somatic symptoms such as "heaviness" in the lower extremities, and heart palpitations. Nevertheless, by the *end* of the treatment, the participant felt relief from her physical discomfort but experienced a sense of "emotional disconnection."

Phase IV: Posttreatment Transference Assessments

Triggering condition.

Qualitative interview findings. In comparison to the pretreatment transference assessment, the participant reported experiencing much less intensity during exposure to the photograph than to the video clip of the most-triggering face. This difference in perceived intensity is understandable, since the participant was only exposed to the photograph of the most-triggering face during the treatment phase. She reported reductions in her somatic symptoms (e.g., chest tightness, "heaviness" in her body, and nausea in her stomach) compared to the pretreatment transference assessment. The severity and intensity of feelings of agitation and anxiety also diminished. With the right (dominant) eye open, her visual distortions were minimized. With the left (nondominant) eye open, some visual perceptual distortion remained, but there was significant reduction in the severity and intensity of her somatic symptoms. The participant stated that she "could hold more easily that there was a 'kindness' there" (associated with the person with the most-triggering face).

Psychometric findings. The participant's posttreatment CADSS score of 8.5 was much lower than her pretreatment triggering condition score of 18.5 and also much lower than the mean scores for PTSD patients (M = 18.9) or PTSD patients with comorbid disorders (M = 19.3; Bremner et al., 1998; see Table 1)

In Bremner et al. (1998), PTSD participants were asked to write a story, letter, or poem of one of the five most traumatic events experienced during their military services. They were then asked to read these scripts aloud to other participants. In response to this triggered condition, PTSD participants demonstrated increases in dissociative symptoms

from the baseline condition (*M* = 21.8) to the pretreatment triggered condition (*M* = 35). Likewise, the CADSS result for the present study revealed that there was an increase in dissociative symptoms from the baseline score of 12 to the pretreatment triggered score of 18.5 (see Table 1). The OEI intervention, however, significantly decreased these symptoms (resulting in a CADSS score of 8.5). This implies that in both the Bremner et al. (1998) study and the present study, dissociative symptoms increased in response to a triggered condition.

On the *appearance* dimension of the TRR, there was a slight colour difference with the left (nondominant) eye open. In terms of *emotional* reaction, the participant's reporting of a slight difference in emotional response with left (nondominant) eye open on the TRR ("emotional disconnection") may be indicative of dissociation. In contrast to the pretreatment triggering state, there were more *somatic* symptoms associated with the right (dominant) eye than with left (nondominant) eye open. In terms of *projected cognitions*, both the eyes were associated with cognitive difficulties. On the TRR, the participant reported no difference in the perceived *proximity* dimension of transference projection between 'left eye open' and 'right eye open' conditions (see Table 4).

Psychophysiological measurements. In this phase, the participant was exposed to the photograph and the one-minute video clip of only the most-triggering face (image 4). In response to the *photograph*, during the 'both eyes-open' condition, the participant's low alpha to high alpha ratio at F_z, and difference in mean alpha amplitudes between F_3 and F_4 were above the normal range; however, her theta to beta ratio at O_1 and her hibeta/beta ratio at F_z were below the normal range. With her 'right eye open', the participant's theta to beta ratio at O_1 and hibeta/beta ratio at F_z were both below the

normative range. During the 'left eye open' condition, her low alpha to high alpha ratio at F_z exceeded the normal range; whereas, her theta to beta ratio at O_1 and her hibeta/beta ratio at F_z were below the normative range. In response to the *video clip*, during the 'both eyes open' condition, the participant's difference in mean beta amplitudes between F_4 and F_3 was above the normal range; and her theta to beta ratio at O_1 and hibeta/beta ratio at F_z were below the normative range. In the other two conditions: (a) 'right eye open' and (b) 'left eye open', her theta to beta ratio at O_1 and hibeta/beta ratio at F_z were below the normative range (see Table 7).

In summary, from the above findings it appears that at posttreatment the participant reported decreases in her somatic symptoms. In comparison to the 'left (nondominant) eye open' condition, there was more reduction in intensity (e.g., visual distortions, projected cognitions) associated with the 'right (dominant) eye open' condition. From the above findings, it can be inferred that in the pretreatment triggering condition, the participant experienced overwhelming somatic and affective states. Decreases in posttreatment triggered intensity, however, suggest that after treatment the participant was able to 'distance herself' from original negative event associated with her transference reactions to the most-triggering face and increase her 'mentalizing' capacity (i.e., ability to stand back and simply observe her reactions, rather than be overwhelmed by them).

Table 7

Swingle Signature Scores during qEEG Recordings at Phase IV with the Male Face Triggering the Strongest Transference Response

Swingle Signature		Photograph			Video		
Location(s)	Frequency(ies)	*BO*	*R*	*L*	*BO*	*R*	*L*
O_1	θ/β	†1.44	†1.41	†0.85	†0.82	†1.33	†0.97
F_Z	Hiβ/β	†0.33	†0.37	†0.32	†0.31	†0.33	†0.38
F_Z	Loα/Hiα	*1.73	1.05	*1.65	1.01	0.94	1.07
F_3/F_4	α	*1.16	0.97	1.02	0.84	1.04	0.94
F_4/F_3	β	1.01	1.02	1.05	*1.21	1.09	1.10

Note. Swingle signatures are ratios of amplitudes: one frequency at two locations or two frequencies at one location O_1 = Electrode placed in the left side of the occipital lobe; F_z = Electrode placed in the frontal midline; F_3 = Electrode placed in the left side of the frontal lobe; F_4 = Electrode placed in the right side of the frontal lobe;; α = Alpha; β = Beta; θ = Theta, Hiβ = High Beta; Loα = Low Alpha; Hiα = High Alpha; BO = Both eyes open; R= Right eye open; L= Left eye open; * = Above the normal range, † = Below the normal range (Swingles, 2008).

Brain Structure Activation as Shown in LORETA Analyses

LORETA analyses were applied to the participant's pre- and post-treatment exposures to visual stimuli (photograph and video clip) associated with the most-triggering face, Since the greatest differences were observed between 'right eye open' and 'left eye open' conditions results from the 'both eyes-open' condition were not discussed here. The purpose of the LORETA analyses was to identify brain structures from which scalp-level EEG waveforms (theta, alpha, and beta; see Table 8) most likely originated, before and after the OEI treatment (see Table 9).

It is important to specify the terminology "activation" before understanding the relationship between activation and emotion-related process. Power in the alpha band (8-12 Hz) is inversely related to brain activation; that is, increases in alpha amplitude reflect decreases in cortical activation (i.e., *hypo*activation) and decreases in number of action potentials from neighboring neurons (Davidson, 1988, 1992). Usually, there is an inverse relationship between theta and beta activation; beta 'suppression' means theta *enhancement* or beta *enhancement* implies theta 'suppression' (Swingle, 2008). The significance of activations in these EEG bandwidths (theta, alpha, and beta) depends on brain location.

Table 8

Frequency Bandwidths used during qEEG Recordings

Frequency	Bandwidth (Hz)
Delta	1-3
Theta	3-7
Low Alpha	8-9
High Alpha	11-12
Low Beta	13-15
Beta	16-25
High Beta	26-28
Gamma	28-40

Table 9

Comparison of Brain Areas activated for different EEG frequencies when presented with the Photograph of the Male Face Triggering the Strongest Transference Response in Phases I and IV

Frequency	Phase I		Phase IV	
	Right Eye Open (R)	*Left Eye Open (L)*	*Right Eye Open (R)*	*Left Eye Open (L)*
θ	Brodman area 23	Brodman area 23	Brodman area 13	Brodman area 24
	Cingulate gyrus	Cingulate gyrus	Right Insula	Anterior cingulate
	Limbic lobe	Limbic lobe	Sub-lobar	Limbic lobe
α	Brodman area 37	Brodman area 37	Brodman area 41,21,22	Brodman area 37
	Right Fusiform gyrus	Right Hippocampus	Right Superior temporal gyrus	Right Inferior temporal gyrus
	Temporal lobe	Subgyrus	Right Middle temporal gyrus	Temporal lobe
β	Brodman area 9	Brodman area 9	Brodman area 31	Brodman area 17
	Middle frontal gyrus	Right Middle frontal gyrus	Precuneus, Cuneus	Left Lingual gyrus
	Frontal lobe	Frontal lobe	Cingulate (posterior)	Occipital lobe

(continued)

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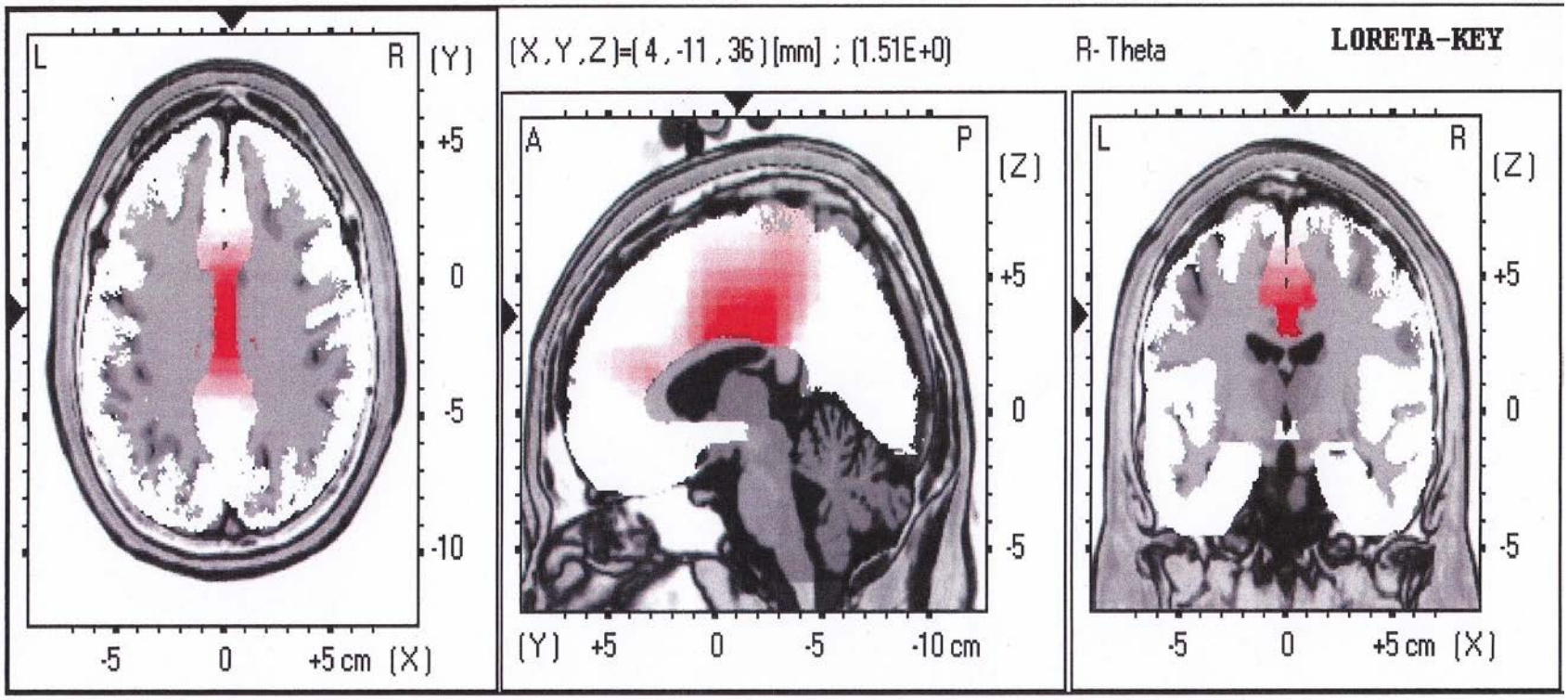
Note. θ = Theta frequency range; α = Alpha frequency range; β = Beta frequency range. The regions of activation are described in three ways: Brodmann's Area, gyrus or structure where the effect is located, and lobe or structure where the effect is located.

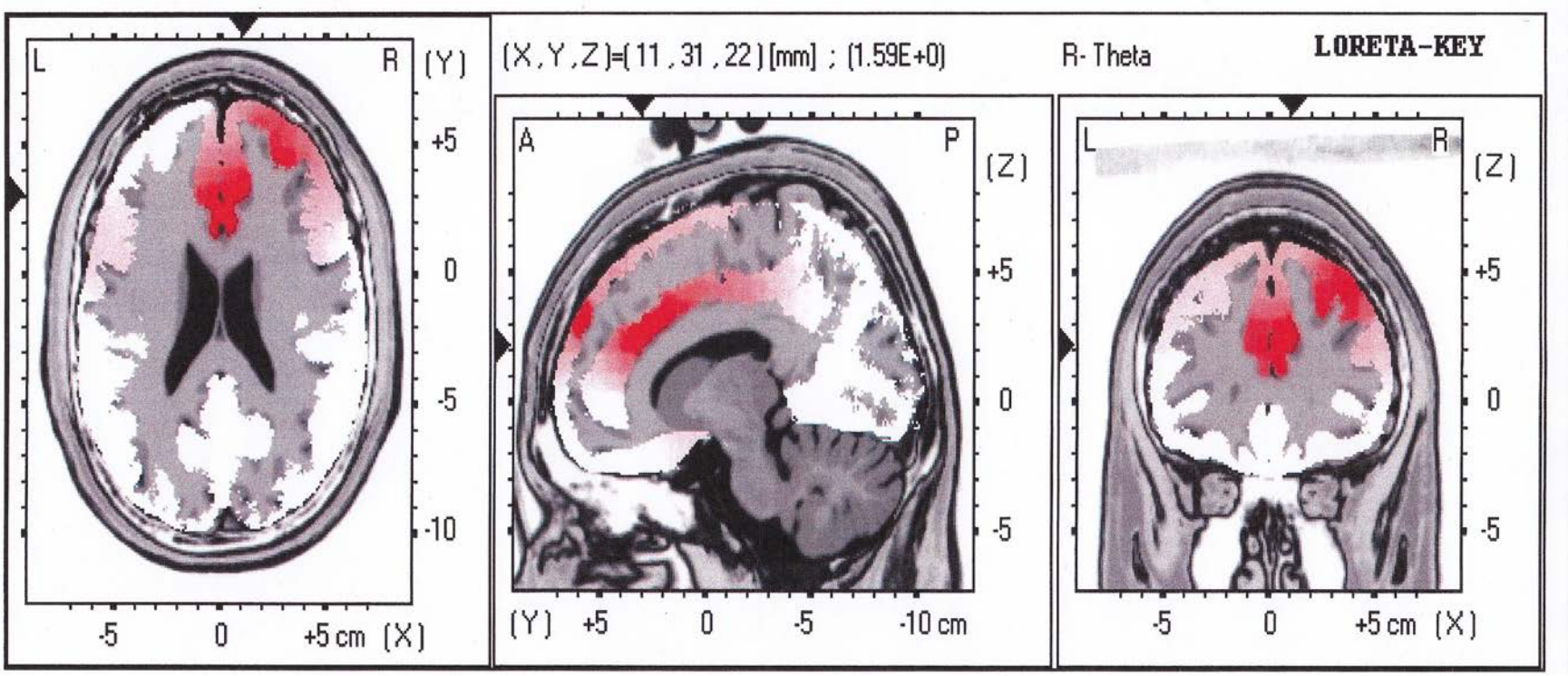
Theta activation. When the *left* eye of the participant was open during exposure to the photograph of the most-triggering face, LORETA analyses revealed that theta activation originated in (primarily posterior) cingulate gyrus (Brodman Area 23; see Figure 1). Following the OEI session, activation occurred in anterior cingulate cortex (Brodman Area 24; see Figure 2). When the *right* eye of the participant was open and the most-triggering face was being perceived before treatment, it appeared that theta activity originated in the cingulate gyrus (Brodman Area 23; see Figure H1). After the OEI treatment, under the same conditions, theta activation appeared to originate in the insula (Brodman Area 13, see Figure H2).

Alpha activation. When the participant had her *left* eye open during exposure to the most-triggering face prior to the OEI session, alpha activation originated in the hippocampus (Brodman Area 37; see Figure 3). In the same condition after the OEI intervention, the source of alpha activation shifted to the inferior temporal gyrus (Brodman Area 37; for detail image, see Figure 4). When the *right* eye was open during exposure to the photograph of the most-triggering face prior to the OEI session, the findings indicated that alpha activity originated in the fusiform gyrus (Brodman Area 37; see Figure H3). Following the OEI session, under the same conditions, alpha activation seemed to originate in the superior temporal gyrus (Brodman Area 41) and middle temporal gyrus (Brodman Area 21, 22; see Figure H4).



A B C

Figure 1. Images of theta activation in Cingulate Gyrus with the male face triggering the strongest transference response when the left eye of the participant was open in Phase I using LORETA-KEY ©® Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in $\mu A/mm^2$.



A B C

Figure 2. Images of theta activation in Anterior Cingulate Cortex with the male face triggering the strongest transference response when the left eye of the participant was open in Phase IV using LORETA-KEY ©®Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in μA/mm^2.

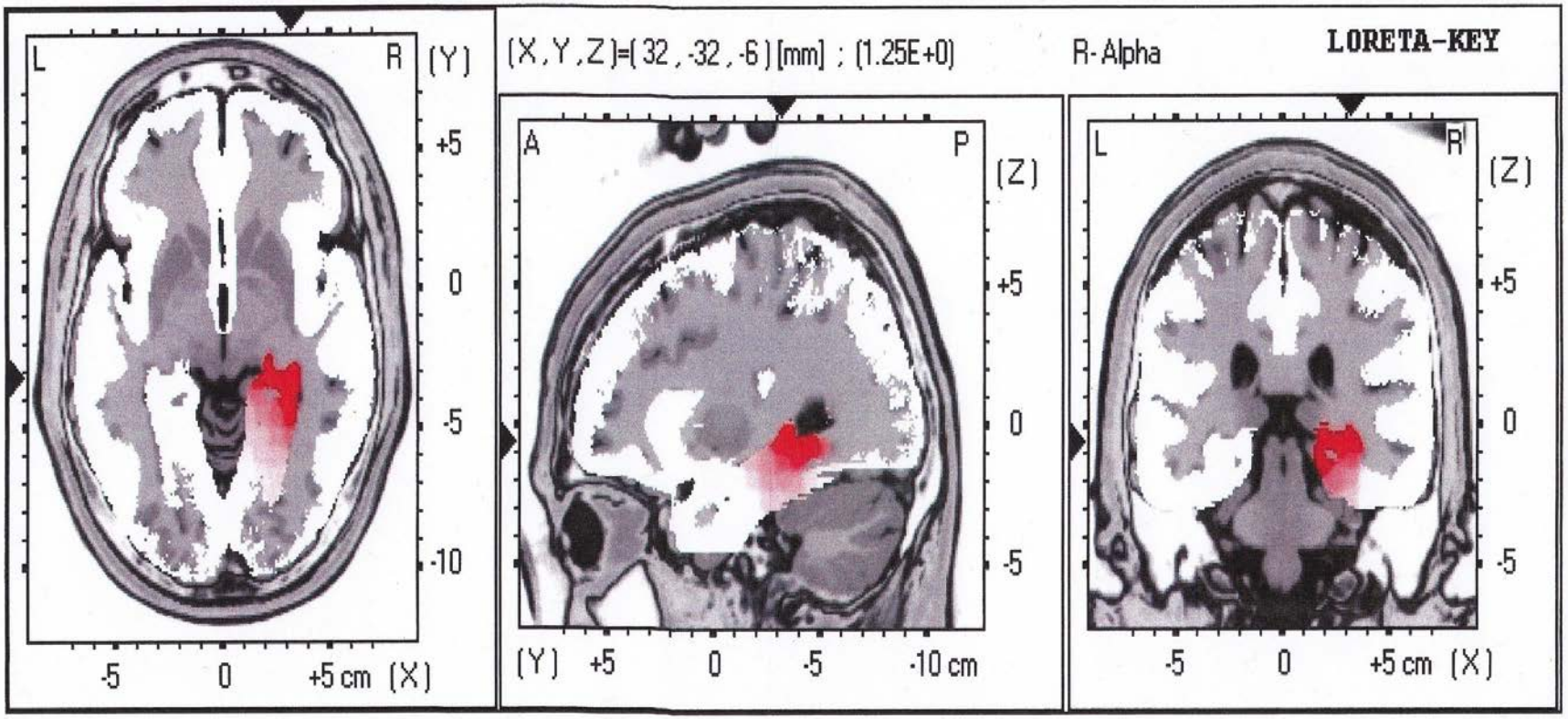


Figure 3. Images of alpha activation in right Hippocampus with the male face triggering the strongest transference response when the left eye of the participant was open in Phase 1 using LORETA-KEY ©®Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in μA/mm^2.

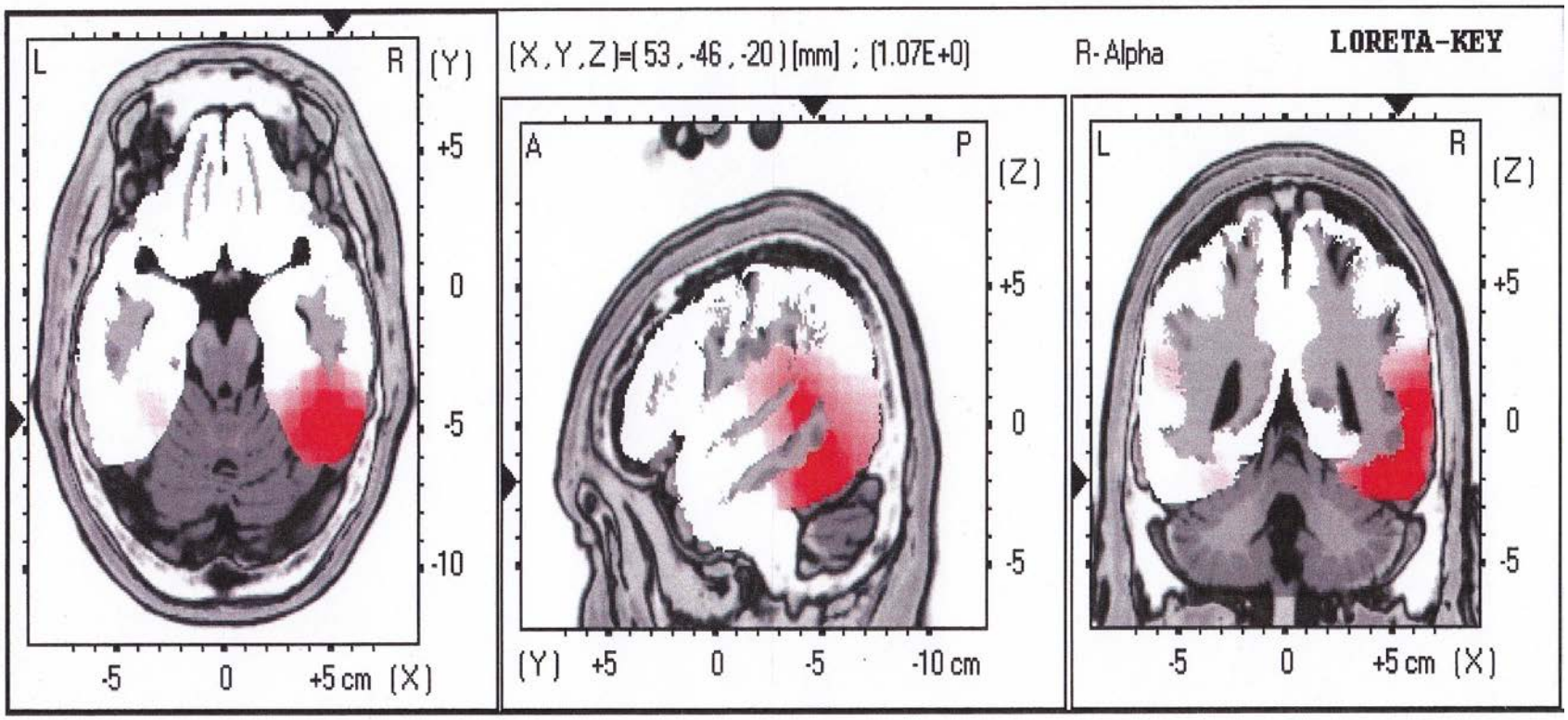


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Figure 4. Images of alpha activation in right Inferior Temporal Gyrus with the male face triggering the strongest transference response when the left eye of the participant was open in Phase IV using LORETA-KEY ©® Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogocal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in $\mu A/mm^2$.

Beta activation. When the participant's *left* eye was open during exposure to the photograph of the most-triggering face, it was found that prior to the OEI session beta activation originated in middle frontal gyrus (Brodman Area 9; see Figure 5). Under the same condition following treatment, the lingual gyrus appeared to be the source of beta activation (Brodman Area 17; see Figure 6). When the participant's *right* eye was open during exposure to the photograph of the most triggering face, LORETA results indicated that prior to the OEI session beta activation originated in the middle frontal gyrus (Brodman Area 9; see Figure H5). Under the same condition following OEI treatment, activation appeared to originate in the precuneus and cuneus (Brodman Area 31; see Figure H6).

These LORETA findings demonstrate shifts in the origins of cortical (qEEG) activity from pre- to post-treatment with OEI. The range of structures involved includes cortical and subcortical areas involved in memory, visuospatial processing, and emotional processing, in response to changes in negative transference reactions.

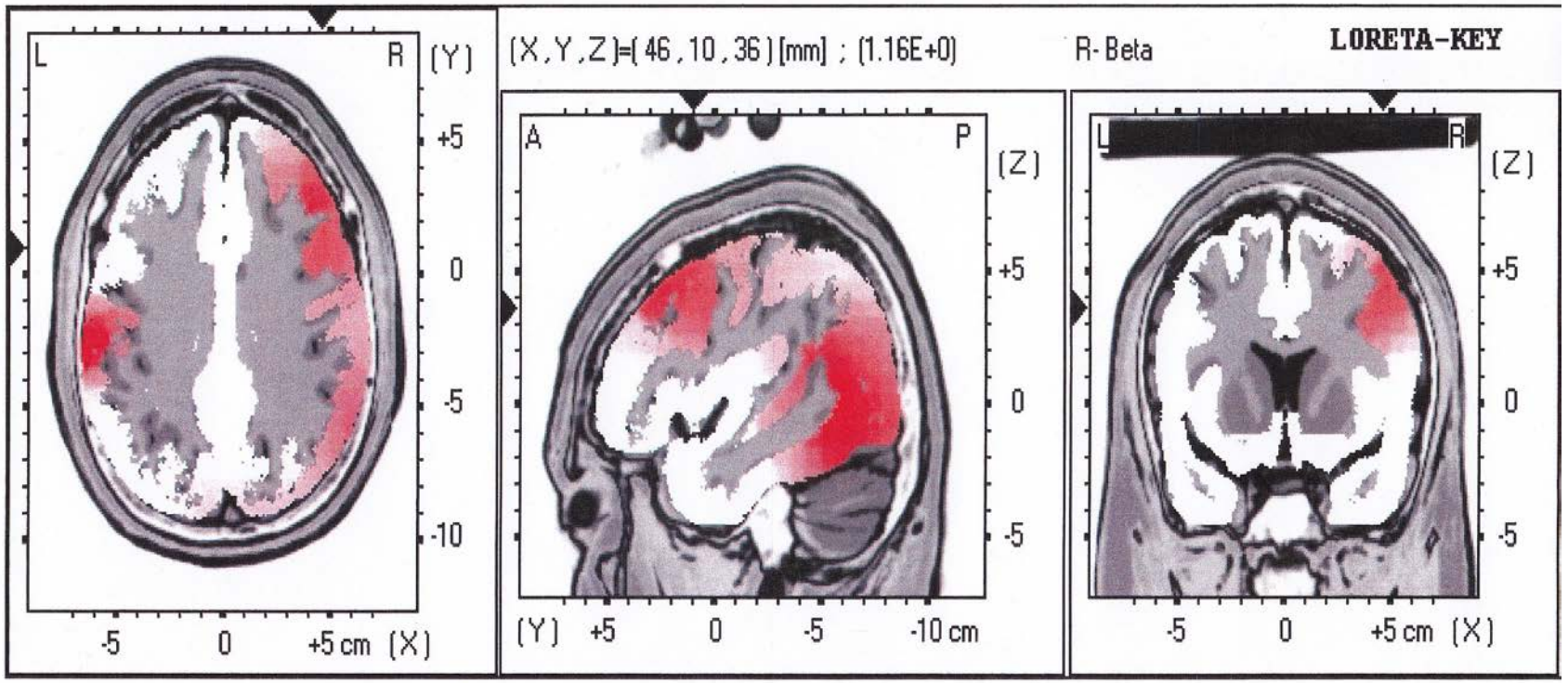

Figure 5. Images of beta activation in right Middle Frontal Gyrus with the male face triggering the strongest transference respone when the left eye of the participant was open in Phase I using LORETA-KEY ©® Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in $\mu A/mm^2$.

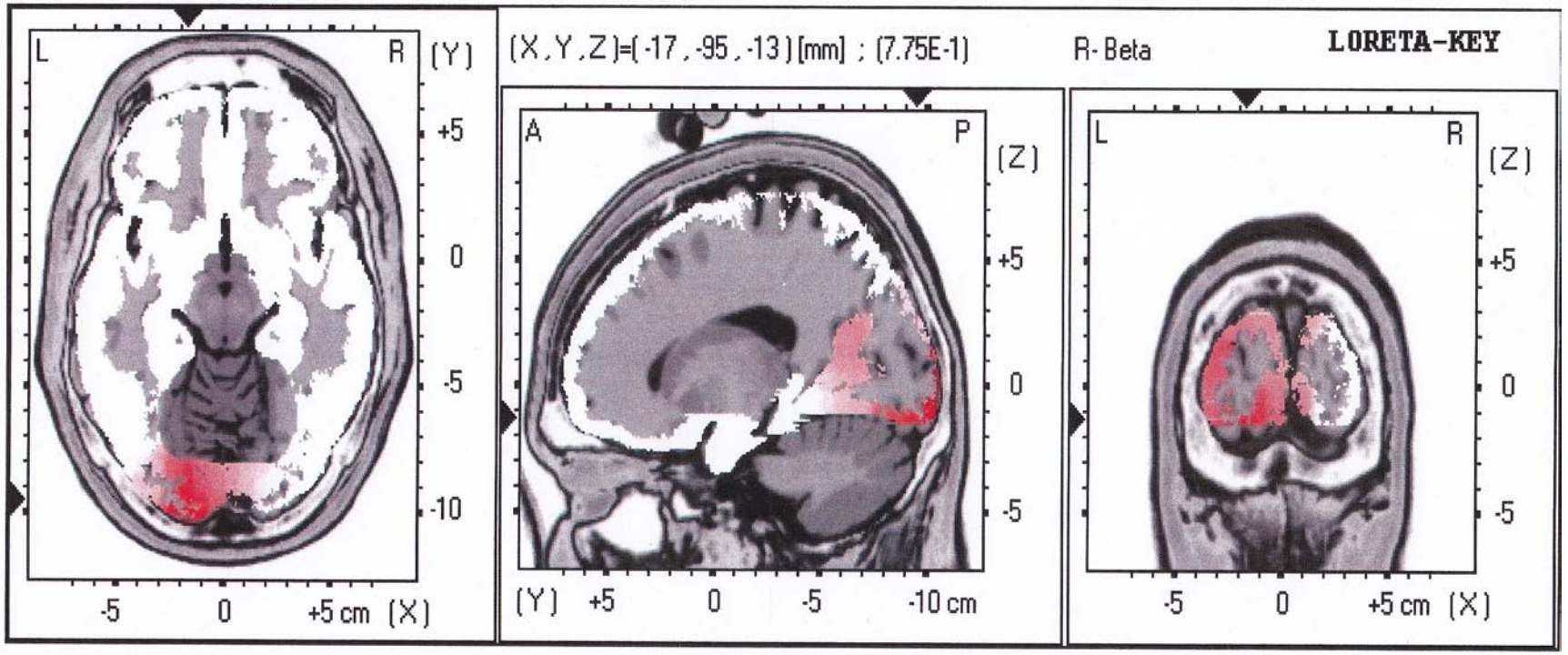

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Figure 6. Images of beta activation in left Lingual Gyrus with the male face triggering the strongest transference response when the left eye of the participant was open in Phase IV using LORETA-KEY ©®Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in μA/mm^2.

CHAPTER 5: DISCUSSION

With the protocol developed and applied in this study, the researcher sought to investigate the neurological concomitants of transference reactions before, during, and after OEI treatment for one participant who exhibited negative transference reactions to one face, particularly with her left eye open. This pilot study was exploratory in nature, intended as an initial test of the protocol. LORETA analyses revealed some overlap with neuronal responses typically associated with individuals suffering from PTSD, and some findings that differed in location from those typically seen in participants with PTSD. The single 90-minute session of OEI appeared to result in major changes in transference reactions, as evidenced in quantitative, qualitative, and psychophysiological findings.

LORETA Changes from Pre- to Post-treatment Assessments

During the pretreatment triggering condition, the participant reported intense somatic symptoms. The authors of numerous other studies report evidence of heightened physiological responses during pretreatment triggering conditions (i.e., exposure to trauma-related imagery), including increases in heart rate, blood pressure, and skin conductance in individuals with PTSD (Carson et al., 2000; Keane et al., 1998; Shin et al., 1997). Investigators in other studies report that somatoform dissociation during triggering of traumatic material is potentially correlated with PTSD symptoms (Näring & Nijenhuis, 2005; Nijenhuis, van der Hart, Kruger, & Steele, 2004).

LORETA analyses. In the present study, LORETA was applied to localize the origins of electrical activity on the scalp in response to a selected visual stimulus (i.e. photograph of the most-triggering face). More negative transference reactions were exhibited when the participant had her ‘left eye open.’ For that reason, discussion in the

following section pertains to comparative pre- to post-treatment brain activations in the 'left eye open' condition versus the 'right eye open' condition (see Table 9).

Left eye open. With her 'left eye open', pretreatment theta activity originated in the *cingulate gyrus*. This brain area is ventral to the surface of the cerebral cortex but dorsal to the midbrain, primarily posterior to the prefrontal cortices (Swingle, 2008). At posttreatment, theta waveforms appeared to originate from the *anterior cingulate gyrus* (ACC). The ACC is part of the frontal lobe, which is involved in both cognitive and emotional processing (Fonzo et al., 2010; Lanius, Bluhm, Lanius, & Pain, 2006; Phillips, Drevets, Rauch, & Lane, 2003). In addition, according to Chen, Li, Xu, and Liu (2009), there are several neuroimaging studies which show that the ACC and the occipital lobe are involved in the retrieval processes of memory. Research results indicate that people with PTSD have *diminished* ACC volume when cued with emotional stimuli (Damsa et al., 2008; Hou et al., 2007; Shin et al., 2006; Woodward et al., 2006). Decreased blood flow in response to emotional stimuli was found in the ACC (Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Staib et al., 2003). According to Hou et al. (2007), "A deactivation of the anterior cingulate gyrus could explain the inability of patients with PTSD to extinguish fear reactions associated with conditioned stimulus when the unconditioned stimulus is no longer occurring" (p. 168). Still other studies have observed *greater* activation of the ACC in participants with PTSD in response to traumatic script-driven imagery than in control group participants (Lanius et al., 2002, 2004; Liberzon et al., 1999). Bergmann (2000) suggested that increased activation in ACC through bilateral stimulation facilitated the processing of traumatic memories into semantic and other cortical networks. From the above discussion, it is apparent that some

discrepancies exist in the results. Thus, activation in the ACC at posttreatment suggests that this brain structure aided the processing of both affective and cognitive aspects of information. Such integration likely prevented the participant from inaccurately perceiving the incoming sensory stimuli as a potential threat.

When the participant was exposed to the most-triggering face prior to OEI treatment, alpha activation appeared to originate in the *hippocampus.* Results from numerous research studies provide evidence of *decreased* hippocampal volume in individuals with PTSD (Bremner, 2006; Shin et al., 2006; van der Kolk, 2001; Wignall et al., 2004). Furthermore, smaller hippocampal volume has been associated with deficits in verbal declarative memory, which leads to inability to accurately identify signals of potential threat under stressful conditions (Bremner, Staib, et al., 1999; Shin et al., 2006). Additionally, alpha waveforms are characteristics of visualization responses (Swingle, 2008, 2009). Findings in the current study show pronounced alpha activation in the right hippocampus when the participant was exposed to the photograph of the most triggering face. This suggests her visual response to the most-triggering face was associated with her experience of fragmented *visual memories* associated with the triggering face. It is plausible that these memories manifested in the participant's negative transference reactions, which typically involve perceptual and affective overlays of past onto present experiences.

After OEI treatment, alpha activation appeared to originate in the *inferior temporal gyrus* on the right. Since the inferior temporal gyrus and the fusiform gyrus have the same Brodman area (37), readers are referred to research findings pertaining to the fusiform gyrus (discussed in pretreatment assessements in the 'right eye open'

condition). These findings suggest that the temporal lobe and limbic structures underlie neuronal responses in PTSD. According to Leon-Carrion et al. (2006), activation in the right inferior temporal gyrus is associated with facial *recognition*. In other words, from pre- to post-treatment, there was a shift in activation from the brain region associated with *distant* visual (facial) *memory* (i.e., the right hippocampus) to the region associated with *current* facial recognition (i.e., inferior temporal gyrus).

Beta activation was most pronounced in the right *middle frontal gyrus* during the 'left eye open' condition of the pretreatment assessment. The middle frontal gyrus is involved in encoding and retrieval of verbal memories; with encoding on the left and retrieval on the right (Tulving et al., 1994 as cited in Bremner, Narayan et al., 1999). Results from previous studies indicate that there was *less* activation in the medial or middle frontal gyrus in PTSD participants when they were cued with emotional stimuli rather than neutral ones (Hou et al., 2007; Lindauer et al., 2004). Other researchers found *increased* activation in the middle frontal gyrus during the retrieval of emotionally valenced words (Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Staib et al., 2003; Flatten et al., 2004). Evidently, variability exists across cases in terms of activation within this same brain structure. Activation of the middle frontal gyrus at pretreatment suggested that the participant was engaged in the process of encoding and retrieving memories, possibly related to the photograph of the most-triggering face.

When the 'left eye' of the participant was open, beta activity occurred in the *lingual gyrus* at posttreatment. The lingual gyrus plays an important role in visual memory, particularly memory of faces (Kapur, Friston, Young, Frith, & Frackowiak, 1995). Results of a study by Bremner, Staib et al. (1999) revealed that *increased* blood

flow in the lingual gyrus was found in participants with PTSD symptoms when they were exposed to traumatic stimuli. At posttreatment, the shift in activated brain area to the occipital lobe or visual association cortex suggests that the participant was reminiscing visual images of upsetting events and faces (Bremner, Narayan et al., 1999). There are very few studies involving activation is this brain area. There is a need for future elucidation of the contributions of the lingual gyrus in the neurocircuitry of PTSD.

Right eye open. After the OEI intervention, it appeared that there was a shift in theta activity from the cingulate gyrus to right *insula*. Research suggests that anterior insular cortex and ACC are involved in limbic sensory processes. These structures are associated with the sensing of internal physiological states (e.g., pain, temperature, itching, and tickling) and emotional states (e.g., anger, fear, and disgust) in the immediate present (Craig, 2009; Fonzo et al., 2010; Simmons et al., 2008). More specifically, the insula plays a crucial role in fear responses with respect to symptom-provoking stimuli (Bremner et al., 2004). Various findings suggest that activation in the insula is associated with working memory, emotional distress, anticipatory processing, and regulation of autonomic arousal; thereby signaling the need to initiate self-preservative actions (Chen et al., 2009; Etkin & Wagner, 2007; Fonzo et al., 2010; Simmons et al., 2008).

In two studies of women exposed to fearful or angry faces, those with PTSD were found to show *increased* activation in the anterior insula relative to comparison subjects when they were processing emotional faces (Fonzo et al., 2010; Simmons et al., 2008). In other studies, hyperactivity has been reported in the insula for participants with PTSD (Etkin & Wagner, 2007; Nagai, Kishi, & Kato, 2007; Rauch et al., 1997). In another brain imaging study, greater activation was found in the insula when participants tried to

suppress their responses to negative stimuli (Butler & James, 2010). It appears that hyperactivity in the insula reflects activation of the neuronal network responsible for generating fear responses to negative emotional cues (Etkin & Wagner, 2007). Simmons, Strigo, Mathews, Paulus, and Stein (2009), however, found that women with PTSD showed *less* activation in the right anterior insula than women in the nontraumatized control group, which implies a mismatch between cognitive and interoceptive states. They suggested that "individuals with PTSD are cognitively aware of an impending shift in physiological/emotional state; they fail to appropriately activate neural circuitry in generating preparatory interoceptive changes" (p. 375). In a recent study on declarative memory, it was found that there was less activation in PTSD participants (relative to individuals in the control group) when they were performing encoding and retrieval memory tasks, suggesting that the insular cortex may be involved in declarative memory via the hippocampus (Chen et al., 2009). In the present study, therefore, theta activation in the insula at posttreatment, in response to the most-triggering face, suggests that the OEI intervention increased the participant's ability to sense her own internal physiological and emotional states. Increased activation in the anterior insula engenders increased capacity to remain aware of oneself, others, and ones' environment. This suggests that in the current study, the participant had an increased activation of the neural correlates of awareness and improved retrieval and depth of encoding, and concomitant improvements in declarative memory in response to OEI treatment (Chen et al., 2009; Craig, 2009; Critchley, Wiens, Rotshtein, Öhman, & Dolan, 2004).

When the 'right eye' of the participant was open during exposure to the photograph of the most-triggering face prior to OEI treatment, alpha waveforms appeared

to originate in *fusiform gyrus*. In the right hemisphere, the fusiform gyrus is involved in the processing of facial images (Haxby et al., 2001; McCarthy, Puce, Gore, & Allison, 1997; Rossion et al., 2000). Research results indicate that activation in the fusiform gyrus occurs when participants view human faces with particular expressions (e.g., angry versus neutral). This suggests that this region is involved in initial orientation, gradual habituation, and activation recovery associated with the viewing of faces (Britton, Shin, Barrett, Rauch, & Wright, 2008).

Results of one study of women with PTSD showed *decreased* blood flow in the fusiform gyrus during retrieval of emotionally valenced words, suggesting that these women may be trying to dispel visual images associated with their traumatic events (Bremner, Narayan et al., 1999; Bremner, Vermetten, Southwick, McGlashan, Staib et al., 2003). In another study, Jatzko, Schmitt, Demirakca, Weimer, and Braus (2006) reported that, in comparison to members of a chronic PTSD group, those in the control group showed *increased* activation in the left fusiform gyrus when they viewed positive emotional visual stimuli. Morey et al. (2009) demonstrated that there was greater activation of the fusiform gyrus in soldiers with PTSD than in those without PTSD during exposure to combat stimuli, relative to noncombat distractors. Results of past imaging studies have shown that the left fusiform gyrus is typically activated when participants are attempting to suppress recall of negative rather than neutral memories (Butler & James 2010). From results of these studies, it can be inferred that activation in the fusiform gyrus during perception of a triggering facial photograph is associated with intrusive emotional recollections and hypervigilant sensory reactions associated with traumatic memories. This pattern of response is consistent with PTSD symptoms (Butler

& James, 2010; Morey et al., 2009). Extreme vigilance may place increased demands on brain areas involved with (a) visuospatial aspects of memory, and (b) planning of responses to potentially threatening stimuli (Bremner, Narayan et al., 1999). Thus, alpha activation in the fusiform gyrus at pretreatment suggests that the participant was involved in the initiation, habituation, activation, and suppression of memories associated with negative transference reactions to the most-triggering face.

At posttreatment, alpha activation was found in the right *superior and middle temporal gyri.* The middle temporal cortex plays a crucial role in fear extinction through inhibition of the amygdala (Bremner, Staib et al., 1999). In normal volunteers these regions are associated with processing and retrieval of autobiographical memories (Fink et al., 1996; Markowitsch, 1995).

Studies have revealed that participants with PTSD show *greater* activation in middle and superior temporal gyri in reaction to neutral scripts than to trauma scripts, compared to activation patterns observed in members of a control group who had experienced prior traumas but were not diagnosed with PTSD (Lindauer et al., 2004). In an fMRI study by Lanius et al. (2002), participants with PTSD who experienced dissociation in response to traumatic script-driven imagery had higher levels of activation in the superior and middle temporal gyri. Increased activation in the superior temporal lobe of a participant with acute PTSD was also reported some studies (Bremner et al., 2004; Flatten et al., 2004). Increased activation in the middle temporal gyrus of a woman with PTSD was found during retrieval of emotionally valenced words (Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Staib et al., 2003).

After OEI treatment, there was a clear difference in brain activation (from the fusiform gyrus to the superior and middle temporal gyri) following confrontation with the most-triggering face. These findings support the notion that during processing of negative transference reactions, OEI treatment leads to differential activation of brain regions. However, it appears that the shifts in brain areas from deeper levels at pretreatment to cortical areas at posttreatment signifies that the participant was engaged in the process of retrieving and encoding autobiographical memories (i.e., she was 'making sense' of memories; Fink et al., 1996; Markowitsch, 1995).

At pretreatment, there was activation in right *middle frontal gyrus* (discussed in pretreatment assessements in the 'left eye open' condition). However, at posttreatment, activation was found to originate in the posterior cingulate cortex (PCC) or *precuneus* and *cuneus*. The PCC, along with other brain structures, is involved in visuospatial processing, assessment of threat, and self-referential processing (Moran, Macrae, Heatherton, Wyland, & Kelley, 2006; Nemeroff et al., 2006). In response to the emotional Stroop task, *decreased* blood flow was found in the precuneus among participants who had been abused but did not have PTSD (Bremner et al., 2004). Research results revealed *decreased* activation in the cuneus among participants with PTSD relative to control group participants in response to the colour Stroop test (Bremner et al., 2004), but another study found an *increased* activation in this area (Lindauer et al., 2004). According to Bremner et al. (2004) "The visual association cortex and cuneus are involved in making visual associations and processing visual imagery and memory. Altered function in these regions might represent a neural correlate of alterations in visual imagery in PTSD" (p. 617). From this discussion, it is

clear that there are inconsistencies among research results. Nevertheless, activation in these areas (precuneus and cuneus) suggests that the participant was engaged in visual processing associated with the most-triggering face.

In summary, the above discussion of cortical areas revealed in LORETA analyses provides a start at interpreting activations of different brain regions before and after OEI treatment. Clearly, there exist some apparent inconsistencies among findings from studies. These inconsistencies in results across studies may be due to individual participant characteristics, differences in imaging technologies, heterogeneity in task designs, duration of PTSD symptoms, extent of comorbidity, or analytic approaches. Contributions of these brain areas to the neuronal networks of individuals with PTSD needs further clarification in future studies.

Treatment assessments. During the treatment process, the participant experienced perceptual distortions with her left (nondominant) eye. These distortions were associated with prominent somatic symptoms such as nausea, heart palpitations, and "heaviness" in her legs. At the beginning of the OEI treatment, she experienced physical agitation and anxiety. By the end of the treatment, she reported significant relief from her physical discomfort and distress, but also experienced a sense of "emotional disconnection". The reported reductions in severity and intensity of her somatic distress are in accordance with what is typically observed in response to OEI treatment (Bradshaw et al., 2011).

Comparisons of results from this study with results from previous studies. As discussed in Chapter 4, the participant in the current study exhibited a brainwave profile characteristic of individuals with low distress tolerance, and interpersonal

passivity (low theta/beta ratios at O_1 and hibeta/beta ratios at F_z). When compared to findings from a previous qEEG study of trauma survivors with PTSD (Faas, 2009), it appears that the participant in the current study had a qEEG profile characteristic of *simple PTSD*, more likely involving adult onset-traumas with little observed dissociation in the nontriggering condition. In future qEEG studies of trauma survivors, it will be important to discern early in the research which PTSD subtype each participant most closely resembles, using Faas' (2009) typology (simple PTSD, complex PTSD, or dissociative PTSD-readers unfamiliar with these subtypes, and the associated qEEG patterns, are referred to Appendix L). Such information will likely be helpful in delivering the most appropriate dosages and treatments depending on participant subtypes.

Professional Implications

Directions for continued research protocol development. Results of this study offer relevant findings for future researchers in Counselling Psychology. Most importantly, the findings provide support for the efficacy of OEI in relieving negative transference reactions (including perceptual distortions with faces), and suggest how brain areas are affected by such therapeutic interventions. Such an evidence-based approach opens several new doors for development of protocol for neurophysiological research related to transference. It is hoped that in the near future such neurophysiological assessments might be accepted by representatives of managed care institutions who seek evidence-based treatments. Many clients could benefit from such a neurophysiological approach to trauma therapy.

This case-based study included an OEI clinician, who was able to hold negative transference reactions for approximately 30 seconds; which seems to be unachievable in

the real world. This may not be appropriate for trauma clients with severe, prolonged, or early-onset relational traumatization, since such individuals would likely be unable to tolerate negative transference reactions for longer than 5-10 seconds (in extreme cases, such clients have even lost consciousness briefly during OEI transference procedures). Such profound and intense transference reactions are also typically associated with other 'side effects' (referred to in OEI as "dissociative artifacts" or "core trauma symptoms"). For this reason, although the inclusion criteria for the study (i.e only OEI clinicians) greatly limited the generalizability of the findings, they were warranted in this study and should be applied in future studies (at least during initial explorations) of OEI transference reactions for ethical reasons.

Implications for clinical practice. The current findings have several implications for clinical practice. Firstly, qEEG and LORETA analyses at each phase of the study (baseline, protocol development & testing, pretreatment transference assessments, treatment assessments, and posttreatment transference assessments) offer valuable information to clinicians regarding OEI therapy process and outcome. For any client, it is evident that varying degrees of transference projections will occur, depending on the facial characteristics, gender, age, hair, and eye colour in the photographs or video footage. Secondly, the manifestation of somatoform dissociative symptoms during this study should alert clinicians to make inquiries of their clients regarding the presence of such symptoms during psychotherapy, since these may not be readily observable by therapists. Such developments can be very challenging clinically, resulting in self-report measures that appear unusual or inconsistent (e.g., clients who report feeling less anxiety emotionally, but more physical numbness or pain). However, combining psychometric

self-report measures with qEEG trauma signatures assessments can illuminate this problem considerably. Thirdly, such simple and brief treatment techniques as OEI transference checking and clearing may be particularly useful in heterogeneous groups where individuals can pair up in dyads with other group members and leaders prior to the first full group meeting. This has been done by psychotherapy group leaders, and has been found to greatly facilitate in resolution of negative transference reactions between group members and between members and leaders. Responses are based on individual differences in experience, and visible personal characteristics (e.g., height, weight, facial hair, eyeglasses, race, and skin colour). These OEI techniques can easily be added to, and combined with, other empirically-based treatments for psychological disorders.

Limitations and Future Directions

Limitations of the current study. The *results* of this study should be viewed in light of the *limitations* of the study design. These include the small sample size ($N = 1$) and the lack of formal assessments for PTSD and/or common comorbid diagnoses (e.g., major depressive disorder, dissociative disorder, or substance use disorder), which weakens the generalizability of results of the study. Formal assessments were not performed in the present study, partly because this investigation was intended to be (a) an initial protocol development and test, and (b) an initial neuropsychological exploration of both negative transference reactions and OEI transference checking and clearing procedures. Future studies using this protocol could include assessments for PTSD and common comorbid disorders such as depression.

This study involved one female participant with mild to moderate experiences of psychological trauma (rather than early, severe, or prolonged trauma), and she received

only one session of OEI. The findings were not complicated by gender, age, culture, or race differences between participants. Likewise, there were no variations in types, degrees, or developmental timelines of traumas, since there was only one participant. Such criteria obviously limit the generalizability of results, but for this exploratory pilot investigation such simplicity was an asset.

Another limitation is that the sample population from which the participant was selected included only (a) OEI clinicians who had experienced negative transference reactions, and (b) Counselling Psychology faculty members at Trinity Western University, in Langley, BC. The study therefore lacks adequate representation of the more general population with respect to age, gender, ethnicity, and trauma-related experiences. Further investigation will be essential to determine whether the findings of the present study can be replicated with, and generalized to, other participants.

In the experimental protocol, the same photograph of the most-triggering face was used at pretreatment, during treatment, and at posttreatment to evaluate the nature and intensity of transference reactions. Although, stimulus consistency was essential to evaluate treatment outcome, it may have resulted in habituation to the visual stimulus. Additionally, it is also possible that because the participant was an OEI clinician and was aware of OEI procedures and typical characteristics of transference reactions, she may have responded in ways that she thought the researchers expected her to respond. This was only a consideration with regard to qualitative interview findings, since it would be much less likely that the participant was capable of intentionally generating responses recorded during qEEG assessments.

Treatment outcome was assessed using self-report measures. Some readers might consider this is a major concern, because the re-experiencing of a traumatic event (associated with a negative transference reaction) might make it difficult for the participant to accurately recall the event and rate the items accurately (McInnes, 2007). Another limitation of the present research design is that it precludes drawing causal inferences. The absence of a comparison group (or an individual) rules out many statistical analyses; especially those that involve inferential statistics.

Recommendations for future studies. The first priority should be to repeat the research protocol with larger group of cases. This will help identify dimensions or factors involved in negative transference reactions and neurobiological concomitants of such experiences. Assessment of transference reactions across at least six individuals may suggest major implications for future clinical assessment and treatment of negative transference reactions. Thus, future research studies should incorporate larger group sizes and eventually include assessment of comparison treatments as well (e.g., CBT, or EMDR).

Visual stimuli such as facial photographs may be less likely to lead to habituation than other visual stimuli, because they possess inherent and persistent strength. Although they are commonly encountered in daily life, they can still trigger negative transference reactions (visual projections or distortions). In this study, familiarity with visual stimuli could have led to stimulus habituation; therefore future protocols should include presentation of a neutral face after each exposure to the most-triggering face. Additionally, visual stimuli were also presented in a fixed order, which may have

contributed to an order effect (Bremner, Staib et al., 1999). In future studies, stimulus order should be randomized to prevent such potential confounds.

To simplify data collection and analysis for each participant, it is suggested that the future research protocol include only responses to *least triggering* and *most-triggering* faces. A series of photographs could be shown to each participant prior to the start of the formal study, to facilitate selection of the most- and the least-triggering faces for each participant.

Somatic numbing was *qualitatively* reported by the participant during this study. For that reason, it is recommended that the Somatoform Dissociation Questionnaire (SDQ-20; Nijenhuis et al., 2004) be added to future studies using this protocol. This will enable future researchers to *quantitatively* assess symptoms of both somatoform and psychoform dissociative symptoms more precisely. Also, since the TRR is an important assessment tool in this protocol, it will be important to establish its reliability and validity during a future study. However, the measure was important to include for comparisons between pre- and post-treatment characteristics of transference reactions (or lack thereof) in this initial exploratory pilot study. No formal assessment of treatment fidelity was completed in this initial pilot study, other than debriefing of the therapist by one of the co-developers of OEI; however, future studies using this protocol should include formal evaluation of treatment fidelity by one of the OEI co-developers or an OEI master trainer.

Finally, EEG technology has limited spatial resolution, and provides only coarse distinctions between anterior/posterior and left/right cortical activations (Rabe et al., 2008). Further understanding of the mechanisms and efficacy of OEI treatment for negative transference reactions is needed, involving both qEEG/LORETA and MEG

technologies in order to permit finer distinctions between left/right and anterior/posterior activations. This study included LORETA evaluation of only theta, alpha, and beta activations in different brain areas. It is suggested that future studies include delta, hibeta, and even gamma activations as well, because these waveforms are crucially important for assessing somatic symptoms and obsessive-compulsive tendencies (Swingle, 2009).

In summary, additional studies are needed to verify, clarify, and extend the findings of the present study. The exploratory nature of the pilot study has certainly provided some interesting and relevant findings, although it will be crucially important to replicate the study with a much larger population, more flexible inclusion/exclusion criteria, and more than one OEI session.

Conclusions

According to van Der Kolk et al. (1997), when an individual receives sensory input from a personally significant event, he or she is able to transcribe these sensations into personal narratives; however, traumatic experiences get stored in the form of intense somatic and emotional representations with impaired semantic processing. Results of this pilot study suggest that visual distortions and other experiences associated with negative transference reactions can be reduced through a new therapy known as Observed & Experiential Integration (OEI). These treatment procedures seem to be associated with changes in cortical representations of transference phenomena, such that visual distortions are reduced, along with reductions in concomitant disturbing affective and somatic symptoms.

The last decade of neuroimaging research has provided important information regarding brain functioning in individuals with PTSD. Results of the present study

contribute to greater understanding of dysfunctions in emotional processing, particularly associated with selected facial images. These distortions may or may not be directly associated with PTSD. Results of the present study offer *preliminary* neurobiological information regarding the phenomenon of negative transference reactions. The differences observed clinically and psychophysiologically between one participant's responses *before* versus *after* OEI treatment suggest neuronal mechanisms that underlie these two distinct states and reactions. Future studies with larger samples will be necessary to replicate these findings and further clarify the neural correlates (i.e., brain localizations) associated with negative transference reactions, pre- and post-treatment. Despite the limitations of this initial case study, the results provide preliminary evidence of the effectiveness of OEI for resolving negative transference reactions, which shows to be clinically promising.

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APPENDIX A: Email and Oral Recruitment Announcement for the Study

Are you interested in finding out more about brain activation during transference projections (perceptual distortions)?

"**Development and pilot testing of a protocol for assesing negative transference reactions duing Observed and Experiential Integration using Electroencephalography and Low resolution brain electromagnetic tomography".**

Purpose: Development and Pilot testing of a protocol to assess transference phenomena via qEEG assessment of cortical activity. Participants will be gazing at visual stimuli and will be observed pre- and post-treatment.

Who: Clinicians who have completed OEI training and have experience providing treatment. We are interested in individuals who know they have in the past experienced transference projections in counselling sessions.

When: One full day to be scheduled between July 15 and August 15, 2010.

Where: Offices of Dr Paul G. Swingle (Swingle & Associates), Neurotherapy Clinic on Melville Street, Vancouver, BC, Canada.

Interested participants are requested to contact Mahima Jacob through email or phone. Selected participants will be screened and contacted through email.

Email: mahima.jacob@mytwu.ca
Phone: 604-996-4537

For further concerns and questions, please contact Dr. Rick Bradshaw via email or phone.
Email: rickphyl@telus.net
Phone: 604-513-2121 (ext 3382)

APPENDIX B: Research Ethics Board (REB) Certificate

APPENDIX C: Qualitative Interview Questions

BASELINE ASSESSMENTS

I am going to ask you a couple of questions to help you process the *baseline condition.* Most of the questions are centered on self-awareness; i.e., your thoughts, feelings, and bodily sensations. The intent is to get an understanding of the process and experience from your perspective. After I ask questions I may add additional prompts. If items apply to you, I will ask you to describe your responses more fully. If you don't understand a question, please feel free to interrupt and ask me about it. I will be glad to clarify items for you by giving examples.

As we talk, some personally sensitive thoughts and feelings may emerge. For that reason, it is important to stress the fact that *you* are in charge of how much detail you want to provide. It is totally up to you to determine the depth of information you disclose. As we progress through the questions, if the process is distressing for you or is making you emotionally upset, please let me know. This will help me to understand your experience and help you process any internal struggles.

1. How was the experience of the baseline condition for you [Eyes open/ eyes closed]? [Were you comfortable or uncomfortable with the process? What was the most uncomfortable thing for you?]

   ---
2. Were you able to quickly and easily calm yourself? [Did it take long to get yourself into a relaxed state?]

   ---
3. Did you find it difficult to focus on the green computer screen? [If yes, what were the hindrances to focusing? Was it an event or situation from the day? Were you thinking of a specific person? Relationship issues? Stress? Concerns? Memories?]

   ---
4. What were your feelings and thoughts while you were concentrating on the green computer screen? [Were you anxious or frightened? Uncertain? Having doubts?]

   ---
5. What did you notice about your body, while you were concentrating on the green computer screen? [About your eyes? Heart? Palms of your hands? Stomach? Chest? Abdomen?]

Phase II: PRETREATMENT TRANSFERENCE ASSESSMENTS

I am going to ask you a couple of questions to help you process the *pretreatment phase*. Most of the questions are centered on self-awareness; i.e., your thoughts, feelings, and bodily sensations. The intent is to get an understanding of the process and experience from your perspective. I will first ask questions about the *non-triggering condition,* and then the *triggering condition*. After I ask questions I may add additional prompts. If items apply to you, I will ask you to describe your responses more fully. If you don't understand a question, please feel free to interrupt and ask me about it. I will be glad to clarify items for you by giving examples.

As we talk, some personally sensitive thoughts and feelings may emerge. For that reason, it is important to stress the fact that *you* are in charge of how much detail you want to provide. It is totally up to you to determine the depth of information you disclose. As we progress through the questions, if the process is distressing for you or is making you emotionally upset, please let me know. This will help me to understand your experience and help you process any internal struggles.

A) Non-Triggering Condition

1. What were you experiencing when you were concentrating visually on the non-triggering face on the screen? [Did it bring to mind particular memories, events, or situations? Were the memories pleasant or unpleasant? In the distant past or recent present?]

   ---
2. What was it about the non-triggered face that reminded you of? Was it the facial appearance of the person? Did it make you think of another individual who resembled the face you were looking at? Did it remind you of a particular place or scenario? A dream? A conversation? A special event? A particular piece of music?]

   ---
3. How often have you thought about that person recently? [Every day? Every week?]
   ---
4. How old do you feel when you see the non-triggering face? [Young child? Adolescent? Young adult? Current age?]

   ---
5. What were your reactions or feelings when you were visually concentrating on the non-triggered face? [No feelings? Negative feelings of worry, anxiousness, or fear? Positive feelings of happiness, joy, or love? How intense were those feelings? Mild? Moderate? Severe?]

   ---
6. What did you notice in your body when you were visually concentrated on the non-triggered face ? [Relaxed or tense in your shoulders? Headache? Visual distortions? How intense were those feelings? Mild? Moderate? Severe?]

B) Triggering Condition

Now, I will be asking questions pertaining to the face that triggered you the most.

1. How did you feel when you were asked to concentrate on a face that triggered you in terms of transference projections? [Were you already feeling tense anxious, scared, or emotionally upset, as you were asked to concentrate on triggered face?]

   ---
2. Did the face bring to mind a traumatic event? [Was it easy or difficult to remember an event associated with the face? Was it in fragments or was it whole? Was the recollection vague or detailed?]

   ---
3. What were you experiencing (or feeling) when you saw the triggered face on the screen?

   ---
4. On a scale of 1 to 10 (1 being least and 10 being most distressful), how distressed or uncomfortable did the face cause you to become?

   ---
5. What was about the triggered face that was most distressful? [Was it facial characteristics, particular aspects of appearance, non-verbals?]

   ---
6. How long did the experience last for the triggered face? [Few seconds? Minutes? Never fades away? Only rare flashbacks?]

   ---
7. How much have recollections of the person this triggered face *reminded* you of interfered with your life? [Every moment? Every day? Only in the presence of triggers?]

   ---
8. Assuming that you understand transference projections, were you aware of these occurring in the moment? [If yes, what made you aware of these projections?]

   ---
9. Did you experience any visual distortions, when transference projections were happening? [If yes, have you been aware of similar visual distortions in the past?]

   ---
10. What was the face of the person (that triggered you the most) like with your dominant eye open? With your non-dominant eyes open?

---

11. What were your thoughts and feelings during this transference check? [Scared, angry, frustrated, helpless, hopeless, ashamed, or confused?]

---

12. What were your physical reactions during this phase? [Change in breathing? Heart racing? Sweating? Feeling shaky? Tense in shoulders? Feeling numb? Chest compression? Nausea in stomach? Cold or tingling feet?]

---

13. What signs will indicate to you that you have passed the triggered state? [Calmness in your body? Getting back to what you were doing before you were triggered or controlled by your emotions? Able to talk to the person whose face was shown on the screen? Not being triggered at all?]

Phase III: TREATMENT ASSESSMENTS

I'm going to ask you some questions about the *treatment phase*. Most of the questions revolve around self-awareness; i.e., your thoughts, feelings, and bodily sensations. The intent is to get an understanding of the process and experience from your perspective. After I ask questions, I may prompt you. If any items are relevant for you, I will ask you to describe them more fully. Again, if you don't understand my questions, please feel free to interrupt and ask me. I will try to clarify the intent and meaning by giving examples

1. What was the treatment process like for you? [Relaxing? Stressful? Emotionally upsetting? Severely distressing?

---

2. As you were going through the treatment, did you notice any change in the transference projections *while* gazing at the triggering face on the screen? [How did you perceive the triggered face with your dominant eye open? With your non-dominant eye open?]

---

3. List one prominent feeling you experienced in the statement "At the beginning of the treatment I was feeling……."

---

4. How strong were your feelings? [None? Mild? Moderate? Severe?]

---

5. What was the most difficult part of this process? [Being triggered by the face of the person repeatedly? Intense emotional intensity? Physical sensations?

Talking about the event?]

---

6. What was the most helpful part of the treatment? [Therapist empathy or encouragement? The way the therapist was you in the moment? Therapist's non-judgmental attitude? OEI treatment itself?

---

7. List one prominent feeling you experienced in the statement "By the end of the treatment, I was feeling……".

---

8. How strong were your feelings by the end of the treatment? [None? Mild? Moderate? Severe?]

---

9. Did you have any expectation or desire before starting the treatment? [If yes, express this in a sentence, for example, "I wish I didn't have to deal with that event any more", or " I hope that event doesn't trouble me anymore in the future"]

Phase IV: POSTTREATMENT TRANSFERENCE ASSESSMENTS

I'm next going to ask you some questions to debrief the *posttreatment phase*. Most of the questions are based on comparisons between pre-treatment and post-treatment phases.

A) Triggering Condition

1. After receiving treatment, what was it like for you to concentrate on the face of the person that previously triggered you (transference projections)?

---

2. Did it bring to mind any traumatic event(s)? If so, was it similar in all aspects to the event you were reminded of by that face before you received treatment? Was there any shift in your awareness about the event? [Less distressing? Less emotional or physically intensity?]

---

3. On a scale from 1 to 10 (1 being least and 10 being most distressing), how much distress or uncomfortableness did the triggered face of person caused?

---

4. What, if anything, was still distressing as you looked at the triggered face and/or reflected upon any related event?[Was it the face of the person? recollection of any actions, nonverbal behaviours or silence or some particular facial characteristics?]

---

5. How long did distress last? [A few seconds? Several minutes? It never faded away? It was only distressful during actual flashbacks?]

6.Did you experience any visual distortions? If yes, how strong were the distortions? [Mild? Moderate?Severe?]

---

7. What was the triggering face of the person like with your dominant eye open? With your non-dominant eye open?

---

8. What were you feeling now that you saw the face of the person that triggered you the most? How intense were those feelings? [Mild? Moderate? Severe?]

---

9. What was your physical reaction during the post treatment phase? [Change in breathing pattern? Racing heart? Sweating? Feeling shaky? Tension in your shoulders? Feeling numb? Chest compression? Nausea in your stomach? Cold or tingling feet?

---

10. What sign (s)would indicate (to you) that you had passed the triggered stage? [Calmness in your body? Getting back to what you were doing before you were triggered? Controlling your emotions? Able to think about talking to the person (whose face was triggered by the one on the screen) without being triggered at all?] Would you say that you have acquired that state? If not, what do you think you still need to do? [More therapy sessions, journaling, or other coping mechanisms?]

APPENDIX D:

Clinician-Administered Dissociative States Scale (CADSS)

0= not at all 4= extremely

Subjective Items

(At this time, in this room)

1. Do things seem to be moving in slow motion?	0----1----2----3----4
2. Do things seem to be unreal to you, as if you are in a dream?	0----1----2----3----4
3.Do you have some experience that separates you from what is happening; for instance, do you feel as if you are in a movie or a play, or as if you are a robot?	0----1----2----3----4
4. Do you feel as if you are looking at things from outside of your body?	0----1----2----3----4
5. Do you feel as if you are watching the situation as an observer or spectator?	0----1----2----3----4
6. Do you feel disconnected from your own body?	0----1----2----3----4
7. Does your sense of your own body feel changed: for instance, does your own body feel unusually large or unusually small?	0----1----2----3----4
8. Do people seem motionless, dead, or mechanical?	0----1----2----3----4
9. Do objects look different than you would expect?	0----1----2----3----4
10.Do colors seem to be diminished in intensity?	0----1----2----3----4
11.Do you see things as if you were in a tunnel, or looking through a wide angle photographic lense?	0----1----2----3----4
12.Does this experience seem to take much longer than you would have expected?	0----1----2----3----4
13.Do things seem to be happening very quickly, as if there is a lifetime in a moment?	0----1----2----3----4
14.Do things happen that you later cannot account for?	0----1----2----3----4
15.Do you space out, or in some other way lose track of what is going on?	0----1----2----3----4
16.Do sounds almost disappear or become much stronger than you would have expected?	0----1----2----3----4
17.Do things seem to be very real, as if there is a special sense of clarity?	0----1----2----3----4
18.Does it seem as if you are looking at the world through a fog, so that people and objects appear far away or unclear?	0----1----2----3----4
19.Do colors seem much brighter than you would have expected?	0----1----2----3----4

Observer Items

20. Did the subject seem eery or strange, or in some other way give you an uncomfortable feeling?	0----1----2----3----4
21. Did the subject blank out or space out, or in some other way appear to have lost track of what was going on?	0----1----2----3----4
22. Did the subject appear to be separated or detached from what is going on, as if not a part of the experience or not responding in a way that you would expect?	0----1----2----3----4
23.Did the subject say something bizarre or out of context, or not speak when you would have expected it?	0----1----2----3----4
24. Did the subject behave in a bizarre, unexpected manner, or show no movement at all, being stiff and wooden?	0----1----2----3----4
25.Did the subject have to be put back on track, or grounded in the here and now, during or soon after the experience?	0----1----2----3----4
26. Did the subject show any unusual twitching or grimacing in the facial musculature?	0----1----2----3----4
27. Did the subject show any unusual rolling of the eyes upward or fluttering of the eyelids?	0----1----2----3----4

Items 1-19 are subjective and items 20-27 are objective.

APPENDIX E

TRANFERENCE REACTION RECORD (TRR)

☐ Pretreatment ☐ Post-treatment Participant----------

Indicate your eye dominance (circle one) R	L

Record Transference Reactions

Proximity
(*difference in distance*)

YES

1-----2-----3----4-----5
near *far*

1-----2----3----4----5
near *far*

NO

Appearance
(*perceived state of other person*)

YES

Emotional state (e.g., anger, fear, sad, worry, shame, downcast eyes)

Anger 1------2-----3-----4----5 Sad

Color (e.g., red, green, grey)

Red 1-----2-----3-----4-----5 Light

Emotional state (e.g., anger, fear, sad, worry, shame, downcast eyes)

Fear 1-- - -2---- 3---- 4---- -5 Worry

Color (e.g., red, green, grey)

Green 1--- -2-----3---- 4------5 Dark

NO

Emotions/Sensations
(Your own)

YES

Feelings (e.g., anger, fear, sad, worry, shame)

Fear 1-----2------3-----4----5 Shame

Bodily sensations (e.g., eyes, head, hands, throat, chest, abdomen)

Head 1---- 2----- -3----4---5 Chest

Feelings (e.g., anger, fear, sad, worry,shame

Sad 1-----2-----3-----4------5 Anger

Bodily sensations (e.g., eyes, head, hands, throat, chest, abdomen)

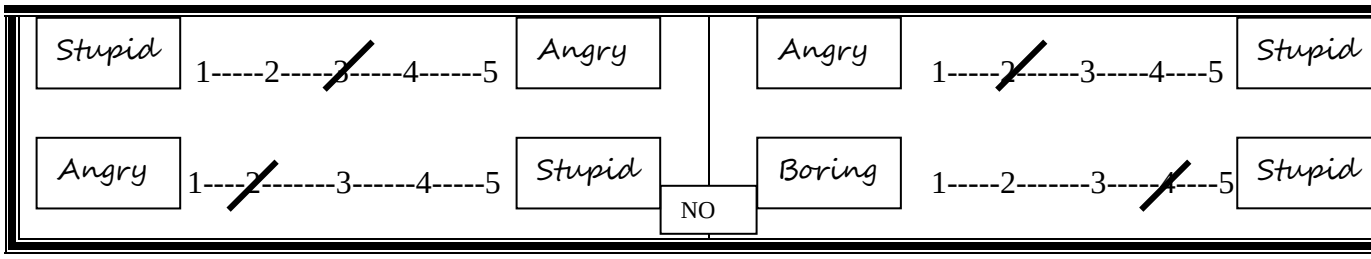
Hands 1-----2------3------4-----5 Throat

NO

Projection of Therapist Thoughts/Feelings
(About you)
(e.g., stupid, boring, angry, irritated)

YES

(e.g., stupid, boring, angry, irritated)



APPENDIX F

Major Steps involved in the Development of the Research Protocol

TIME	VISUAL STIMULI	PROCEDURES	ASSESSMENTS
Baseline condition	Computer screen	3 minutes eyes open and 3 minutes eye closed	Qualitative Interview, CADSS, qEEG and LORETA
Phase I: *Stimulus source comparisons*	i)Photograph ii)Video-clip iii)Liveperson	Stimuli was perceived for approximately 30 seconds	qEEG and LORETA
Phase II: *Pretreatment transference assessments*	i)Photograph ii)Video-clip of four target faces	Stimuli was perceived with both eyes-open,right eye open, and left eye open for for approximately 30 seconds	a)Non-triggering condition:qualitative interview, TRR, qEEG and LORETA b)Triggering condition: qualitative interview, TRR, CADSS,qEEG and LORETA
Phase III: *Treatment assessments*	Photograph of the most-triggering face	Approximately 90-minutes of OEI session	Qualitative interview and TRR
PhaseIV:*Posttreatment transference assessments*	i)Photograph ii)Video-clip of the most triggering face	Stimuli was perceived with both eyes open,right eye open, and left eye open for approximately 30 seconds	a) Triggering condition: Qualitative Interview, TRR, CADSS, qEEG and LORETA

APPENDIX G

LORETA Images of Theta, Alpha, and Beta Activation

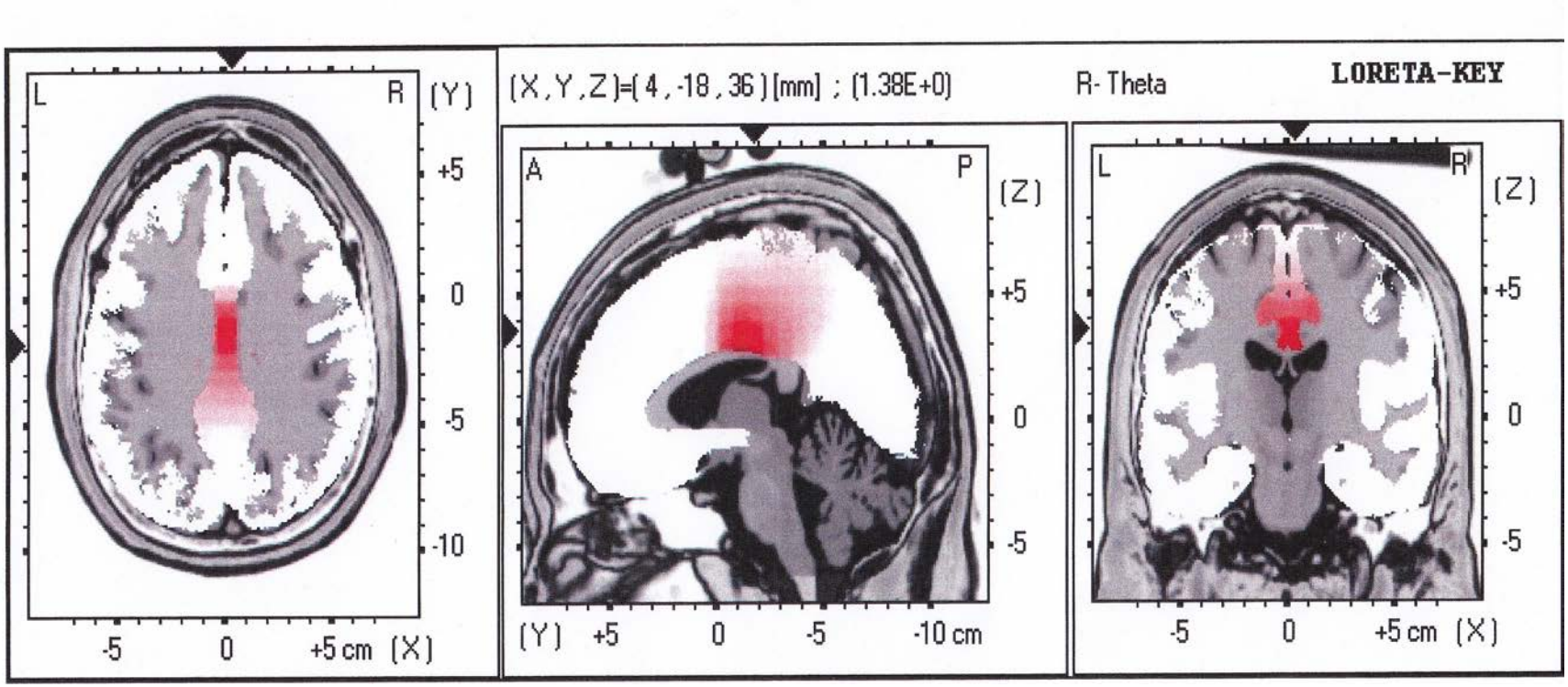
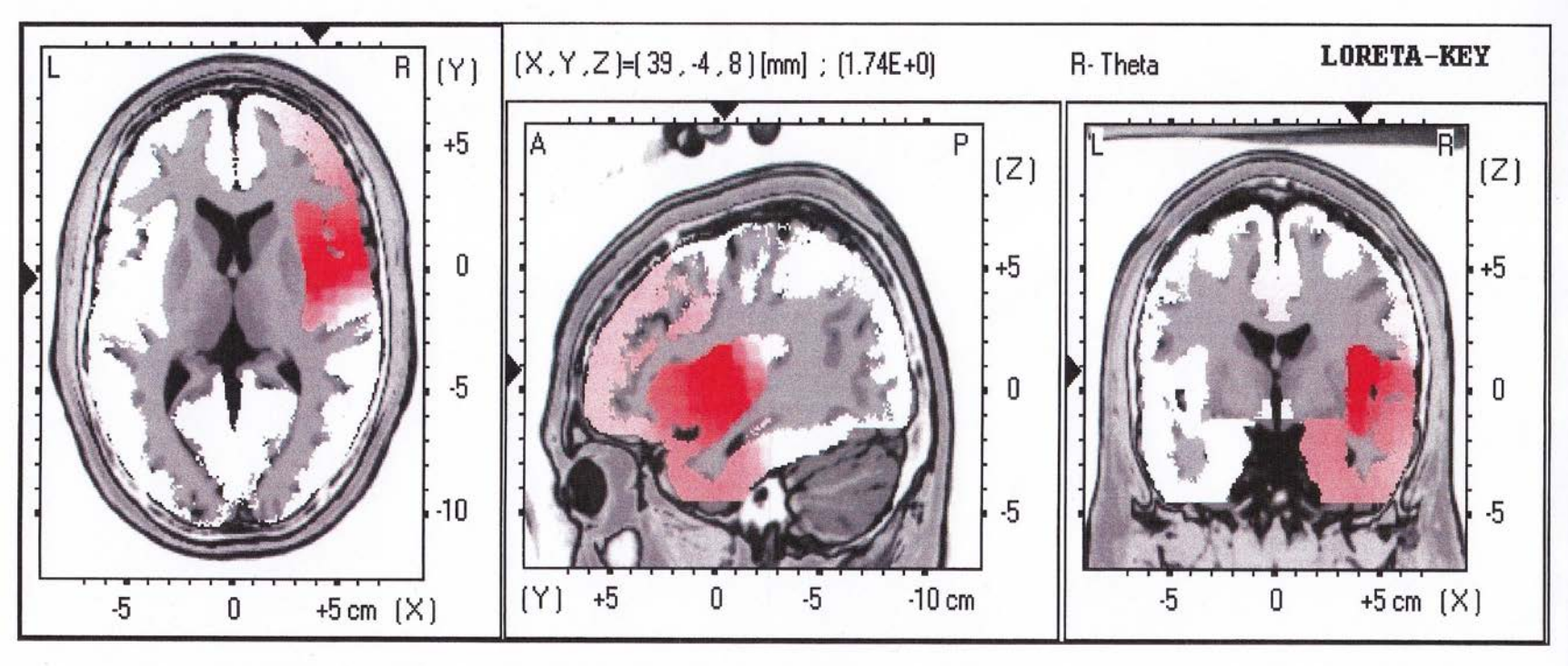
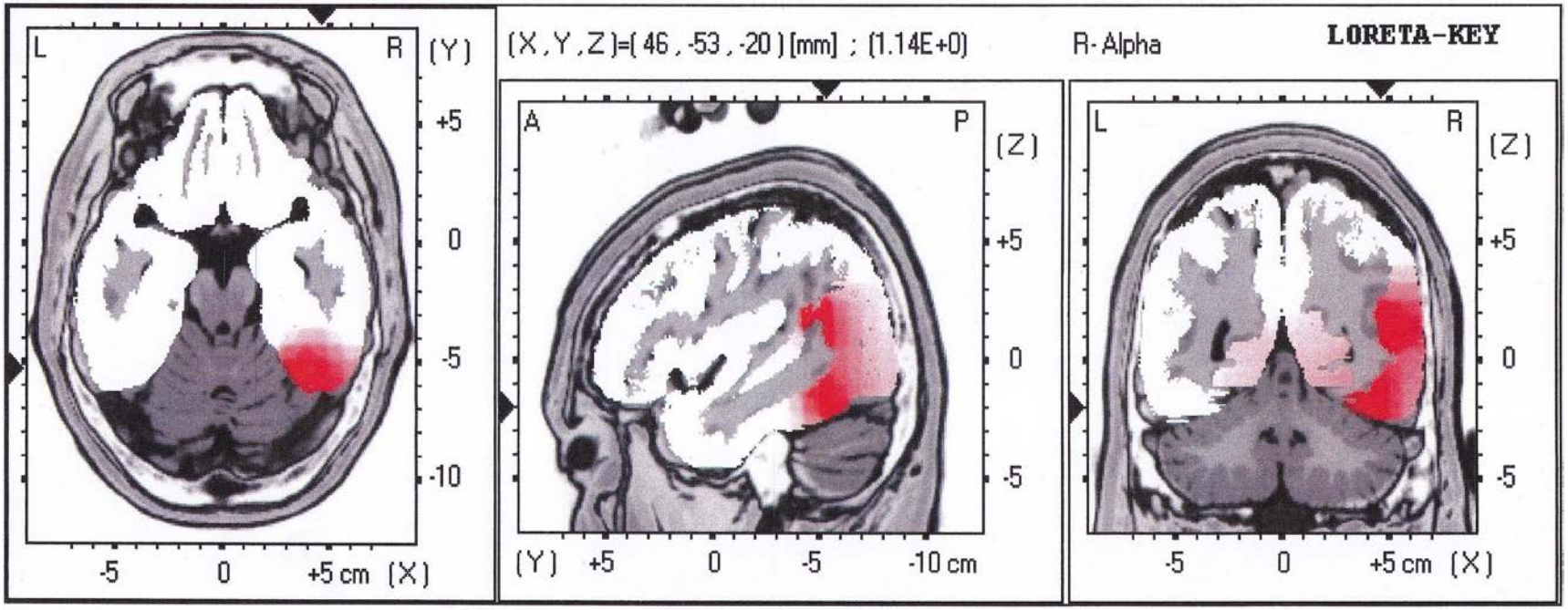


FIGURE G1: Images of theta activation in the Cingulate Gyrus with the male face triggering the strongest transference response when right eye of the participant was open in Phase I using LORETA-KEY ©®Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in μA/ mm^2.



A B C

FIGURE G2: Images of theta activation in right Insula with the male face triggering the strongest transference response when the right eye of the participant was open in Phase IV using LORETA-KEY ©® Software Package. Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in $\mu A/mm^2$.



A B C

FIGURE G3: Images of alpha activation in right Fusiform Gyrus with the male face triggering the strongest transference response when right eye of the participant was open in Phase I using LORETA-KEY ©®Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in μA/ mm^2.

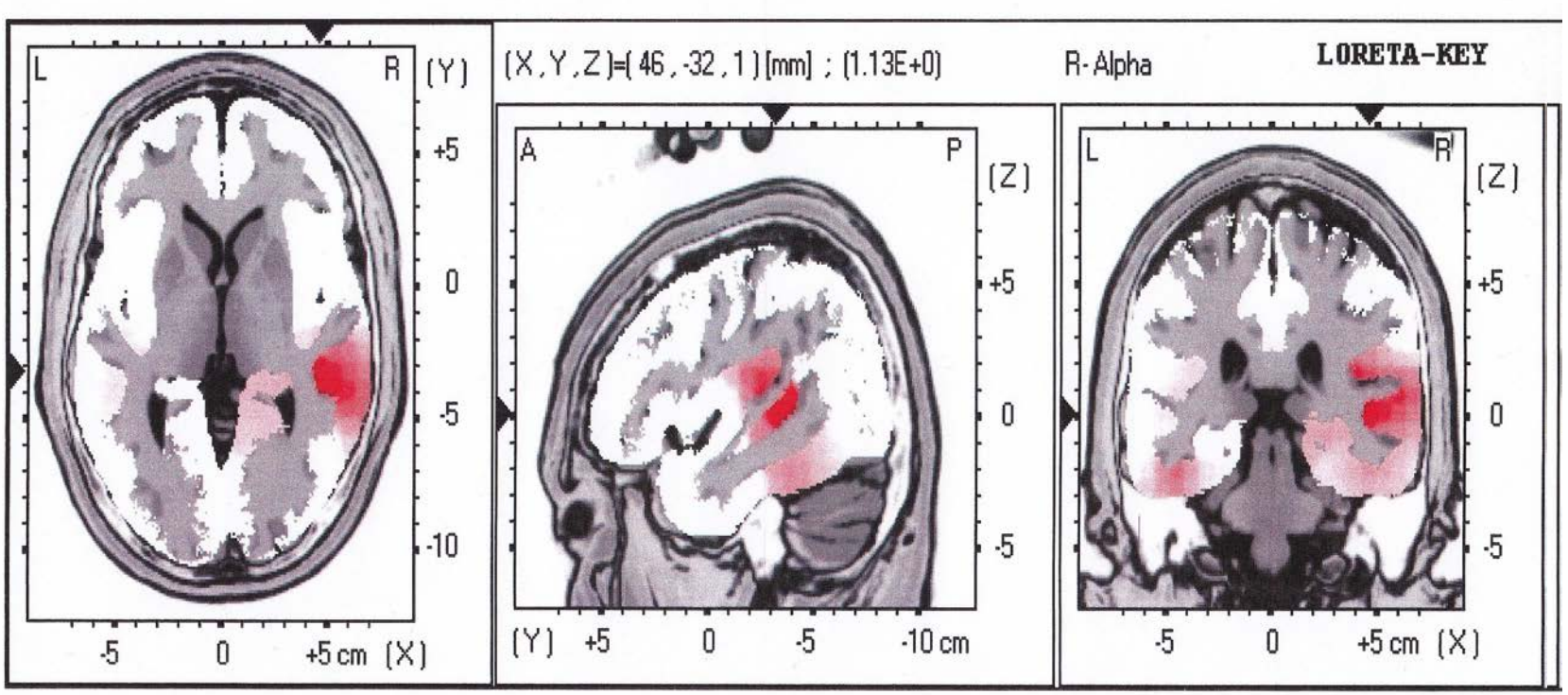
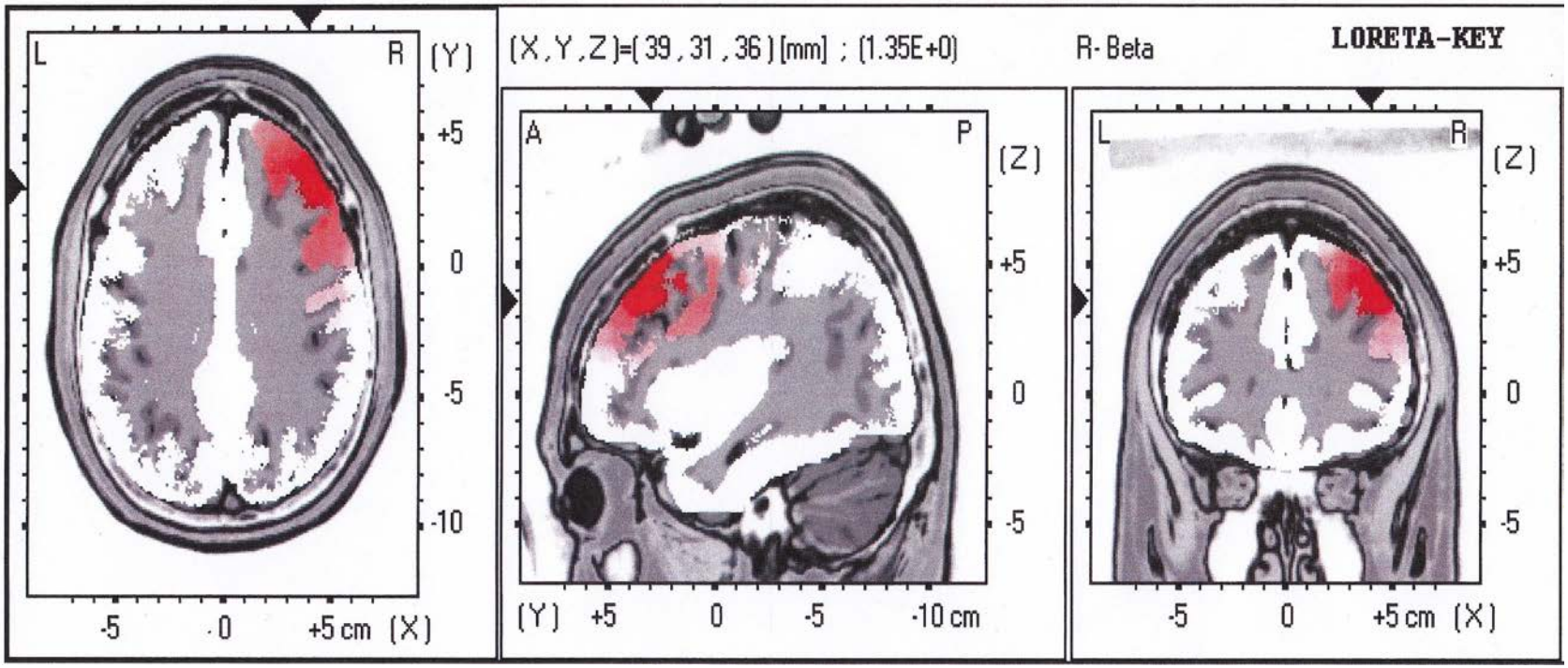
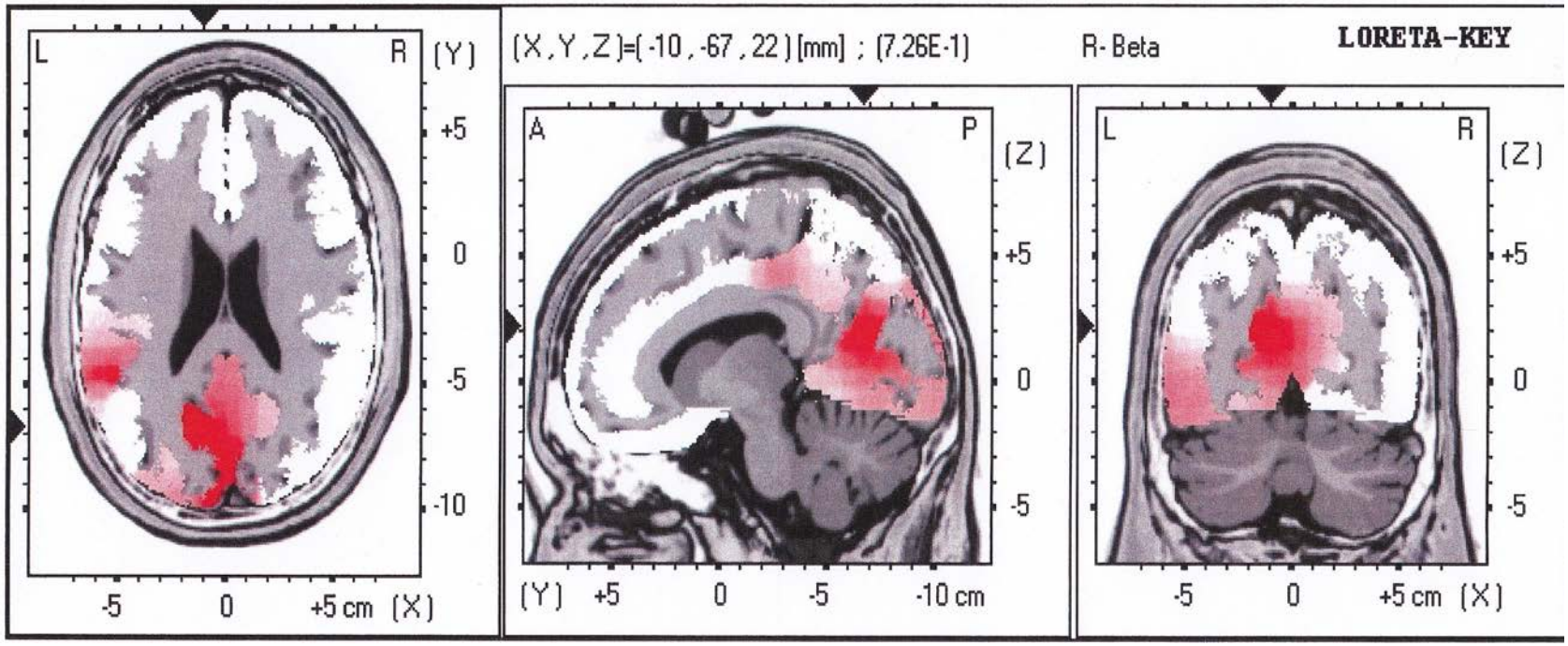


FIGURE G4: Images of alpha activation in right Superior Temporal Gyrus and right Middle Temporal Gyrus with the male face triggering the strongest transference response when the right eye of the participant was open in Phase IV using LORETA-KEY ©®Software Package. Hyperactivated region (indicated in red) is plotted onto three orthogonal slices by: (A) Horizontal Plane, viewed from the top,(B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in μA/mm^2.



A B C

FIGURE G5: Images of beta activation in right Middle Frontal Gyrus with the male face triggering the strongest transference in response when the right eye of the participant was open in Phase I using LORETA-KEY ©®Software Package**.** Hyperactivated region (indicated in red) is plotted onto three slices by: (A) Horizontal Plane, viewed from the top, (B) Sagittal Plane, viewed from the left, and (C) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in $\mu A/mm^2$.



A B C

FIGURE G6: Images of beta activation in Precuneus and Cuneus with the male face triggering the strongest transference response when the right eye of the participant was open in Phase IV using LORETA-KEY ©® Software Package**.** Hyperactivated region (indicated in red) is plotted onto three orthogocal slices by: (a) Horizontal Plane, viewed from the top, (b) Sagittal Plane, viewed from the left, and (c) Coronal Plane, viewed from the back. The activated brain area indicated in Talairach coordinates: X from left (L) to right (R); Y from posterior (P) to anterior (A); and Z from inferior to superior. The black triangle along the axes indicates the point through which all three orthogonal slices were made. The color key depicts the current density in μA/mm^2.

APPENDIX H

Tables of EEG Power Spectra

within Selected Bandranges

TABLE H1:*Table of EEG Power Spectra for all Bandranges at Baseline when Both of the Participant's Eyes were Open*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.295	0.98	1.585	2.93	1.273	7.81	1.298	16.11	0.929	12.94	0.547	27.83	0.607	7.81	0.585	11.96
Fp2-Ref	2.312	1.46	1.607	2.93	1.219	7.81	1.343	16.11	0.915	12.94	0.610	27.83	0.617	7.81	0.529	11.96
F7-Ref	2.062	0.98	1.344	2.93	1.216	7.81	1.246	15.87	0.851	13.67	0.609	28.56	0.623	7.81	0.487	11.96
F3-Ref	2.974	0.98	2.160	2.93	1.626	7.81	1.684	17.33	1.210	12.94	0.577	27.83	0.867	7.81	0.731	11.72
Fz-Ref	3.168	1.22	2.480	2.93	1.679	7.81	1.684	15.87	1.207	12.94	0.497	27.83	0.901	7.81	0.753	11.72
F4-Ref	2.919	1.22	2.248	4.64	1.548	7.81	1.858	17.33	1.082	12.94	0.807	27.83	0.809	7.81	0.684	11.72
F8-Ref	2.478	0.98	1.658	3.17	1.271	9.52	1.357	16.11	0.848	13.43	0.714	28.81	0.642	7.81	0.487	11.96
T3-Ref	2.057	0.98	1.397	3.17	1.407	10.01	1.509	15.87	1.119	13.67	0.813	30.27	0.675	7.81	0.627	10.99
C3-Ref	3.114	0.98	2.197	3.17	2.001	7.81	1.705	16.11	1.341	12.94	0.513	28.81	1.243	7.81	0.773	11.72
Cz-Ref	3.646	0.98	2.750	3.17	1.985	7.81	1.832	15.87	1.360	12.94	0.568	27.83	1.117	7.81	0.904	11.96
C4-Ref	3.215	1.46	2.420	3.42	1.864	7.81	1.838	15.87	1.318	13.18	0.527	27.83	1.045	7.81	0.873	11.72
T4-Ref	2.285	0.98	1.668	4.64	1.507	9.52	1.239	15.87	0.936	13.43	0.512	30.27	0.659	8.79	0.730	10.99
T5-Ref	2.212	1.22	1.683	3.17	1.666	7.81	1.724	15.87	1.242	13.18	0.871	29.05	0.904	7.81	0.834	11.47
P3-Ref	2.865	0.98	2.060	3.17	1.973	7.81	1.847	15.87	1.324	13.18	0.588	29.05	1.217	7.81	0.848	11.96
Pz-Ref	3.198	0.98	2.295	3.17	2.078	7.81	1.908	15.87	1.433	13.67	0.570	29.54	1.202	7.81	0.947	11.96
P4-Ref	3.010	0.98	2.154	3.42	1.902	7.81	1.755	15.87	1.422	13.67	0.565	29.54	1.052	7.81	0.912	11.96
T6-Ref	2.451	0.98	1.725	3.42	1.673	11.47	1.514	15.87	1.274	13.18	0.642	33.20	0.759	7.81	0.927	11.47
O1-Ref	2.356	0.98	1.776	3.17	1.782	7.81	1.847	15.87	1.511	13.67	0.910	33.69	0.943	7.81	0.907	11.96
O2-Ref	2.409	0.98	1.614	2.93	1.681	7.81	1.735	15.87	1.618	13.67	0.723	27.83	0.882	7.81	0.838	11.96

TABLE H2*: Table of EEG Power Spectra for all Bandranges with the Photograph of the Most Triggering Face when Both of the Participant's Eyes were Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	4.912	0.98	2.611	3.42	1.925	8.54	2.713	16.36	1.149	13.67	2.437	32.23	1.066	8.54	0.787	10.99
Fp2-Ref	4.573	0.98	2.718	3.42	1.829	8.54	3.731	18.55	1.149	13.43	3.526	38.33	1.164	8.54	0.893	10.99
F7-Ref	3.520	0.98	1.959	3.66	1.219	8.54	1.356	16.11	0.688	12.94	0.680	29.54	0.801	8.54	0.530	10.99
F3-Ref	3.353	0.98	2.714	3.42	1.586	7.81	2.093	17.33	0.871	13.43	1.188	27.83	0.890	7.81	0.576	10.99
Fz-Ref	2.863	1.71	3.032	3.42	1.757	7.81	1.725	15.87	0.841	13.43	0.875	29.05	1.078	7.81	0.619	10.99
F4-Ref	2.413	0.98	2.790	3.42	1.843	7.81	2.690	19.29	0.999	13.43	1.803	29.30	1.169	7.81	0.740	10.99
F8-Ref	3.396	0.98	2.083	3.17	1.390	7.81	1.806	15.87	0.784	13.92	0.796	29.05	0.967	7.81	0.445	10.99
T3-Ref	2.178	0.98	1.702	3.66	1.072	11.96	1.770	17.82	0.756	12.94	1.069	32.47	0.620	7.81	0.581	11.96
C3-Ref	2.996	1.22	2.612	3.42	1.676	7.81	1.820	17.33	0.836	12.94	0.574	29.30	1.179	7.81	0.442	11.96
Cz-Ref	2.758	1.95	3.006	3.42	1.734	7.81	1.953	17.33	0.873	14.89	0.679	29.30	1.036	7.81	0.541	11.72
C4-Ref	2.576	0.98	2.678	3.42	1.613	7.81	1.860	15.87	0.768	14.89	0.691	29.30	0.859	7.81	0.573	11.96
T4-Ref	1.802	0.98	1.895	3.42	1.435	7.81	1.232	15.87	0.697	13.92	0.657	32.71	0.830	7.81	0.619	11.23
T5-Ref	2.574	0.98	1.985	3.66	1.457	11.96	1.674	17.33	1.110	13.67	1.156	32.47	0.797	8.54	0.679	11.96
P3-Ref	3.640	0.98	2.591	3.42	1.653	9.28	1.879	17.33	1.041	13.43	0.668	32.47	0.975	7.81	0.534	11.96
Pz-Ref	3.954	0.98	2.683	3.42	1.741	9.28	2.051	17.33	1.037	13.43	0.670	29.54	0.850	8.79	0.654	11.96
P4-Ref	3.250	0.98	2.406	3.42	1.537	9.28	1.772	17.33	0.841	13.43	0.790	29.54	0.750	8.54	0.651	11.96
T6-Ref	2.483	0.98	1.819	3.42	1.833	7.81	1.595	15.87	0.942	14.40	0.904	29.54	0.981	7.81	0.776	11.23
O1-Ref	3.533	0.98	2.202	2.93	1.345	8.54	1.646	17.33	0.927	13.92	0.878	29.54	0.763	8.54	0.455	11.72
O2-Ref	3.305	1.46	2.197	3.42	1.606	8.54	1.753	17.33	1.166	14.16	1.062	29.54	0.838	8.54	0.605	11.72

TABLE H3: *Table of EEG Power Spectra for all Bandranges with the Photograph of the Most Triggering Face when the Right Eye of the Participant was Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.027	0.98	1.614	3.66	1.583	11.47	1.757	19.04	0.946	12.94	0.983	31.25	0.808	8.79	0.845	11.47
Fp2-Ref	1.893	1.46	1.593	3.42	1.634	8.79	1.702	17.82	0.919	12.94	1.083	31.25	0.784	8.79	0.733	11.47
F7-Ref	2.213	1.46	1.575	3.66	1.282	8.54	1.266	17.58	0.734	14.89	0.673	32.96	0.732	8.54	0.618	11.47
F3-Ref	2.556	1.46	2.200	3.42	1.820	10.50	1.971	16.60	1.008	14.89	0.848	27.83	0.929	8.54	0.808	10.99
Fz-Ref	2.743	1.46	2.281	3.42	1.881	7.81	1.892	16.60	0.929	13.67	0.596	27.83	0.953	7.81	0.788	10.99
F4-Ref	2.519	1.46	2.220	3.42	1.778	8.79	2.478	16.60	1.015	14.89	1.461	27.83	0.844	8.79	0.767	10.99
F8-Ref	1.920	1.71	1.519	2.93	1.375	10.25	1.363	18.07	0.723	12.94	0.993	28.81	0.680	8.79	0.523	11.47
T3-Ref	1.827	1.71	1.285	3.66	1.136	8.79	1.834	21.00	0.890	14.89	1.082	30.52	0.616	8.79	0.513	11.96
C3-Ref	2.533	1.46	2.090	6.84	1.908	7.81	1.822	16.85	1.204	13.67	0.574	29.05	1.097	7.81	0.649	11.96
Cz-Ref	3.297	1.46	2.276	3.42	1.954	7.81	1.953	16.60	1.117	13.67	0.663	29.05	1.096	7.81	0.814	11.72
C4-Ref	2.950	1.46	2.052	6.84	1.611	11.96	1.868	18.07	1.078	13.67	0.718	29.79	0.765	7.81	0.766	11.96
T4-Ref	2.126	1.95	1.925	3.42	1.301	11.96	1.406	17.33	0.742	12.94	0.717	29.05	0.491	7.81	0.586	11.96
T5-Ref	2.066	1.71	1.618	2.93	1.570	7.81	1.756	16.36	1.149	14.89	1.205	29.05	0.804	7.81	0.828	11.47
P3-Ref	2.605	1.71	2.000	6.84	1.930	7.81	1.818	16.36	1.309	14.89	0.703	29.05	1.210	7.81	0.763	11.47
Pz-Ref	2.977	1.46	2.023	6.84	1.900	7.81	1.879	19.29	1.483	13.92	0.691	28.81	1.180	7.81	0.810	11.47
P4-Ref	2.866	1.46	2.096	6.84	1.669	7.81	1.928	17.33	1.269	13.92	0.818	28.81	0.917	7.81	0.718	11.96
T6-Ref	2.309	1.71	2.021	2.93	1.841	9.28	2.039	17.58	1.013	13.67	1.209	28.81	0.660	7.81	0.862	11.96
O1-Ref	2.179	0.98	1.801	3.17	1.728	7.81	1.856	15.87	1.484	13.92	1.048	29.30	0.984	7.81	0.723	11.47
O2-Ref	2.431	0.98	1.732	4.39	1.813	7.81	2.172	15.87	1.585	13.92	1.246	28.81	0.976	7.81	0.872	10.99

TABLE H4: *Table of EEG Power Spectra for all Bandranges with the Photograph of the Most Triggering Face when the Left Eye of the Participant was Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	3.126	1.71	1.609	3.66	1.435	9.03	1.372	16.85	0.835	14.65	0.707	28.56	0.761	8.79	0.491	10.99
Fp2-Ref	2.241	0.98	1.480	2.93	1.487	8.54	1.142	16.85	0.860	14.65	0.694	33.45	0.786	8.54	0.518	10.99
F7-Ref	2.143	0.98	1.238	3.66	1.144	9.03	1.125	17.09	0.742	14.40	0.643	29.30	0.495	8.79	0.459	10.99
F3-Ref	3.064	0.98	1.879	3.17	1.463	7.81	1.497	16.85	1.102	14.40	0.700	27.83	0.829	7.81	0.492	10.99
Fz-Ref	3.421	0.98	2.270	4.64	1.587	7.81	1.454	16.85	0.972	14.65	0.605	27.83	0.906	7.81	0.540	10.99
F4-Ref	3.012	0.98	2.196	3.42	1.658	8.79	1.806	16.85	0.885	14.65	0.961	29.54	0.836	8.79	0.652	11.72
F8-Ref	2.088	1.22	1.741	4.39	1.522	8.79	1.210	19.53	0.854	14.16	0.752	34.18	0.740	8.79	0.600	11.72
T3-Ref	2.330	0.98	1.264	3.17	1.278	11.72	1.964	19.29	1.162	14.40	1.272	30.27	0.431	8.79	0.700	11.72
C3-Ref	3.423	0.98	1.963	3.42	1.488	7.81	1.637	16.85	1.074	14.40	0.671	30.52	0.752	7.81	0.489	11.72
Cz-Ref	4.361	1.71	2.439	4.64	1.686	7.81	1.613	16.85	1.042	14.40	0.785	30.52	1.049	7.81	0.601	10.99
C4-Ref	3.898	1.71	2.489	5.62	1.665	11.72	2.101	16.36	1.028	14.16	1.260	30.76	0.842	8.54	0.827	11.72
T4-Ref	2.378	1.22	2.220	3.66	1.982	8.79	2.202	15.87	1.276	14.16	1.545	28.08	0.857	8.79	1.152	10.99
T5-Ref	2.314	1.71	1.694	3.42	1.448	11.72	2.410	19.29	1.467	13.18	1.860	31.49	0.589	8.06	0.754	11.72
P3-Ref	3.552	1.71	2.026	3.42	1.557	9.52	1.733	19.29	1.128	14.16	0.769	29.54	0.791	8.54	0.568	11.72
Pz-Ref	4.136	1.71	2.048	6.59	1.728	9.52	1.642	19.29	1.064	13.67	0.774	29.54	0.988	8.54	0.661	10.99
P4-Ref	3.657	1.71	2.212	3.42	1.786	8.54	1.833	19.04	1.034	14.16	1.182	30.76	0.942	8.54	0.896	11.72
T6-Ref	2.746	2.44	2.402	3.42	2.195	11.72	3.269	18.55	1.815	14.16	2.511	31.98	0.807	8.54	1.337	11.72
O1-Ref	2.846	1.71	1.586	2.93	1.710	9.52	1.595	18.07	0.985	13.18	0.945	30.76	0.827	8.54	0.719	11.72
O2-Ref	2.865	1.71	1.806	3.42	1.817	10.50	1.824	19.04	1.119	14.16	1.145	30.76	0.845	8.54	0.606	10.99

TABLE H5: *Table of EEG Power Spectra for all Bandranges with the Video of the Most Triggering Face when Both of the Participant's Eyes were Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.048	0.98	1.807	2.93	1.485	7.81	2.293	24.90	0.962	12.94	1.423	28.08	0.718	7.81	0.797	10.99
Fp2-Ref	2.152	1.71	1.882	2.93	1.752	10.99	2.138	23.44	1.048	12.94	1.650	28.08	0.764	7.81	0.888	10.99
F7-Ref	1.863	0.98	1.598	2.93	1.086	7.81	1.328	16.85	0.694	13.92	0.594	29.05	0.540	7.81	0.505	11.23
F3-Ref	2.686	1.22	2.181	2.93	1.662	10.01	1.790	17.58	1.181	13.92	0.810	29.30	0.809	7.81	0.628	11.96
Fz-Ref	2.412	1.22	2.331	2.93	1.735	7.81	1.837	16.85	1.139	13.92	0.602	29.05	0.934	7.81	0.609	11.23
F4-Ref	2.121	2.93	2.068	2.93	1.869	7.81	2.491	17.58	1.216	13.92	1.526	33.45	0.978	7.81	0.715	11.47
F8-Ref	2.241	1.46	1.668	2.93	1.276	8.06	1.433	20.02	0.730	13.92	0.874	28.32	0.727	8.06	0.555	11.96
T3-Ref	1.789	1.22	1.299	2.93	1.348	8.79	1.206	16.60	0.610	14.89	0.489	30.52	0.629	8.79	0.456	11.96
C3-Ref	2.793	0.98	2.042	6.84	1.902	8.06	1.537	16.36	1.045	14.89	0.525	29.30	1.009	8.06	0.709	11.47
Cz-Ref	2.819	2.20	2.451	6.84	2.032	10.01	1.780	16.85	1.276	13.92	0.647	29.30	1.034	8.06	0.680	11.47
C4-Ref	2.102	1.71	2.001	2.93	1.716	7.81	1.846	17.58	1.352	13.92	0.649	29.30	0.991	7.81	0.697	11.47
T4-Ref	2.288	0.98	1.464	4.64	1.457	7.81	1.247	15.87	0.780	13.92	0.521	28.08	0.808	7.81	0.634	11.72
T5-Ref	2.785	1.71	1.350	6.84	1.465	8.06	1.707	16.60	0.781	13.18	0.814	28.08	0.653	8.06	0.643	11.47
P3-Ref	3.208	1.22	1.983	6.84	2.050	8.06	1.561	16.36	1.069	13.92	0.590	28.08	1.005	8.06	0.886	11.47
Pz-Ref	3.186	0.98	2.118	6.84	1.965	8.06	1.521	16.85	1.348	13.92	0.620	29.30	1.009	8.06	0.774	11.47
P4-Ref	2.187	1.71	1.757	6.84	1.651	8.06	1.414	18.55	1.462	13.92	0.673	29.79	0.811	8.06	0.698	11.47
T6-Ref	1.837	0.98	1.354	2.93	1.554	10.50	1.433	17.33	1.056	13.92	0.858	28.81	0.677	7.81	0.759	11.96
O1-Ref	3.266	1.46	1.678	4.64	1.769	8.06	1.332	16.36	1.263	13.43	0.779	28.08	0.819	8.06	0.740	11.23
O2-Ref	2.859	1.71	1.682	4.64	1.702	8.06	1.496	18.55	1.472	13.92	0.986	27.83	0.841	8.06	0.587	11.96

TABLE H6: *Table of EEG Power Spectra for all Bandranges with the Video of the Most Triggering Face when the Right Eye of the Participant was Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.547	0.98	1.598	3.42	1.228	10.01	1.456	16.60	1.139	14.16	0.783	31.49	0.515	8.30	0.450	10.99
Fp2-Ref	1.671	0.98	1.631	3.42	1.194	10.01	1.497	16.60	1.002	14.65	0.762	32.47	0.494	8.06	0.394	11.96
F7-Ref	1.372	2.69	1.572	3.42	1.139	10.01	1.150	16.36	0.832	14.40	0.545	29.79	0.516	8.30	0.452	10.99
F3-Ref	1.871	2.69	2.193	3.66	1.635	9.52	1.702	17.33	1.121	14.65	0.702	30.03	0.907	8.30	0.455	11.96
Fz-Ref	1.871	1.46	2.192	3.66	1.769	8.30	1.652	16.60	1.168	14.65	0.576	31.49	1.097	8.30	0.462	11.96
F4-Ref	1.858	2.69	2.079	3.66	1.547	9.28	2.107	16.60	1.002	14.65	1.229	31.25	0.883	8.06	0.420	11.96
F8-Ref	1.899	0.98	1.512	5.37	1.108	9.28	1.364	23.68	0.759	13.43	0.734	32.47	0.445	8.79	0.382	11.96
T3-Ref	1.517	0.98	1.525	2.93	1.314	9.52	1.270	16.11	0.987	13.67	0.669	28.81	0.643	8.79	0.476	11.23
C3-Ref	1.860	1.46	2.174	3.91	2.018	8.30	1.663	18.80	1.257	14.40	0.573	29.79	1.292	8.30	0.567	11.47
Cz-Ref	2.411	1.46	2.480	3.91	2.090	9.28	1.692	18.80	1.222	14.40	0.597	29.79	1.225	8.30	0.605	11.96
C4-Ref	1.909	1.46	2.257	2.93	1.744	9.52	1.765	18.80	1.110	14.65	0.530	32.71	0.967	7.81	0.539	10.99
T4-Ref	1.501	0.98	1.382	2.93	1.734	9.28	1.319	19.53	0.767	14.65	0.545	30.52	0.731	7.81	0.768	10.99
T5-Ref	2.041	1.71	1.702	3.42	1.845	9.52	1.916	20.51	1.039	14.40	1.249	31.49	0.916	8.54	0.755	11.23
P3-Ref	2.410	0.98	2.303	3.17	2.056	9.28	1.688	18.55	0.911	14.40	0.685	29.54	1.158	8.30	0.749	11.47
Pz-Ref	2.678	1.22	2.418	3.17	2.145	9.28	1.716	18.55	0.898	13.43	0.629	28.81	1.154	8.30	0.749	11.72
P4-Ref	2.352	1.22	2.280	2.93	1.964	9.52	1.788	17.33	0.911	13.43	0.638	28.81	1.069	8.54	0.728	10.99
T6-Ref	1.843	1.46	1.714	3.17	2.059	10.74	1.761	19.53	1.002	14.40	0.986	30.52	0.839	8.54	0.987	10.99
O1-Ref	2.347	0.98	1.990	3.17	1.903	9.28	1.579	18.55	0.888	14.40	1.022	29.54	0.929	8.30	0.656	10.99
O2-Ref	2.399	1,22	2.032	3.17	1.861	8.54	1.791	18.55	0.877	13.43	0.989	28.56	0.942	8.54	0.708	10.99

TABLE H7: *Table of EEG Power Spectra for all Bandranges with the Video of the Most Triggering Face when the Left Eye of the Participant was Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.294	1.71	1.504	3.42	1.234	8.30	1.294	18.55	0.787	14.65	0.701	28.56	0.793	8.30	0.579	11.23
Fp2-Ref	2.448	1.22	1.468	3.66	1.335	7.81	1.279	18.55	0.838	14.65	0.587	29.79	0.835	7.81	0.597	10.99
F7-Ref	1.534	1.22	1.302	3.66	1.113	8.54	1.117	17.33	0.663	14.65	0.584	27.83	0.658	8.54	0.511	11.23
F3-Ref	2.588	1.95	2.185	3.66	1.588	8.30	1.534	19.04	0.858	13.18	0.679	28.56	1.016	8.30	0.730	10.99
Fz-Ref	2.908	1.22	2.194	3.66	1.702	8.30	1.576	18.55	1.014	14.65	0.571	27.83	1.194	8.30	0.724	11.23
F4-Ref	2.965	1.46	1.924	3.66	1.685	8.54	1.657	18.55	0.968	13.18	0.750	29.54	1.102	8.54	0.794	11.23
F8-Ref	2.236	1.22	1.361	2.93	1.243	7.81	1.321	18.55	0.741	12.94	0.607	29.54	0.710	7.81	0.481	10.99
T3-Ref	1.881	1.95	1.362	3.17	1.334	7.81	1.082	17.33	0.659	13.18	0.541	29.05	0.757	7.81	0.545	11.23
C3-Ref	2.549	1.95	2.442	3.42	1.886	7.81	1.551	16.85	1.045	14.65	0.552	27.83	1.204	7.81	0.856	10.99
Cz-Ref	3.024	1.95	2.521	3.66	1.903	7.81	1.625	18.55	1.241	14.65	0.602	28.08	1.284	7.81	0.843	11.23
C4-Ref	3.113	1.46	2.258	2.93	1.665	7.81	1.568	18.55	1.132	14.89	0.555	28.08	1.147	7.81	0.678	11.23
T4-Ref	1.999	0.98	1.492	3.42	1.372	9.03	1.129	16.36	0.859	13.92	0.492	29.05	0.669	8.30	0.660	11.23
T5-Ref	2.289	1.95	1.887	3.42	1.543	7.81	1.830	17.33	0.887	12.94	1.100	29.79	0.922	7.81	0.557	10.99
P3-Ref	2.648	1.95	2.256	2.93	1.669	7.81	1.601	16.85	1.065	14.40	0.598	29.79	1.092	7.81	0.629	11.96
Pz-Ref	2.798	1.95	2.395	2.93	1.663	7.81	1.590	16.85	1.085	14.65	0.565	28.08	1.064	7.81	0.703	11.23
P4-Ref	2.750	1.46	2.234	2.93	1.608	7.81	1.571	16.85	1.169	14.65	0.599	28.08	1.047	7.81	0.667	11.96
T6-Ref	2.289	1.46	1.812	2.93	1.672	9.03	1.428	18.31	0.943	13.67	0.718	28.56	0.790	7.81	0.838	11.23
O1-Ref	2.524	1.95	2.038	2.93	1.463	7.81	1.466	18.31	0.926	14.40	0.786	30.03	0.891	7.81	0.614	11.47
O2-Ref	2.509	0.98	2.004	2.93	1.549	7.81	1.544	18.31	0.877	14.16	0.819	30.03	0.824	7.81	0.660	11.23

TABLE H8*: Table of EEG Power Spectra for all Bandranges with the Live Person when Both of the Participant's Eyes were Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.072	1.46	1.382	4.15	1.466	7.81	2.710	18.31	0.936	14.89	2.073	27.83	0.777	7.81	0.708	11.23
Fp2-Ref	2.223	1.22	1.663	2.93	1.665	10.74	3.292	19.29	1.205	13.67	2.668	27.83	0.761	7.81	0.800	10.99
F7-Ref	1.935	1.46	1.726	3.91	1.320	9.77	1.210	16.85	0.669	13.18	0.645	29.05	0.732	8.30	0.552	11.23
F3-Ref	2.496	0.98	2.362	4.15	1.977	8.54	1.999	20.51	1.183	13.43	1.147	30.76	1.037	8.54	0.898	11.96
Fz-Ref	2.939	0.98	2.440	4.15	1.974	8.54	1.744	16.11	0.934	13.43	0.736	30.76	1.034	8.54	0.833	11.96
F4-Ref	2.820	0.98	2.330	3.17	2.186	8.79	3.481	22.71	1.361	14.65	2.331	28.56	0.993	8.79	1.120	11.23
F8-Ref	2.546	1.22	1.641	3.17	1.466	8.79	1.699	19.29	0.724	13.18	0.959	27.83	0.790	8.79	0.596	11.96
T3-Ref	1.832	0.98	1.706	3.91	1.396	9.77	2.048	19.04	0.829	13.18	1.381	28.81	0.655	8.30	0.593	10.99
C3-Ref	2.354	0.98	2.422	4.15	1.956	9.77	1.438	19.04	1.094	13.18	0.702	30.76	1.068	8.54	0.719	11.96
Cz-Ref	3.071	2.20	2.454	4.15	2.095	9.52	1.616	16.11	1.026	13.67	0.730	30.76	1.106	8.54	0.781	11.96
C4-Ref	2.629	2.20	2.077	2.93	2.166	8.79	1.651	15.87	0.860	12.94	0.837	28.32	1.053	8.79	0.865	11.72
T4-Ref	2.768	1.22	1.654	2.93	1.982	10.74	1.764	20.26	0.758	12.94	1.334	30.76	0.775	8.79	0.965	11.72
T5-Ref	2.465	0.98	2.195	3.42	2.055	10.74	3.046	16.11	1.420	14.65	2.220	28.08	0.755	8.79	0.841	11.96
P3-Ref	2.646	0.98	2.271	3.66	1.995	9.77	1.765	18.80	1.182	13.18	0.953	35.40	1.041	8.30	0.750	10.99
Pz-Ref	2.710	0.98	2.298	2.93	2.213	8.30	1.637	16.85	0.998	13.18	0.780	29.30	1.276	8.30	0.677	11.72
P4-Ref	2.227	2.20	2.100	2.93	2.397	8.54	1.797	15.87	0.998	12.94	1.003	29.30	1.143	8.54	0.901	11.72
T6-Ref	2.316	2.20	1.831	4.39	3.030	10.74	3.526	20.26	1.485	12.94	2.749	29.79	0.761	8.79	1.774	11.23
O1-Ref	2.688	0.98	1.808	2.93	1.924	10.25	1.911	16.85	1.180	14.89	1.298	31.74	0.814	8.79	0.817	11.96
O2-Ref	2.279	0.98	1.698	2.93	2.211	10.25	1.968	16.85	1.130	12.94	1.215	28.32	0.886	8.79	1.060	11.96

TABLE H9: *Table of EEG Power Spectra for all Bandranges with the Live Person when the Right Eye of the Participant was Open in Phase I*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.672	1.71	1.427	4.64	1.384	10.25	1.897	20.02	1.186	13.43	1.366	31.01	0.476	8.06	0.719	10.99
Fp2-Ref	3.279	1.71	1.675	4.88	1.717	10.25	3.237	23.68	1.357	13.92	1.873	29.54	0.933	8.06	0.595	11.72
F7-Ref	2.504	1.71	1.436	2.93	1.019	11.72	1.201	17.33	0.702	13.43	0.646	28.81	0.392	8.79	0.572	11.72
F3-Ref	3.470	1.71	1.914	2.93	1.780	10.25	1.786	16.60	1.041	14.65	0.913	28.56	0.741	8.79	0.806	11.72
Fz-Ref	3.533	1.46	1.935	2.93	1.690	10.25	1.525	16.60	0.822	12.94	0.659	28.81	0.777	8.79	0.622	11.72
F4-Ref	3.887	1.71	1.846	2.93	1.719	10.25	3.079	20.02	1.135	12.94	1.661	28.56	0.832	8.06	0.797	11.72
F8-Ref	3.491	1.22	1.342	5.13	1.207	10.25	1.445	23.93	1.064	12.94	0.915	30.76	0.563	8.54	0.520	11.72
T3-Ref	2.101	1.46	1.203	3.66	1.368	9.28	1.761	20.02	1.054	14.89	1.310	28.56	0.651	8.06	0.598	11.72
C3-Ref	3.199	2.69	2.001	2.93	1.838	8.79	1.474	16.11	0.741	13.18	0.687	27.83	0.835	8.79	0.583	11.96
Cz-Ref	3.678	2.69	2.312	2.93	2.187	8.79	1.642	16.11	0.714	12.94	0.704	29.30	1.082	8.79	0.804	11.72
C4-Ref	4.081	1.46	1.952	2.93	1.824	10.25	1.540	17.58	1.006	12.94	0.781	33.94	0.776	8.79	0.744	10.99
T4-Ref	2.716	1.22	1.521	5.13	1.627	10.01	1.828	19.53	1.265	14.65	1.194	33.94	0.516	7.81	0.794	11.72
T5-Ref	2.417	0.98	1.528	5.37	1.932	8.06	2.852	21.48	1.898	13.18	2.632	27.83	1.014	8.06	0.888	11.72
P3-Ref	3.053	2.69	1.774	2.93	1.811	8.79	1.438	16.11	0.969	13.43	0.832	27.83	0.973	8.79	0.688	11.72
Pz-Ref	3.116	2.69	1.802	2.93	1.948	8.79	1.461	18.31	0.811	14.65	0.707	34.18	0.984	8.79	0.851	10.99
P4-Ref	3.372	1.71	1.652	2.93	1.880	10.74	1.621	19.53	0.906	14.65	0.946	34.18	0.782	8.79	0.900	10.99
T6-Ref	2.494	2.20	1.628	2.93	2.656	11.72	4.180	19.29	2.026	14.65	2.707	33.94	0.609	8.54	1.289	11.72
O1-Ref	2.818	0.98	1.701	2.93	1.719	8.79	1.822	24.66	0.855	13.43	1.097	32.71	0.943	8.79	0.811	10.99
O2-Ref	2.574	2.69	1.959	2.93	1.665	10.99	1.774	24.66	0.855	13.43	1.153	32.96	0.765	8.06	1.003	10.99

TABLE H10: Table of EEG Power Spectra for all Bandranges with the Live Person when the Left Eye of the Participant was Open in Phase I

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.914	1.71	1.937	3.17	1.479	8.54	2.517	18.31	1.382	13.43	1.755	29.30	0.805	8.54	0.612	11.23
Fp2-Ref	2.498	0.98	1.788	3.17	1.725	8.54	3.121	20.26	1.638	13.18	2.382	29.79	0.869	8.54	0.747	11.23
F7-Ref	1.707	1.22	1.463	4.64	1.062	8.30	1.167	21.00	0.641	13.67	0.634	32.71	0.607	8.30	0.383	11.96
F3-Ref	2.588	1.22	2.198	4.64	1.459	8.30	1.891	16.36	0.872	14.16	0.900	29.54	0.967	8.30	0.545	11.96
Fz-Ref	2.927	1.22	2.262	4.64	1.862	8.54	1.688	16.36	0.831	13.67	0.687	29.54	1.273	8.54	0.647	11.23
F4-Ref	2.652	1.22	2.102	3.17	1.970	8.54	2.796	18.80	1.085	14.89	1.699	28.08	1.264	8.54	0.743	11.47
F8-Ref	2.427	0.98	1.637	3.17	1.707	8.54	1.526	16.36	0.809	13.18	0.815	28.56	0.900	8.54	0.637	11.23
T3-Ref	1.951	1.95	1.523	3.91	1.484	9.03	2.002	19.78	0.910	14.65	1.457	29.54	0.686	8.79	0.418	11.23
C3-Ref	3.102	0.98	2.412	3.42	1.657	8.54	1.608	16.36	0.813	14.16	0.666	29.05	1.089	8.54	0.526	11.96
Cz-Ref	3.466	1.71	2.356	4.64	2.142	8.54	1.877	16.36	0.964	14.40	0.695	29.54	1.474	8.54	0.693	11.96
C4-Ref	3.151	0.98	2.019	3.42	1.983	8.54	1.784	16.36	0.918	13.67	0.714	29.54	1.234	8.54	0.717	11.23
T4-Ref	2.166	1.22	1.490	3.17	1.967	8.54	1.746	16.11	0.847	14.65	1.106	30.52	0.991	8.54	0.974	11.23
T5-Ref	2.447	1.71	2.040	3.17	1.956	9.52	2.782	20.51	1.540	14.65	2.064	28.56	0.844	8.54	0.893	11.72
P3-Ref	3.343	0.98	2.420	3.17	1.801	8.54	1.700	16.85	0.924	13.92	0.862	29.30	1.169	8.54	0.564	11.96
Pz-Ref	3.666	0.98	2.325	3.91	1.997	8.54	1.792	16.36	1.009	13.67	0.719	29.30	1.338	8.54	0.644	11.72
P4-Ref	3.384	0.98	2.070	3.91	2.017	8.54	1.772	18.80	1.109	13.18	0.891	29.05	1.283	8.54	0.793	11.72
T6-Ref	2.415	0.98	1.571	6.84	2.360	8.79	2.918	18.80	1.649	13.18	2.096	30.52	1.104	8.79	1.276	11.47
O1-Ref	3.065	0.98	2.153	3.17	1.894	8.54	1.655	19.78	0.866	13.67	1.194	28.08	1.065	8.54	0.802	11.72
O2-Ref	3.530	1.71	2.065	3.17	2.038	8.54	1.688	17.82	1.147	13.67	1.143	27.83	1.120	8.54	0.919	11.72

TABLE H11: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 1 when Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.140	2.69	1.857	2.93	1.377	11.47	1.574	18.31	0.784	14.89	0.895	27.83	0.586	8.06	0.666	11.47
Fp2-Ref	2.105	1.46	1.896	3.17	1.388	11.72	1.979	16.85	0.757	14.89	1.265	28.32	0.525	8.79	0.730	11.72
F7-Ref	1.797	1.46	1.654	2.93	1.055	11.72	1.114	19.04	0.608	12.94	0.527	29.79	0.370	8.79	0.513	11.72
F3-Ref	2.494	2.69	2.479	3.17	1.700	11.72	1.560	15.87	1.009	13.18	0.646	28.81	0.882	8.06	0.788	11.72
Fz-Ref	2.666	2.69	2.757	3.17	1.846	9.77	1.608	15.87	0.975	13.18	0.579	28.81	0.996	8.30	0.844	11.72
F4-Ref	2.388	0.98	2.345	3.17	1.860	8.30	2.229	18.80	1.040	13.18	1.070	29.79	1.015	8.30	0.846	11.72
F8-Ref	1.822	0.98	1.536	4.15	1.252	10.25	1.458	19.29	0.720	13.67	0.793	28.08	0.598	8.30	0.607	11.47
T3-Ref	1.595	2.69	1.619	2.93	1.060	10.01	1.361	15.87	0.776	14.65	0.765	36.38	0.459	8.79	0.400	11.96
C3-Ref	2.555	2.69	2.606	2.93	1.901	7.81	1.656	15.87	1.354	13.43	0.547	28.81	1.074	7.81	0.764	11.72
Cz-Ref	3.251	1.46	3.051	2.93	2.260	8.30	1.802	15.87	1.459	13.43	0.699	29.05	1.333	8.30	0.959	11.72
C4-Ref	2.618	0.98	2.525	3.17	1.978	8.30	1.702	15.87	1.227	13.18	0.647	28.81	1.126	8.30	0.911	11.72
T4-Ref	1.671	0.98	1.588	4.15	1.636	10.01	1.260	16.36	0.826	13.67	0.631	28.81	0.848	8.30	0.724	11.96
T5-Ref	2.123	2.93	1.798	2.93	1.188	10.50	1.597	15.87	0.984	13.67	0.791	29.54	0.478	8.79	0.454	11.72
P3-Ref	2.717	2.93	2.373	2.93	1.804	7.81	1.785	15.87	1.469	13.43	0.603	30.03	1.038	7.81	0.694	11.72
Pz-Ref	3.066	1.46	2.548	2.93	2.026	7.81	1.824	15.87	1.603	13.43	0.613	30.03	1.183	7.81	0.907	11.72
P4-Ref	2.378	1.22	2.391	2.93	1.933	7.81	1.647	15.87	1.361	13.43	0.693	28.56	1.173	7.81	0.848	11.47
T6-Ref	1.525	0.98	1.892	3.17	1.944	10.74	1.745	17.82	1.061	13.43	0.844	28.81	0.975	8.30	0.870	10.99
O1-Ref	2.749	1.22	1.927	2.93	1.413	11.47	1.673	15.87	1.304	13.92	0.810	30.03	0.707	7.81	0.824	11.47
O2-Ref	2.489	1.22	1.963	2.93	1.649	7.81	1.648	15.87	1.279	13.92	0.986	29.79	0.926	7.81	0.860	11.47

TABLE H12: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 1 when the Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.537	1.46	1.409	5.13	1.592	8.30	1.419	17.09	0.863	12.94	0.586	27.83	1.155	8.30	0.404	11.96
Fp2-Ref	2.909	0.98	1.476	6.84	1.454	8.30	1.534	19.04	0.684	14.16	0.698	32.23	1.040	8.30	0.434	11.23
F7-Ref	1.677	1.71	1.080	5.37	1.491	8.06	1.211	17.33	0.967	14.16	0.504	28.56	1.078	8.06	0.411	11.96
F3-Ref	2.746	0.98	1.716	3.17	2.086	8.06	1.831	17.09	1.173	14.16	0.557	29.30	1.610	8.06	0.330	11.96
Fz-Ref	3.465	0.98	2.019	3.17	2.114	8.06	1.935	17.09	1.177	13.92	0.560	27.83	1.625	8.06	0.400	11.96
F4-Ref	3.394	0.98	2.075	3.17	1.852	8.30	2.184	19.04	0.951	13.18	0.915	31.98	1.256	8.30	0.602	10.99
F8-Ref	2.451	0.98	1.595	3.17	1.267	8.30	1.492	17.09	0.687	14.89	0.630	28.56	0.751	8.30	0.478	11.23
T3-Ref	1.740	1.46	1.171	3.66	1.686	8.06	1.338	18.80	1.053	13.18	0.645	31.74	1.210	8.06	0.569	11.23
C3-Ref	2.627	2.44	1.913	3.42	2.394	8.06	1.782	17.09	1.199	13.18	0.642	28.56	1.810	8.06	0.398	11.96
Cz-Ref	3.931	1.71	2.288	3.42	2.418	8.06	2.098	17.09	1.183	13.18	0.699	28.56	1.846	8.06	0.469	11.23
C4-Ref	3.427	0.98	2.373	3.17	1.979	8.06	1.968	19.53	1.018	13.18	0.614	31.98	1.368	8.06	0.701	11.23
T4-Ref	2.336	0.98	1.901	3.17	1.807	9.28	1.284	19.53	0.810	14.89	0.492	29.54	0.688	8.79	0.871	11.23
T5-Ref	1.980	1.71	1.507	3.42	1.997	8.06	1.641	18.80	0.960	13.18	0.832	29.30	1.393	8.06	0.512	11.96
P3-Ref	2.711	1.22	2.205	3.42	2.511	8.06	1.660	18.07	1.067	13.18	0.654	29.30	1.906	8.06	0.413	11.96
Pz-Ref	3.247	1.22	2.333	3.42	2.511	8.06	1.735	17.09	1.183	13.18	0.645	29.05	1.933	8.06	0.613	11.23
P4-Ref	3.161	0.98	2.431	3.17	2.280	8.06	1.670	19.53	1.018	13.18	0.606	29.05	1.568	8.06	0.766	11.23
T6-Ref	2.402	0.98	2.005	3.17	2.087	10.74	1.527	19.53	1.159	13.92	0.583	29.05	0.933	8.79	0.858	10.99
O1-Ref	3.024	0.98	1.801	3.42	2.166	8.06	1.707	20.02	0.843	14.89	0.859	30.52	1.462	8.06	0.680	11.72
O2-Ref	3.050	0.98	1.865	3.17	2.198	8.06	1.653	15.87	0.881	14.40	0.839	28.81	1.358	8.06	0.836	11.23

TABLE H13: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 1 when the Left Eye of the Participant was Open in Phase II.*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.652	0.98	1.530	4.39	1.118	9.77	1.356	15.87	0.801	14.89	0.579	27.83	0.528	8.79	0.499	10.99
Fp2-Ref	2.505	0.98	1.677	2.93	1.197	10.99	1.515	15.87	0.724	14.89	0.545	28.56	0.583	8.79	0.582	10.99
F7-Ref	1.967	0.98	1.310	6.84	1.173	8.79	1.015	15.87	0.619	13.67	0.520	32.23	0.597	8.79	0.437	11.96
F3-Ref	2.544	1.22	2.152	3.42	1.476	8.54	1.682	15.87	0.759	14.89	0.534	28.81	0.869	8.54	0.471	10.99
Fz-Ref	2.875	0.98	2.518	2.93	1.609	8.79	1.876	15.87	0.828	14.89	0.482	32.23	0.967	8.79	0.534	10.99
F4-Ref	2.957	1.71	2.427	2.93	1.477	8.79	1.944	15.87	0.813	14.89	0.609	30.03	0.779	8.79	0.618	10.99
F8-Ref	2.214	1.95	1.606	2.93	1.299	10.01	1.332	17.82	0.661	13.18	0.563	30.27	0.815	7.81	0.427	10.99
T3-Ref	1.710	0.98	1.515	6.59	1.643	8.54	1.665	18.80	0.617	14.65	0.946	31.49	0.795	8.54	0.756	11.72
C3-Ref	2.477	2.69	2.459	2.93	1.637	8.54	1.741	16.11	0.826	14.40	0.523	28.81	1.033	8.54	0.474	11.96
Cz-Ref	2.937	2.20	2.711	2.93	1.820	8.54	1.888	16.60	0.875	14.89	0.561	28.81	1.240	8.54	0.557	11.96
C4-Ref	2.775	0.98	2.440	2.93	1.648	8.54	1.871	17.58	0.880	13.18	0.567	29.79	1.022	8.54	0.573	11.96
T4-Ref	2.378	1.71	1.925	2.93	1.800	10.01	1.428	17.58	0.600	13.18	0.737	27.83	0.859	7.81	0.607	11.72
T5-Ref	2.090	2.44	1.976	6.35	1.779	8.54	2.247	18.55	0.886	13.67	1.429	31.49	0.987	8.54	0.937	11.72
P3-Ref	2.455	2.93	2.773	2.93	1.743	8.54	1.742	16.11	0.961	14.40	0.607	28.32	1.250	8.54	0.587	11.96
Pz-Ref	2.449	2.93	2.671	2.93	1.889	8.54	1.647	16.11	0.955	12.94	0.574	29.30	1.386	8.54	0.623	11.96
P4-Ref	2.137	2.93	2.571	2.93	1.742	8.54	1.677	17.58	0.752	12.94	0.638	27.83	1.242	8.54	0.622	11.96
T6-Ref	1.907	2.93	2.166	2.93	1.861	7.81	2.468	19.78	0.752	14.65	1.276	27.83	0.957	7.81	0.856	11.72
O1-Ref	2.269	2.44	2.251	2.93	1.654	8.54	1.501	16.11	0.776	13.92	0.773	28.08	1.134	8.54	0.541	11.72
O2-Ref	2.235	2.93	2.157	2.93	1.801	8.54	1.457	19.29	0.774	13.18	0.750	33.20	1.228	8.54	0.606	10.99

TABLE H14: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 2 when Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.230	1.71	1.651	3.17	1.175	8.79	1.143	19.04	0.682	12.94	0.662	29.79	0.549	8.79	0.674	11.23
Fp2-Ref	2.167	1.22	1.660	3.42	1.118	8.79	1.374	16.60	0.848	14.16	0.815	29.05	0.436	8.79	0.661	11.47
F7-Ref	1.373	1.22	1.459	3.17	1.164	8.79	1.053	20.75	0.572	12.94	0.537	29.79	0.496	8.79	0.594	11.23
F3-Ref	2.485	1.22	2.107	3.17	1.591	7.81	1.360	16.60	0.919	12.94	0.608	29.05	0.828	7.81	0.897	11.23
Fz-Ref	2.489	1.22	2.299	3.17	1.620	8.30	1.518	16.60	0.949	12.94	0.492	33.69	0.977	8.30	0.916	11.47
F4-Ref	2.518	1.22	2.156	2.93	1.654	8.30	1.658	19.04	0.846	12.94	1.089	31.01	0.899	8.30	0.963	11.47
F8-Ref	2.316	1.22	1.688	3.42	1.295	11.47	1.372	17.09	0.762	14.89	0.711	28.81	0.403	8.79	0.701	11.47
T3-Ref	1.613	1.22	1.298	2.93	1.377	10.01	0.843	15.87	0.657	12.94	0.471	29.79	0.633	8.54	0.652	11.47
C3-Ref	2.581	1.22	2.544	2.93	2.121	7.81	1.477	15.87	1.065	12.94	0.540	29.79	1.308	7.81	0.868	11.47
Cz-Ref	3.317	1.22	2.400	2.93	1.833	8.30	1.746	16.85	1.278	12.94	0.565	29.79	1.040	8.30	1.108	11.47
C4-Ref	2.771	1.22	2.253	2.93	1.644	8.30	1.710	16.60	1.087	12.94	0.540	29.05	0.815	8.30	0.985	11.47
T4-Ref	2.346	1.22	1.658	3.66	1.575	10.50	1.213	20.02	0.774	13.92	0.467	30.27	0.460	8.79	0.695	11.72
T5-Ref	3.130	1.46	1.669	2.93	1.778	8.54	1.147	15.87	0.691	14.89	0.813	30.76	1.036	8.54	0.645	11.96
P3-Ref	3.433	1.22	2.331	2.93	2.130	8.54	1.657	19.04	0.945	14.65	0.643	29.79	1.363	8.54	0.686	11.96
Pz-Ref	3.944	1.22	2.260	2.93	1.849	7.81	1.695	19.04	1.204	13.18	0.590	29.79	1.043	7.81	0.791	11.47
P4-Ref	3.582	0.98	2.045	2.93	1.587	8.30	1.478	17.82	1.219	13.92	0.593	29.79	0.784	8.30	0.802	11.47
T6-Ref	2.460	0.98	1.736	2.93	1.352	8.30	1.318	20.02	0.926	13.92	0.638	30.03	0.743	8.30	0.511	11.96
O1-Ref	3.615	1.22	1.920	2.93	1.691	8.54	1.561	15.87	0.810	14.89	0.939	30.76	1.052	8.54	0.666	11.47
O2-Ref	3.437	1.22	1.725	2.93	1.523	8.30	1.259	17.82	1.011	13.43	0.887	30.03	0.928	8.30	0.566	11.47

TABLE H15: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 2 when the Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.893	1.46	1.661	3.17	1.228	7.81	1.148	17.09	0.707	12.94	0.458	29.54	0.880	7.81	0.517	11.23
Fp2-Ref	2.063	0.98	1.710	3.17	1.134	8.06	1.194	17.09	0.667	13.18	0.446	27.83	0.689	8.06	0.578	11.23
F7-Ref	1.146	1.71	1.408	3.42	1.173	7.81	1.071	18.31	0.708	13.18	0.431	28.56	0.782	7.81	0.405	10.99
F3-Ref	1.849	1.71	2.089	3.17	1.507	7.81	1.706	17.09	0.938	13.18	0.551	28.08	1.082	7.81	0.494	11.96
Fz-Ref	2.431	0.98	2.291	3.17	1.588	7.81	1.681	17.09	1.094	13.43	0.508	30.03	1.157	7.81	0.633	11.23
F4-Ref	2.839	1.22	2.145	3.17	1.375	8.06	1.538	18.07	0.974	13.18	0.651	27.83	0.873	8.06	0.656	11.23
F8-Ref	2.262	0.98	1.517	3.17	1.153	8.79	1.273	17.09	0.616	13.18	0.582	27.83	0.460	8.79	0.537	11.23
T3-Ref	1.864	0.98	1.399	3.66	1.322	7.81	1.321	21.24	0.849	13.18	0.581	36.38	0.734	7.81	0.598	11.47
C3-Ref	2.924	0.98	2.063	6.59	1.814	7.81	1.765	17.09	1.287	13.18	0.486	27.83	1.236	7.81	0.667	11.96
Cz-Ref	3.297	0.98	2.228	6.59	1.914	7.81	1.786	17.33	1.181	13.43	0.582	30.03	1.391	7.81	0.702	11.96
C4-Ref	3.488	1.22	1.877	6.84	1.615	8.06	1.518	17.09	1.167	13.18	0.583	27.83	1.084	8.06	0.657	11.23
T4-Ref	2.805	1.22	1.692	6.59	1.492	9.77	1.053	17.09	0.850	13.43	0.588	27.83	0.490	8.79	0.657	11.72
T5-Ref	2.740	0.98	1.815	3.17	1.851	11.47	1.863	20.75	1.085	13.18	0.868	27.83	0.856	7.81	1.123	11.47
P3-Ref	3.875	0.98	2.092	6.84	1.980	7.81	1.740	19.29	1.396	13.18	0.560	32.23	1.305	7.81	0.894	11.47
Pz-Ref	3.579	0.98	2.117	3.66	2.033	7.81	1.620	17.33	1.668	13.43	0.539	31.25	1.335	7.81	0.796	10.99
P4-Ref	3.951	0.98	1.790	6.84	1.735	7.81	1.431	17.33	1.511	13.43	0.576	31.49	1.015	7.81	0.691	10.99
T6-Ref	3.176	1.22	1.879	6.84	1.492	9.77	1.188	17.33	1.186	13.92	0.599	28.81	0.699	7.81	0.696	11.47
O1-Ref	3.767	1.95	1.750	3.66	1.530	7.81	1.529	16.60	1.204	13.92	0.756	32.23	0.819	7.81	0.776	11.47
O2-Ref	3.773	1.46	1.762	3.17	1.502	7.81	1.324	17.58	1.447	13.67	0.734	31.49	0.829	7.81	0.639	10.99

TABLE H16: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 2 when the Left Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.078	1.46	1.613	2.93	1.275	10.01	1.160	18.55	0.617	14.89	0.590	28.56	0.640	8.30	0.554	10.99
Fp2-Ref	2.112	1.46	1.889	2.93	1.293	10.01	1.210	18.55	0.651	14.16	0.598	28.56	0.618	8.30	0.486	10.99
F7-Ref	1.430	0.98	1.267	5.62	1.168	10.01	1.077	17.58	0.515	14.89	0.462	28.81	0.674	8.06	0.402	11.96
F3-Ref	1.959	1.22	1.990	3.91	1.725	10.01	1.447	18.55	0.696	14.89	0.559	28.81	1.032	8.06	0.665	11.96
Fz-Ref	2.058	1.71	2.411	4.64	1.904	8.30	1.544	18.55	0.767	14.89	0.592	28.56	1.141	8.30	0.720	10.99
F4-Ref	2.224	1.46	2.406	3.91	1.629	8.30	1.526	18.55	0.701	13.92	0.749	31.01	0.874	8.30	0.661	10.99
F8-Ref	1.571	1.22	1.724	5.86	1.260	9.77	1.297	18.31	0.465	13.18	0.592	27.83	0.640	8.30	0.435	11.47
T3-Ref	1.366	1.71	1.379	4.88	1.272	8.79	1.182	16.11	0.678	14.89	0.580	29.30	0.679	8.79	0.445	10.99
C3-Ref	2.067	1.22	2.275	4.64	1.981	8.06	1.596	15.87	0.828	14.89	0.588	28.81	1.141	8.06	0.760	10.99
Cz-Ref	2.921	1.71	2.626	4.64	2.274	10.01	1.696	18.55	0.789	14.89	0.668	28.08	1.201	8.06	0.823	11.96
C4-Ref	2.594	1.22	2.388	3.66	2.075	8.06	1.753	16.85	0.646	12.94	0.629	29.05	1.268	8.06	0.677	10.99
T4-Ref	2.237	1.71	1.572	2.93	1.568	9.52	1.199	18.31	0.559	13.18	0.569	31.49	0.766	8.06	0.613	11.47
T5-Ref	1.773	2.44	1.575	3.66	1.754	8.06	1.424	15.87	0.938	14.89	0.852	31.01	1.031	8.06	0.789	10.99
P3-Ref	2.283	1.22	2.300	4.88	2.168	8.06	1.650	16.11	0.969	14.89	0.629	28.81	1.319	8.06	0.667	10.99
Pz-Ref	2.873	1.71	2.081	4.64	2.203	8.06	1.706	17.82	1.042	12.94	0.619	28.08	1.261	8.06	0.721	10.99
P4-Ref	2.569	1.71	1.835	3.66	2.153	8.06	1.562	16.85	0.816	14.89	0.601	29.79	1.442	8.06	0.595	10.99
T6-Ref	2.324	1.71	1.638	3.66	1.781	8.06	1.341	17.58	0.732	14.16	0.689	28.32	1.082	8.06	0.552	10.99
O1-Ref	2.254	1.71	1.586	3.66	1.815	8.06	1.492	16.11	1.142	14.89	0.803	28.08	1.186	8.06	0.525	10.99
O2-Ref	2.251	1.71	1.626	3.66	1.863	8.06	1.486	16.85	1.137	12.94	0.819	31.25	1.339	8.06	0.375	11.96

TABLE H17: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 3 when the Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.548	1.22	1.344	3.66	1.337	7.81	1.383	20.75	0.700	13.18	0.668	28.32	0.844	7.81	0.341	11.72
Fp2-Ref	2.114	1.22	1.415	5.13	1.475	8.06	1.020	16.11	0.718	13.43	0.796	28.32	0.966	8.06	0.469	11.72
F7-Ref	2.047	0.98	1.503	3.66	1.241	7.81	1.177	18.55	0.600	13.18	0.591	28.81	0.677	7.81	0.541	11.23
F3-Ref	3.087	0.98	1.707	3.66	1.596	8.79	1.746	20.26	0.813	13.92	0.770	30.52	0.963	8.79	0.596	11.96
Fz-Ref	3.182	1.22	1.896	3.66	1.690	7.81	1.521	17.82	0.836	13.43	0.455	32.71	1.074	7.81	0.546	10.99
F4-Ref	2.525	1.22	1.863	3.17	1.800	8.30	2.077	17.82	0.794	12.94	1.199	28.08	1.195	8.30	0.582	11.96
F8-Ref	1.898	1.22	1.412	5.13	1.552	8.06	1.255	24.41	0.556	13.67	0.739	27.83	1.024	8.06	0.506	11.47
T3-Ref	2.217	1.22	1.321	3.66	1.205	10.74	1.072	17.58	0.861	13.67	0.532	32.47	0.331	8.06	0.670	11.47
C3-Ref	2.928	0.98	2.003	2.93	1.381	8.79	1.778	17.58	1.104	13.67	0.540	27.83	0.671	8.79	0.649	10.99
Cz-Ref	3.719	0.98	2.379	2.93	1.703	8.79	1.726	17.82	0.933	14.40	0.577	32.71	0.820	8.79	0.815	10.99
C4-Ref	2.612	1.22	1.979	2.93	1.795	8.30	1.564	17.82	0.802	14.40	0.518	32.71	1.119	8.30	0.792	10.99
T4-Ref	2.022	1.46	1.195	2.93	1.575	8.06	1.129	17.82	0.998	13.43	0.448	31.25	0.849	8.06	0.658	11.23
T5-Ref	2.121	1.46	1.632	3.66	1.091	9.52	1.544	17.58	1.152	13.67	0.771	32.47	0.382	8.79	0.435	10.99
P3-Ref	2.258	1.46	2.154	2.93	1.387	9.52	1.874	17.58	1.468	14.40	0.683	29.30	0.696	8.79	0.684	10.99
Pz-Ref	2.759	0.98	2.457	2.93	1.669	10.99	1.912	17.82	1.224	14.40	0.718	29.30	0.921	8.30	0.877	10.99
P4-Ref	2.250	1.46	2.246	2.93	1.561	8.30	1.808	17.58	0.984	14.16	0.721	29.30	0.949	8.30	0.817	10.99
T6-Ref	1.829	1.46	1.648	2.93	1.406	9.77	1.542	17.58	1.109	14.16	0.700	29.30	0.744	8.30	0.597	11.96
O1-Ref	1.753	0.98	2.022	2.93	1.281	8.06	1.678	17.58	1.062	14.40	0.941	29.30	0.780	8.06	0.561	11.72
O2-Ref	2.068	1.46	1.776	2.93	1.492	8.06	1.707	17.58	1.068	14.40	1.176	31.98	0.947	8.06	0.678	11.72

TABLE H18: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 3 when the Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.790	1.95	1.049	3.17	1.326	7.81	1.025	20.02	0.615	12.94	0.507	33.69	0.680	7.81	0.538	10.99
Fp2-Ref	1.579	1.95	1.236	3.17	1.415	7.81	1.178	17.33	0.607	12.94	0.549	28.81	0.780	7.81	0.509	10.99
F7-Ref	1.618	0.98	0.959	3.42	1.100	7.81	0.831	19.29	0.519	13.92	0.419	35.64	0.576	7.81	0.634	11.72
F3-Ref	2.494	0.98	1.468	3.42	1.673	7.81	1.326	17.58	0.748	13.92	0.530	29.54	1.006	7.81	0.652	11.72
Fz-Ref	2.849	0.98	1.737	3.91	2.025	7.81	1.573	17.82	0.774	13.92	0.449	29.05	1.260	7.81	0.576	11.72
F4-Ref	2.274	0.98	1.845	3.91	1.967	7.81	1.755	17.82	0.725	14.16	0.668	27.83	1.159	7.81	0.533	11.72
F8-Ref	1.691	0.98	1.194	3.42	1.552	9.03	1.136	20.26	0.541	12.94	0.564	32.96	0.590	8.79	0.519	11.47
T3-Ref	1.943	1.22	1.175	3.17	1.177	11.47	1.145	19.29	0.902	13.92	0.585	31.25	0.513	7.81	0.778	11.47
C3-Ref	2.717	1.22	1.513	5.86	1.864	7.81	1.414	15.87	0.973	13.92	0.464	29.79	1.275	7.81	0.599	11.47
Cz-Ref	3.614	1.22	1.850	4.39	2.252	7.81	1.490	16.11	1.052	13.92	0.565	29.79	1.511	7.81	0.431	11.72
C4-Ref	2.792	1.22	1.645	3.91	1.865	9.52	1.667	16.11	1.040	14.16	0.538	31.25	1.009	7.81	0.397	11.47
T4-Ref	1.663	1.71	1.412	5.13	1.807	9.28	1.203	18.31	0.727	12.94	0.478	28.81	0.481	8.79	0.557	11.96
T5-Ref	1.772	1.22	1.268	3.42	1.349	8.54	1.467	16.36	0.706	14.40	0.826	31.01	0.853	8.54	0.436	10.99
P3-Ref	2.407	1.22	1.561	3.42	1.800	8.30	1.365	18.07	0.962	13.92	0.515	27.83	1.257	8.30	0.363	10.99
Pz-Ref	3.042	1.22	1.623	5.13	2.012	9.28	1.451	16.36	1.055	13.92	0.529	29.54	1.279	8.06	0.382	11.96
P4-Ref	2.727	1.22	1.660	5.13	1.792	9.28	1.432	16.36	1.078	13.92	0.531	28.56	0.929	8.30	0.518	11.47
T6-Ref	1.809	0.98	1.590	3.66	1.679	9.28	1.305	16.36	0.874	13.43	0.562	34.18	0.687	7.81	0.462	11.96
O1-Ref	2.183	1.22	1.390	5.86	1.488	8.30	1.320	16.36	0.626	13.18	0.709	29.54	0.965	8.30	0.496	11.23
O2-Ref	2.330	1.22	1.553	3.42	1.523	9.28	1.322	16.36	0.634	14.89	0.827	28.08	0.816	8.30	0.623	11.96

TABLE H19*: Table of EEG Power Spectra for all Bandranges with the Photograph of Image 3 when the Left Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.845	1.46	1.168	5.62	1.080	7.81	0.994	17.09	1.087	13.67	0.601	31.98	0.596	7.81	0.619	11.47
Fp2-Ref	2.644	1.46	1.152	5.62	1.256	7.81	1.239	17.09	1.194	13.67	0.563	27.83	0.558	7.81	0.478	11.72
F7-Ref	2.217	1.22	1.027	5.62	0.949	11.47	0.925	17.09	0.868	13.67	0.505	28.56	0.504	7.81	0.636	11.47
F3-Ref	2.523	0.98	1.658	3.91	1.709	7.81	1.305	18.31	1.355	14.89	0.538	28.56	0.916	7.81	0.764	11.47
Fz-Ref	3.064	2.69	2.020	4.88	1.847	7.81	1.487	18.31	1.445	13.67	0.544	31.98	0.915	7.81	0.756	11.47
F4-Ref	2.727	2.69	1.846	4.64	1.782	9.52	1.641	21.00	1.115	13.67	0.759	32.23	0.771	7.81	0.733	11.72
F8-Ref	2.005	1.46	0.955	4.64	1.388	9.28	1.528	17.33	0.961	14.40	0.764	30.27	0.498	7.81	0.304	11.72
T3-Ref	1.784	0.98	1.109	3.17	1.082	7.81	0.648	15.87	0.863	13.43	0.453	29.05	0.751	7.81	0.457	11.47
C3-Ref	2.514	1.71	1.700	5.37	1.745	7.81	1.296	16.11	1.520	13.67	0.509	29.05	0.995	7.81	0.640	11.72
Cz-Ref	3.089	1.71	1.844	5.37	2.118	9.28	1.714	17.58	1.275	13.67	0.765	28.08	1.085	7.81	0.774	11.72
C4-Ref	2.706	2.69	1.692	4.15	2.036	9.28	1.892	17.58	0.914	14.89	0.588	28.08	0.933	7.81	0.531	11.72
T4-Ref	2.143	2.69	1.347	4.88	1.961	9.28	1.309	17.58	0.751	13.92	0.444	28.32	0.456	7.81	0.471	11.72
T5-Ref	2.100	2.69	1.598	2.93	1.322	9.03	1.424	16.11	1.063	13.92	0.812	32.23	0.706	8.79	0.528	11.23
P3-Ref	2.981	1.71	1.956	2.93	1.740	9.03	1.840	16.11	1.554	13.92	0.465	29.05	0.989	7.81	0.481	11.23
Pz-Ref	3.520	1.71	1.553	6.35	1.581	7.81	1.642	17.58	1.413	13.67	0.478	28.08	0.883	7.81	0.402	10.99
P4-Ref	2.966	1.71	1.707	3.17	1.488	9.52	1.630	19.29	1.235	14.89	0.533	28.08	0.548	7.81	0.326	11.96
T6-Ref	2.292	2.69	1.926	2.93	1.749	9.28	1.314	18.80	0.804	14.89	0.607	28.08	0.323	8.30	0.470	11.23
O1-Ref	2.609	1.71	1.811	2.93	1.256	11.23	1.514	16.11	1.093	13.92	0.483	31.25	0.624	8.30	0.768	11.23
O2-Ref	2.417	1.71	1.829	3.17	1.294	9.52	1.393	19.04	1.187	14.16	0.609	31.25	0.394	8.79	0.522	11.23

TABLE H20: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 4 when Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	3.639	1.46	1.903	3.66	1.114	10.25	1.818	18.07	0.733	13.92	0.913	29.05	0.536	8.54	0.196	11.96
Fp2-Ref	2.959	1.46	1.520	3.66	1.100	9.52	2.161	16.11	0.833	13.67	1.354	30.52	0.512	8.30	0.459	11.96
F7-Ref	2.414	1.46	1.657	3.91	1.035	10.74	1.235	16.11	0.722	13.67	0.554	29.05	0.453	8.54	0.438	11.47
F3-Ref	3.259	1.46	2.056	6.10	1.512	9.03	1.679	16.11	0.789	13.67	0.641	28.08	0.774	8.79	0.452	11.47
Fz-Ref	4.070	1.46	2.306	6.10	1.360	8.06	1.617	16.11	0.873	13.67	0.588	28.08	0.723	8.06	0.468	10.99
F4-Ref	3.259	1.46	1.958	6.10	1.198	9.52	2.124	16.36	0.702	13.92	1.446	28.08	0.661	8.06	0.449	10.99
F8-Ref	1.928	1.46	1.095	6.59	1.084	9.52	1.649	18.07	0.550	14.65	1.002	29.05	0.515	8.30	0.408	10.99
T3-Ref	2.280	1.22	1.551	5.13	0.791	8.30	1.188	16.11	0.728	13.92	0.388	29.05	0.480	8.30	0.438	11.47
C3-Ref	3.746	1.46	2.025	3.91	1.211	9.03	1.818	16.11	0.994	13.67	0.494	29.05	0.658	8.06	0.476	11.47
Cz-Ref	4.949	1.46	2.485	6.10	1.253	11.47	1.966	16.11	0.856	13.67	0.556	28.08	0.594	8.06	0.572	11.47
C4-Ref	4.025	1.46	1.950	3.91	1.090	9.52	1.771	16.11	0.935	13.67	0.550	28.08	0.551	8.30	0.417	11.47
T4-Ref	2.006	1.46	1.136	2.93	1.240	9.52	1.536	18.07	0.828	12.94	0.518	29.05	0.492	8.30	0.504	11.47
T5-Ref	2.305	1.22	1.770	3.91	0.897	9.03	1.876	16.11	0.963	13.92	0.560	36.38	0.354	8.30	0.294	11.96
P3-Ref	4.355	1.46	2.353	3.91	1.241	10.74	2.412	16.11	1.133	13.67	0.473	36.38	0.263	8.06	0.471	10.99
Pz-Ref	4.744	1.46	2.431	3.91	1.491	11.47	2.541	16.11	1.070	13.67	0.496	30.27	0.399	8.06	0.839	11.47
P4-Ref	4.035	1.46	2.037	3.91	1.454	9.52	2.307	16.11	0.922	13.18	0.555	34.42	0.421	8.30	0.857	11.47
T6-Ref	2.476	0.98	1.674	2.93	1.393	11.23	1.702	18.07	1.036	14.40	0.627	38.33	0.450	7.81	0.868	11.23
O1-Ref	3.564	1.22	1.910	3.91	0.904	11.96	1.929	16.11	1.040	13.18	0.827	36.38	0.227	7.81	0.237	11.96
O2-Ref	3.445	0.98	1.685	3.91	1.153	11.96	2.030	16.11	0.917	13.18	0.988	38.33	0.315	8.79	0.658	11.96

TABLE H21: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 4 when Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.514	1.46	1.297	6.35	1.192	10.99	1.151	19.53	0.439	14.89	0.551	33.20	0.447	8.79	0.640	10.99
Fp2-Ref	1.669	1.46	1.223	5.37	1.207	10.50	1.122	19.53	0.373	14.65	0.613	29.54	0.491	8.79	0.642	10.99
F7-Ref	1.486	0.98	1.119	5.37	1.212	10.74	1.307	20.75	0.638	13.43	0.631	30.52	0.420	8.79	0.566	10.99
F3-Ref	2.292	0.98	1.895	5.37	1.461	11.23	1.532	19.78	0.514	14.89	0.674	29.54	0.600	8.79	0.739	11.23
Fz-Ref	2.386	0.98	2.219	5.13	1.583	7.81	1.607	19.78	0.548	14.89	0.589	29.54	0.822	7.81	0.695	11.23
F4-Ref	1.955	1.46	1.859	5.37	1.404	7.81	2.128	19.53	0.786	14.40	0.743	29.54	0.759	7.81	0.623	11.23
F8-Ref	1.525	1.46	1.057	4.64	0.866	11.47	1.345	19.29	0.585	14.89	0.846	29.54	0.447	7.81	0.420	11.47
T3-Ref	2.179	1.22	1.479	5.13	1.465	9.03	2.032	20.02	1.080	12.94	1.118	31.25	0.357	8.79	0.656	11.47
C3-Ref	2.707	0.98	2.338	3.66	1.832	9.03	1.844	18.55	0.662	14.89	0.804	29.54	0.739	8.79	0.819	10.99
Cz-Ref	3.074	0.98	2.449	5.86	2.039	7.81	1.949	16.36	0.776	14.65	0.688	29.54	1.153	7.81	0.822	10.99
C4-Ref	2.150	0.98	2.172	4.15	1.521	7.81	1.872	16.36	0.807	14.40	0.777	29.54	0.813	7.81	0.646	11.23
T4-Ref	1.514	1.71	1.715	4.64	1.237	7.81	1.189	17.33	0.764	12.94	0.708	29.30	0.769	7.81	0.457	10.99
T5-Ref	2.212	2.20	2.024	3.91	1.150	9.77	1.822	18.31	0.828	13.92	1.022	28.08	0.495	8.79	0.510	11.47
P3-Ref	2.743	2.44	2.532	3.91	1.748	9.28	1.778	18.07	0.806	13.18	0.758	29.54	0.686	8.79	0.774	10.99
Pz-Ref	2.726	0.98	2.519	5.86	2.011	9.28	1.922	16.36	0.805	14.40	0.678	29.54	1.099	7.81	0.837	10.99
P4-Ref	2.532	2.44	2.421	4.15	1.679	9.28	1.628	17.33	0.815	13.92	0.651	34.67	0.952	7.81	0.632	10.99
T6-Ref	1.726	2.44	1.834	3.42	1.239	9.03	1.257	17.33	0.851	14.40	0.735	34.67	0.714	8.54	0.430	11.96
O1-Ref	2.144	2.44	2.183	3.17	1.484	9.28	1.504	17.33	0.864	13.18	0.880	28.08	0.711	8.30	0.527	10.99
O2-Ref	2.255	2.44	2.252	3.42	1.588	9.28	1.602	17.33	0.734	13.92	1.002	28.08	0.842	8.30	0.513	10.99

TABLE H22: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 4 when the Left Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.581	0.98	1.847	3.91	1.171	8.79	1.224	18.31	0.637	14.89	0.497	27.83	0.724	8.79	0.496	10.99
Fp2-Ref	2.064	0.98	1.766	3.91	1.185	8.79	1.178	18.55	0.580	14.89	0.466	34.67	0.718	8.79	0.497	10.99
F7-Ref	1.730	1.95	1.698	3.42	1.185	11.47	0.945	18.31	0.794	14.16	0.474	30.52	0.626	8.54	0.711	11.47
F3-Ref	1.970	0.98	2.122	3.42	1.511	8.79	1.430	18.31	0.742	14.16	0.452	30.52	0.887	8.79	0.678	11.47
Fz-Ref	2.544	0.98	2.069	2.93	1.597	8.79	1.558	18.31	0.771	14.16	0.491	34.42	0.948	8.79	0.618	10.99
F4-Ref	2.567	0.98	1.848	3.91	1.513	8.79	1.446	18.80	0.842	13.67	0.582	29.54	0.909	8.79	0.584	10.99
F8-Ref	2.460	0.98	1.451	3.91	1.306	9.03	1.165	18.31	0.750	13.67	0.550	29.79	0.757	8.79	0.480	10.99
T3-Ref	1.821	0.98	1.300	3.42	1.431	10.25	1.203	16.36	0.953	13.92	0.625	29.30	0.669	8.54	0.697	10.99
C3-Ref	2.544	1.71	2.081	2.93	1.483	9.52	1.419	17.33	0.909	13.92	0.515	29.05	0.771	8.79	0.652	10.99
Cz-Ref	2.796	1.95	2.466	2.93	1.714	8.79	1.653	18.80	0.835	13.92	0.642	29.54	0.992	8.79	0.641	10.99
C4-Ref	2.341	1.46	2.022	2.93	1.447	9.03	1.796	18.80	0.784	14.89	0.744	28.81	0.776	8.79	0.638	11.72
T4-Ref	2.926	1.22	1.341	3.91	1.572	9.03	1.332	18.80	0.944	13.18	0.860	29.79	0.790	8.06	0.534	11.72
T5-Ref	2.271	1.71	1.823	2.93	1.335	10.25	1.435	21.73	0.858	13.92	0.761	27.83	0.477	7.81	0.449	10.99
P3-Ref	3.195	1.71	2.159	3.91	1.437	10.25	1.405	17.33	0.993	13.92	0.609	28.81	0.511	8.54	0.762	10.99
Pz-Ref	2.982	1.95	2.391	2.93	1.519	10.25	1.557	17.33	0.917	13.92	0.593	28.81	0.708	8.06	0.817	10.99
P4-Ref	2.951	1.46	2.305	3.66	1.245	11.72	1.759	18.80	0.933	13.92	0.589	28.81	0.532	8.06	0.762	11.72
T6-Ref	3.687	1.22	2.108	3.66	1.238	10.50	1.473	18.80	1.055	13.18	0.685	31.49	0.536	8.30	0.620	11.47
O1-Ref	2.420	1.95	1.791	2.93	1.268	10.25	1.352	16.60	0.860	14.40	0.723	27.83	0.354	7.81	0.628	11.47
O2-Ref	2.651	1.46	1.777	2.93	1.124	10.25	1.598	17.09	0.792	13.92	0.807	29.54	0.427	8.79	0.704	11.47

TABLE H23: *Table of EEG Power Spectra for all Bandranges with the Video of Image 1 when Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.046	0.98	1.307	3.91	1.750	10.74	1.669	17.33	0.903	14.40	0.973	31.74	0.719	8.30	0.731	10.99
Fp2-Ref	1.772	1.46	1.457	3.91	1.575	9.77	2.018	17.33	0.665	14.65	1.142	27.83	0.747	8.30	0.595	11.96
F7-Ref	1.931	1.46	1.063	3.42	1.316	10.74	1.303	17.33	0.728	14.40	0.655	37.11	0.557	8.06	0.627	11.96
F3-Ref	3.274	0.98	2.355	3.91	1.701	9.77	1.769	20.51	0.944	14.40	0.827	28.08	0.911	8.06	0.525	11.96
Fz-Ref	3.140	0.98	2.681	3.66	2.089	9.77	1.851	17.33	1.064	14.65	0.687	28.08	1.150	8.06	0.616	11.96
F4-Ref	2.502	0.98	2.712	3.66	2.102	8.30	3.023	21.24	1.286	13.92	1.460	28.08	1.332	8.30	0.922	11.96
F8-Ref	1.975	1.22	1.520	3.66	1.491	11.96	1.606	24.17	0.735	14.65	1.138	28.08	0.774	8.79	0.600	11.96
T3-Ref	1.404	1.46	1.366	4.64	1.467	9.52	1.223	17.33	0.663	13.92	0.468	30.52	0.679	8.06	0.509	10.99
C3-Ref	2.949	0.98	2.794	4.64	2.777	8.06	1.749	17.33	0.985	14.65	0.531	27.83	1.566	8.06	0.699	11.96
Cz-Ref	4.009	0.98	2.817	2.93	2.395	11.96	1.720	17.33	1.312	13.67	0.775	29.30	1.190	8.06	0.609	11.96
C4-Ref	2.763	0.98	3.004	2.93	2.230	11.96	1.587	18.55	1.447	14.65	0.635	29.54	1.374	8.30	1.082	11.96
T4-Ref	1.844	0.98	1.790	3.66	1.817	10.99	1.172	15.87	1.044	14.65	0.588	29.30	0.716	8.30	1.251	10.99
T5-Ref	1.898	0.98	1.821	3.17	1.948	10.50	1.498	17.33	0.917	13.67	0.614	30.76	0.890	8.06	0.805	11.96
P3-Ref	3.113	0.98	2.482	3.17	2.604	10.74	1.797	17.33	0.792	14.89	0.593	31.98	1.256	8.06	1.023	11.96
Pz-Ref	3.762	0.98	2.544	3.17	2.354	11.96	1.692	17.82	1.126	13.67	0.639	31.49	1.028	8.06	0.833	11.96
P4-Ref	3.447	0.98	2.683	3.17	1.838	11.96	1.556	18.31	1.363	14.40	0.697	29.30	0.727	8.30	0.899	11.96
T6-Ref	2.600	0.98	1.865	5.13	1.623	11.96	1.287	20.02	1.742	13.18	0.729	29.54	0.408	7.81	1.133	11.96
O1-Ref	2.890	0.98	2.285	3.17	1.356	10.74	1.933	16.60	0.830	13.18	0.774	31.49	0.563	7.81	0.633	11.96
O2-Ref	3.748	0.98	2.523	3.17	1.390	11.96	2.043	16.60	1.256	13.18	0.833	39.06	0.591	7.81	0.845	11.96

TABLE H24: *Table of EEG Power Spectra for all Bandranges with the Video of Image 1 when the Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.300	0.98	1.894	3.91	1.459	8.06	1.530	16.11	0.676	13.43	0.655	28.56	0.912	8.06	0.530	10.99
Fp2-Ref	2.135	0.98	1.881	3.91	1.596	8.79	1.609	16.11	0.757	12.94	0.730	27.83	0.936	8.79	0.663	10.99
F7-Ref	2.413	0.98	1.393	2.93	1.249	8.06	1.138	16.11	0.813	13.18	0.455	31.98	0.774	8.06	0.392	10.99
F3-Ref	3.505	0.98	2.126	6.59	1.943	7.81	1.725	16.11	0.976	13.18	0.549	28.56	1.298	7.81	0.606	10.99
Fz-Ref	3.322	0.98	2.340	6.59	2.230	7.81	1.780	16.11	1.136	14.16	0.540	28.56	1.511	7.81	0.646	10.99
F4-Ref	2.851	0.98	2.248	3.91	2.079	7.81	2.017	17.33	1.245	14.16	0.876	30.03	1.365	7.81	0.632	10.99
F8-Ref	1.886	2.44	1.599	2.93	1.650	9.77	1.462	19.78	0.654	12.94	0.703	27.83	0.806	8.79	0.570	10.99
T3-Ref	1.819	1.95	1.389	2.93	1.460	8.79	0.940	17.58	0.907	13.18	0.385	28.81	0.767	8.79	0.456	11.23
C3-Ref	3.159	1.95	2.024	6.59	2.324	8.06	1.636	17.58	1.528	13.67	0.560	28.56	1.545	8.06	0.498	10.99
Cz-Ref	3.281	0.98	2.026	6.59	2.418	8.06	1.811	20.02	1.363	13.18	0.652	30.76	1.635	8.06	0.621	11.72
C4-Ref	3.043	0.98	1.833	6.59	2.109	7.81	1.890	18.07	1.102	13.18	0.561	27.83	1.294	7.81	0.678	11.72
T4-Ref	2.147	1.71	1.492	2.93	1.583	10.01	1.359	18.55	0.767	12.94	0.479	27.83	0.685	7.81	0.668	11.23
T5-Ref	2.541	1.95	1.725	2.93	1.812	8.79	1.442	17.33	1.153	13.92	0.613	30.76	0.911	8.79	0.603	11.23
P3-Ref	3.367	1.95	2.222	2.93	2.550	8.79	1.665	17.33	1.667	13.92	0.490	28.81	1.351	8.79	0.792	11.23
Pz-Ref	3.519	1.95	2.248	2.93	2.611	8.79	1.651	17.58	1.595	12.94	0.528	28.81	1.324	8.79	0.938	11.72
P4-Ref	3.736	1.95	2.000	2.93	2.572	8.79	1.647	20.51	1.235	12.94	0.524	28.56	1.187	8.79	1.164	11.72
T6-Ref	2.984	1.95	1.689	2.93	2.000	11.72	1.406	19.78	0.913	13.67	0.459	29.79	0.892	8.79	1.136	11.72
O1-Ref	3.626	1.95	2.126	2.93	1.938	8.79	1.470	17.33	1.306	13.92	0.520	28.08	0.877	8.79	0.727	11.23
O2-Ref	3.542	1.95	1.870	2.93	2.044	8.79	1.602	18.31	1.092	13.43	0.543	30.27	0.860	8.79	0.950	11.72

TABLE H25: *Table of EEG Power Spectra for all Bandranges with the Video of Image 1 when the Left Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.881	1.22	1.567	3.66	1.417	7.81	1.146	15.87	0.913	14.16	0.620	27.83	0.882	7.81	0.498	10.99
Fp2-Ref	2.101	1.71	1.623	2.93	1.369	10.25	1.357	18.55	0.884	14.65	0.622	31.74	0.834	7.81	0.508	11.47
F7-Ref	1.796	1.46	1.278	3.42	1.172	7.81	0.984	18.31	0.769	14.16	0.524	31.01	0.626	7.81	0.443	11.96
F3-Ref	2.824	1.46	2.005	3.66	1.542	7.81	1.416	18.31	1.052	14.16	0.563	31.01	0.977	7.81	0.584	11.96
Fz-Ref	2.851	1.46	2.456	3.66	1.725	7.81	1.577	15.87	1.086	14.16	0.526	27.83	1.183	7.81	0.572	11.96
F4-Ref	2.596	1.71	2.283	2.93	1.690	7.81	1.882	20.75	1.095	13.43	0.912	28.08	1.096	7.81	0.594	11.96
F8-Ref	1.976	1.71	1.458	6.84	1.187	8.79	1.339	21.00	0.806	13.43	0.663	27.83	0.695	8.79	0.431	11.47
T3-Ref	2.145	1.71	1.154	3.42	1.260	9.77	1.119	17.33	0.676	14.16	0.619	32.71	0.562	8.79	0.553	10.99
C3-Ref	2.916	1.46	1.909	3.66	1.749	10.74	1.488	17.33	1.031	14.16	0.522	27.83	1.025	7.81	0.678	11.96
Cz-Ref	2.838	1.46	2.258	3.66	1.963	10.74	1.766	20.02	1.255	14.16	0.601	29.54	1.169	8.30	0.706	11.96
C4-Ref	2.377	2.69	2.201	2.93	1.990	7.81	1.619	20.75	1.240	13.43	0.632	27.83	1.265	7.81	0.669	11.96
T4-Ref	2.028	0.98	1.516	2.93	1.280	9.52	1.041	15.87	0.761	13.43	0.631	29.05	0.501	8.79	0.519	11.96
T5-Ref	2.596	0.98	1.337	3.42	1.600	8.79	1.351	18.55	0.914	13.18	0.776	28.32	0.688	8.79	0.666	10.99
P3-Ref	3.195	1.22	1.812	2.93	1.712	10.25	1.560	17.09	0.872	13.43	0.478	32.71	0.925	8.79	0.636	10.99
Pz-Ref	2.929	1.46	1.873	3.17	1.712	10.74	1.701	17.09	1.056	13.18	0.485	32.71	0.858	8.30	0.678	10.99
P4-Ref	2.563	1.46	1.789	3.17	1.768	10.74	1.617	20.75	1.168	13.43	0.530	33.20	0.928	7.81	0.681	10.99
T6-Ref	2.195	0.98	1.628	3.42	1.457	9.52	1.371	20.26	0.901	13.43	0.668	33.20	0.610	8.79	0.655	11.96
O1-Ref	2.743	1.22	1.291	2.93	1.448	8.79	1.306	17.09	1.022	13.43	0.601	32.71	0.711	8.79	0.463	10.99
O2-Ref	2.486	0.98	1.335	6.84	1.516	10.25	1.384	16.11	0.978	14.89	0.587	30.03	0.709	8.79	0.517	10.99

TABLE H26: *Table of EEG Power Spectra for all Bandranges with the Video of Image 2 when Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.972	0.98	1.348	4.39	1.341	8.30	2.026	15.87	1.088	13.67	1.180	32.71	0.815	8.30	0.800	11.23
Fp2-Ref	1.797	0.98	1.555	4.39	1.345	8.30	2.030	15.87	1.036	13.67	1.277	30.52	0.858	8.30	0.733	11.47
F7-Ref	1.429	0.98	1.018	4.15	1.027	8.30	1.200	16.85	0.837	14.40	0.495	28.08	0.654	8.30	0.543	11.47
F3-Ref	1.848	0.98	1.668	4.39	1.412	8.30	1.771	15.87	0.967	14.65	0.667	29.54	1.017	8.30	0.695	11.23
Fz-Ref	2.304	0.98	1.921	4.39	1.572	8.06	1.681	15.87	0.974	14.89	0.500	29.30	1.116	8.06	0.854	11.47
F4-Ref	2.000	1.95	2.002	4.39	1.445	8.30	2.724	17.09	1.117	13.18	1.224	30.52	1.000	8.30	0.663	11.47
F8-Ref	1.739	0.98	1.404	4.39	1.105	11.47	1.186	15.87	0.691	13.67	0.690	28.32	0.644	8.06	0.588	11.47
T3-Ref	1.202	0.98	1.201	5.62	1.129	8.06	1.015	16.36	0.731	13.67	0.429	30.03	0.660	8.06	0.550	11.23
C3-Ref	1.798	0.98	2.058	4.39	1.741	8.06	1.566	17.09	1.031	13.18	0.502	29.79	1.151	8.06	0.925	11.23
Cz-Ref	2.639	1.71	2.310	4.39	1.913	8.06	1.650	16.85	1.191	14.65	0.583	29.05	1.329	8.06	0.960	11.23
C4-Ref	2.311	1.71	2.031	4.39	1.577	8.06	1.681	15.87	1.328	13.18	0.497	30.52	1.044	8.06	0.814	11.23
T4-Ref	1.703	0.98	1.356	4.39	1.211	11.47	1.017	15.87	0.886	13.43	0.466	28.08	0.571	8.06	0.645	11.47
T5-Ref	2.005	0.98	1.490	2.93	1.451	8.79	1.230	16.11	0.926	14.16	0.681	29.05	0.856	8.79	0.600	10.99
P3-Ref	2.291	2.20	2.006	2.93	1.927	8.06	1.443	17.82	1.352	14.40	0.568	28.32	1.337	8.06	0.874	11.23
Pz-Ref	3.088	0.98	2.176	2.93	1.956	8.06	1.491	15.87	1.462	14.40	0.544	28.32	1.414	8.06	0.904	11.23
P4-Ref	2.345	2.20	1.885	4.39	1.664	8.06	1.513	15.87	1.407	13.18	0.595	32.71	1.191	8.06	0.751	11.96
T6-Ref	2.159	0.98	1.461	2.93	1.177	8.06	1.215	15.87	1.090	12.94	0.618	32.71	0.856	8.06	0.439	11.96
O1-Ref	2.832	1.22	1.255	2.93	1.616	8.06	1.636	15.87	1.518	14.40	0.769	29.05	1.035	8.06	0.661	11.96
O2-Ref	2.376	1.46	1.360	2.93	1.433	8.06	1.704	15.87	1.644	14.40	0.671	29.05	1.024	8.06	0.565	11.96

TABLE H27: *Table of EEG Power Spectra for all Bandranges with the Video of Image 2 when the Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.817	0.98	1.761	3.42	1.096	8.30	1.297	16.36	0.706	14.65	0.518	28.56	0.611	8.30	0.551	11.47
Fp2-Ref	1.722	1.46	1.435	3.42	1.269	8.30	1.464	18.07	0.550	14.65	0.684	29.30	0.764	8.30	0.614	11.47
F7-Ref	1.591	0.98	1.267	3.17	1.179	10.01	1.143	18.07	0.526	14.65	0.398	29.30	0.425	8.79	0.485	11.47
F3-Ref	2.328	0.98	2.061	3.17	1.514	8.30	1.391	17.82	0.889	14.65	0.444	29.54	0.859	8.30	0.778	11.47
Fz-Ref	2.769	0.98	2.067	3.17	1.771	8.30	1.519	17.82	0.781	14.65	0.436	29.30	1.060	8.30	0.806	11.47
F4-Ref	2.204	0.98	1.736	3.17	1.783	9.03	1.850	18.31	0.630	14.65	0.847	28.56	0.930	8.30	0.877	11.47
F8-Ref	1.745	1.95	1.170	4.39	1.507	9.03	1.468	23.68	0.539	14.16	0.705	28.56	0.701	8.79	0.635	11.23
T3-Ref	1.866	1.46	1.155	3.17	1.321	9.77	1.078	17.33	0.565	13.18	0.388	29.54	0.510	8.79	0.535	10.99
C3-Ref	2.415	1.22	1.958	3.17	1.781	8.30	1.400	18.31	0.872	14.40	0.443	27.83	1.138	8.30	0.765	11.96
Cz-Ref	3.227	1.71	2.267	3.17	2.135	8.79	1.529	18.80	0.987	14.65	0.512	31.01	1.229	8.79	0.866	11.47
C4-Ref	2.732	1.95	1.719	6.84	1.994	9.03	1.727	17.09	0.969	13.18	0.478	27.83	1.063	8.79	0.811	11.96
T4-Ref	1.745	1.46	1.281	6.59	1.814	9.52	1.244	17.09	0.691	12.94	0.467	33.45	0.671	8.79	0.666	11.96
T5-Ref	2.328	1.95	1.536	3.17	1.504	9.77	1.195	17.58	0.681	13.18	0.718	29.79	0.821	8.79	0.547	10.99
P3-Ref	2.854	1.95	2.048	3.17	1.973	8.30	1.277	17.58	1.009	13.18	0.541	30.03	1.287	8.30	0.756	10.99
Pz-Ref	3.224	1.71	2.046	3.17	2.083	8.79	1.466	17.58	1.158	13.18	0.529	28.81	1.339	8.79	0.812	11.72
P4-Ref	3.166	1.95	1.764	6.84	2.104	9.03	1.525	19.78	1.042	13.18	0.543	28.81	1.256	8.79	0.827	11.72
T6-Ref	2.498	1.95	1.400	6.84	1.931	9.03	1.436	17.09	0.797	12.94	0.560	28.56	1.032	8.79	0.732	11.96
O1-Ref	2.724	1.95	2.082	3.17	1.680	8.79	1.284	17.58	1.030	14.16	0.709	32.71	1.036	8.79	0.444	10.99
O2-Ref	2.770	1.95	1.902	2.93	1.791	8.79	1.412	17.58	0.843	12.94	0.738	28.56	1.085	8.79	0.605	11.96

TABLE H28: *Table of EEG Power Spectra for all Bandranges with the Video of Image 2 when the Left Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.298	1.46	1.330	3.17	1.196	8.54	1.250	15.87	0.729	14.89	0.711	28.81	0.650	8.54	0.561	11.23
Fp2-Ref	1.577	1.95	1.312	4.88	1.374	9.52	1.547	23.93	0.669	14.89	0.667	29.79	0.736	8.54	0.601	11.23
F7-Ref	1.268	1.46	1.091	3.17	0.874	9.03	1.041	15.87	0.460	14.89	0.422	31.74	0.512	7.81	0.329	11.96
F3-Ref	1.850	1.46	1.742	3.17	1.261	9.03	1.438	15.87	0.625	14.89	0.520	27.83	0.774	8.06	0.485	11.23
Fz-Ref	1.857	1.46	2.250	3.17	1.578	9.03	1.652	16.11	0.807	14.89	0.453	29.30	0.921	8.30	0.635	11.23
F4-Ref	1.981	2.20	2.129	4.88	1.630	9.52	1.989	19.29	1.061	14.89	0.786	29.30	0.881	8.06	0.703	11.47
F8-Ref	1.648	1.71	1.519	3.42	1.506	8.54	1.327	22.22	0.709	14.89	0.538	31.01	0.833	8.54	0.564	11.72
T3-Ref	1.875	1.46	1.562	3.17	0.954	8.06	0.921	19.53	0.513	14.89	0.380	27.83	0.581	8.06	0.359	11.96
C3-Ref	2.556	1.46	2.014	3.17	1.525	9.03	1.512	22.22	0.954	14.16	0.434	27.83	0.894	8.06	0.627	11.72
Cz-Ref	2.395	1.46	2.479	3.17	1.826	9.03	1.837	16.11	1.077	14.65	0.545	30.03	0.930	8.30	0.730	11.47
C4-Ref	2.474	2.20	2.540	3.17	1.770	9.52	1.904	19.29	1.273	13.43	0.517	32.47	0.981	8.30	0.684	11.72
T4-Ref	1.882	1.46	1.869	6.35	1.814	9.52	1.207	22.22	0.907	13.43	0.423	28.08	0.785	8.30	0.886	11.72
T5-Ref	1.853	1.95	1.695	3.17	1.149	9.77	1.359	16.85	0.676	14.40	0.775	29.54	0.557	8.06	0.436	10.99
P3-Ref	2.676	1.46	2.169	3.17	1.567	9.77	1.621	16.85	0.955	14.16	0.519	32.71	0.836	8.06	0.681	11.47
Pz-Ref	2.935	1.46	2.198	2.93	1.895	9.52	1.681	21.00	1.164	14.65	0.548	28.32	0.976	8.54	0.783	10.99
P4-Ref	2.578	2.20	2.334	3.42	1.916	9.52	1.496	18.55	1.308	14.40	0.521	28.32	1.052	7.81	0.749	10.99
T6-Ref	2.302	1.46	2.016	6.35	1.627	9.52	1.266	15.87	0.977	13.43	0.476	28.32	0.853	8.30	0.775	10.99
O1-Ref	2.312	2.20	1.712	2.93	1.190	9.77	1.303	16.85	0.771	13.18	0.798	32.71	0.537	7.81	0.503	11.47
O2-Ref	2.180	2.20	1.715	6.35	1.362	9.77	1.411	18.80	0.801	13.18	0.624	30.27	0.660	7.81	0.520	10.99

TABLE H29: *Table of EEG Power Spectra for all Bandranges with the Video of Image 3 when Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.672	0.98	1.454	3.17	1.359	11.72	1.699	17.82	0.632	14.89	1.196	30.52	0.516	7.81	0.907	11.72
Fp2-Ref	2.311	0.98	1.368	4.39	1.284	11.72	1.938	20.75	0.597	14.89	1.216	28.56	0.548	8.79	0.842	11.72
F7-Ref	1.941	0.98	1.226	6.59	0.918	9.52	1.163	21.00	0.643	13.67	0.629	30.03	0.386	7.81	0.489	11.23
F3-Ref	2.631	0.98	1.812	3.42	1.613	9.28	1.513	20.75	0.956	13.18	0.790	28.32	0.814	8.06	0.750	11.72
Fz-Ref	2.885	0.98	1.967	6.84	1.794	8.06	1.512	20.26	0.854	13.18	0.555	28.32	0.952	8.06	0.756	11.72
F4-Ref	2.601	0.98	1.771	6.84	1.664	8.06	1.907	20.26	0.917	13.43	1.289	29.05	0.999	8.06	0.657	11.72
F8-Ref	1.840	0.98	1.436	4.39	1.142	8.79	1.317	20.75	0.732	13.18	1.062	28.32	0.625	8.79	0.590	11.47
T3-Ref	1.796	0.98	1.569	6.35	1.288	9.52	1.722	22.71	0.932	13.43	1.107	31.25	0.529	8.30	0.715	11.47
C3-Ref	2.829	0.98	1.782	3.42	1.827	9.28	1.771	16.11	1.008	13.67	0.744	36.38	0.866	7.81	0.676	11.47
Cz-Ref	3.872	0.98	2.179	3.17	2.056	9.28	1.636	16.11	1.047	13.67	0.591	28.56	0.962	8.06	0.899	11.47
C4-Ref	3.279	0.98	1.826	3.17	1.873	9.28	1.607	20.75	0.984	13.18	0.696	29.05	1.009	7.81	0.733	11.47
T4-Ref	1.852	0.98	1.407	4.39	1.534	9.03	1.229	20.51	0.908	13.43	0.695	28.81	0.805	8.79	0.503	10.99
T5-Ref	1.634	1.46	1.721	6.59	1.440	9.28	1.312	20.26	0.830	14.40	0.851	32.23	0.550	7.81	0.798	10.99
P3-Ref	2.901	1.46	1.949	3.42	1.648	9.28	1.541	16.11	1.100	14.65	0.676	29.05	0.791	7.81	0.589	11.72
Pz-Ref	3.481	1.46	2.018	3.42	1.765	9.28	1.637	21.24	1.127	14.65	0.629	29.05	0.947	7.81	0.563	11.47
P4-Ref	3.151	0.98	1.734	3.42	1.853	9.28	1.575	21.97	1.120	14.65	0.611	29.05	1.041	7.81	0.590	11.23
T6-Ref	2.079	0.98	1.488	3.42	1.847	10.50	1.244	16.60	1.021	13.43	0.565	34.18	0.893	7.81	0.639	10.99
O1-Ref	2.417	1.46	1.767	3.17	1.520	7.81	1.631	16.11	1.275	14.65	0.886	33.94	0.970	7.81	0.610	11.23
O2-Ref	2.340	0.98	1.536	3.42	1.727	7.81	1.617	16.60	1.051	13.18	0.761	33.94	1.111	7.81	0.534	11.72

TABLE H30: *Table of EEG Power Spectra for all Bandranges with the Video of Image 3 when the Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.647	1.95	1.637	5.13	0.999	10.74	1.423	18.07	0.557	13.92	0.734	28.08	0.278	8.79	0.460	11.72
Fp2-Ref	1.923	1.71	1.494	2.93	0.807	10.50	2.151	21.24	0.814	13.92	1.132	31.74	0.403	7.81	0.249	11.23
F7-Ref	2.138	0.98	1.306	5.13	1.081	11.72	0.836	18.07	0.538	13.92	0.365	28.56	0.290	7.81	0.663	11.72
F3-Ref	1.984	1.95	2.073	6.84	1.159	11.72	1.375	18.07	0.906	13.92	0.517	32.23	0.283	8.79	0.728	11.72
Fz-Ref	2.616	1.71	2.278	6.84	1.159	11.23	1.471	18.07	0.909	13.92	0.437	32.23	0.393	7.81	0.726	11.23
F4-Ref	2.570	1.71	1.890	6.84	1.031	7.81	2.025	18.31	1.027	13.92	1.005	32.23	0.481	7.81	0.605	11.72
F8-Ref	2.345	1.46	1.459	5.62	0.986	9.77	1.855	18.07	0.624	13.43	0.692	37.11	0.409	7.81	0.370	11.96
T3-Ref	1.422	1.22	0.918	6.84	1.076	11.72	0.815	17.82	0.680	13.18	0.347	30.76	0.351	7.81	0.757	11.72
C3-Ref	2.316	2.44	1.740	6.84	1.355	11.72	1.435	17.82	0.994	13.43	0.454	28.32	0.594	7.81	0.935	11.72
Cz-Ref	2.949	1.95	2.196	6.84	1.364	11.23	1.638	16.60	0.991	13.43	0.518	28.32	0.677	7.81	0.901	11.23
C4-Ref	2.788	2.44	1.739	6.84	1.190	7.81	1.466	17.58	0.959	14.65	0.551	28.08	0.709	7.81	0.725	11.23
T4-Ref	2.240	1.46	1.295	6.84	1.403	9.77	1.192	17.82	0.639	14.65	0.511	31.25	0.697	7.81	0.542	11.96
T5-Ref	1.457	1.95	1.333	3.17	1.108	11.72	1.155	16.11	0.673	14.40	0.782	27.83	0.580	7.81	0.680	11.72
P3-Ref	2.011	1.95	1.490	6.84	1.322	7.81	1.553	17.58	0.650	13.43	0.648	30.03	0.771	7.81	0.733	11.72
Pz-Ref	2.765	1.95	1.439	6.84	1.276	8.06	1.404	17.58	0.834	13.43	0.567	30.03	0.780	8.06	0.822	11.47
P4-Ref	2.559	1.95	1.303	6.84	1.201	7.81	1.416	17.58	1.035	14.65	0.523	31.98	0.876	7.81	0.648	11.96
T6-Ref	2.014	1.95	1.072	6.84	1.297	11.96	1.225	17.09	0.856	14.65	0.616	31.98	0.759	7.81	0.727	11.96
O1-Ref	2.502	1.95	1.469	6.59	1.211	8.30	1.563	16.11	0.660	13.43	0.961	27.83	0.893	8.30	0.505	10.99
O2-Ref	2.464	1.95	1.310	6.59	1.166	11.96	1.468	16.11	0.995	13.18	0.844	29.30	0.893	8.06	0.589	11.96

TABLE H31*: Table of EEG Power Spectra for all Bandranges with the Video of Image 3 when the Left Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.406	1.71	1.595	2.93	1.346	11.47	1.323	18.31	0.673	14.40	0.697	29.30	0.766	8.30	0.668	11.47
Fp2-Ref	2.388	1.46	1.537	2.93	1.380	11.47	1.634	21.48	0.811	13.18	0.871	31.98	0.696	8.54	0.703	11.47
F7-Ref	1.437	1.46	1.498	2.93	1.190	8.30	1.076	19.04	0.575	14.40	0.501	29.54	0.695	8.30	0.615	11.47
F3-Ref	2.053	2.20	1.833	2.93	1.652	8.30	1.538	19.04	0.692	14.40	0.555	27.83	0.975	8.30	0.805	11.47
Fz-Ref	2.581	1.46	1.971	3.17	1.845	8.54	1.556	19.04	0.728	14.89	0.490	29.54	1.090	8.54	0.820	11.47
F4-Ref	2.557	1.46	1.946	3.17	1.809	8.79	1.717	18.55	0.828	13.67	0.919	31.98	1.024	8.79	0.752	11.47
F8-Ref	1.939	1.46	1.702	3.17	1.369	8.79	1.119	18.55	0.780	12.94	0.620	32.71	0.747	8.79	0.625	10.99
T3-Ref	1.502	2.20	1.234	3.17	1.422	11.72	1.108	17.58	0.631	14.40	0.421	28.08	0.593	8.30	0.795	11.72
C3-Ref	2.387	2.20	1.922	4.88	1.806	8.54	1.653	16.60	0.879	14.89	0.487	28.32	0.993	8.54	0.850	11.47
Cz-Ref	3.334	1.71	2.221	4.88	2.109	8.79	1.834	16.85	0.942	14.89	0.611	28.32	1.071	8.79	0.910	11.47
C4-Ref	2.906	1.95	2.135	4.88	1.980	8.79	1.915	16.60	0.859	14.89	0.587	33.45	1.031	8.79	0.995	11.47
T4-Ref	2.202	1.46	1.752	3.42	1.888	9.77	1.187	16.60	0.724	14.89	0.609	31.98	0.944	8.54	0.806	11.23
T5-Ref	2.159	1.46	1.381	3.42	1.399	9.52	1.387	16.85	1.059	14.65	0.679	30.76	0.607	8.54	0.488	11.96
P3-Ref	2.853	2.20	1.984	3.42	1.774	9.77	1.551	16.85	1.231	14.65	0.560	28.32	0.902	8.54	0.772	11.96
Pz-Ref	3.201	2.20	2.247	4.88	1.952	8.54	1.668	16.85	1.165	12.94	0.522	28.32	0.999	8.54	0.962	11.23
P4-Ref	2.993	0.98	2.247	4.88	2.054	8.79	1.666	19.53	0.986	12.94	0.500	31.98	1.088	8.79	1.047	11.23
T6-Ref	2.522	0.98	1.822	3.42	1.817	11.47	1.373	18.31	1.110	14.40	0.584	31.98	0.908	8.79	1.021	11.47
O1-Ref	2.851	0.98	1.568	2.93	1.538	9.77	1.415	16.85	1.408	13.67	0.718	27.83	0.658	8.79	0.417	11.23
O2-Ref	2.958	0.98	1.922	3.42	1.791	9.77	1.635	19.53	1.345	14.40	0.695	28.81	0.872	8.79	0.751	11.23

TABLE H32: *Table of EEG Power Spectra for all Bandranges with the Video of Image 4 when Both of the Participant's Eyes were Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.349	0.98	1.630	6.84	1.063	9.28	1.342	17.09	0.769	14.89	0.630	28.81	0.450	7.81	0.500	11.23
Fp2-Ref	2.234	0.98	1.580	6.84	1.134	9.28	1.831	18.07	0.845	14.89	1.110	28.56	0.503	8.30	0.548	11.96
F7-Ref	2.066	0.98	1.602	3.42	1.095	7.81	1.220	16.36	0.664	14.40	0.472	28.81	0.653	7.81	0.401	11.23
F3-Ref	2.709	0.98	2.152	5.37	1.485	7.81	1.804	17.09	0.836	14.89	0.553	28.56	0.804	7.81	0.721	11.23
Fz-Ref	2.848	0.98	2.276	6.84	1.419	10.50	1.713	17.09	1.098	13.92	0.555	28.56	0.664	7.81	0.712	11.96
F4-Ref	2.890	0.98	2.082	6.84	1.491	10.50	2.289	19.78	1.352	13.67	1.309	28.32	0.716	7.81	0.682	11.96
F8-Ref	2.431	0.98	1.592	6.84	1.379	9.28	1.270	16.11	0.776	13.18	0.775	29.79	0.688	8.30	0.646	11.72
T3-Ref	1.704	0.98	1.488	3.66	1.735	9.77	1.059	16.36	0.653	12.94	0.434	29.79	0.934	8.54	0.640	11.72
C3-Ref	2.069	0.98	2.138	3.66	2.276	7.81	1.822	16.36	0.940	14.89	0.529	27.83	1.491	7.81	0.847	11.72
Cz-Ref	2.834	1.71	2.601	3.66	1.809	10.74	1.841	16.36	1.101	14.89	0.592	28.56	0.952	8.06	0.890	10.99
C4-Ref	2.567	1.71	2.231	6.84	1.697	10.50	1.781	20.26	1.412	13.18	0.550	28.56	0.906	8.06	0.809	11.96
T4-Ref	2.007	0.98	1.450	2.93	1.765	9.28	1.058	16.11	0.807	14.40	0.441	29.79	0.604	8.79	0.796	11.96
T5-Ref	1.596	1.71	1.710	3.66	2.093	8.79	1.308	19.53	0.976	12.94	0.652	28.56	1.024	8.79	0.822	11.72
P3-Ref	1.971	1.71	2.019	3.66	2.554	8.06	1.699	16.36	1.093	13.92	0.640	28.56	1.435	8.06	1.240	11.72
Pz-Ref	2.616	1.71	1.982	6.84	2.192	11.72	1.725	16.36	1.430	13.92	0.614	28.56	1.009	8.06	1.367	11.72
P4-Ref	2.784	1.95	1.809	6.84	1.978	11.72	1.490	19.78	1.654	13.92	0.583	31.98	0.784	8.06	1.314	11.72
T6-Ref	2.502	1.95	1.440	2.93	1.686	10.74	1.166	19.53	1.337	13.92	0.590	27.83	0.527	8.79	0.976	11.96
O1-Ref	2.147	1.95	1.515	3.42	1.897	11.72	1.544	16.60	1.289	13.67	0.797	28.56	0.691	8.79	1.228	11.72
O2-Ref	2.467	1.71	1.613	3.42	1.984	11.72	1.521	16.60	1.572	13.92	0.801	31.98	0.637	8.06	1.421	11.72

TABLE H33: *Table of EEG Power Spectra for all Bandranges with the Video of Image 4 when the Right Eye of the Participant was Open in Phase II*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.702	1.95	1.810	3.17	1.412	7.81	1.284	16.11	1.034	13.92	0.521	31.01	0.768	7.81	0.490	10.99
Fp2-Ref	1.681	1.95	1.713	2.93	1.354	7.81	1.335	16.11	0.969	14.89	0.572	28.81	0.782	7.81	0.477	10.99
F7-Ref	1.653	1.95	1.467	6.10	1.155	7.81	1.198	16.85	0.926	14.40	0.423	28.81	0.743	7.81	0.367	10.99
F3-Ref	2.227	1.95	2.359	2.93	1.745	7.81	1.564	16.85	1.137	13.92	0.530	27.83	1.229	7.81	0.441	11.96
Fz-Ref	2.219	2.93	2.564	2.93	1.979	7.81	1.590	15.87	1.165	14.89	0.548	31.01	1.389	7.81	0.603	10.99
F4-Ref	2.066	2.93	2.156	2.93	1.772	7.81	1.771	16.11	1.061	14.89	0.712	30.27	1.128	7.81	0.678	10.99
F8-Ref	1.795	1.71	1.253	2.93	1.416	8.79	1.280	15.87	0.739	14.89	0.586	30.03	0.647	8.79	0.500	11.23
T3-Ref	2.288	1.46	1.373	2.93	1.438	7.81	1.030	17.09	0.982	14.40	0.448	28.32	1.062	7.81	0.629	11.47
C3-Ref	2.806	0.98	2.510	2.93	2.335	7.81	1.513	17.09	1.144	14.40	0.560	31.49	1.916	7.81	0.791	11.47
Cz-Ref	3.113	2.44	2.662	2.93	2.190	7.81	1.549	15.87	1.171	14.16	0.632	30.27	1.565	7.81	0.720	10.99
C4-Ref	2.509	2.69	2.160	2.93	2.103	7.81	1.572	17.33	1.194	13.67	0.575	31.25	1.456	7.81	0.755	11.47
T4-Ref	1.880	1.46	1.401	4.88	1.917	9.52	1.240	17.82	0.811	13.67	0.515	27.83	0.763	8.79	0.512	11.72
T5-Ref	2.884	0.98	1.587	2.93	2.132	7.81	1.442	17.09	1.077	13.92	0.761	27.83	1.568	7.81	0.949	11.23
P3-Ref	3.381	0.98	2.267	2.93	2.461	7.81	1.564	17.09	1.228	14.65	0.603	28.32	1.914	7.81	1.042	11.47
Pz-Ref	3.500	0.98	2.326	2.93	2.256	7.81	1.598	16.85	1.198	14.65	0.568	28.32	1.561	7.81	0.988	11.47
P4-Ref	2.845	0.98	2.155	2.93	2.022	7.81	1.618	17.58	1.227	13.92	0.604	30.03	1.207	7.81	0.972	11.47
T6-Ref	2.237	0.98	1.798	2.93	1.970	9.77	1.461	17.58	1.111	12.94	0.538	33.45	0.843	8.06	0.793	11.47
O1-Ref	2.927	0.98	1.803	2.93	1.884	8.06	1.494	15.87	1.159	14.65	0.760	28.32	1.228	8.06	0.978	11.23
O2-Ref	2.709	0.98	1.803	2.93	1.916	11.47	1.583	17.58	1.306	12.94	0.762	28.08	0.987	8.06	1.097	11.47

TABLE H34*: Table of EEG Power Spectra for all Bandranges with the Video of Image 4 when the Left eye of the Participant was Open in Phase II*.

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	3.769	0.98	2.212	3.91	1.342	11.47	1.306	19.29	0.909	14.65	0.634	33.20	0.667	8.54	0.702	11.47
Fp2-Ref	2.614	1.22	1.854	4.39	1.239	10.50	1.659	23.19	0.805	14.40	0.815	30.76	0.476	8.06	0.674	11.47
F7-Ref	2.176	1.22	1.849	5.37	1.179	8.79	0.965	19.53	0.738	14.40	0.417	31.25	0.597	8.79	0.561	11.96
F3-Ref	2.693	2.20	2.542	5.37	1.496	10.50	1.405	19.53	0.927	13.18	0.602	28.56	0.686	8.30	0.658	11.47
Fz-Ref	2.946	2.20	2.910	4.39	1.527	11.47	1.675	19.29	1.029	14.40	0.528	28.56	0.741	8.30	0.736	11.47
F4-Ref	3.019	1.22	2.645	4.39	1.504	10.50	1.760	20.02	0.985	14.40	0.770	28.81	0.529	7.81	0.810	11.47
F8-Ref	1.995	1.22	1.909	3.91	1.215	8.06	1.306	18.55	0.734	14.65	0.467	32.96	0.585	8.06	0.645	11.47
T3-Ref	2.186	2.20	1.704	3.91	1.473	11.23	1.069	17.82	0.866	13.43	0.445	28.56	0.591	8.79	0.963	11.23
C3-Ref	2.694	2.20	2.538	5.37	1.844	10.50	1.769	18.55	0.944	14.40	0.548	28.56	0.879	8.30	0.831	11.47
Cz-Ref	2.965	2.20	2.733	4.15	1.854	10.50	1.918	18.55	1.024	14.40	0.614	28.56	0.919	7.81	0.827	11.47
C4-Ref	2.842	1.22	2.636	4.15	1.674	11.47	1.643	17.33	0.959	14.40	0.556	28.56	0.823	8.30	0.882	11.47
T4-Ref	1.736	1.22	1.538	4.15	1.415	8.06	1.323	17.33	0.743	13.18	0.432	28.56	0.736	8.06	0.658	11.47
T5-Ref	2.227	0.98	1.915	3.66	1.598	10.74	1.543	18.80	1.127	13.43	0.737	28.56	0.613	8.79	0.790	11.23
P3-Ref	2.731	0.98	2.431	5.62	1.985	10.50	1.991	18.55	0.915	13.43	0.572	28.56	0.903	8.30	0.642	11.72
Pz-Ref	2.867	0.98	2.526	6.84	1.980	10.50	2.088	18.55	1.023	13.67	0.577	28.56	0.801	8.06	0.714	10.99
P4-Ref	2.933	0.98	2.664	5.62	1.856	10.50	1.956	17.33	0.966	13.92	0.588	28.56	0.939	8.54	0.897	11.47
T6-Ref	2.402	0.98	2.009	5.62	1.612	8.06	1.645	17.33	0.994	13.92	0.506	28.56	0.982	8.06	0.804	11.23
O1-Ref	2.467	0.98	2.112	3.66	1.718	10.50	1.903	16.60	1.317	13.67	0.803	30.52	0.868	8.06	0.510	11.72
O2-Ref	2.800	0.98	2.232	4.15	1.799	8.06	1.855	17.33	1.382	13.67	0.685	30.52	0.999	8.06	0.772	11.96

TABLE H35: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 4 when Both of the Participant's Eyes were Open in Phase IV*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.005	1.95	1.533	3.66	1.404	10.50	1.850	18.55	0.915	12.94	1.288	30.27	0.619	8.54	0.572	10.99
Fp2-Ref	2.222	1.46	1.552	3.66	1.438	10.50	1.937	18.31	0.824	12.94	1.247	33.69	0.599	8.79	0.675	11.96
F7-Ref	1.838	1.22	1.624	3.66	1.022	7.81	1.357	17.58	0.756	13.18	0.661	30.03	0.623	7.81	0.324	11.47
F3-Ref	3.061	1.22	2.328	3.66	1.814	8.54	1.975	17.82	0.957	13.67	0.987	28.08	1.000	8.54	0.782	11.72
Fz-Ref	3.222	1.71	2.563	3.66	1.849	8.54	1.652	16.60	0.849	13.67	0.544	28.08	1.070	8.54	0.618	10.99
F4-Ref	3.320	0.98	2.219	3.66	1.567	8.54	1.991	16.85	0.963	13.67	0.960	28.32	0.887	8.54	0.618	11.96
F8-Ref	2.246	1.46	1.538	3.17	1.286	9.28	1.772	18.31	0.782	12.94	1.092	28.56	0.569	8.54	0.573	11.47
T3-Ref	2.136	1.22	1.670	3.66	1.253	11.47	1.065	19.78	0.757	14.16	0.491	32.96	0.651	8.79	0.595	11.47
C3-Ref	3.166	1.22	2.530	3.66	1.964	8.54	1.430	15.87	1.065	14.40	0.571	28.08	1.219	8.54	0.699	11.72
Cz-Ref	3.555	1.22	2.907	3.66	2.113	8.54	1.646	16.60	1.191	13.67	0.657	28.08	1.186	8.54	0.840	11.96
C4-Ref	2.882	1.71	2.451	2.93	1.870	8.79	1.642	17.09	1.210	13.67	0.584	27.83	1.009	8.79	0.788	11.72
T4-Ref	1.879	2.20	1.779	2.93	1.670	8.54	1.175	18.31	0.822	13.67	0.466	33.45	0.980	8.54	0.729	11.47
T5-Ref	2.377	1.22	1.781	3.42	1.608	8.54	1.308	16.60	0.897	14.16	0.756	31.01	0.883	8.54	0.720	11.47
P3-Ref	3.215	1.22	2.368	3.66	2.000	8.79	1.429	16.36	1.188	14.40	0.655	28.08	1.109	8.79	0.811	11.72
Pz-Ref	3.577	1.22	2.484	2.93	2.073	8.79	1.520	16.36	1.348	12.94	0.658	28.08	1.119	8.79	0.912	11.72
P4-Ref	3.128	1.22	2.313	2.93	1.954	8.79	1.524	16.36	1.180	13.67	0.715	28.08	1.028	8.79	0.870	11.72
T6-Ref	2.391	1.46	1.936	2.93	1.854	8.79	1.380	19.78	0.896	13.43	0.803	32.47	0.930	8.79	0.973	11.72
O1-Ref	2.898	1.22	1.930	2.93	1.748	8.79	1.344	17.09	1.107	14.40	1.013	28.08	0.897	8.79	0.756	11.72
O2-Ref	2.991	1.22	2.092	3.66	2.037	8.79	1.555	17.09	1.017	14.40	1.102	28.08	1.079	8.79	0.842	10.99

TABLE H36: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 4 when the Right Eye of the Participant was Open in Phase IV*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.092	0.98	1.642	4.15	1.121	10.99	1.047	15.87	0.715	14.16	0.564	29.54	0.501	7.81	0.644	10.99
Fp2-Ref	2.632	0.98	1.644	4.15	1.334	10.99	1.127	15.87	0.934	14.40	0.509	28.08	0.615	8.54	0.763	10.99
F7-Ref	2.300	1.71	1.576	6.59	1.116	10.74	0.955	16.60	0.590	12.94	0.471	29.54	0.613	8.54	0.564	10.99
F3-Ref	2.522	0.98	2.112	4.15	1.627	7.81	1.435	16.60	0.899	14.89	0.588	29.54	0.948	7.81	0.780	10.99
Fz-Ref	2.753	0.98	2.358	4.15	1.741	7.81	1.510	16.36	1.094	14.16	0.556	29.54	0.909	7.81	0.868	10.99
F4-Ref	2.776	0.98	2.144	4.15	1.684	7.81	1.462	16.36	1.032	14.16	0.589	29.54	0.770	7.81	0.914	10.99
F8-Ref	2.771	0.98	1.692	5.13	1.205	8.79	1.205	15.87	0.785	14.40	0.566	31.74	0.594	8.79	0.594	11.96
T3-Ref	1.750	1.22	1.478	3.42	1.510	8.54	1.215	16.36	0.715	12.94	0.508	33.20	0.907	8.54	0.416	11.23
C3-Ref	2.518	0.98	2.091	4.15	1.758	8.54	1.694	16.36	0.874	12.94	0.535	29.54	1.106	8.54	0.542	11.96
Cz-Ref	2.830	0.98	2.647	4.15	1.882	7.81	1.867	16.36	1.143	12.94	0.630	28.81	0.996	7.81	0.961	10.99
C4-Ref	2.862	0.98	1.984	4.15	1.552	11.96	1.647	16.36	1.012	12.94	0.557	31.74	0.696	7.81	0.871	11.96
T4-Ref	2.014	0.98	1.481	6.84	1.584	9.28	1.132	16.60	0.576	14.89	0.452	31.74	0.794	8.30	0.708	11.47
T5-Ref	1.809	0.98	1.662	4.39	1.656	8.54	1.449	17.09	0.742	14.40	0.754	28.56	0.991	8.54	0.550	11.23
P3-Ref	2.330	2.44	2.253	4.15	1.951	8.54	1.718	16.36	0.966	14.40	0.568	29.30	1.366	8.54	0.520	10.99
Pz-Ref	2.864	0.98	2.446	4.15	1.954	8.54	1.913	17.58	1.006	14.89	0.580	29.05	1.352	8.54	0.690	11.96
P4-Ref	2.808	0.98	2.215	4.15	1.708	8.54	1.631	16.36	0.897	14.89	0.623	31.74	1.016	8.54	0.833	10.99
T6-Ref	1.885	0.98	1.554	6.84	1.420	10.50	1.302	16.60	0.776	14.89	0.709	28.32	0.534	8.06	0.687	10.99
O1-Ref	2.172	0.98	1.913	3.66	1.759	8.54	1.353	16.36	1.107	14.40	0.806	36.38	1.096	8.54	0.520	10.99
O2-Ref	2.249	2.44	2.028	4.15	1.685	8.54	1.399	20.51	1.120	14.89	0.789	31.74	1.041	8.54	0.561	10.99

TABLE H37: *Table of EEG Power Spectra for all Bandranges with the Photograph of Image 4 when the Left Eye of the Participant was Open in Phase IV*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.987	1.46	1.312	6.84	1.172	8.30	1.259	17.33	0.660	14.89	0.551	28.08	0.784	8.30	0.450	11.96
Fp2-Ref	2.084	1.71	1.462	6.35	1.211	8.30	1.344	17.33	0.710	14.65	0.553	28.56	0.730	8.30	0.564	10.99
F7-Ref	1.951	1.71	1.266	6.35	1.016	8.30	1.078	17.58	0.629	13.18	0.488	29.05	0.650	8.30	0.358	11.47
F3-Ref	2.748	1.46	1.837	6.35	1.623	8.30	1.594	17.58	0.653	14.89	0.566	31.25	1.055	8.30	0.626	11.72
Fz-Ref	2.661	1.46	2.151	6.35	1.777	8.30	1.708	17.33	0.792	14.16	0.546	31.01	1.119	8.30	0.680	11.72
F4-Ref	2.601	1.46	2.272	6.35	1.588	8.54	1.669	17.33	0.729	14.65	0.570	31.01	0.884	8.54	0.651	10.99
F8-Ref	2.251	0.98	1.462	3.42	1.244	8.30	1.383	17.33	0.664	14.16	0.551	29.79	0.628	8.30	0.557	10.99
T3-Ref	1.654	1.22	1.301	3.42	1.091	11.96	1.104	19.29	0.696	13.18	0.527	28.32	0.398	8.30	0.506	11.96
C3-Ref	2.576	1.46	1.975	6.35	1.518	8.30	1.692	18.80	0.734	14.16	0.560	28.08	0.983	8.30	0.614	11.72
Cz-Ref	2.738	1.46	2.409	3.17	2.019	9.52	2.019	17.33	0.821	14.89	0.640	27.83	1.169	8.30	0.787	11.72
C4-Ref	2.804	1.46	2.034	3.17	1.769	11.72	1.839	17.33	0.762	14.89	0.552	31.25	0.839	8.30	0.801	11.72
T4-Ref	1.998	1.46	1.481	3.42	1.662	9.28	1.293	15.87	0.536	14.89	0.506	27.83	0.720	8.54	0.649	11.96
T5-Ref	2.333	1.22	1.537	3.17	1.342	8.54	1.542	16.60	0.836	13.18	0.818	29.54	0.714	8.54	0.655	11.96
P3-Ref	2.788	1.22	1.875	3.17	1.631	8.30	1.718	16.60	0.786	14.16	0.590	28.56	1.089	8.30	0.628	11.96
Pz-Ref	3.157	1.46	1.955	3.17	1.859	9.52	1.724	16.60	0.773	14.40	0.592	30.52	1.067	8.30	0.731	11.96
P4-Ref	3.133	1.22	1.906	3.17	1.732	10.01	1.679	15.87	0.835	13.18	0.635	31.01	0.790	8.30	0.780	11.96
T6-Ref	2.639	1.46	1.533	3.17	1.799	9.28	1.586	15.87	0.829	13.18	0.701	30.03	0.600	8.54	0.905	10.99
O1-Ref	3.026	1.22	1.488	3.17	1.427	9.28	1.747	22.95	0.744	13.67	0.931	28.56	0.804	8.30	0.671	11.72
O2-Ref	3.036	1.22	1.488	3.17	1.525	9.28	1.808	20.02	0.842	14.16	0.985	28.56	0.620	8.54	0.707	11.96

TABLE H38: *Table of EEG Power Spectra for all Bandranges with the Video of Image 4 when Both of the Participant's Eyes were Open in Phase IV*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.475	0.98	1.382	2.93	1.698	9.28	1.706	16.11	0.926	12.94	0.702	31.25	0.761	8.06	0.763	10.99
Fp2-Ref	2.225	0.98	1.488	2.93	1.777	9.28	1.658	20.51	0.925	12.94	0.793	29.54	0.591	7.81	0.898	11.47
F7-Ref	1.973	1.46	1.392	3.66	1.453	9.28	1.242	17.09	0.748	12.94	0.460	28.08	0.770	8.06	0.559	10.99
F3-Ref	1.975	1.46	2.124	3.66	1.862	9.03	1.848	17.58	1.029	12.94	0.608	28.08	0.949	7.81	0.759	10.99
Fz-Ref	1.811	2.93	2.416	3.66	2.164	9.03	1.903	17.58	1.120	12.94	0.582	30.03	1.065	8.06	1.055	11.47
F4-Ref	2.203	0.98	2.209	3.66	2.218	11.47	2.232	16.11	1.045	12.94	0.868	30.03	1.044	8.06	1.100	11.47
F8-Ref	2.570	1.46	1.523	2.93	1.643	9.28	1.537	22.95	0.813	12.94	0.757	29.79	0.529	7.81	0.758	11.47
T3-Ref	2.147	1.46	1.610	2.93	1.030	7.81	1.117	16.11	0.860	12.94	0.509	35.64	0.533	7.81	0.495	11.96
C3-Ref	2.603	1.46	2.292	2.93	1.751	10.25	1.828	20.51	1.394	12.94	0.509	29.79	0.817	8.30	0.844	11.47
Cz-Ref	2.290	2.93	2.810	2.93	2.529	11.47	2.151	16.11	1.520	12.94	0.646	29.30	1.350	8.30	1.306	11.47
C4-Ref	1.901	0.98	2.311	2.93	2.671	9.03	2.176	16.85	1.885	14.40	0.635	29.54	1.337	8.30	1.331	11.47
T4-Ref	1.653	0.98	1.349	2.93	2.043	9.03	1.060	16.85	0.868	12.94	0.514	29.79	0.933	8.06	1.119	11.72
T5-Ref	2.561	1.46	1.612	2.93	1.695	9.28	1.537	17.33	1.117	14.16	0.659	31.01	0.767	8.30	0.700	11.23
P3-Ref	3.252	2.69	1.988	2.93	2.246	10.25	2.123	17.58	1.397	12.94	0.564	30.03	0.853	8.30	1.199	10.99
Pz-Ref	3.521	1.95	2.002	6.84	2.494	11.23	2.270	17.58	1.559	14.89	0.581	30.03	1.112	8.30	1.351	11.23
P4-Ref	2.919	1.22	1.680	3.17	2.435	8.30	2.087	17.82	1.487	14.65	0.606	28.08	1.323	8.30	1.186	11.47
T6-Ref	2.472	1.22	1.426	4.15	2.328	9.52	1.459	16.11	1.126	14.65	0.688	28.08	1.208	8.30	1.226	11.72
O1-Ref	3.127	2.69	1.843	2.93	2.022	10.50	2.257	16.36	1.151	14.89	0.834	30.03	0.953	8.30	0.945	10.99
O2-Ref	2.889	1.46	1.685	6.35	2.343	8.30	2.164	17.82	1.310	14.89	0.855	30.27	1.342	8.30	0.979	11.96

TABLE H39: *Table of EEG Power Spectra for all Bandranges with the Video of Image 4 when the Right Eye of the Participant was Open in Phase IV*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	2.008	0.98	1.902	2.93	1.284	11.47	1.481	16.60	0.712	13.67	0.797	30.03	0.513	8.79	0.745	11.47
Fp2-Ref	1.971	0.98	1.994	3.17	1.278	10.99	1.600	18.31	0.754	13.67	0.841	32.96	0.577	8.79	0.682	10.99
F7-Ref	1.865	1.46	1.688	2.93	1.116	9.77	1.166	16.85	0.622	13.43	0.527	30.76	0.344	8.79	0.411	11.96
F3-Ref	2.033	2.93	2.444	2.93	1.710	11.47	1.550	16.85	0.869	13.92	0.680	30.27	0.807	8.30	0.816	11.47
Fz-Ref	2.315	2.93	2.801	2.93	1.814	11.47	1.743	16.85	0.852	13.67	0.567	30.76	0.885	8.30	0.945	11.47
F4-Ref	2.391	0.98	2.405	2.93	1.647	9.28	1.684	16.85	0.916	13.67	0.765	30.76	0.754	8.54	0.866	11.47
F8-Ref	2.046	0.98	2.121	2.93	1.048	10.99	1.692	21.97	0.765	12.94	0.796	28.56	0.398	8.54	0.592	10.99
T3-Ref	1.892	1.71	1.497	3.17	1.476	9.52	1.280	18.07	0.862	13.92	0.634	30.76	0.447	8.79	0.611	11.96
C3-Ref	2.243	0.98	2.392	2.93	2.167	9.52	1.595	17.09	1.145	13.92	0.566	30.76	1.026	8.30	0.831	10.99
Cz-Ref	2.674	1.71	2.743	2.93	2.385	9.28	1.780	16.85	1.049	13.67	0.694	30.76	1.294	8.30	1.008	11.47
C4-Ref	2.441	1.95	2.331	2.93	1.884	9.28	1.559	16.85	1.061	13.67	0.637	28.08	0.919	8.30	0.886	11.47
T4-Ref	1.895	1.71	1.811	3.17	1.395	10.25	1.153	15.87	0.825	12.94	0.540	31.49	0.612	8.30	0.469	11.96
T5-Ref	2.203	1.71	1.599	5.86	1.771	9.52	1.420	20.02	1.024	13.92	0.884	30.76	0.686	8.30	0.704	10.99
P3-Ref	2.670	2.69	2.486	3.17	2.172	9.52	1.517	16.11	1.181	13.92	0.637	28.08	1.103	8.30	0.750	10.99
Pz-Ref	2.670	0.98	2.363	3.17	2.381	8.30	1.535	16.11	1.085	13.92	0.654	28.08	1.397	8.30	0.912	11.47
P4-Ref	2.884	0.98	2.104	2.93	2.103	9.28	1.379	20.75	1.183	13.67	0.644	28.32	1.175	8.30	0.727	10.99
T6-Ref	2.452	0.98	1.585	3.17	1.977	10.25	1.380	20.02	1.058	13.67	0.706	28.32	0.887	8.30	0.535	11.96
O1-Ref	2.455	2.20	1.939	5.86	1.731	9.52	1.457	16.85	1.072	14.16	0.836	28.32	0.817	8.30	0.508	10.99
O2-Ref	2.697	2.20	1.956	6.10	1.782	8.30	1.478	16.60	1.162	14.16	0.884	28.32	1.025	8.30	0.524	10.99

TABLE H40: *Table of EEG Power Spectra for all Bandranges with the Video of Image 4 when the Left Eye of the Participant was Open in Phase IV*

	Delta		Theta		Alpha		Beta		SMR		Hi Beta		Lo ALpha		Hi ALpha	
	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz	uV	Hz
Fp1-Ref	1.784	2.44	1.699	3.66	1.261	10.99	1.149	20.02	0.508	14.16	0.602	29.05	0.627	8.06	0.706	10.99
Fp2-Ref	2.389	1.22	1.711	3.66	1.308	10.99	1.206	18.31	0.571	13.92	0.574	29.05	0.670	8.30	0.722	10.99
F7-Ref	2.202	1.71	1.497	3.91	1.155	10.99	1.055	18.31	0.515	13.67	0.494	28.32	0.500	8.06	0.696	10.99
F3-Ref	2.366	1.71	2.209	3.91	1.539	10.99	1.463	20.02	0.711	14.16	0.608	29.79	0.804	8.06	0.846	10.99
Fz-Ref	2.475	0.98	2.249	3.42	1.779	10.99	1.557	20.02	0.710	14.16	0.587	29.79	0.953	8.06	0.891	10.99
F4-Ref	2.342	0.98	2.103	3.42	1.641	9.77	1.608	18.07	0.782	13.92	0.634	29.54	0.922	8.30	0.758	10.99
F8-Ref	1.928	2.44	1.474	4.88	1.223	11.96	1.393	21.00	0.858	13.92	0.665	31.01	0.659	8.79	0.622	11.96
T3-Ref	1.909	1.95	1.391	3.91	1.292	10.74	1.244	18.07	0.815	13.43	0.685	28.56	0.519	8.06	0.493	10.99
C3-Ref	2.225	0.98	2.197	3.91	1.565	8.06	1.651	18.07	0.964	13.43	0.560	30.27	0.863	8.06	0.712	10.99
Cz-Ref	2.864	1.46	2.766	3.17	1.882	9.77	1.728	17.33	0.840	13.67	0.633	30.27	0.961	7.81	0.867	10.99
C4-Ref	2.573	1.46	2.382	3.17	1.674	9.52	1.742	16.11	0.998	13.67	0.698	29.05	0.958	8.30	0.780	10.99
T4-Ref	1.807	1.71	1.500	3.91	1.524	8.06	1.351	18.55	1.038	13.18	0.661	29.05	0.865	8.06	0.695	11.23
T5-Ref	2.243	0.98	1.581	3.91	1.397	8.06	1.594	17.82	0.866	13.67	0.875	31.98	0.878	8.06	0.394	10.99
P3-Ref	2.679	1.22	2.180	3.17	1.779	8.06	1.778	17.33	1.028	12.94	0.637	32.71	1.159	8.06	0.720	11.23
Pz-Ref	2.987	0.98	2.446	3.42	1.928	8.06	1.739	17.33	1.037	12.94	0.610	29.79	1.240	8.06	0.846	11.23
P4-Ref	2.751	1.46	2.352	3.42	1.811	8.06	1.763	17.33	1.123	12.94	0.654	29.79	1.090	8.06	0.847	11.23
T6-Ref	2.546	0.98	1.554	3.42	1.875	8.06	1.385	16.11	1.103	13.67	0.708	27.83	1.010	8.06	0.971	11.23
O1-Ref	2.493	0.98	1.879	3.42	1.815	8.06	1.930	16.85	1.192	12.94	0.870	27.83	1.085	8.06	0.841	11.23
O2-Ref	2.416	0.98	1.813	3.42	1.934	8.06	1.987	16.11	1.466	12.94	0.970	27.83	1.030	8.06	1.018	11.23

APPENDIX I

Topographic Maps of EEG Power

Spectra at Selected Bandranges

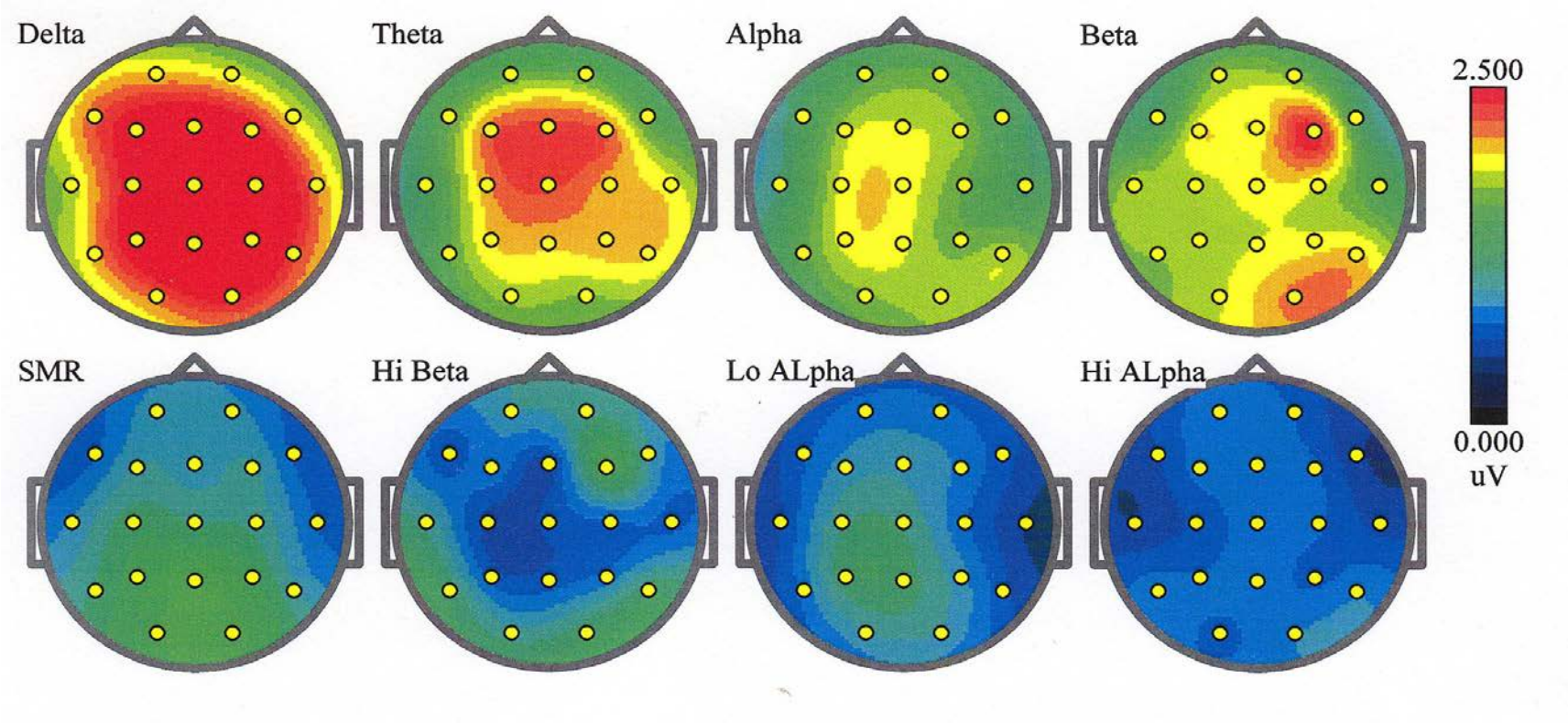


FIGURE I1: Topographic maps of EEG power spectra for all bandranges in response to the photograph of the male face triggering the strongest transference response when the right eye of the participant was open in Phase I. The amplitude areas are shown by different colours in accordance with the key in the upper right corner of the figure. The colour code of green represents the norm, while the colour codes red and blue represent levels of brain activity that are above or below the norm, respectively.

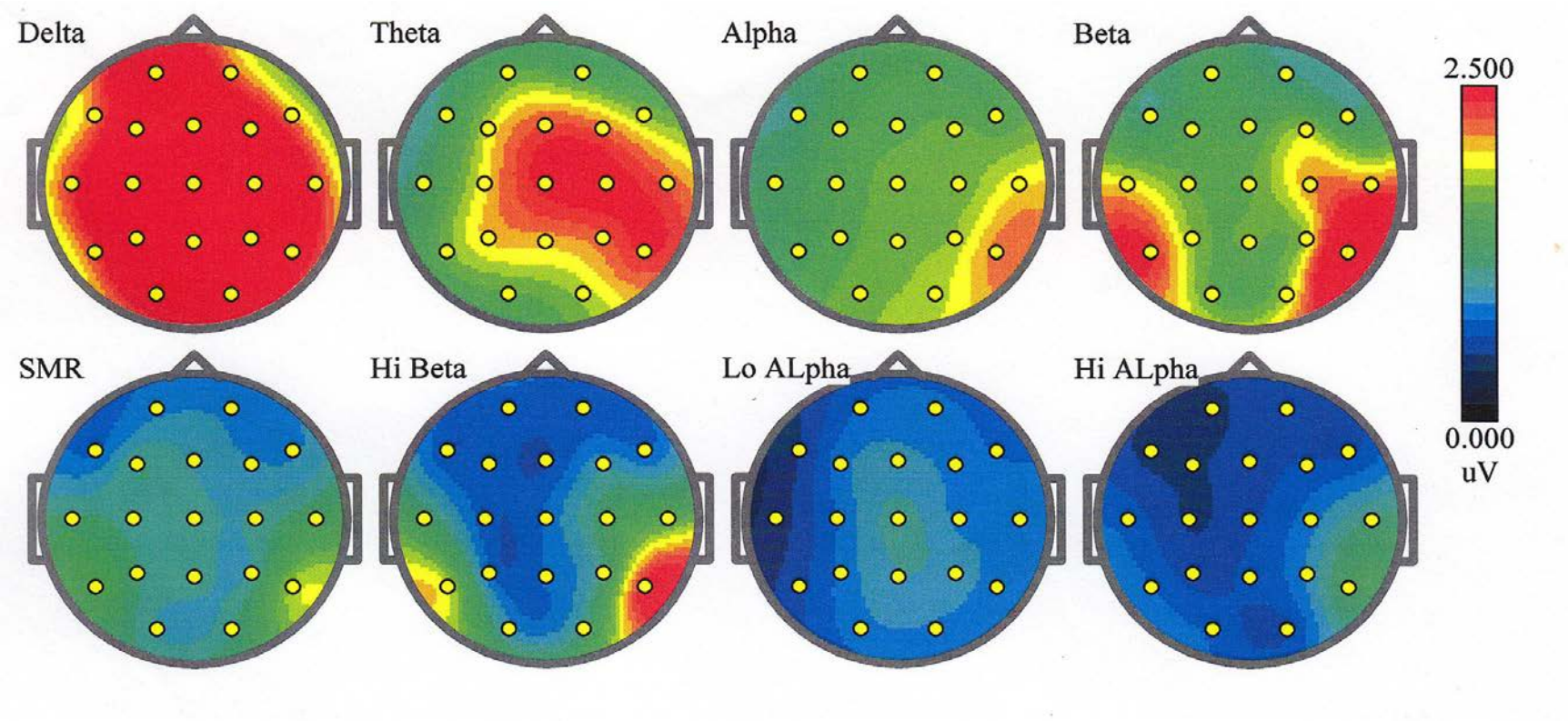


FIGURE I2: Topographic images of maps of EEG power spectra for all bandranges in response to the photograph of the male face triggering the strongest transference response when the left eye of the participant was open in Phase I The amplitude areas are shown by different colours in accordance with the key in the upper right corner of the figure. The colour code of green represents the norm, while the colour codes red and blue represent levels of brain activity that are above or below the norm, respectively.

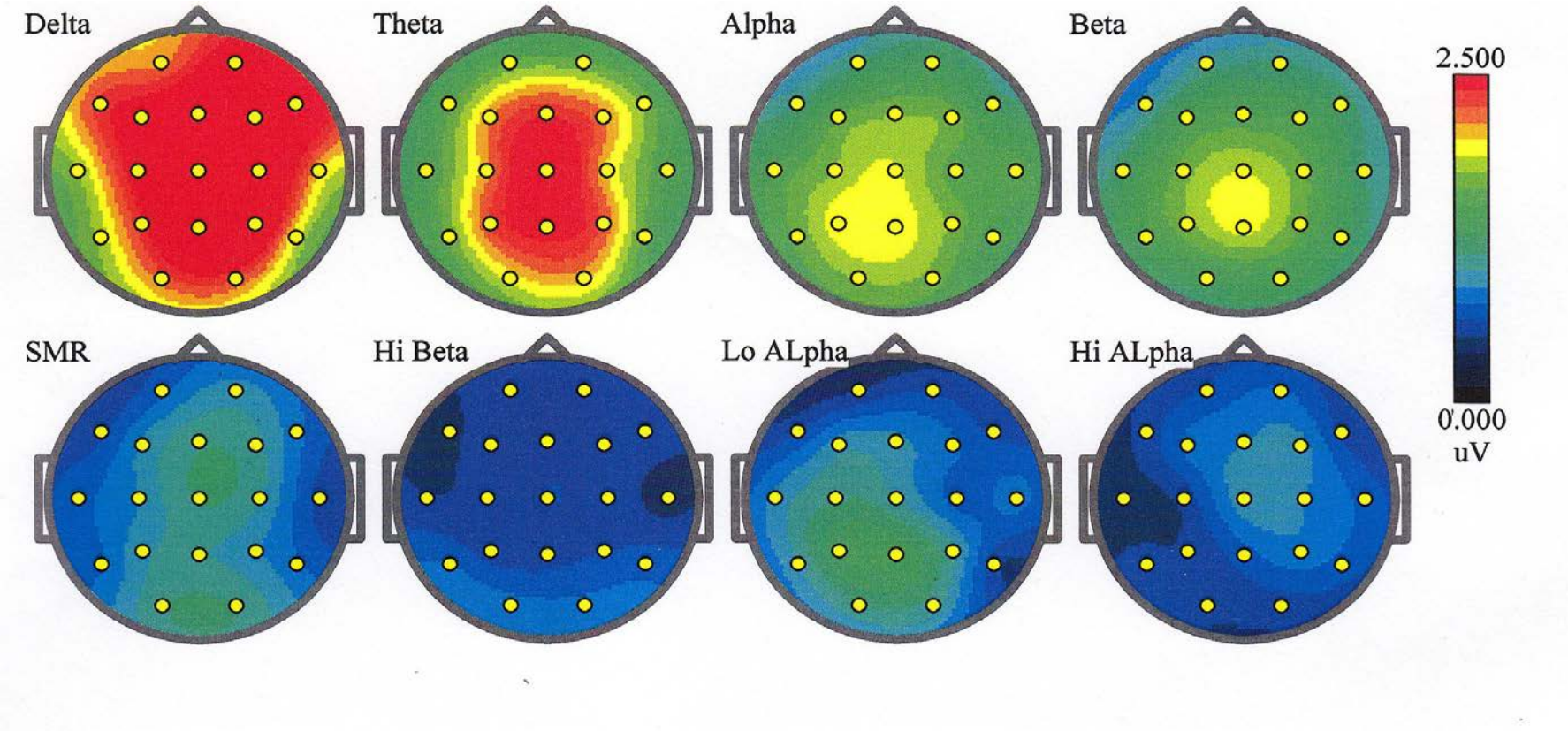


FIGURE I3: Topographic maps of EEG power spectra for all bandranges in response to the photograph of the male face triggering the strongest transference response when the right eye of the participant was open in Phase IV The amplitude areas are shown by different colours in accordance with the key in the upper right corner of the figure. The colour code of green represents the norm, while the colour codes red and blue represent levels of brain activity that are above or below the norm, respectively.

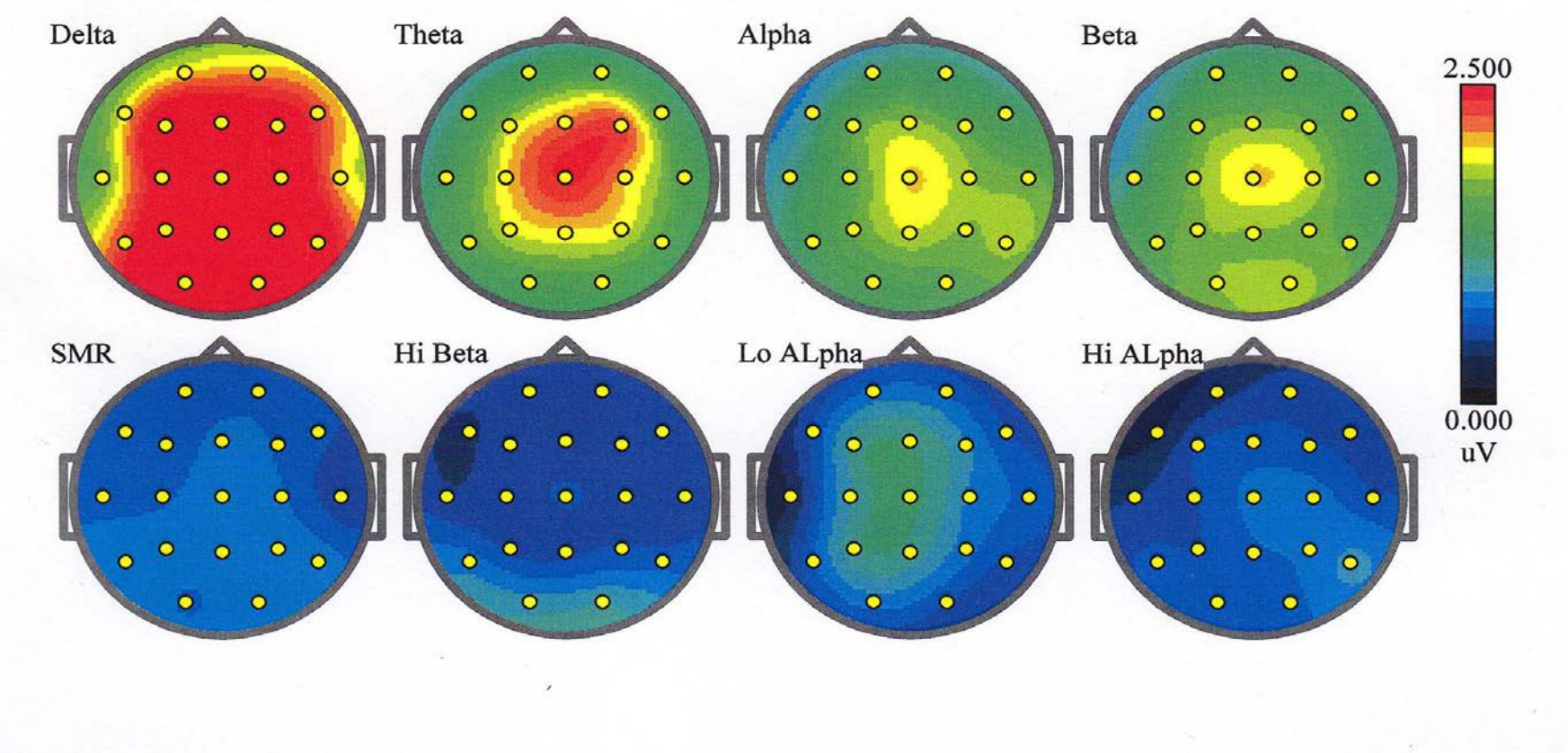


FIGURE I4: Topographic maps of EEG power spectra for all bandranges in response to the photograph of the male face triggering the strongest transference response when the left eye of the participant was open in Phase IV The amplitude areas are shown by different colours in accordance with the key in the upper right corner of the figure. The colour code of green represents the norm, while the colour codes red and blue represent levels of brain activity that are above or below the norm, respectively.

APPENDIX J: Informed Consent Form

OEI Transference Project
REB Approval Date: April 25, 2010

Development and Pilot Testing of a Protocol for assessing negative transference reactions during Observed and Experiential Integration usinqEEG and LORETA

One of the emerging new psychotherapies for psychological trauma is Observed & Experiential Integration (OEI). In the last 15 years, Dr Rick Bradshaw and his colleagues have observed the manifestation of transference phenomena during OEI. In psychoanalysis, transference is defined as unconsciously projecting feelings for one person onto another. In OEI, transference has been observed during Switching (alternate complete covering and uncovering of one eye at a time). It was also noted that these perceptual distortions were greatly diminished while Switching and gazing at visual stimuli (such as therapist, partner, child, or even one's own image in a mirror).

The purpose of this pilot study is to develop a protocol to assess the transference phenomenon in response to visual stimuli. In addition to this, qEEG assessment of cortical activity while gazing at visual stimuli will be done pre- and post-treatment. Participant will complete standardized questionnaires [Clinician Administered Dissociative States Scale (CADSS)], Transference Reaction Record [TRR], qualitative interviews and brain imaging [quantitative Electroencephalography (qEEG) with Low Resolution Electromagnetic Tomography (LORETA)]. Assessment and treatment sessions will be video recorded. Administration of questionnaires and other assessments, and provision of treatment, will require a commitment of one full day by the participant.

All data collected through questionnaires, interviews, video recordings, and qEEG assessments will be stored on CDs kept in a locked file cabinet in a locked office in the Department of Counselling Psychology at Trinity Western University and on a password-protected computer in the same department. For future use, these data will be stored for 10 years from the date of study completion.

All information shared by you will be strictly confidential. No information will be released without your consent, other than due to standard legal limitations of confidentially that include imminent danger to yourself or others, suspected child abuse or neglect, and court subpoena.

Psychotherapy involves both risks and benefits. Recalling past traumatic events may evoke uncomfortable thoughts, feelings, and memories. At the same time, OEI treatment usually provides insight and improves relationships. Greater understanding of brain activity associated with transference phenomena will provide insights regarding the neuropsychological bases of perceptual distortions. Finally, having observed transference projections and perceptual distortions in your own practice, it is hoped that this pilot study is less threatening. Breathing and relaxation techniques will be encouraged during debriefing to ensure your emotional and psychological stability before leaving the clinic.

Your participation is completely voluntarily and at any point during the study you have the right to refuse or withdraw from participation. Even if you choose to withdraw during the research process, however, it is imperative that you complete debriefing and engage in breathing and relaxation exercises before you leave the research site, for your own emotional wellbeing. Data from participants who have chosen to withdraw will be destroyed.

Your signature implies that you have read, understood, and consented to, participation in this study. Your signature below indicates that you have had your questions about the study answered to your satisfaction, and that you have received a copy of this consent form for your own records.

_____

Your Full Name (Please Print)

_____

Your Signature Date

_____

Witness Date

If you have any question or desire further information with respect to this study, you may contact any one of the following persons.

Mahima Jacob (TWU Graduate Student)	604-996-4537	mahima.jacob@mytwu.ca
Dr Rick Bradshaw (TWU Faculty Member)	604-513-2121 (Ext 3382)	rickphyl@telus.net
Dr Marvin McDonald (Program Director)	604-513-2121 (Ext 3223)	mcdonald@twu.ca

If you have any concerns about your treatment or your rights as a research participant, you may contact Ms. Sue Funk in the Office of Research, Trinity Western University, at 604-513-2142 or sue.funk@twu.ca

APPENDIX K: Glossary

Alpha suppression: Limited or negative increase in alpha from eyes open to eyes closed condition at C_z and O_1 locations. At C_z, an increase of less than 30% is considered alpha suppression. At O_1, an increase of less than 50% is considered alpha suppression (Swingle, 2009).

Complex PTSD: A trauma subgroup characterizes by some observed dissociation, alpha asymmetries in the parietal regions, and theta/beta ratios in the occipital region (Faas, 2009).

Dissociative PTSD: A trauma subgroup characterizes by early/extensive-trauma histories, vague physical complaints, consistent observed dissociation, alpha suppression and some high theta/beta ratios at O_1 (Faas, 2009).

HiBetaGamma/Beta: Activity over the anterior cingulate cortex (F_z) in the frequency range 28-40Hz/16-25Hz. A higher ratio indicates rumination, racing thoughts and fretting, whereas lower ratio is associated with passivity (Swingle, 2009).

Simple PTSD: A trauma subgroup characterized by single incident, adult-onset trauma histories, very little observed dissociation, low theta/beta ratios at O_1, and low hibeta/beta ratio at F_z (Faas, 2009).

Swingle signature: Clinically informed, brainwave patterns and ratios at various scalp locations and frequency ranges in accordance to Dr. Paul Swingle.

Theta/Beta Ratio: It is the activity in the left side of occipital lobe (O_1) in the frequency range 3-7 Hz/16-25 Hz. A low theta/beta ratio indicates sleep difficulties and inability to ‘quiet’ the brain. A high ratio is associated with dissociative fugue state (Swingle, 2009).