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EEG Patterns of TBI Patients with Attention Deficits During Cognitive Tasks and Second Resting Baseline

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To the Graduate Council:

I am submitting herewith a dissertation written by Stamatina Stathopoulou entitled "EEG Patterns of TBI Patients with Attention Deficits During Cognitive Tasks and Second Resting Baseline." I have examined the final electronic copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Psychology.

Joel F. Lubar, Major Professor

We have read this dissertation and recommend its acceptance:

Eric Sundstrom, Richard Saudargas, Michael Sims

Accepted for the Council:
Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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EEG PATTERNS OF TBI PATIENTS WITH ATTENTION DEFICITS DURING COGNITIVE TASKS AND SECOND RESTING BASELINE

A
Dissertation
Presented for the Doctor of Philosophy
Degree
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Stamatina Stathopoulou

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ABSTRACT

According to previous research, different regions of the brain are activated when a person is required to use different types of attention like selective, alternating, focused, sustained and divided attention. The frontal, prefrontal and parietal areas especially in the right hemisphere, seem to be the most frequently activated areas. Little research has addressed differences in the electroencephalogram (EEG) between traumatic brain injured (TBI) patients, with different types of attentional deficits because of their injury, and normal population. This study focuses on differences in magnitude in five brain regions between TBI patients and normal population, during recording of one cognitive task (ADT task) and the after tasks eyes-open baseline (EO2). All matched controls' psychometrics, and eyes-closed EEG are representative of an average person without neurological deficits.

Four frequencies are examined. The attention skills of the experimental (TBI) and matched for age and gender control individuals are assessed through a variety of psychometrics as well as through scaled self-reports. Their EEG is recorded during eyes-open, eyes-closed, six cognitive tasks (taken from the software program Captain's Log), and a second eyes-open baseline. The EEG of one out of the six cognitive tasks is statistically examined as well as the second baseline. Only one task is statistically analyzed, an auditory task, discriminating types of melody. It is hypothesized that the topography, frequencies and direction of significant changes from task (ADT) minus first resting baseline (EO1) will be different between the clinical and the matched control individuals. The same hypothesis, that there will be differences between control and clinical cases, holds also for the second eyes-open baseline minus the first baseline comparison. A third hypothesis is that, there will be consistent EEG patterns, depending on the type of attention deficit. All hypotheses are supported. Low alpha and low beta in frontal and right posterior areas constitute the frequencies and brain regions respectively, which show the consistent EEG patterns for each type of attention deficit.

The EEG results of this study will serve as a diagnostic tool for each type of attention deficit. It is possible that the different types of attention deficit are not easily shown through an eyes-open or an eyes-closed recording but during recording of cognitive tasks or in the after tasks (second eyes-open baseline) recording.

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ADD	Attention Deficit Disorder
ADHD	Attention Deficit Hyperactivity Disorder
EEG	Electroencephalogram
DAI	Diffuse Axonal Injury
IVA	The Integrated Visual and Auditory test
IQ	Intelligence Quotient
PASAT	Paced Auditory Serial Addition Test
QEEG	Quantified Electroencephalogram
TBI	Traumatic Brain Injury
UT	University of Tennessee
WAIS-R	Wechsler Adult Intelligence Scale-Revised

Section I

INTRODUCTION

In the current study, different types of attention are compared between participants with some attention deficits and their matched controls while performing a cognitive task (ADT task) and a second eyes-open resting baseline (EO2). These differences are depicted in the electroencephalogram (EEG) and assessed by four psychometrics.

Types of attentional dysfunction and their representation on the brain

Sturm et al. (1999), in his project studied alertness, probably the prerequisite for the more complex and capacity demanding domains of attention selectivity. Typical tasks to assess optimal levels of intrinsic alertness are simple reaction time measurements without preceding warning stimuli. In this study, PET activation in 15 right-handed young healthy male was found in an extended right hemisphere network including frontal (anterior cingulate-dorsolateral cortical)-inferior parietal-thalamic (pulvinar and the reticular nucleus) and brainstem (ponto-mesencephalic tegmentum, possibly involving the locus coeruleus), when subjects waited for and rapidly responded to a centrally presented white dot by pressing a response key with the right-hand thumb. Induced alpha rhythms are analogous to the gamma-band rhythms induced by moving stripes for example. In the study where Basar et al. (1989) demonstrated when a target is anticipated, gamma band energy is emitted. On the other hand, a well-trained subject emitted time-locked bursts of alpha band for up to a full second before the delivery of an expected target. By contrast to the modest evidence of the gamma band, the alpha burst in anticipation of the target, was more robust and highly statistically significant (Basar et al. 1989)**.**

There are different types of attention which may be disrupted: The "focused attention" of the individual, that is his/her ability to respond discretely to specific stimuli like auditory, verbal, visual or tactile, constitutes one of them. Other types of attention are the "sustained attention", involving the duration over time one is able to maintain performance, as well as the consistency of performance over that period; the "selective attention", defined as the ability to focus on relevant stimuli in the presence of distracting stimuli and select information for conscious processing; the "alternating attention", constituting the ability to switch from one stimulus or activity to another; and the "divided attention", defined as the ability to either do more than one activity simultaneously, or to attend multiple stimuli (Ashley & Krych, 1995).

In general, the frontal and parietal (usually right-lateralized) cortices and the thalamus are most often associated with the source of attentional modulation (Coull, 1998). Moreover, locus coeruleus (LC), via its massively divergent efferent projections, participates in generating a generalized brain state that can be characterized as "alertness." It seems that that LC activation can convert the EEG activity of the forebrain from patterns characteristic of a non-alert state to those characteristic of an alert state (Foote, 1991).

Starting with the category of sustained attention, it has been noted that prefrontal and parietal areas, preferentially in the right hemisphere are frequently engaged (Lewin et al., 1996; Pardo et al., 1991; Haxby et al.1994).

Selective attention is characterized by increased activity in parietal region involved in stimulus processing. Different regions seem to be involved depending on the specific attribute that is attended to (Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1990). Recent examples of attentional modulation of auditory regions are provided in Woodruff et al. (1996) and Pugh et al. (1996) and modulation of activity in the lingual and fusiform gyri during a color attention task was demonstrated by Clark et al. (Clark et al., 1997). Cox et al. (1997) showed that attending to motion activated a region in occipito-temporal cortex and Buchel et al. (1998b) extended these findings by showing that, in addition to extrastriate regions, attention to motion increased activity in several higher order areas as well. It was argued that activity in extrastriate regions may be modulated by prefrontal, parietal and thalamic regions. Similarly, Heinze et al. (1994) suggested that modulation of activity in specific posterior regions is mediated by regions in parietal and anterior cingulate cortices, as well as the pulvinar. A role of parietal cortex, especially the inferior parietal lobe, in control of selective attention is suggested by the findings of Pugh et al (1996). Rees at al.'s study pointed to a role of prefrontal cortex in attentional modulation. Allen et al. (1997), suggest also that cerebellum may be part of this network as well. Several of the studies on Selective Attention are based on the Stroop test, which is associated with activations in the anterior cingulate cortex and the left prefrontal cortex (Taylor et al., 1997).

Coull et al. (1998) investigated the hypothesis that right frontal and parietal cortices provide the neuroanatomical location of the functional interaction between sustained attention and the process of selectively monitoring for target objects. Six healthy volunteers performed one of two tasks which required either selective or non-selective responding. In the task which took place 3 times of 18 min. each time, 12 PET measurements of regional cerebral blood flow (rCBF) were obtained for each subject. The right inferior frontal and parietal cortices were differentially activated by increasing time on task during the selective (S) vs non-selective (NS) task. Specifically, rCBF decreased with increasing time spent performing the NS task but not the S task.. Thus, it seems that Coull et al. identified the neuroanatomical correlates of each process separately, and confirmed earlier reports of prefrontal cortex and anterior cingulate activation associated with selective responding, and a fronto-parietal-thalamic network associated with sustained attention.

As far as divided attention is concerned, it seems that when two tasks are performed simultaneously, performance often deteriorates, with simultaneous increases in reaction time and error rate. Three potential neurophysiological mechanisms behind this deterioration in performance have been considered in Klingberg's study (1998): a) dual-task performance requires additional cognitive operations and activation of cortical areas in addition to those active during single-task performance; b) two tasks interfere if they require activation of the same part of cortex; and c) cross-modal inhibition causes interference between two tasks involving stimuli from different sensory modalities. Positron emission tomography was used to measure regional cerebral blood flow (rCBF) during performance of an auditory working memory (WM) task, a visual WM task, both WM tasks (dual task) and a control condition. Compared to the control condition, the auditory and visual WM tasks activated sensory-specific areas in the superior temporal gyrus and occipital pole respectively. Both WM tasks also activated overlapping parts of

cortex in the dorsolateral prefrontal, inferior parietal and cingulate cortex. There was no separate cortical area which was activated only in the dual task, and thus no area which could be associated with any dual task specific cognitive process. Decrease in rCBF in one WM task did not overlap with the areas of rCBF increase in the other WM task. However, an inhibitory mechanism could not be ruled out, since the rCBF increase in sensory specific areas was smaller in the dual-task condition than in the single-task conditions. The cortical activity underlying WM was to a large extent organized in a non-sensory specific way, and the results are consistent with the hypothesis that concurrent tasks interfere with each other if they demand activation of the same part of the cortex.

Finally, both spatial and temporal orienting are found to activate a number of brain regions, including prefrontal and parietal brain regions. More detailed analyses revealed that activations in the intraparietal sulcus were right lateralized for spatial attention and left lateralized for temporal attention (Corbetta, Miezin, Shulman, & Petersen, 1995). Moreover, simultaneous spatial and temporal attention activated mainly parietal regions, suggesting that parietal cortex, especially in the right hemisphere, is a site for interaction between different attentional processes. In addition, in the study of Le at al. (1998), the cerebellum was implicated in attention shifting. Table 1. and figure 1 summarize the four types of attention dysfunction, their representation on the brain and the best tests to measure these specific types of attention.

A Nosology of Disorders of Attention

Impairment of attention is a common symptom of neuropsychiatric disorder and merits systematic attempt and classification. All symptoms of impaired attention do not stem from the same cause. Instead, they may have a variety of causes that comprise a number of broad categories. The etiology can be familial or genetic, metabolic and environmental. Other (non-specified) causes can be sleep/breathing disorders or eating disorders (Mirsky, 1995)**.**

Some examples of familial/genetic etiologies which result in impaired attention are schizophrenia (Mirsky, Lochhead & Jones, 1992), absence epilepsy (Ottman, Hauser &Susser, 1985), autism, narcolepsy and Attention Deficit Disorder (ADD) and Attention Deficit Hyperactivity Disorder (ADHD) (Barkley, 1999). More specifically, as far as petit mal seizures are concerned, their primary symptom is a brief interruption of consciousness or attention that occurs in conjunction with bilaterally symmetrical and synchronous spike-and- wave EEG discharges. Although a genetic mutation of the human lymphocyte antigens (HLA) region of chromosome 6 has been implicated for the impairment of sustained attention in all the above disorders, with the possible consequence of an abnormal firing in thalamic neurons, it seems that each one of a number of genes make a subtle contribution to a person's susceptibility to a disease (Weissbecker, Durner & Janz, 1991).

Some examples of impaired attention with metabolic etiologies are phenylketonouria and uremia (Andersen & Siegel, 1967). End-stage uremia is sometimes accompanied by an EEG pattern that resembles that seen in absence epilepsy. It appears that toxins associated with kidney failure attack the same structures implicated in absence epilepsy (Penfield & Jasper, 1954).

Examples of environmental reasons for impaired attention can be malnutrition, lead intoxication, pregnancy or birth complications, fetal alcohol syndrome, neurocysticercosis or other parasitic infections and lack of intellectual stimulation. Whereas some of the problems do not require poverty (e.g. maternal drinking, pregnancy or birth complications), there is a greater likelihood that poor people will suffer from them, (Cravioto, Delicardie & Birch, 1966). Moreover, cerebral insults like head injuries, brain infections or tumors produce impairments in attention (Greenblatt, 1986).

Finally, the deleterious effects of sleep breathing disorders (apnea) on attention are perhaps not unexpected in view of the interrupted sleep patterns of persons with this disorder. Prolonged loss of sleep may result in brief transitions from wakefulness to slow-wave sleep that intrude upon wakefulness. The individual at the time of those intrusions is less responsive to external stimuli and exhibits attention lapses. Attention which is closely related to arousal and wakefulness and extends from general alerting, as in the orienting reflex to specific alerting, targeting a specific modality, originates in the medial portions of the brainstem reticular formation and pons and ascends till the reticular formation of the thalamus, with widespread connections to most of the forebrain cortical areas, (Mirsky & Cardon, 1962).

Although there is a considerable heterogeneity of in the attentional deficits associated with neuropsychiatric disorders, there is a substantial degree of homogeneity as well. Of the sixteen etiologies mentioned above there is evidence of impaired sustained attention in eleven of them and in alternating attention in eight of them. There are though many aspects of attention like working memory or selective attention that remain to be investigated for most of the etiologies (Mirsky, 1995).

The similarities of signs and symptoms on attentional deficits create though problems related to the correct diagnosis of the disorder, suggesting a need for a comprehensive medical evaluation for each disorder suspected. For example there are other medical and neurological conditions which simulate ADHD, like learning disabilities , Tourette Syndrome (Comings, 2001), epilepsy, fragile X syndrome, (Borghgraef, Fryns, Van Den Berghe, 1990), pervasive developmental and autistic disorders, hyperthyroidism or hypothyroidism (Weiss & Stein, 1999).

In case of someone suffering from attention deficits, ADD or ADHD seems to be the most frequently diagnosed disorder. It is a common, genetically transmitted neurological disorder, with onset in childhood, probably mediated by decreased brain dopaminergic functioning (Wender, Wolf, & Wasserstein, 2001). Anatomical imaging studies of individuals with ADHD consistently point to the involvement of the frontal lobes, basal ganglia, corpus callosum and cerebellum. Total brain size in ADHD subjects is 5% smaller than in age and gender matched controls. Moreover, smaller globus pallidus and anterior corpus callosum have been consistently found in ADHD subjects. Smaller anterior corpus callosum areas are consistent with involvement of prefrontal cortical regions: Although, normally, the right anterior brain is slightly larger than the left, significant decreases of this asymmetry in ADHD have been reported. Finally there is a trend towards greater cerebellar atrophy in adults with prior history of hyperkinetic dysfunction. It is speculated that dysfunction of the cerebello-thalamo-prefrontal circuit may underlie the motor control, inhibition, and executive function deficits encountered in ADHD.

When PET was used in adults with ADHD, decreased frontal cerebral metabolism was demonstrated. More specifically, decreased blood flow has been found in the striatum and prefrontal regions (Giedd, Blumenthal, Molloy & Castellanos, 2001).

The current study will concentrate on attention deficits due to Traumatic Brain Injury (TBI). Despite the fact that some research already exists, concerning types of attention and their representation on the brain using EEG, little research has addressed the issue of types of attention deficits in TBI population and their EEG patterns while these individuals are engaging in cognitive tasks. The purpose of this study is to investigate this almost new area, by comparing TBI individuals with different types of attention deficits with their matched for age and gender healthy control participants during EEG recording while engaging in an auditory cognitive task and an after tasks resting baseline.

Traumatic Brain Injury

The National Head Injury Foundation defines head injury as a traumatic insult to the brain capable of producing physical, intellectual, emotional, social, and vocational changes. This definition implies brain damage and associated dysfunction such as inability to coordinate movements, speak, remember, reason, or modulate behavior. While not denying that pathology may be diffuse, research suggests that frontal and temporal damage is a common and relatively more severe consequence of TBI (Solberg & Mateer, 1988). Lacerations, contusions and hemorrhages as well as diffuse axonal injury (DAI)- which is the diffuse degeneration of the cerebral white matter- are prominent in the frontal and temporal regions after brain injury. Finally, the shearing, tearing, and stretching of axons may also result in a true disconnection between prefrontal, limbic, and association cortices leading to disturbances of the cognitive and executive processes.

Moreover, different frequencies (Basar, 1998) in the EEG have been found to be emitted in different areas of the brain depending on the cognitive activity the person engages each time**.**

Theta Band

According to Miller (1992), for example, there are structures in the brain that produce theta activity following a sensory or cognitive event that may be either external or internal. There is for example some evidence that the midline prefrontal region of the cortex can generate theta activity in certain cognitive states. This was reported by Mizuki et al. (1980): EEG rhythms of 5-5.5 Hz frequency appeared with some regularity during the performance of simple repetitive mental arithmetic tasks. In the human frontal cortex a theta enhancement increase of 50% was recorded while a subject paid attention to a target that was expected 100% . In addition, Lange et al. (1978), showed that theta frequencies (3-7Hz) increased during motor or verbal learning tasks. Miller argues that all these data are compatible with the hypothesis that theta activity in frontal regions is associated with a theta activity in the hippocampus. It might be expected that the prefrontal cortex should emit theta activity at the same time as the hippocampus, in view of the strong connections between the two structures. The experiments of Adey (1960, 1969) were the first to indicate that induced theta rhythms in the limbic system and in hippocampus are significantly correlated with cognitive processes in the central nervous

system. According also to Basar (1999), during selective attention processes, large theta enhancements occur in frontal and parietal areas. Frontal midline theta rhythm (Fm theta), recognized as distinct theta activity on EEG in the frontal midline area, reflects mental concentration as well as meditative state or relief from anxiety. Attentional network in anterior frontal lobes including anterior cingulate cortex is suspected to be the generator of this activity, and the regulative function of the frontal neural network over autonomic nervous system (ANS) during cognitive process is suggested (Kubota, 2000).

Alpha and Beta Band

The alpha frequency has been studied even more extensively. The general view is that in comparison with a resting period, task demands tend to attenuate or desynchronize alpha rhythms (Berger, 1929;). When someone closes his eyes in a lighted room, alpha band burst appears. However, it immediately disappears when for example the subject follows the instruction "multiple 11 by 13" and it reappears as soon as the answer is delivered. However, the experiments by Osaka et al. (1984) showed that only for difficult but not for easy tasks, alpha frequency increases selectively in the hemisphere that is dominant for a particular task. Evidence of a positive relationship between memory performance and increase and synchronized alpha activity was indicated by Basar et al. (1985, 1987, 1989). Again, clear evidence for a positive relationship between memory performance and mean alpha was reported by Klimesch et al. (1990).

As far as memory and alpha desynchronization are concerned, in Klimesch's experimental findings (1996, 1997), the following hypothesis was supported: Episodic memory demands lead to a synchronization in the theta band, whereas semantic memory demands lead to a task-specific desynchronization in the upper alpha band. Klimesch further suggests that episodic memory processes are reflected by oscillations in an anterior limbic system, whereas semantic memory processes are reflected by oscillations in a posterior thalamic system. Moreover, according to Foxe et al. (1999) parieto-occipital approximately 10 Hz activity reflects anticipatory state of visual attention mechanisms.

Moreover, when a subject has been visually stimulated, large alpha enhancements are recorded in the mesencephalic reticular formation, lateral geniculate nucleus and hippocampus. There are also large alpha enhancements in the visual and association cortices. Upon visual stimulation large theta enhancements are recorded in the thalamus, hippocampus, primary cortex, and association cortices including the frontal lobes. Alpha enhancements are not observed in the cortical auditory areas and in the medial geniculate nucleus upon visual stimulation. However, theta enhancements are present in these structures. During the first second following a visual stimulus there are large and dominant alpha responses in the occipital cortex. The alpha information then reaches the parietal cortex and via the association areas the frontal cortex, but not the auditory areas (Basar, 1989).

Upon an auditory stimulation, alpha enhancements dominate the structure in the auditory pathways, like reticular formation and hippocampus. Large alpha responses also occur in the medial geniculate nucleus and auditory cortex. Again it is noteworthy that reticular formation and hippocampus show large alpha responses, whereas lateral geniculate nucleus and visual cortex depict only theta responses. Theta enhancements are

present in all structures, regardless of whether the stimulus is adequate or inadequate. The largest alpha enhancements are marked in temporal, parietal and occipital areas. If an auditory stimulation generates 10Hz activity in the reticular formation, this signal can be hypothetically transferred to hippocampus and from there to prefrontal cortex and nonlimbic association cortex. In this case there is also the possibility that the signal can reach the primary sensory areas too, via the nonlimbic association cortex. (Basar,1999).

Induced alpha rhythms are analogous to the gamma-band rhythms induced by moving stripes for example. In the study where Basar et al. (1989) demonstrated when a target is anticipated, gamma band energy is emitted. On the other hand, a well-trained subject emitted time-locked bursts of alpha band for up to a full second before the delivery of an expected target. By contrast to the modest evidence of the gamma band, the alpha burst in anticipation of the target, was more robust and highly significantly statistically. As far as another comparison between alpha and beta is concerned, according to Gomez et al. (1998) statistically significant differences were found in the decrease of alpha (9-11 Hz) and the increase of beta (15-17 Hz) frequencies during the attention condition with respect to the unattended condition of a spatial selective task.

In another study Gutierre and Corsi-Cabrera (1988) recorded EEG activity of 8 male volunteers at P3 and P4 during four resting periods and during the performance of three series of cognitive tasks: one verbal, one spatial and one demanding verbal and spatial processing or "mixed" task. Beta, alpha and theta relative power were compared between successful and unsuccessful trials, between start and end of performance interval and among resting periods and tasks. There were no significant differences between successful and unsuccessful trials, nor between start and ending of performance period. The effect of tasks and hemispheres on relative power showed different results for each band: beta was responsive to hemispheres while alpha and theta were sensitive to tasks; beta relative power was significantly higher in the left parietal and the same pattern of asymmetry was maintained during the three series of tasks; alpha relative power decreased and theta increased significantly during the three series of tasks regardless of their cognitive nature as compared to baseline.

In another study, Ramos, Corsi-Cabrera, Guevara and Arce (1985) recorded EEG activity of 20 female volunteers at P3, P4, C3 and C4 during four resting periods and three series of cognitive tasks: one analytic, one spatial and one demanding analytical and spatial processing or mixed task. Relative power and inter and intrahemispheric correlations were analysed. Beta relative power was significantly higher during the resting periods at the right parietal and the same pattern of asymmetry was maintained during the three series of tasks. Alpha relative power decreased and theta increased during the three series of tasks regardless of their cognitive nature as compared to baseline.

Ray and Cole (1985) conducted a research on two areas: lateralization of electrocortical processing of cognitive material and psychophysiological information processing studies related to foci of attentional demands. Eighteen subjects on each of three separate days were presented with tasks considered to be 'right hemispheric' or 'left hemispheric'. These tasks were paired in a 2 X 2 design with an attentional factor requiring attention to the environment or to internal processing. All subjects received all types of tasks. Bilateral EEG measures were taken from the frontal and parietal areas

referenced to linked ears. The results suggest that task factors (left vs. right hemisphere tasks) and attentional demands (internal vs. external) are differentially represented in terms of EEG functioning. In general the higher beta frequencies were more sensitive to the hemispheric tasks demands and the middle frequencies (alpha and low beta) more sensitive to the attentional demands especially in the parietal areas.

In two other experiments conducted by Ray and Cole (1979), the effects of attentional demands on the electroencephalogram were examined during cognitive and emotional tasks. They found an interaction of task with hemisphere as well as more overall parietal alpha for tasks not requiring attention to the environment, such as mental arithmetic, than for those requiring such attention. Differential hemispheric activation for beta was found most strongly in the temporal areas for emotionally positive or negative tasks and in the parietal areas for cognitive tasks.

Gamma Band

Several papers have handled the importance of 40Hz activity in states of attention and motivation. Tiitinen at al. (1993) reported that auditory selective attention enhances the 40Hz response in humans, especially over the frontal and central areas. The 40Hz was larger when subjects paid attention to stimuli rather than ignoring them, so the authors proposed a physiological correlation between selective attention and the 40 Hz response in humans. Muller (2000) found increased gamma band power at posterior electrode sites when subjects also perceived an object. In Gruber et al. (1999), investigated the attentional modulation of gamma band responses in a visual spatial attention task where a moving stimulus is attended: Colored rectangles were presented on a screen. After 500 ms an arrow indicated whether subjects had to shift their attention to the left or right half of the screen to detect target stimuli. During the task, either the attended half of the screen rotated horizontally while the unattended part remained motionless, or vice versa. When subjects attended the rotating stimulus, they found significantly higher power in a specific gamma band from 35-51 Hz on parieto-occipital electrode sites contralateral to the stimulation side. In addition, after the onset of the arrow which indicated what side subjects should direct their attention to, the 35-51 Hz response shifted from a broad posterior distribution to an increase of power at parieto-occipital sites contralateral to the to-be-attended side. Furthermore, the rotating stimulus elicited higher gamma band power as compared to the standing stimulus at electrode locations, which may be related to the activity of underlying cortical structures specialized for motion processing.

Lutzenberger et al. (1995) wrote that visual stimulation alters local 40Hz responses in the EEG. Their results show that area-specific 40Hz responses are correlated with the perception of coherent visual patterns in humans. Tallon et al. (1995) tested the hypothesis that synchronized activity in the gamma band plays a role in visually binding coherent static objects. The authors used two coherent Kaninza triangles (one with real contours and one with illusory contours) and a noncoherent one. They found a specific 30Hz power increase only in the case of coherent stimulation, no matter whether the real or the illusory triangle was used. This finding supports the hypothesis of a code for coherency representation. Pulvermuller et al. (1994) suggested stronger gamma responses to words compared to pseudowords. Goertz et al. (1994) presented results showing that 40Hz activity is related to stimulus evaluation in sensorimotor processing

during a visual discrimination task. The results of Basar –Eroglu et al. (1996) show also that viewing multistable patterns leads to an enhancement of 40Hz activity. Although the 40Hz increase is highest at fronto-central locations, it is also observed in parietal and occipital areas. This means that the 40Hz is enhanced over widely spread regions of the cortex, which is compatible with the concept of a distributed gamma response system in the brain.

Sheer (1989) interpreted the ongoing 40 Hz rhythm as a sign of focused cortical arousal. Van der Tweel and Spekreije (1969) found a visual high-frequency response after sinusoidal light stimulation in the occipital recordings of the human brain. Evoked gamma is tightly time-locked to an external stimulus. This type of gamma activity is recorded from the human scalp during stimulation with auditory clicks or flashing lights (Pantev et al. 1991). According to these studies it can be argued that the 40Hz activity might represent an important general information processing of the brain similar to the 10Hz activity.

Different authors like Steriades et al (1990) or Basar-Eroglu and Basar (1991), point to the fact that brain structures like the thalamic nuclei, hippocampus, the reticular formation or even the cerebellar cortex have an ability to respond to the gamma band at the same time. According to Basar (1999) the 10Hz responses are mostly a sign of adequate sensory excitement. This also seems to be the case for structures like thalamic relay nuclei and primary cortices. In lower structures, such as the reticular formation, large alpha enhancements are observed for all types of stimuli.

Related to gamma is also the basal ganglia hypothesis which supports that the basal ganglia support basic attentional mechanism which binds input to output in the executive forebrain. It provides the automatic link between voluntary effort, sensory input, and the calling up and operation of a sequence of motor programs or thoughts. The physiological basis for this attentional mechanism may lie in the tendency of distributed, but related, cortical activities to synchronize in the gamma (30 to 50 Hz) band, as occurs in the visual cortex.

Cognitive tests and EEG as assessment measures for TBI and ADD

Several kinds of cognitive tests, or subtests are currently used in order to assess attention deficits. The WAIS-R Digit Span Subtest for example, is commonly used to test immediate or working memory, the WAIS-R Digit Symbol Subtest, to test information processing speed performance, the Paced Auditory Serial Addition Test (PASAT), to assess divided attention skills and the Stroop Test, to test distractibility attention capacities. The Integrated Visual and Auditory (IVA) test finally, is supposed to test all types of attention deficits (Solberg & Mateer, 1988).

On the other hand, the quantitative electroencephalogram (QEEG)- the recordings from an electrode of the electric brain potentials- constitutes one among the different methodologies used for the study of the dynamic functional aspects of brain function (Christensen & Uzzell, 1994). QEEG is a highly validated method, for assessing among other, attention deficit disorders.

Chabot et al. studied (1998), a sample of 130 children with attention deficit disorders were evaluated with Conners' and DSM III rating scales and with neurometric QEEG, before and 6-14 months after treatment with stimulants. Significant QEEG differences

were found between the normal control group and the children with attention problems ($p<.001$). QEEG abnormalities involved increased theta or alpha power greatest in frontal regions, frontal theta/alpha hypercoherence, and posterior interhemispheric power asymmetry; coherence is analogous to a cross-correlation coefficient in the frequency domain and thus is a metric of the amount of shared activity between the two regions, while phase is the measure of the lead or lag of shared rhythms between two regions. The degree of correspondence between behavioral and QEEG changes after the stimulant treatment was at 78.5%. Pre-treatment clinical and QEEG features could predict treatment response with a sensitivity of 83.1% and a specificity of 88.2%.

In another study Clarke, Barry, McCarthy, Selikowitz and Brown (2002) investigated the presence of EEG clusters within a sample of children with the inattentive type of attention-deficit/hyperactivity disorder (ADHD). The participants consisted of 100 boys with ADHD and 40 age-matched controls. EEG was recorded from 21 sites during an eyes-closed resting condition. Two distinct EEG clusters of children with the inattentive type of ADHD were found. These were characterised by (a) increased high-amplitude theta with deficiencies of delta and beta activities, and (b) increased slow wave and deficiencies of fast wave activity.

Similarly, in a study conducted in 1991, by Thatcher, Cantor, McAlaster, Geisler, and Krause, a number of variables were used to study the development of prognostic equations for patients with closed-head injury*, studied early after injury. These were, EEG recording from 19 scalp locations, CT scan, Glaskow Coma Score (GCS) and the Rappaport Disability Rating Scale (DRS). According to the results, the best predictors of outcome in both the discriminant analyses and the regression analyses were the EEG measures ,coherence and phase.

It seems that a technique that has promise for the detection and quantification of diffuse axonal injury and thus the detection of mild cerebral injury, is the power spectral analyses of coherence and phase from the human EEG. According to Thacher et al. (1989), head injured patients show increased coherence and decreased phase in frontal and frontal temporal regions, decreased power differences between posterior and anterior cortical regions and reduced alpha power in posterior cortical regions.

On the other hand, prior research regarding QEEG and TBI has shown that the head injured group displays higher amplitudes and greater variance than the control subjects in the occipital and especially the temporal placements. Frequency analysis has revealed also increased amplitude within all frequency bands except the alpha band; (the slow bands are the Delta (0-4Hz) and Theta bands (4-8Hz). The rest of the bands are the Alpha (8-12Hz), Beta (12-32Hz) and Gamma (32-42Hz) bands). The increased amplitudes, amplitude variance, and reduced correlation coefficients at the temporal sites of the closed head injured patients in the research study conducted by Randolph and Miller (1988), are presumed to reflect areas of dysfunctional cortex. The same patients when asked to participate in cognitive tasks demanding increased arousal showed increases in delta and beta bands and decreases in alpha and theta bands. Increased theta power in brain injured patients was also reported by Montgomery et al. (1991) even after a six months period after the accident. A predominance of slow waves was also reported by Enomoto et al. (1986) from 280 cases of minor head injured patients.

Research on Attention Deficit (ADD) patients moreover, has shown an increase of the slow wave theta, and more specifically an increase in the theta-beta ratio in the frontal or central regions-depending on the age (Monastra et al., 1999).

Rationale for the study

There are no obvious EEG consistent patterns in amplitude among those with the same type of attention deficit depending on their eyes-open or eyes-closed recordings. These conclusions are taken from the results of my Master's thesis " EEG Changes of TBI Patients with Attention Deficits after Implementation of Cognitive Rehabilitation" (2001) and the preliminary analysis of this study on eyes-open and eyes-closed EEG recording of the TBI participants. The rationale for the study is that people with the same type of attention deficit will probably demonstrate the same EEG pattern, not during an eyes-open or eyes-closed EEG, but during a cognitive task or a second eyes-open baseline. Since the magnitude of an EEG recording depends also on various factors like thickness of scalp, a task minus the first eyes-open baseline EEG recording (as well as the second eyes-open baseline minus the first eyes-open baseline) were considered more appropriate ways to compare TBIs with "healthy" matched individuals.

Purpose of the study

The purpose of this study is to look at participants with different areas of brain injury and their respective type of attention deficit (like deficit in sustained, alternating, divided or selective attention) and find the EEG patterns which correspond to each type of attention deficit while the person engages into a cognitive task and the after tasks baseline.

Hypotheses for the study

It is hypothesized that the topography, direction and significance of changes from baseline to task will be different between the clinical (TBI) and matched for age and gender control individual. The same holds also for the second eyes-open baseline when compared to the first eyes-open baseline. A third hypothesis is that there will be consistent EEG patterns depending on the type of attention deficit.

Section II

METHOD

Research Design

In a series of 10 single subject experiments conducted in the Brain Research and Neuropsychology laboratory of the University of Tennessee, Knoxville, Traumatic Brain Injured (TBI) patients with attention deficits who responded to the lab's announcement to the Disability Service of the University of Tennessee, and the local newspaper "Knoxville News -Sentinel", were compared with their matched for age and gender "healthy" participants.

Participants

The proposal was distributed to the Disability Service of the University of Tennessee and to the local paper, the "Knoxville News-Sentinel" in order to attract people for the TBI group. The TBI participants who showed interest for the study had first to show evidence of their disorder and attention deficits through their medical records, a thorough interview and four psychometrics. The matched for age and gender participants of the control group were undergraduate students from the University of Tennessee, Knoxville who participated in exchange of $20 reward (from the money acquired from the departmental dissertation support grant) and/or course credit The matched for age and gender controls were assessed for "normality" through a thorough interview -where any type of neurological history had to be excluded -and through the same four psychometrics testing for attention deficits. All participants who came in contact for the project, were instructed about the scope and the procedures of the study, and signed the consent form. Ages ranged from 18 to 46. All participants were native speakers. The age and the time of the accident of the TBI participants mentioned below is their age and accident time accounted at the time of the study. The TBI participants were the following:

DH is a 46 years old female. She had her motor accident 15 years ago. Damage occurred mainly in the frontal lobes and especially in the right hemisphere. She stayed in coma for 3 days and in the emergency room for 5 days. She reports having had post-traumatic amnesia.

DS is a 38 years old female. She had a motor accident 23 years ago. Damage occurred in her frontal lobe, especially the left frontal, and the left temporal. She also had some lacerations in the occipital area. She did not stay in coma but was out of awareness of her surroundings for 7 days. She stayed in the Intensive Care Unit for 7 days and in the hospital for 14 days. She had post-traumatic amnesia for 7 days.

FM is a 23 years old male. She had his motor accident 4 years ago. Damage occurred mainly in the bilateral thalamic areas with the left greater than the right, the left basal ganglia, and left frontal vertex. He stayed in the hospital's rehabilitation center for 6 months.

GS is a 23 years old male. He had his motor accident 6 years ago. Damage occurred mainly in the left temporal and parietal-frontal lobe. He remained in a comatose and semi comatose state for about 10 days. He stayed in a rehabilitation center for 8 months.

MF is a 48 years old female. She had a motor accident one and a half year ago. She had her left hemisphere injured. She did not stay in coma, she stayed in the emergency

room for 4 hours and in the hospital for 2 months and a half. She had post-traumatic amnesia the first two months.

RM is a 32 years old male. He had his motor accident 14 years ago. Damage occurred to the right hemisphere, frontal lobe and optical nerve. He had a massive right to left shift, with a right subdural hematoma 2 cm in diameter. He later developed a small subdural hydroma in the left frontal region and in the right occipital region. He stayed in coma for 10 weeks, in an emergency room for 6 weeks and in the hospital for one year and a half. At the present time he suffers both from anterograde and retrograde amnesia.

RQ is a 20 years old female. She had a motor accident 6 years ago. Damage occurred mainly in the right posterior lobe and left thalamic area. She stayed in coma for 24 days, in the emergency room for 3 hours, in the Intensive Care Unit for 72 hours and in the hospital for 4 months. She reports having had post-traumatic amnesia.

SM is a 40 years old male. He had a motor accident 8 years ago. Damage occurred in the left temporal/hippocampal area. He stayed in coma for 3 months and in the hospital for 4 months and a half. He reports having had post-traumatic amnesia.

SJ is a 28 years old male. He had had multiple motor race accidents (23), with the most serious having taken place two years ago. He has lost consciousness for more than 1 minute nine times while in his most serious accident, he lost consciousness for 15 minutes. He stayed in the hospital for four weeks. Damage occurred mainly in the frontal and central areas and especially in the right hemisphere. He reports having had post-traumatic amnesia for four months.

WH is a 46 years old female. She had her motor accident 6 years ago. She lost consciousness for half an hour, she stayed in the emergency room for 1 day and in the hospital for 2 weeks and the rehabilitation center for 1 month. Damage occurred mainly in the left hemisphere. She reports having had posttraumatic amnesia.

Additionally they were 20 matched for age and gender controls, two for each experimental participant. Only one of those two was chosen for each case, the one whose psychometrics and eyes-closed EEG was the most representative of an average, "normal" person. All ten matched controls' psychometrics, and eyes-closed EEG were representative of an average non-clinical control.

Setting

The room for the psychometrics assessment and the recording of EEG, in the Brain Research lab of UT, are specifically designed to be free of visual and auditory distraction. There are no windows. Two chairs, a table and two computers exist in the room. During the assessment and recording no one else was allowed to get in the room except the participant and the experimenter.

Materials

All participants were assessed with the WAIS-R Digit Span Subtest, the WAIS-R Digit Symbol Subtest, the Paced Auditory Serial Addition Test (PASAT), and the Integrated Visual and Auditory (IVA) test. Digit Span assesses working memory, short-term memory, sequential processing and learning ability. Digit Symbol assesses perceptual organization, sequential processing, learning ability, visual short-term memory and visual-motor coordination. PASAT assesses information processing skills and the IVA all

different types of attention capacities for both audition and vision. More specifically its scales Prudence and Vigilance assess Focused Attention; the scales Stamina, Consistency and Focus assess Sustained Attention; the scales Prudence, Vigilance and Comprehension assess Selective Attention; the scales Speed, Balance, Readiness, Consistency and Focus assess Alternating Attention, and the scales Prudence and Speed assess Divided Attention.

More specifically- regarding the subscales in IVA- Prudence measures the ability to stop, think and not automatically react; Consistency measures the ability to perform in a generally reliable manner over time; Stamina looks at increase or decrease in a person's reaction time speed during the test and can be useful in identifying difficulties in maintaining an effort over time; Vigilance looks at failure to make a response to a target during "rare blocks"; Focus looks at momentary losses of focus in attention; Speed measures discriminatory mental processing speed. It may reflect mental slowness; Balance tests whether the person is relatively faster in terms of mental processing speed for one modality or the other; Readiness shows how a person reacts to frequent versus rare stimuli; and Persistence how much the person complies with the test demands throughout the entire test.

From the above tests, in the WAIS-R Digit Span subtest, the orally presented 3-9 digits have to be orally reproduced forward and backwards. In the WAIS-R Digit Symbol there are nine symbols paired with nine digits. The examinee has 1 1/2 minutes to fill in as many symbols as he can, under the numbers on the answer sheet. In the PASAT test, numbers are orally presented from a tape. The individual has to add each number he/she hears to the just previous number and orally present the resulting number. In the IVA test, the participant hears or sees on the screen either the number "1" or the number "2" and must click the mouse only when he hears or sees number "1".

The six cognitive tasks are taken from the Captain's Log software program -measuring and training different types of attention- and each one of them lasts for 3 minutes: In the first task the person listens to two patterns of rhythm and has to chose whether they are the same or different and click on the respective ("same" or "different") box. In the second, and the only statistically analyzed, task the person listens to two patterns of melody and has to chose whether they are the same or different and click on the respective ("same" or "different") box. In the third task the person has to click the mouse each time two of three boxes (the center one with each of the lateral ones) match in color. In the fourth task the person clicks the mouse each time the box matches in color with the rectangular border line. In the fifth task the person sees a series of numbers/letters appearing the one after the other and has to click the mouse each time he/she sees the number or letter designed as target from the beginning. In the sixth task the person clicks the mouse each time he/she sees a box of particular size but not when boxes of other sizes appear.

Apparatus

EEG was recorded with a Lexicor Neurosearch 24 EEG recorder, using an electro cap with electrodes set according to the 10/20 international standard. The IVA as well as the cognitive tasks from the Captain's Log program were presented in a computer screen

50cm from the participants' eyes. The participants responded using the computer's mouse. Speakers with adjustable volume were also used.

Procedure

Participants, who came in contact for the project either the Disability Service of UT, or through the local newspaper, came to the Brain Research and Neuropsychology laboratory of the University of Tennessee. After signing the consent form, and orally respond to a questionnaire, their assessment tests –which last for about one hour-were given individually to each one of them. The instructions for all the tests last 10 minutes, as well as the discussion and disassembly.

An eyes open/eyes closed EEG baseline, a recording during six cognitive tasks and a second post-task eyes-open recording took place. EEG activity was recorded using a 19-channel electrode cap. The preparation period lasts for 40 minutes, the learning period to familiarize with an eyes- open and eyes-closed baseline recording lasts for about 5 minutes and the actual recording for about 35 minutes.

EEG was recorded with a 19 electrode cap, according to the 10/20 system, which is connected to a Lexicor Neurosearch 24 EEG recorder. Cap electrodes are filled with electrolyte gel, gently rubbed into the scalp until impedance reaches less than 5Kohms. Recording is referred to the two ear lobes with additional electrodes. Cortical location is measured at all 19 bands. In the eyes-open baseline recording, the participants have to fixate their eyes on a point on the screen for three minutes, while in the eyes-closed baseline they just have to close their eyes and relax. In the cognitive tasks the have to be as still as possible and always try to eye-blink as little as possible.

Independent or semi-independent variables

a) Brain injured individuals versus their matched for age and gender control individual.
b) Type of frequency
c) Brain region
d) Type of condition (ADT task or EO2) .
e) Type of attention deficit.

Dependent variables

Magnitude differences:
a) Auditory Discrimination Task (ADT) minus First Resting Baseline (EO1).
b) Second Resting Baseline (EO2) minus First Resting Baseline (EO1).

Statistical Analyses

In the analysis, the average magnitude of the first eyes-open baseline for each channel and frequency is subtracted from each epoch of the ADT task and of EO2. Four frequencies were analyzed: theta (4-8 Hz), low alpha (8-10 Hz), high alpha (10-12 Hz) and low beta (12-21 Hz). Afterwards, the channels were grouped into five brain regions for each frequency. The five grouped regions consist of the right frontal area (channels F8, F4), the left frontal area (F7, F3), the central area (FZ, CZ, PZ, C3, C4), the right

posterior area (P4, T6, O2), and the left posterior area (P3, T5, O1). The channels F1, F2, T3 and T4 were omitted due to muscle artifacts.
Individual epochs for each condition (ADT and EO2) minus baseline were used as individual observations of the sample (complete EEG recording). Statistical tests compared EEG recordings between each TBI and his/her matched control. Before conducting the comparison, recordings were tested for normality and homogeneity of variance using the Shapiro and the Levene test respectively.

These tests rejected normality and homogeneity of variance, and the use of various transformations did not improve the distribution of the data. Therefore, the Mann-Whitney non-parametric tests were used to compare the TBIs with the controls. Wherever the variances of the two samples were found homogeneous, the Mann-Whitney test was used to compare the means. Wherever homoscedasticity was rejected, this was accepted as a valid measure of difference between the two samples.

P-values (2-tailed) were corrected for multiple comparisons using a stepwise adjustable Bonferroni method. For the four frequencies, the five brain regions and the two conditions (ADT task and EO2) the alpha level was adjusted to 0.001. The smallest p-value was compared to 0.001 and if it was found smaller, then 0.001 was multiplied by the number of the significantly different tests plus one (e.g., after the first rejection, 0.001 was multiplied by 2, after the second rejection it was multiplied by 3, and so on), in order to avoid a type II error (of ignoring significant differences). The direction of the differences is shown in figures, where the means for the TBI and his/her matched control are portrayed.

Section III

RESULTS

Psychometrics

The psychometric results for all TBI participants are found in table 2. More specifically, for DH the most pronounced deficit is sustained attention. More specifically, she shows one standard deviation below average difference in Consistency Auditory and Stamina Visual and one and a half standard deviations below average in Digit Symbol. In general, her psychometrics demonstrate a deficiency in alternating and sustained Attention and visual-motor coordination.

For DS the most pronounced deficits are working memory and divided attention. More specifically, DS shows two standard deviations below average difference in PASAT and Speed Auditory.

For FM the most pronounced deficits are working memory, focused and selective attention. More specifically, FM shows one standard deviation below average difference in Consistency Auditory, Prudence and Consistency Visual, Focus and Speed Auditory and Visual; two standard deviations below average difference in PASAT and Digit Symbol and three standard deviations below average difference in Vigilance Auditory. In general, his psychometrics demonstrate a deficiency in all types of attention: focused, sustained, selective, divided and alternating Attention, in working memory and visual-motor coordination.

For GS the most pronounced deficits are focused and selective attention, impulsivity and short-term memory. More specifically, GS shows a slight below average difference in Prudence Auditory and Vigilance Auditory, as well as in Digit Span; one standard deviation below average difference in Consistency and Focus Auditory and in Digit Symbol; three standard deviations below average difference in Prudence visual and PASAT. In general, his psychometrics demonstrate a deficiency in short-term and working memory, in learning ability, perceptual organization, visual motor coordination, focused, selective and alternating attention.

For MF the most pronounced deficit is divided attention. More specifically, MF shows one standard deviation below average difference in Speed Auditory and two standard deviations below average difference in Speed Visual.

For RM the most pronounced deficits are focused and selective attention. More specifically, RM shows one standard deviation below average difference in Vigilance and Focus Visual and one and a half standard deviations below average difference in Digit Span and Digit Symbol; five standard deviations below average difference in Stamina Auditory, Vigilance Auditory and Speed Auditory. His psychometrics show a deficiency in all types of attention: focused, sustained, selective, divided and alternating attention, in short-term and working memory and visual-motor coordination.

For RQ the most pronounced deficits are short-term and working memory as well as divided attention. More specifically, RQ shows one standard deviation below average difference in Digit Symbol, two standard deviations in Digit Span, three standard deviations in PASAT and five standard deviations below average difference in Speed Auditory and Visual. Her psychometrics demonstrate a deficiency in visual short-term

memory and visual coordination, in short-term and working memory as well as in divided attention.

For SM the most pronounced deficits are working memory and divided attention. More specifically, SM shows one standard deviation below average difference in Speed Auditory and Visual and Consistency Visual; two standard deviations below average difference in PASAT and five standard deviations in Prudence Auditory and Visual. His psychometrics demonstrate a deficiency in sustained, alternating, divided and selective attention as well as in working memory

For SJ the most pronounced deficits are focused and selective attention. More specifically, SJ shows one standard deviation below average difference in Focused Auditory, two standard deviations below average difference in Consistency Auditory and three standard deviations below average difference in Vigilance Visual. His psychometrics demonstrate a deficiency in Sustained, Alternating, focused and selective attention..

For WH the most pronounced deficits are sustained, alternating and divided attention as well as impulsivity. More specifically, WH shows one standard deviation below average difference in Prudence Auditory, Focus Visual and Auditory; two standard deviations below average difference in Consistency Visual and Speed Auditory and three standard deviations below average difference in Consistency Auditory. She also shows severe hyperactivity. In general, her psychometrics demonstrate a deficiency in all types of attention: focused, sustained, selective, divided and alternating attention.

EEG

DH: Auditory Discrimination Tone (ADT) task-Baseline

During the ADT task, DH and her matched control significantly differ in the following frequencies and brain regions: in theta, low alpha, high alpha (in right posterior region) and low beta (in all areas except left posterior region). More specifically, DH decreased magnitude as compared to initial resting baseline in all frequencies, while her matched control increased it (Table 2, Figure 2-5).

DH: Eyes-Open Two (EO2) -Baseline

During EO2 there are significant differences between DH and her matched control in the following bands and brain areas: low alpha (right frontal and both posterior areas), high alpha and low beta (all areas except right posterior region). More specifically, DH increased low alpha and high alpha magnitude less than her matched control, as compared to initial resting baseline. Concerning low beta, DH decreased magnitude as compared to initial resting baseline, while her matched control increased it. In general, comparing EO2 to EO1, DH did not increase magnitude as much as her matched control, in all four frequencies (Table 3, Figure 6-9).

DS: Auditory Discrimination Tone (ADT) task-Baseline

During ADT task, there are significant differences in several areas between the TBI participant *DS* and her matched control during the ADT task: the left frontal and right posterior area in low alpha, the right frontal, central and posterior areas in high alpha, and

all five brain regions in low beta. More specifically, comparing ADT task to EO1, DS increased magnitude whereas her matched control decreased it (Table 4, Figure 10-13) .

DS: Eyes-Open Two (EO2) -Baseline

During EO2, there are significant differences in theta (in frontal regions) and low beta (all areas except right posterior), between the TBI participant *DS* and her matched control. More specifically, DS increased more theta magnitude as compared to her initial resting baseline, than her matched control. Concerning low beta, DS increased magnitude as compared to initial resting baseline, whereas her matched control decreased it. In general, comparing EO2 to EO1, DS increased magnitude more than her matched control (Table 5, Figure 14-17) .

FM: Auditory Discrimination Tone (ADT) task-Baseline

During ADT, there are significant differences between FM and his matched control in the following frequencies and brain regions: in theta (left posterior region), in low alpha (posterior areas), in high alpha (left posterior) and all areas of low beta. More specifically, in theta and low alpha FM decreased magnitude as compared to initial resting baseline, while his matched control increased it. In high alpha FM increased it less than his control. In low beta, FM increased magnitude more as compared to initial resting baseline, than his matched control. In general, comparing the ADT task to EO1, FM did not increase theta, low alpha and high alpha frequencies as much as his matched control but increased more low beta (Table 6, Figure 18-21).

FM: Eyes-Open Two (EO2) -Baseline

During EO2, there are significant differences between FM and his matched control in the following frequencies and brain regions: in theta, low alpha (all areas except left frontal), in high alpha (right frontal) and low beta (right posterior). More specifically, in theta and low alpha as well as in right posterior area in low beta, FM increased magnitude more as compared to initial resting baseline, than his matched control. The opposite holds for the right frontal region in high alpha. In general, comparing EO2 to EO1, FM increased magnitude more in all frequencies with the exception of right frontal region in high alpha, as compared to his matched control (Table 7, Figure 22-25).

GS: Auditory Discrimination Tone (ADT) task-Baseline

During ADT task, GS and his matched control significantly differ in the following frequencies and brain regions: in theta (except right frontal area), high alpha (all areas except right frontal), low beta (all areas except left posterior). More specifically, GS decreased theta magnitude as compared to initial resting baseline, while his matched control increased it. The same holds for high alpha, except in right posterior area where it increased magnitude less as compared to initial resting baseline, than his matched control. In low beta, GS increased magnitude less, as compared to initial resting baseline, than his matched control. In general, comparing ADT task to EO1, GS did not increase magnitude as much as his matched control (Table 8,Figure 26-29) .

GS: Eyes-Open Two (EO2) -Baseline

During EO2, GS and his matched control significantly differ in the following frequencies and brain regions: in low alpha (left frontal and both posterior regions), high alpha (left frontal), and all areas in low beta. More specifically, when compared to initial resting baseline, GS increased low alpha magnitude less in the left frontal region but increased it more in the posterior ones, than his matched control. In high alpha (left frontal) and all areas in low beta, GS increased magnitude more, as compared to initial resting baseline, than his matched control. In general, comparing EO2 to EO1,GS increased magnitude more than his matched control, in all four frequencies except in the left frontal region of low alpha (Table 9, Figure 30-33).

MF: Auditory Discrimination Tone (ADT) task-Baseline

During ADT task, there are significant differences in all analyzed frequencies (theta, low alpha, high alpha and low beta) between the TBI participant *MF* and her matched control. MF increased theta magnitude less, as compared to her initial resting baseline, than her matched control. Concerning low alpha and high alpha, MF decreased magnitude as compared to initial resting baseline, whereas her matched control increased it. As far as low beta is concerned, MF increased magnitude in the frontal brain regions, as compared to initial resting baseline, whereas her matched control decreased it. The opposite occurs in the posterior brain areas: MF decreased magnitude as compared to initial resting baseline, whereas her matched control increased it. In the central area, she increased low beta magnitude less, as compared to her initial resting baseline, than her matched control. In general, comparing ADT task to EO1, MF did not increase magnitude as much as her matched control, with the exception of frontal brain regions of low beta (Table 10, Figure 34-37).

MF: Eyes-Open Two (EO2) -Baseline

During EO2, there are significant differences between the TBI participant MF and her matched control. These are in all five brain regions of high alpha and low beta, as well as in theta (left frontal area) and low alpha (in both frontal regions). More specifically, MF increased theta and low alpha magnitude more, as compared to her initial resting baseline, than her matched control. However, the opposite occurs in high alpha: MF increased high alpha magnitude less, as compared to her initial resting baseline, than her matched control. Concerning low beta, MF increased magnitude more in the left frontal area, as compared to her initial resting baseline, than her matched control, but decreased it more in the right frontal and central area and even decreased it in posterior regions, whereas her matched control increased it. In general, comparing EO2 to EO1, MF increased magnitude more in the frontal regions of theta, low alpha and low beta, as compared to her matched control, but did not increase it as much as her control, in high alpha and the rest brain regions of low beta, (Table 11, Figure 38-41).

RM: Auditory Discrimination Tone (ADT) task-Baseline

During the ADT task, RM and his matched control significantly differ in the following frequencies and brain regions: in theta (right frontal and posterior areas), in low alpha (in central and posterior areas), in high alpha (in posterior regions) and low beta (in frontal and posterior areas). More specifically, RM decreased theta magnitude more in all areas, as compared to initial baseline (except right posterior region), than his matched control. But decreased it less, as compared to initial baseline, in central and posterior regions in low alpha. The same holds for high alpha in the posterior areas. On the contrary, he decreased more low beta, as compared to initial baseline, than his matched control. In general, comparing ADT to EO1, RM did not increase low alpha, high alpha and right posterior theta as much as his matched control but increased more theta and beta magnitude (Table 12, Figure 42-45).

RM: Eyes-Open Two (EO2) -Baseline

During EO2, RM and his matched control significantly differ in the following frequencies and brain regions: in theta (posterior regions), in low and high alpha (central and posterior areas), and in low beta (in right frontal and both posterior areas). More specifically, RM increased theta magnitude less, as compared to initial resting baseline, than his matched control. In low alpha, RM decreased magnitude, as compared to initial resting baseline, while his matched control increased it. In the central and posterior areas of high alpha and in low beta , RM decreased magnitude less, as compared to initial resting baseline, than his matched control. In general, comparing EO2 to EO1, RM did not increase theta, low alpha and low beta magnitude as much as his matched control, but increased more high alpha (Table 13, Figure 46-49).

RQ: Auditory Discrimination Tone (ADT) task-Baseline

During ADT task, there are significant differences in several areas between the TBI participant RQ and her matched control: These are all brain areas in high alpha; the posterior and central regions in low alpha; the posterior right area in theta and the left frontal and right posterior region in low beta. More specifically, RQ increased theta magnitude more, as compared to her initial resting baseline, than her matched control. In low alpha, she increased magnitude less, as compared to her initial resting baseline, than her matched control in frontal and central regions and even decreased magnitude as compared to initial baseline, in the posterior . The same holds for low beta and high alpha (except in posterior left area). In general, comparing ADT to EO1, RQ did not increase magnitude as much as her matched control, with the exception of theta and left posterior high alpha (Table 14, Figure 50-53).

RQ: Eyes-Open Two (EO2) -Baseline

During EO2, the significant differences between the TBI participant RQ and her matched control, exist in theta, low alpha (except left frontal), in high alpha (in central, right frontal and right posterior areas) and in low beta (central region). More specifically, RQ increased theta magnitude more in posterior right area, as compared to her initial resting baseline, but increased it less in all other areas. The same pattern holds for low alpha. She also increased high alpha and low beta magnitude less, as compared to her

initial resting baseline, than her matched control. In general, comparing EO2 to EO1, RQ shows did not increase as much as her matched control, with the exception of posterior right theta (Table 15, Figure 54-57).

SM: Auditory Discrimination Tone (ADT) task-Baseline

During ADT task, the significant differences between the TBI participant SM and his matched control exist in low beta, low alpha (except the central area), high alpha (except in central and left posterior regions) and in low alpha (posterior areas).

More specifically, SM increased theta and low alpha magnitude less in posterior areas, as compared to his initial resting baseline, than his matched control, but increased more, as compared to his initial resting baseline, in the frontal regions. In high alpha and low beta SM decreased magnitude, as compared to initial resting baseline, whereas his matched control increased it. In general, comparing the ADT task to EO1, SM did not increase magnitude as much as his matched control, with the exception of posterior areas in low alpha (Table 16, Figure 58-61).

SM: Eyes-Open Two (EO2) -Baseline

During EO2, there are significant differences between the TBI participant SM and his matched control, in right frontal and posterior theta, in right frontal and both posterior areas in low alpha, in both frontal and right posterior region in high alpha and in all areas of low beta.

More specifically, SM increased theta and low alpha magnitude more in frontal areas, as compared to his initial resting baseline, than his matched control but decreased it more, as compared to his initial resting baseline, in the posterior regions. In all areas of high alpha SM increased magnitude more, as compared to his initial resting baseline, than his matched control. In low beta, SM decreased magnitude as compared to initial resting baseline in right frontal, central and posterior regions, whereas his matched control increased them. The opposite holds for the other two areas.

In general, comparing EO2 to EO1, SM increased magnitude more than his matched control in all areas of high alpha and the frontal areas of the rest of the frequencies, but did not increase as much as his matched control the magnitude in the central and posterior areas (Table 17,. Figure 62-65).

SJ: Auditory Discrimination Tone (ADT) task-Baseline

During ADT task, the significant differences between the TBI participant SJ and his matched control, occur in theta frequency (right frontal and posterior region), in low alpha (left frontal, central and posterior areas), high alpha (posterior areas) and low beta (frontal and posterior regions).

More specifically, in theta, SJ decreased magnitude as compared to initial resting baseline in frontal regions, whereas his matched control increased them. The opposite holds for the central and posterior areas. In low alpha, SJ decreased magnitude more in the frontal areas, as compared to initial resting baseline, than his matched control but less in the central and posterior regions. In high alpha and low beta, SJ increased magnitude as compared to initial resting baseline, whereas his matched control decreased them. In general, comparing the ADT task to EO1, SJ increased magnitude more than his matched

control- in all frequencies and brain regions, with the exception of the frontal regions of theta and low alpha (Table 18, Figure 66-69).

SJ: Eyes-Open Two (EO2) -Baseline

During EO2, SJ shows significant differences with his matched control in all brain regions in theta, high alpha, and low beta (except left frontal area) and in central and posterior areas in low alpha. More specifically, SJ decreased theta magnitude, as compared to initial resting baseline , whereas his matched control increased them. In low alpha there is less increase of magnitude as compared to initial resting baseline, than his matched control. In high alpha and low beta , SJ decreased magnitude in frontal areas- as compared to initial baseline- whereas his matched control increased it. The opposite holds for the central and posterior areas. In general, comparing EO2 to EO1, SJ did not increase magnitude as much as his matched control, with the exception of the posterior areas in high alpha and low beta (Table 19,. Figure 70-73).

WH: Auditory Discrimination Tone (ADT) task-Baseline

During the ADT task, WH shows significant differences with his matched control in all brain regions and all four frequencies: theta, low and high alpha, and low beta. In theta frequency, WH decreased magnitude in all areas- as compared to initial baseline- whereas his matched control increased it. The same holds for low alpha, as well as in high alpha and low beta with the exception of the left frontal region. In general, comparing ADT to EO1, WH did not increase magnitude as much as her matched control, with the exception of the left frontal area in high alpha and low beta (Table 20, Figure 74-77).

WH: Eyes-Open Two (EO2) -Baseline

During EO2, there are significant differences between WH and her matched control in all brain regions and all four frequencies with the exception of central and right posterior area in theta, left frontal in low alpha, and the frontal areas in low beta. In general, comparing EO2 to EO1 did not increase magnitude as much as her matched control (Table 21, Figure 78-81).

Section IV

DISCUSSION

General Results of all Ten Cases

In nine out of ten TBI participants most of the significant differences from their matched controls occurred during the ADT task and less during EO2. This implies that a cognitive task may be more necessary in order to show differences between a person with attention deficits and one without than a second resting baseline.

Moreover, during the ADT task most of the significant differences between TBIs and controls occur in low alpha and low beta frequencies and less in theta and high alpha. During EO2, most of the significant differences between TBIs and controls, occur in high alpha and low beta frequencies and less in theta and low alpha.

More specifically, during the ADT task, most of the significant differences between TBIs and controls, in theta, low alpha, and high alpha occur in the posterior brain areas, less in the frontal and even less in the central ones. On the other hand, significant differences between the TBI and control groups occur with the same frequency in frontal and posterior brain regions, in low beta (during the ADT task), theta, low and high alpha (during EO2). In low beta (during EO2) significant differences between the two groups occur with the same frequency in posterior and central brain regions (Table 22, 23).

There is at least one consistent significant difference across bands and brain regions between each TBI case and his/her matched control. This consistent difference, across bands and brain regions, probably implies the most pronounced deficit for each case. Moreover, looking at these consistent EEG differences across all TBIs one can arrive to the following conclusions: First, both cases (DH, WH) with sustained attention deficit show their most consistent (across frequencies) difference from their matched control in the right frontal area. Second, all cases (FM, GS and SM) with focused and selective attention deficit show their most consistent (across frequencies) difference from their matched controls in the left frontal area. Third, all cases (DS, MF, RQ, SJ, WH) with divided attention deficit show their most consistent (across frequencies) difference from their matched control in the right posterior region, while some of them (DS, MF, WH) show it also in the frontal areas. I did not find any consistent patterns among those having alternating attentional deficit, probably because cerebellum is mostly involved into this kind of attention and of course not able to be recorded.

The TBI participants with either sustained or divided attention show their EEG difference from their matched controls consistently in the low alpha frequency. However, TBI participants with focused and selective attention deficits show their EEG difference consistently in the low beta frequency.

Comparing either the ADT task or EO2 to EO1, the TBI participants with sustained and selective attention deficits, did not increase as much their low alpha or low beta magnitude as their matched controls. There are mixed results for those with divided attention deficit with a tendency though to show an increased magnitude in comparison to their matched controls.

In general, in all types of attentional deficits the most significant differences are seen in alpha and low beta especially in frontal and especially the posterior areas. What explanation could be given to that? According to a study conducted by Ray et al. (1985), alpha and low beta are more sensitive to attentional demands especially in the parietal areas (Ray et al., 1985). Moreover, the frontal and right posterior areas are the ones who most consistently get activated when a task requires attention (Lewin et al., 1996; Pardo et al., 1991; Haxby et al.1994).

As far as the direction of the magnitude is concerned, TBIs, in general, did not increase as much their low alpha, theta, or low beta magnitude as their matched controls. One hypothesis which could explain this phenomenon comes from a study by Foxe et al.(1999) which shows that
alpha burst occurs in anticipation of a target . The auditory task performed in this study had highly anticipated sounds. The control participants showed a higher burst of alpha probably indicating a higher anticipation of the target. As it is well known, TBI patients suffer from lacerations, contusions and hemorrhages as well as diffuse axonal injury (DAI)- which is the diffuse degeneration of the cerebral white matter- mostly in the frontal and temporal regions after brain injury. Moreover, the shearing, tearing, and stretching of axons may also result in a true disconnection between prefrontal, limbic, and association cortices leading to disturbances of the cognitive and executive processes such as disturbances in memory or planning (Solberg & Mateer, 1988). These disturbances could affect negatively the prediction or anticipation of each new target. The same explanation could be offered to for theta frequency based on the study by Mizuki et al. (1980) which shows that theta burst occurs in the frontal cortex in anticipation of a target.

What about beta activity? A deficiency of beta activity when ADD patients perform a cognitive task is demonstrated by Monastra et al. (1999) and Clarke et al. (2002). Low beta activity is emitted during difficult cognitive tasks (Ramos et al. 1985). TBIs with attentional deficits may have impaired low beta activity which disrupts their ability to perform in difficult cognitive tasks.

Looking more specifically at each type of attention (figures 82-87), the most consistent finding in those with selective attentional deficit is the decreased magnitude in theta activity in the right frontal area. What explanation could be given to that? According to Basar (1999), during selective attention processes, large theta enhancements occur in the frontal areas. It is possible that impairment in theta activity causes selective attentional dysfunction.

Another finding is the increased low beta and high alpha activity in the right posterior area in those with divided attentional deficit. One explanation which could be given to this pattern is overactivation of the right posterior area because of the combination of two opposite tasks activating the same brain region. The area activated during a task which requires divided attention depends on the task. The task was an auditory one (where the temporal area is mostly activated) asking for discrimination of tones (where right hemisphere is mostly activated). However, looking at the screen and following written guidelines is a visual task (occipital area mostly activated). Right posterior area appears to be the brain area where the activity of the two tasks overlap.

But maybe this overactivation is not due to the specific tasks. The right posterior area as well as the frontal areas are the ones who get activated due to attentional demands for any type of task. Maybe increased attentional demand was required for those with divided attentional deficit, using with extreme intensity the right posterior area.

What about the decreased alpha during EO2? Usually task demands attenuate alpha rhythms, and alpha burst appears after performance of tasks (Berger, 1929; Ramos at al., 1985). Probably those with attentional deficits function, even during resting periods, with the same intensity as almost doing a cognitive task.

Although significant differences between TBIs and controls are seen during eyes-open or eyes-closed conditions (Montgomery et al. 1991; Enomoto et al. 1986; Thacher et al 1991; Thacher et al, 1989), specific EEG patterns indicating specific type of attentional deficit are only seen during a cognitive task or in the after tasks baseline. Probably when someone is at rest, he has the liberty to have a diverse range of feelings, thoughts or sensations. But when the person has to push himself to a more active and specific state, like that of a cognitive task, specific modalities have to be used and different types of attention are required in order to perform well in the task. Thus, the specific types of attentional deficit are also easier to be observed.

Why all ten TBIs show any type of attentional deficit regardless of area of injury? In case the lesion has occurred cortically, it should be expected that the local area damaged would affect a specific cognitive function. However, if the lesion has occurred subcortically, then the fibers which connect other brain areas are cut and these areas are affected as well.

It could be possible too that the change of firing in a local area causes change of firing into other areas too. According to Luria, the brain works as a synthetic organ and not just a collection of specialized functions independently the one from the other. Seen it also from a more theoretically cognitive perspective, a deficiency in attention for example can cause some deficiency in memory too, since in order to retain some information the person has first to pay attention to it.

A third explanation to that could be that some brain areas recover faster or slower regardless if they are or not the most immediate and prominent areas of injury. Maybe the need to use more one type of attention than another promotes the increase of synapses in the related brain area, or the lack of motivation or need to use another type of attention may delay the increase in other areas. Personal motivation (or lack of motivation) and environmental enrichment (or deprivation) may contribute in a more prominent way than expected.

Out of any similar uncontrollable and unfortunate situation such as brain injury, that any one of us can find himself accidentally, there is maybe some little hope, a hope based on the personal and environmental contribution which may change to some degree the speed and quality of recovery.

Limitations and strengths

A limitation of this study is the limited number of patients. This means that the results may not generalize to a greater population. The heterogeneity of the sample, in terms of age, gender, attention deficit, duration and area of injury, constitutes theoretically another limitation. However, the consistency of the EEG patterns depending on the type of

attention deficit, regardless if this heterogeneity, constitutes a strong proof of their validity.

Implications

Research implications

In case of replication of this study, it would be worthwhile adding more TBI participants with attention deficits and working with a more homogeneous sample. It would also be interesting statistically analyzing more cognitive tasks and frequencies (delta and high beta). It would also be interesting subtracting the second resting baseline (EO2) from the tasks.

Some types of memory could also be examined like short-term and working memory. According to the current research for example, those with a deficit in working memory (DS, FM) both display their most consistent difference in their left frontal region, by increasing their low alpha magnitude more, when compared with their matched controls.

Finally, the different results of low and high alpha or low and high beta, imply the usefulness of separating the frequencies into smaller units.

Practical implications

Low alpha and low beta could be used as the main frequencies testing for attention deficits. Moreover, the topography (frontal and posterior areas) of the magnitude differences between the TBI participants with attention deficits and their matched controls coincides with the topography on attention deficits, shown by previous research using PET scans. These overlapping findings between EEG and PET scans indicate to some degree the validity of EEG. The current results could motivate experimenters and clinicians to use EEG, which is a less expensive and intrusive mechanism, more frequently than before for the testing of attentional deficits.

Conclusions

The three hypotheses, that the topography, direction and significance of changes from the first baseline to task (as well as of the first baseline to the second baseline) would be different between the TBI and the matched control individuals, or that there would be consistent EEG patterns depending on the type of attention deficit, were supported.

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APPENDICES

Table 1. Types of attention, their representation on the brain and associated tests on attention.

Types of Attention	**Representation on the Brain**	**Test to Measure Specific Type of Attention**
Sustained Attention	Prefrontal and Parietal in Right hemisphere	IVA- scales on Stamina, Consistency and Focus-, PASAT and Digit Span
Selective Attention	Visual: Anterior cingulate cortex, left prefrontal cortex, inferior parietal lobe, Attending to motion: Occipito -temporal cortex modulated by prefrontal, parietal regions Spatial: Right prefrontal and parietal Temporal: Left prefrontal and parietal	IVA- scales on Prudence, Vigilance, and Comprehension-, and Digit Symbol
Divided Atttention	Depends on cognitive task	IVA-scales on Prudence and Speed
Alternating Attention	Prefrontal cortex, cerebellum	IVA- scales on Speed, Balance, Readiness, Consistency,and Focus- and PASAT

Table 2. Psychometric Results

	Psychometric Results														
	IVA results*												Pasat **	Digit Span***	Digit Symbol****
	Auditory results						Visual results								
Ss	Prudence	Consistency	Stamina	Vigilance	Focus	Speed	Prudence	Consisten	Stamina	Vigilance	Focus	Speed			
DH	111	74	90	105	81	86	104	85	71	106	83	84	166	10	7
DS	109	114	93	94	111	50	92	124	96	100	121	83	106	11	9
GS	80	67	122	82	77	128	47	101	100	106	102	111	93	9	8
FM	106	80	112	40	77	76	72	72	96	106	85	76	111	12	6
MF	118	117	92	105	124	58	84	114	94	106	97	80	129	11	10
RM	117	153	0	0	143	38	85	110	88	80	79	88	166	7	7
RQ	109	113	100	83	121	27	106	90	108	105	97	34	67	6	8
SM	37	91	103	86	90	65	38	70	83	105	89	75	97	10	10
SJ	88	61	86	108	71	103	109	88	79	40	97	92	140	12	10
WH	75	40	117	89	71	55	111	58	104	98	65	75	133	13	12

*IVA: (m:100; st.d:15)
**PASAT(m:135; st.d.: 15)
***Digit Span (m: 10; st.d.:2)
****Digit Symbol (m:10;st.d.:2)

Table 3. DH_ADT

ADT_DH	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-1.01671	-1.08172	-0.83101	-2.61375	-2.09378	-0.56175	-0.71032	-0.92386	-1.66667	-2.81802	-1.51812	-0.14392	-0.24605	-1.35098	-3.17085	-3.45874	-4.06222	-2.86444	-1.85237	-4.2572
p-value MV	0.309289	0.279379	0.405966	0.008956	0.036279	0.574285	0.477506	0.355557	0.09558	0.004832	0.128983	0.885564	0.80564	0.176703	0.00152	**0.000543**	**4.86E-05**	**0.004177**	0.063973	**2.07E-05**
p-value Le	**0.003233**	**0.005974**	**0.002799**	**0.000108**	**0.006223**	**0.00301**	**0.001588**	0.000373	**3E-08**	**3.58E-06**	0.019612	0.059255	0.150416	0.513855	**0.00045**	0.130033	0.300515	0.344688	0.284534	0.116862

Table 4. DH_EO2

EO2_DH	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-0.99753	-1.09608	-0.48383	-1.52914	-1.11698	-0.01792	-0.66601	-1.87856	-3.08215	-1.40369	-4.41122	-2.75364	-6.37039	-8.06974	-6.57945	-5.41768	-3.15682	-3.11202	-4.12447	-1.40668
p-value MV	0.31851	0.273045	0.62851	0.126231	0.264002	0.985703	0.505404	0.060305	0.002055	0.16041	1.03E-05	0.005894	1.89E-10	7.04E-16	4.72E-11	**6.04E-08**	**0.001595**	**0.001858**	**3.72E-05**	0.159522
p-value Le	0.013164	0.405103	0.392013	0.29352	0.680056	0.348354	**0.006792**	0.007355	**0.000304**	**5.76E-07**	**8.4E-05**	**3.04E-05**	**1.78E-15**	**9.54E-12**	**8.51E-11**	0.422482	0.064334	0.614276	0.7328	0.329891

Table 5. DS_ADT

ADT_DS	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-0.66292	-0.25437	-0.47791	-1.68813	-0.54729	-1.66501	-2.22772	-2.37416	-1.87312	-2.03501	-0.53959	-0.62438	-1.78061	-1.61104	-1.74207	-3.67688	-5.41128	-2.62082	-1.98874	-1.24874
p-value MV	0.507384	0.799207	0.632712	0.091387	0.584181	0.095911	0.025899	0.017589	0.061052	0.04185	0.589483	0.53238	0.074975	0.10717	0.081496	0.000236	**6.26E-08**	0.008772	0.04673	0.211759
p-value Le	0.311329	0.799742	0.835764	0.743088	0.184649	**1.61E-05**	0.602163	0.098082	0.094882	**1.71E-06**	**0.000175**	0.081817	0.006315	**1.66E-08**	**2.05E-09**	**5.44E-07**	0.006133	**1.43E-06**	**1.34E-06**	**0.000264**

Table 6. DS_EO2

ADT_DS	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-0.93348	-3.64759	-1.51623	-1.77524	-1.00362	-1.15471	-0.82556	-1.8022	-0.9065	-1.3004	-0.42628	-0.50181	-1.35975	-1.10075	-0.70686	-5.05053	-6.20522	-0.67448	-1.56479	-0.75002
p-value MV	0.35057	**0.000265**	0.129462	0.075859	0.31556	0.248209	0.409052	0.071513	0.364672	0.193466	0.669905	0.615799	0.173909	0.271004	0.479655	4.41E-07	5.46E-10	0.500008	0.117632	0.453242
p-value Le	**0.003922**	0.014391	0.815778	0.090917	0.213595	0.113416	0.406055	0.282148	0.281132	0.550972	0.010579	0.035637	0.503628	0.230193	0.748543	**2.37E-08**	**2.09E-05**	**0.003512**	**1.39E-06**	0.005469

Table 7. FM_ADT

ADT_FM	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-1.34096	-0.87472	-1.87321	-2.92947	-1.3946	-0.0949	-2.00525	-0.83758	-4.02288	-4.72016	-2.5375	-0.48275	-1.76181	-1.40697	-0.01238	-7.69501	-9.50635	-8.87093	-8.9287	-9.22166
p-value MV	0.179933	0.381728	0.061039	**0.003395**	0.163138	0.924395	0.044936	0.402265	**5.75E-05**	**2.36E-06**	0.011165	0.629276	0.078102	0.159436	0.990124	**1.41E-14**	**1.97E-21**	**7.25E-19**	**4.31E-19**	2.93E-20
p-value Le	0.073345	0.10384	0.013018	0.133562	0.762147	0.324724	0.07157	0.029237	0.037024	0.003441	0.027162	0.649477	0.276805	**0.000107**	0.164014	0.468553	0.000618	0.23909	0.010569	**0.000186**

Table 8. FM_EO2

EO2_FM	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-1.21929	-1.43162	-0.95548	-1.00052	-2.87931	-0.40857	-1.73403	-0.02895	-1.90132	-3.18172	-0.5823	-2.00427	-1.63107	-1.57959	-2.20371	-1.07774	-1.68898	-0.21555	-1.05843	-3.09165
p-value MV	0.222736	0.152254	0.339336	0.31706	0.003985	0.682853	0.082913	0.976901	0.05726	0.001464	0.560366	0.045041	0.102875	0.1142	0.027545	0.281152	0.091224	0.829342	0.289862	**0.00199**
p-value Le	**0.000113**	**0.00019**	**7.68E-06**	**1.54E-06**	**0.000262**	0.596768	**0.000698**	**1.04E-05**	**1.36E-12**	**5.28E-11**	0.079112	**0.00399**	0.85295	0.491317	0.058898	0.478785	0.846846	0.674443	0.34904	0.065889

Table 9. GS_ADT

ADT_GS	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-0.53535	-0.18836	-2.13149	-4.71405	-3.13775	-0.3916	-0.65432	-2.11166	-0.91208	-0.70389	-0.57005	-2.27525	-0.50065	-4.54552	-3.3509	-0.61466	-2.44874	-1.99765	-1.96295	-4.00026
p-value MV	0.592408	0.850591	0.033049	**2.43E-06**	**0.001703**	0.695353	0.512906	0.034716	0.361727	0.481502	0.568643	0.022891	0.616617	**5.48E-06**	**0.000805**	0.538778	0.014336	0.045755	0.049652	**6.33E-05**
p-value Le	**0.005296**	0.138168	**0.003372**	0.59505	0.462075	0.400724	0.342607	0.187746	0.018435	0.918099	**0.000573**	0.009201	**0.004963**	0.000407	0.007518	**2.07E-07**	**0.005593**	**0.000127**	0.065055	0.322869

Table 10. GS_EO2

EO2-GS	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-1.96888	-0.50103	-0.64977	-1.03728	-0.32097	-1.7262	-1.65574	-0.32097	-2.84959	-2.61082	-0.97465	-0.23877	-1.75751	-1.31911	-0.66151	-7.07311	-7.57802	-2.48164	-8.07513	-5.96535
p-value MV	0.048967	0.616352	0.515841	0.299606	0.748233	0.084312	0.097775	0.748234	0.004378	0.009032	0.329732	0.811283	0.078831	0.187133	0.508284	**1.51E-12**	3.51E-14	0.013078	**6.74E-16**	**2.44E-09**
p-value Le	0.509731	0.301896	0.279398	0.538146	0.576237	**1.96E-05**	0.377162	0.461076	**4.23E-05**	**1.61E-06**	**0.002199**	0.616897	0.010996	0.024764	0.92841	0.00094	**0.000122**	**9.94E-05**	0.565919	0.273745

Table 11. MF_ADT

ADT_MF	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R.
Z-MW	-0.13992	-0.17852	-0.06272	-1.13384	-1.46194	-1.59705	-0.03377	-2.1374	-2.50409	-3.44017	-0.97946	-0.21712	-2.41725	-2.01197	-4.25071	-4.05769	-2.91903	-0.70925	-0.7382	-4.21209
p-value MV	0.888722	0.858314	0.949987	0.256863	0.143759	0.110254	0.973057	0.032565	0.012277	0.000581	0.327353	0.828115	0.015638	0.044223	**2.13E-05**	**4.96E-05**	0.003511	0.478168	0.460392	**2.53E-05**
p-value Le	**0.000103**	**0.001536**	**0.001289**	**0.000222**	**0.005446**	**1.88E-07**	**6.05E-05**	**1.6E-05**	**1.09E-08**	**6.83E-08**	**0.002331**	**0.00285**	0.022393	0.010634	0.001343	0.003351	**2.42E-06**	**0.000396**	**0.000227**	0.002423

Table 12. MF_EO2

EO2_MF	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R.
Z-MW	-2.48985	-1.6447	-1.27988	-0.32529	-0.67794	-2.57498	-4.01598	-1.48053	-0.03344	-1.43797	-3.45661	-4.61489	-6.67304	-6.26871	-5.87957	-5.40229	-1.1978	-2.34392	-5.19859	-2.86074
p-value MV	0.01278	0.100032	0.200586	0.74496	0.497807	0.010024	**5.92E-05**	0.138732	0.973323	0.150442	0.000547	3.93E-06	2.51E-11	3.64E-10	4.11E-09	6.58E-08	0.230994	0.019082	2.01E-07	0.004227
p-value Le	0.402719	0.171695	0.438198	0.562187	0.733055	**2.39E-05**	0.287349	0.25657	0.765045	0.923668	**8.64E-05**	**1.94E-11**	**1.49E-12**	**3.6E-07**	**2.29E-07**	**1.86E-05**	**4.64E-07**	**3.63E-06**	**1.11E-05**	**3.81E-09**

Table 13. RM_ADT

ADT_RM	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R.
Z-MW	-2.12256	-3.45326	-0.81383	-0.67086	-3.60723	-0.54988	-0.35192	-2.84841	-3.53027	-5.75177	-0.68186	-0.022	-0.51689	-1.17676	-2.51848	-3.07936	-5.44384	-0.09898	-2.05657	-0.98979
p-value MV	0.033791	**0.000554**	0.415742	0.502312	**0.000309**	0.5824	0.724895	0.004394	0.000415	8.83E-09	0.495326	0.982451	0.605231	0.239293	0.011786	**0.002074**	**5.21E-08**	0.921154	0.039727	0.322278
p-value Le	0.171185	0.207439	0.473335	0.062755	0.079639	0.009299	0.712455	**0.000116**	**1.24E-06**	**3.21E-06**	0.418156	0.512345	0.044626	**0.000155**	**3.55E-05**	0.17098	0.955498	0.12034	**0.002258**	**0.001006**

Table 14. RM_EO2

EO2_RM	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R.
Z-MW	-1.60819	-0.49799	-0.47515	-0.23757	-0.18275	-1.57621	-3.19354	-0.09137	-1.02339	-0.86805	-0.13706	-1.28382	-1.07822	-1.59448	-3.43111	-0.02741	-4.67836	-1.54422	-4.82455	-2.57675
p-value MV	0.107794	0.618492	0.634684	0.812213	0.854996	0.114977	**0.001405**	0.927195	0.306124	0.385364	0.890982	0.199205	0.280937	0.110829	0.000601	0.978131	**2.89E-06**	0.122534	1.4E-06	0.009973
p-value Le	0.507465	0.153484	0.623035	**0.001478**	**4.43E-06**	0.046434	0.128505	**3.03E-06**	**4.66E-10**	**2.21E-14**	0.177055	0.396173	**0.001363**	**2.72E-06**	**1.57E-07**	0.560507	0.04643	0.022368	**0.00095**	**2.61E-06**

Table 15. RQ_ADT

ADT_RQ	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.F	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L	L.B.Post.R.
Z-MW	-1.70266	-0.62813	-0.05136	-0.67553	-0.19357	-1.2049	-1.33132	-1.76587	-3.18803	-0.94417	-2.74563	-2.51647	-2.75348	-1.05478	-2.72978	-3.58309	-0.4464	-1.38662	-0.53331	-3.15643
p-value M\	0.088632	0.529921	0.959042	0.499339	0.84651	0.228243	0.183082	0.077418	0.001432	0.345085	**0.006039**	0.011854	0.005897	0.291526	0.006338	**0.00034**	0.655306	0.165559	0.593817	**0.001597**
p-value Le\	0.10838	0.050383	0.112154	0.100457	**0.000431**	**0.000989**	**4.51E-06**	**0.002309**	**4.68E-05**	**5.26E-09**	0.053514	**1.13E-06**	**9.98E-08**	**5.89E-05**	**1.32E-05**	0.399681	0.140493	0.044345	0.870104	0.753152

Table 16. RQ_EO2

EO2_RQ	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.F	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L	L.B.Post.R.
Z-MW	-2.55277	-0.32853	-1.58909	-2.59171	-0.06084	-1.12673	-1.64751	-1.28733	-3.24391	-1.65724	-1.84219	-3.06382	-4.0275	-1.80325	-1.86165	-0.04624	-2.31915	-2.68418	-1.75944	-1.40414
p-value M\	0.010687	0.742512	0.11204	0.00955	0.951488	0.259856	0.099453	0.197978	0.001179	0.097472	0.065447	0.002185	**5.64E-05**	0.071349	0.062652	0.963121	0.020387	**0.007271**	0.078503	0.160276
p-value Le\	0.022145	**5.4E-05**	**0.00097**	**0.005526**	**0.003862**	0.036825	**4.88E-11**	**2.22E-09**	**7.86E-11**	**3.11E-10**	0.358871	**0.000148**	0.031159	0.014572	**0.000296**	0.086305	0.225282	0.399329	0.472768	0.986248

Table 17. SM_ADT

ADT_SM	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.F	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L	L.B.Post.R.
Z-MW	-1.73486	-0.41178	-0.41853	-2.8352	-2.8892	-3.78706	-1.80238	-2.36941	-5.13034	-5.1641	-0.81681	-2.38967	-2.40316	-2.19391	-3.63176	-1.74837	-3.30775	-2.0184	-6.12268	-4.4688
p-value M\	0.082765	0.680502	0.675562	**0.00458**	**0.003862**	**0.000152**	0.071486	0.017817	**2.89E-07**	**2.42E-07**	0.414035	0.016864	0.016254	0.028242	**0.000281**	0.080399	0.00094	0.04355	**9.2E-10**	7.87E-06
p-value Le\	0.733945	0.191501	0.70649	0.487833	0.511399	0.018599	**3.89E-05**	0.18558	0.021134	0.001573	**0.000256**	**7.85E-05**	0.065797	0.736406	0.199833	**1.41E-06**	**1.8E-05**	**2.31E-09**	0.061178	**6.97E-07**

Table 18. SM_EO2

EO2_SM	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.F	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L	L.B.Post.R.
Z-MW	-0.80234	-0.70508	-0.466	-2.2976	-3.33902	-1.85592	-1.41827	-1.39801	-3.95496	-4.6479	-2.99459	-0.57542	-1.47095	-2.79198	-0.53084	-0.11346	-5.59611	-2.20035	-1.98964	-1.72219
p-value M\	0.422358	0.480757	0.641213	0.021585	**0.000841**	0.063465	0.156111	0.162109	**7.65E-05**	**3.35E-06**	0.002748	0.56501	0.141305	**0.005239**	0.595529	0.909664	2.19E-08	0.027782	0.046631	0.085034
p-value Le\	**0.003028**	0.056931	0.09831	0.824449	0.780778	0.596488	**0.005921**	0.171546	0.005864	0.001786	**2.18E-05**	**6.11E-11**	0.014319	0.033386	0.015042	**1.72E-13**	**8.66E-15**	**8.26E-09**	**0.004702**	**1.07E-08**

Table 19. SJ_ADT

ADT_SJ	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-2.12256	-3.45326	-0.81383	-0.67086	-3.60723	-0.54988	-0.35192	-2.84841	-3.53027	-5.75177	-0.68186	-0.022	-0.51689	-1.17676	-2.51848	-3.07936	-5.44384	-0.09898	-2.05657	-0.98979
p-value MV	0.033791	**0.000554**	0.415742	0.502312	**0.000309**	0.5824	0.724895	0.004394	0.000415	8.83E-09	0.495326	0.982451	0.605231	0.239293	0.011786	**0.002074**	**5.21E-08**	0.921154	0.039727	0.322278
p-value Le	0.171185	0.207439	0.473335	0.062755	0.079639	**0.009299**	0.712455	**0.000116**	**1.24E-06**	**3.21E-06**	0.418156	0.512345	0.044626	**0.000155**	**3.55E-05**	0.17098	0.955498	0.12034	**0.002258**	**0.001006**

Table 20. SJ_EO2

EO2_SJ	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-5.35517	-7.49945	-3.98425	-0.04467	-1.28434	-1.18104	-2.1443	-1.195	-0.90462	-0.8795	-0.19265	-3.64085	-2.79764	-4.8805	-4.24671	-2.26156	-8.34823	-1.544	-5.09828	-0.16752
p-value MV	8.55E-08	6.41E-14	6.77E-05	0.964368	0.199022	0.237587	0.032009	0.232088	0.365665	0.379133	0.847232	**0.000272**	0.005148	1.06E-06	2.17E-05	0.023724	**6.93E-17**	0.122588	3.43E-07	0.866958
p-value Le	**8.67E-06**	**6.04E-07**	**9.05E-14**	**0**	**0**	0.047285	0.105877	**3.83E-08**	**0**	**0**	**0.008799**	0.003319	**0.004175**	**1.51E-06**	**0.00021**	0.751626	0.199059	**0.000404**	**1.99E-06**	**6.07E-11**

Table 21. WH_ADT

ADT_WH	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-4.78416	-5.09758	-5.12122	-3.12241	-4.12772	-3.45358	-3.08103	-8.26729	-6.84208	-7.53991	-3.21704	-0.71556	-6.13246	-3.29391	-6.88351	-7.04315	-0.62685	-6.20341	-1.24778	-5.14488
p-value MV	1.72E-06	3.44E-07	3.04E-07	0.001794	3.66E-05	0.000553	0.002063	1.37E-16	7.81E-12	4.7E-14	0.001295	0.474266	**8.65E-10**	**0.000988**	5.84E-12	**1.88E-12**	0.530759	**5.53E-10**	0.212111	2.68E-07
p-value Le	**0.000365**	**0.000149**	**2.21E-05**	**4.21E-05**	**0.000381**	**0.009167**	**8.39E-06**	**1.51E-07**	**2.86E-11**	**2.51E-13**	0.325727	**4.44E-06**	0.023049	0.069107	**0.00026**	0.64645	**5.49E-06**	0.00113	**0.000452**	**4.27E-05**

Table 22. WH_EO2

EO2_WH	Frequency/Brain area																		
Test	T.Fr.L.	T.Fr.R.	T.Cen	T.Post.L.	T.Post.R.	L.A.Fr.L.	L.A.Fr.R.	L.A.Cen	L.A.Post.L.	L.A.Post.R	H.A.Fr.L.	H.A.Fr.R.	H.A.Cen	H.A.Post.L	H.A.Post.R	L.B.Fr.L.	L.B.Fr.R.	L.B.Cen	L.B.Post.L.	L.B.Post.R
Z-MW	-2.32185	-4.77643	-3.35677	-0.3982	-1.66396	-3.53762	-3.16441	-7.90043	-7.36949	-7.47338	-4.03391	-3.68765	-7.53878	-7.0925	-7.17714	-1.72552	-0.98683	-5.59974	-2.34108	-0.0327
p-value MV	0.020241	1.78E-06	0.000789	0.690485	0.096121	0.000404	**0.001554**	**2.78E-15**	**1.71E-13**	**7.82E-14**	**5.49E-05**	0.000226	4.74E-14	1.32E-12	7.12E-13	0.084434	0.323725	**2.15E-08**	0.019228	0.973912
p-value Le	**5.94E-06**	**0.00411**	0.012706	**0.002264**	0.766046	0.011553	0.801758	0.746224	0.048388	0.002492	0.655519	**7.43E-07**	**1.28E-08**	**1.25E-07**	**1.12E-07**	0.012691	0.209085	0.013129	0.207912	**0.000188**

Table 23. Combined results of TBIs_ADT

Ss	ADT Gender/Age	Area of injury	Type of problem in attention/memory	Duration of Injury	Theta	Low Alpha	High Alpha	Low Beta
DH	F/46	Fr., R.hem.	Sustained attention	15	All	All	R.post	Fr., Cen., R.post
DS	F/38	L.hem., Fr., Occ.	Working mem.,Divided attention	23		L.fr., R.post.	L.Fr., Cen., Post	All
FM	M/23	L.fr., L.Basal ganglia	Working mem.,Focused/Selective attention	4	R.post.	Post.	L.post.	All
GS	M/23	L.hem.	Short-term mem., Focused/selective attention, Impulsivity	6	L.fr.,Cen.,Post		L.fr., Cen., Post.	Fr., Cen., R.post
MF	F/46	L.hem.	Divided attention	2	All	All	Fr., R.post	All
RM	M/32	R.hem.,R.post., L.fr.	Focused/Selective attention	14	L.fr., R.post	Cen.,Post.	Post.	Fr.,Post.
RQ	F/20	R.Post., L.thalamus	Working and short-term mem., Divided attention	6	R.post.	All	All	L.fr., R.post
SM	M/40	L.temp., L.hippoc.	Focused/Selective attention	8	Post.	Fr., Post.	Fr., R.post	All
SJ	M/28	Fr., Cen., R.hem.	Working mem., Divided attention	2	R.fr., R.post.	L.fr., Cen.,Post.	Cen., Post	Fr., Post.
WH	F/46	L.hem.	Sustained/Alternating/Divided attention, impulsivity	6	All	All	All	All

Table 24. Combined results of TBIs_EO2

Ss	EO2 Gender/Age	Area of injury	Type of problem in attention/memory	Time of Injury	Theta	Low Alpha	High Alpha	Low Beta
DH	F/46	Fr., R.hem.	Sustained attention	15		R.fr., Post.	All	Fr.,Cen., L.post.
DS	F/38	L.hem., Fr., Occ.	Working mem.,Divided attention	23	Fr.			Fr.,Cen., L.post.
FM	M/23	L.fr., L.Basal ganglia	Working mem.,Focused/Selective attention	4		L.fr., Ce., R.post	L.fr.	R.post
GS	M/23	L.hem.	Short-term mem., Focused/selective attention, Impulsivity	6		L.fr., Post	L.fr.	All
MF	F/46	L.hem.	Divided attention	2		Fr.	All	All
RM	M/32	R.hem.,R.post., L.fr.	Focused/Selective attention	14	Post.	Cen., Post.	Cen., Post.	R.fr., Post.
RQ	F/20	R.Post., L.thalamus	Working and short-term mem., Divided attention	6	R.fr., Cen., Post.	R.fr., Cen., Post.	R.fr., Cen.,R.post.	Cen.
SM	M/40	L.temp., L.hippoc.	Focused/Selective attention	8	Fr., R.post.	R.fr., Post.	Fr., L.post.	All
SJ	M/28	Fr., Cen., R.hem.	Working mem., Divided attention	2	All	Cen., Post.	R.fr.,Post.	R.fr., Cen., Post
WH	F/46	L.hem.	Sustained/Alternating/Divided attention, impulsivity	6	Fr., L. post	R.fr., Cen., Post.	All	Cen., R.post

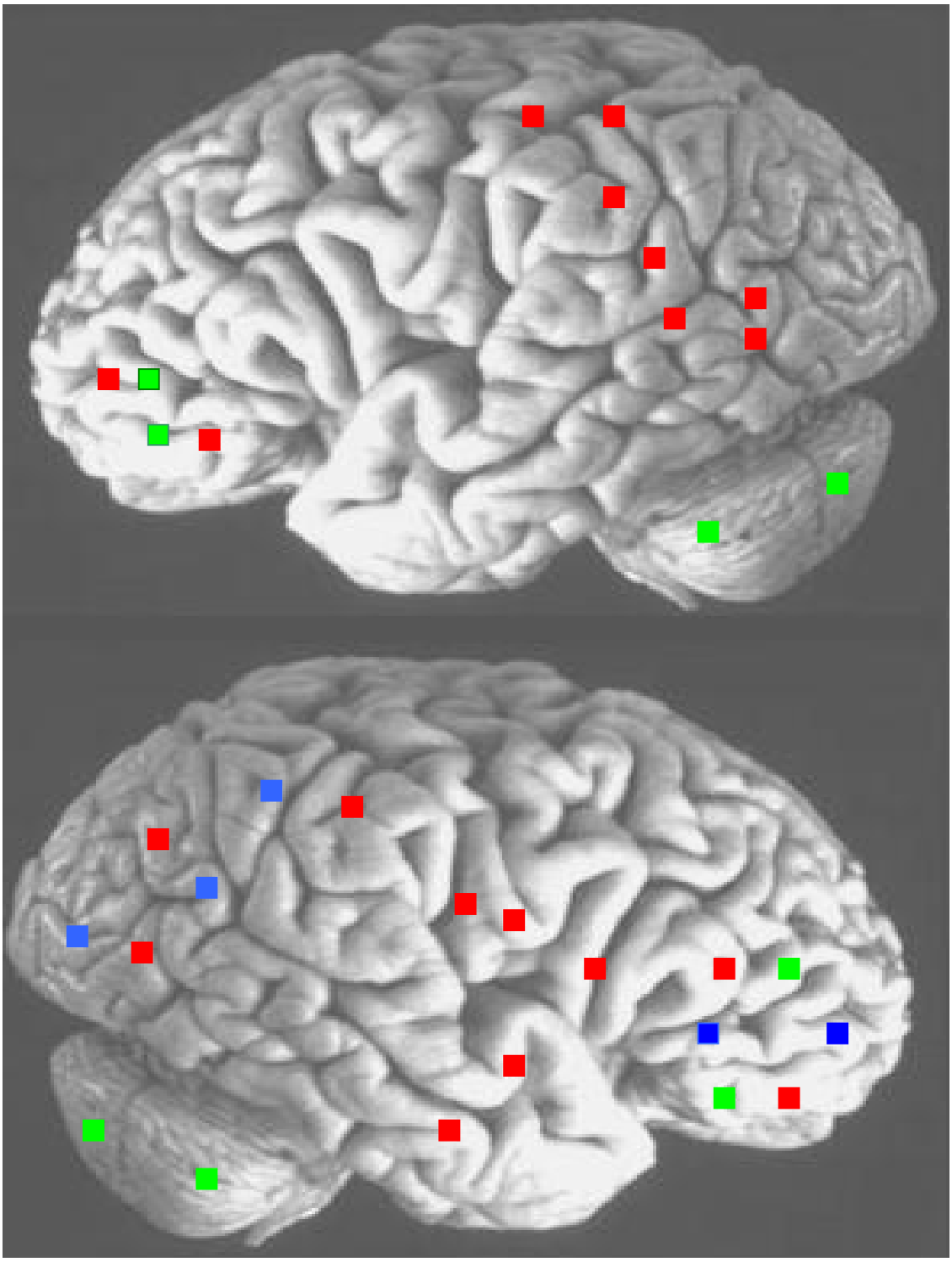


Figure 1. Types of attention represented on two hemispheres. a)Upper image: Left hemisphere; b)Lower image: Right hemisphere

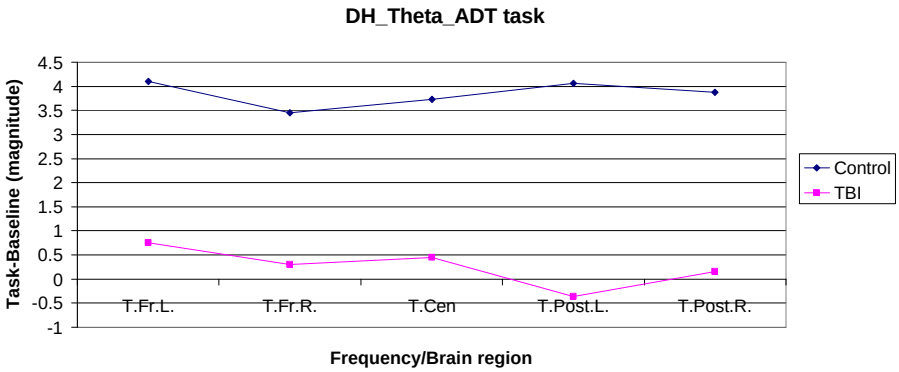


Figure 2. DH_ADT_Theta

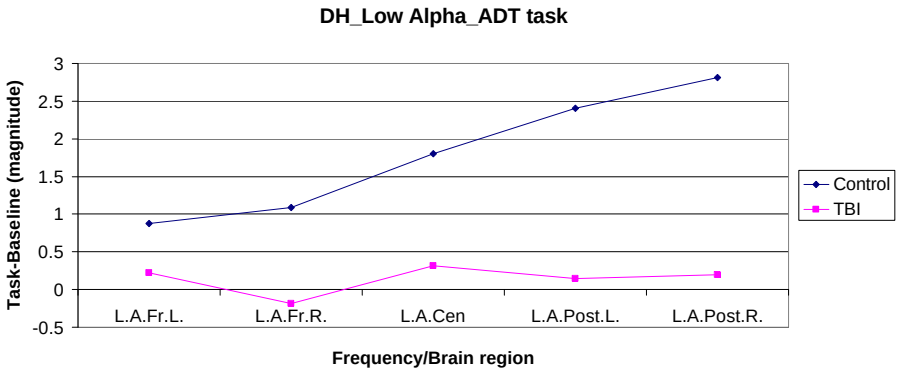


Figure 3. DH_ADT_Low Alpha

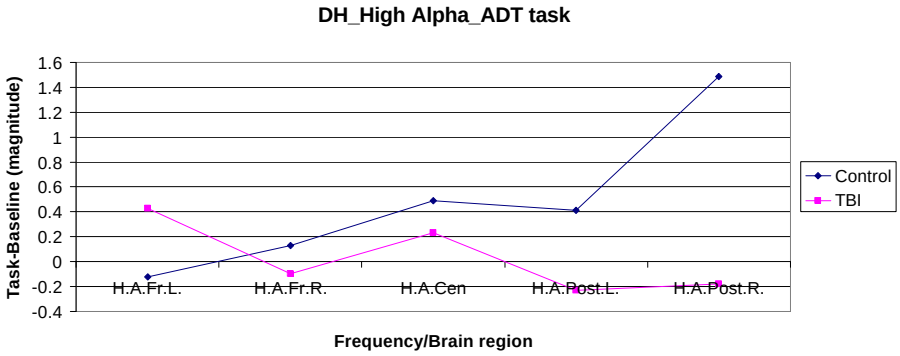


Figure 4. DH_ADT_High Alpha

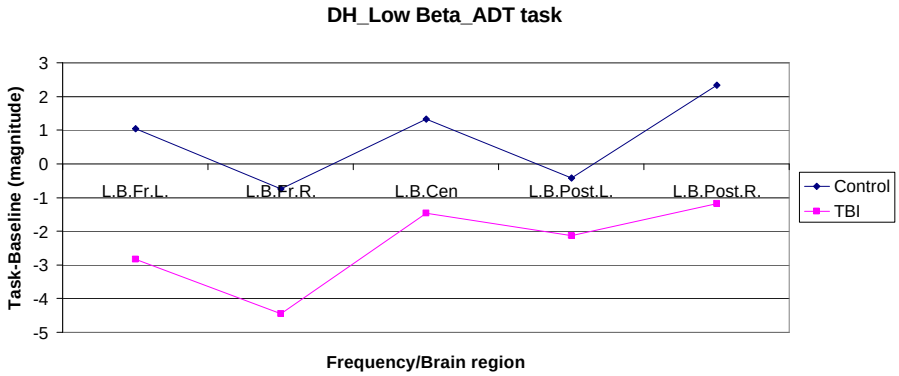


Figure 5. DH_ADT_Low Beta

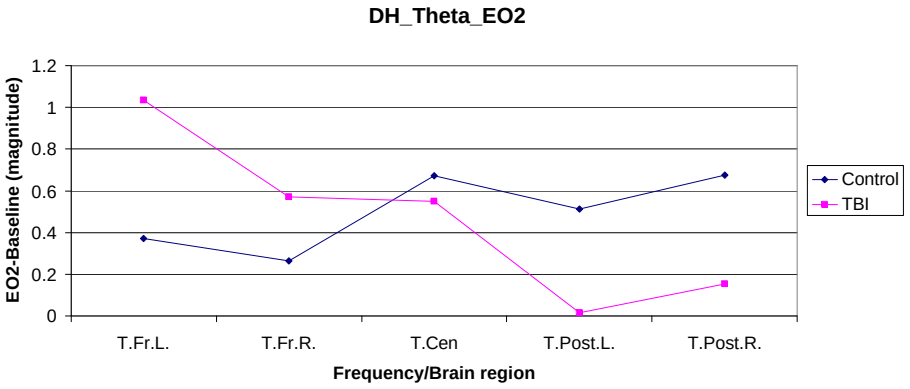


Figure 6. DH_EO2_Theta

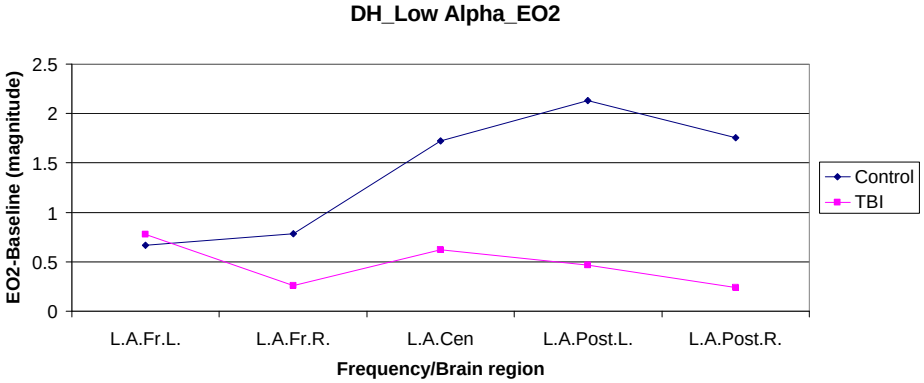


Figure 7. DH_EO2_Low Alpha

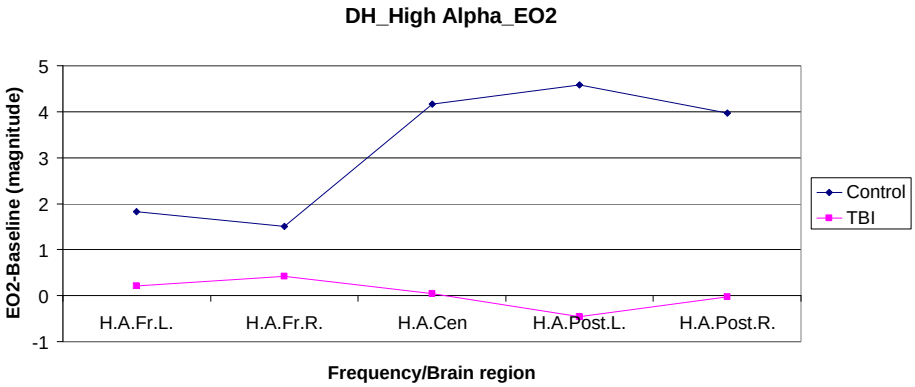


Figure 8. DH_EO2_High Alpha

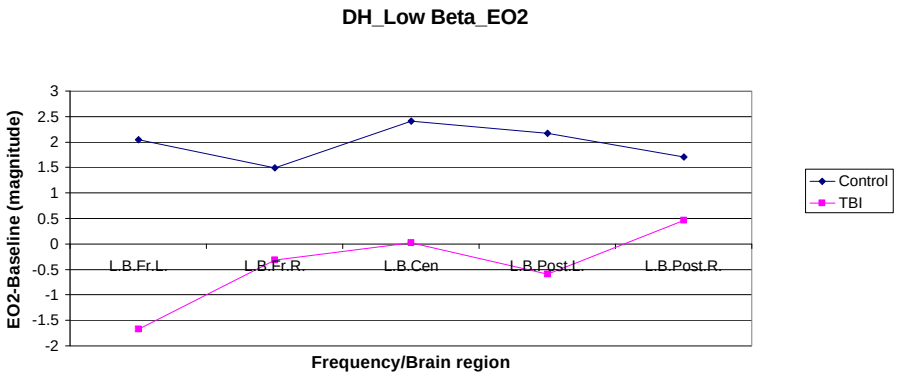


Figure 9. DH_EO2_Low Beta

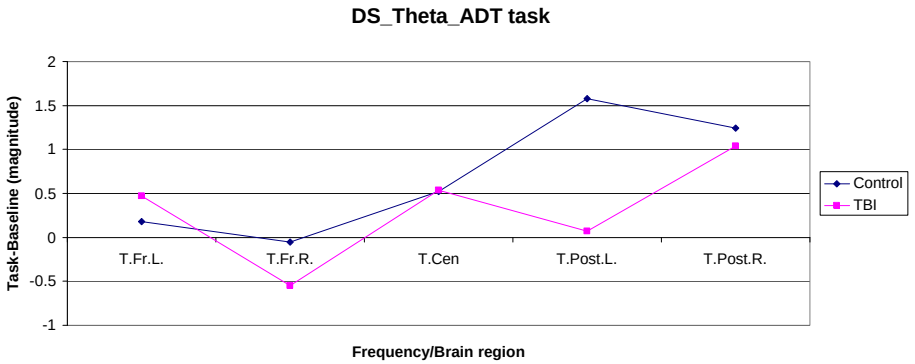


Figure 10. DS_ADT_Theta

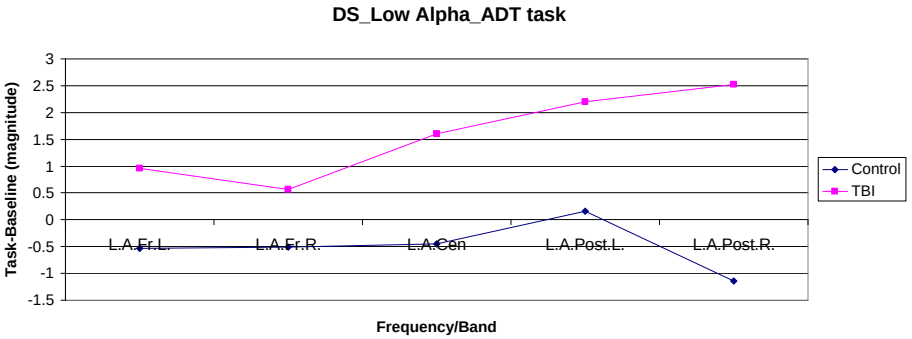


Figure 11. DS_ADT_Low Alpha

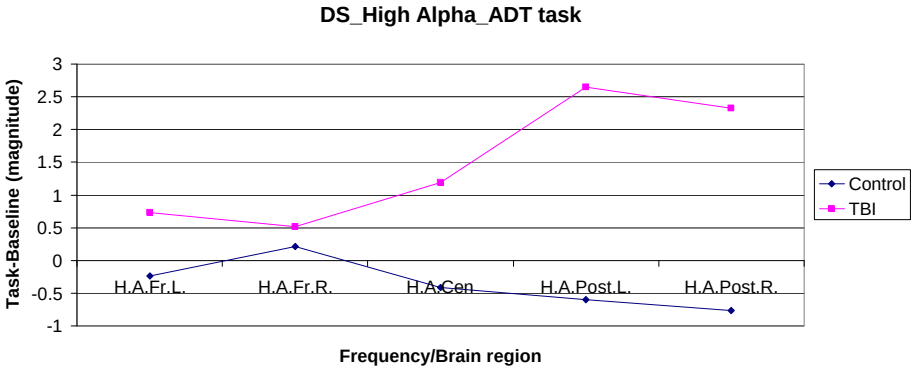


Figure 12. DS_ADT_High Alpha

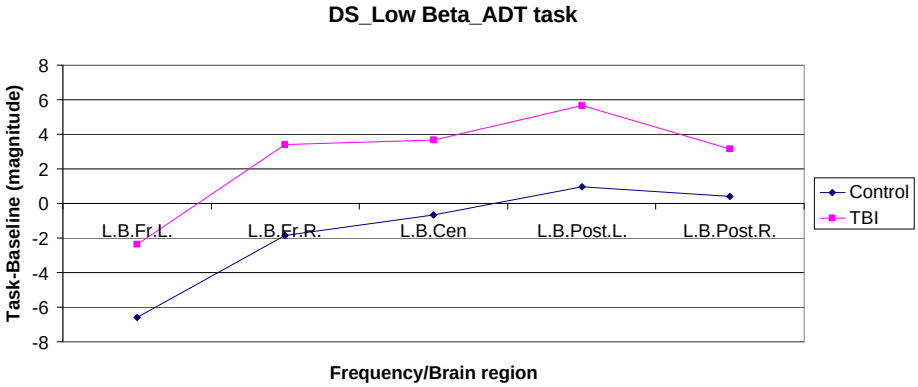


Figure 13. DS_ADT_Low Beta

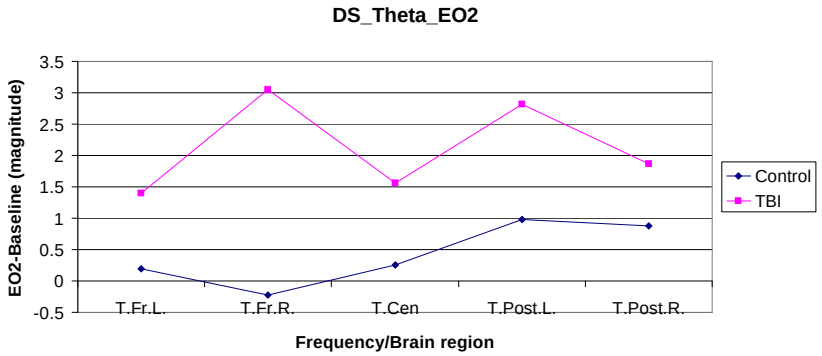


Figure 14. DS_EO2_Theta

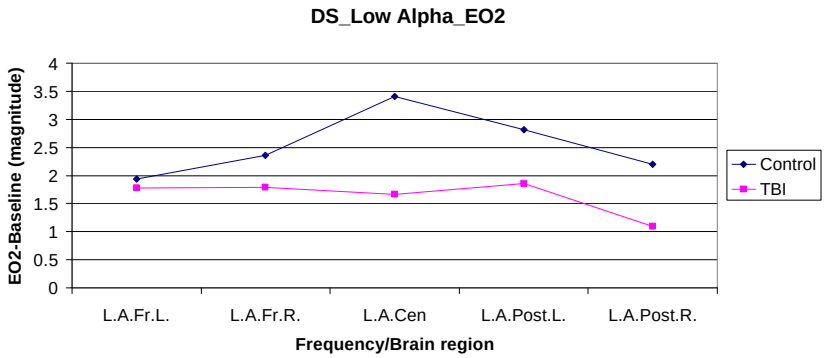


Figure 15. DS_EO2_Low Alpha

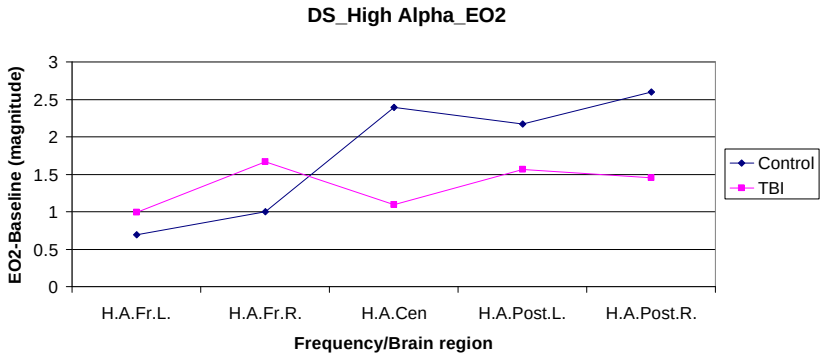


Figure 16. DS_EO2_High Alpha

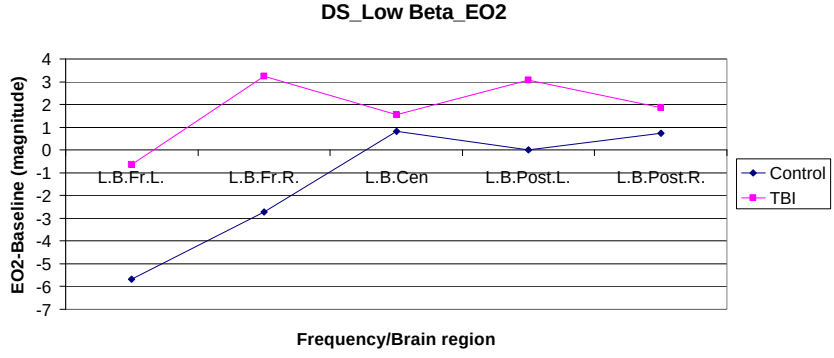


Figure 17. DS_EO2_Low Beta

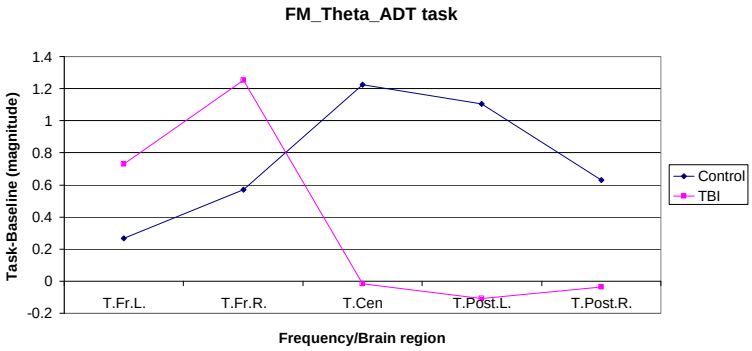


Figure 18. FM_ADT_Theta

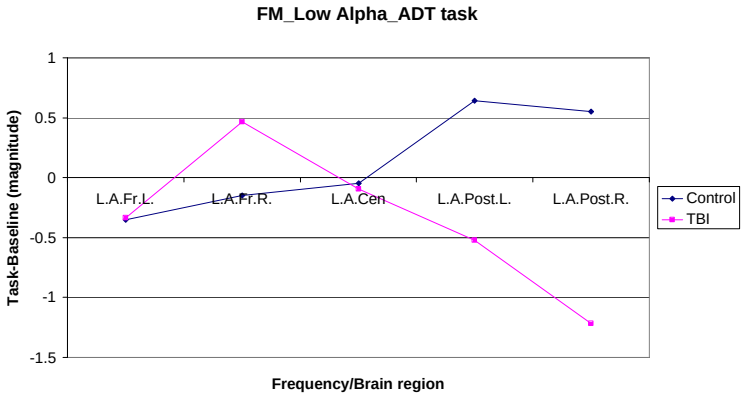


Figure 19. FM_ADT_Low Alpha

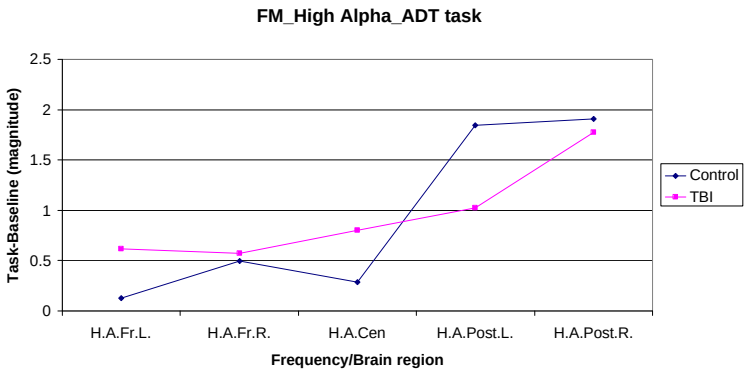


Figure 20. FM_ADT_High Alpha

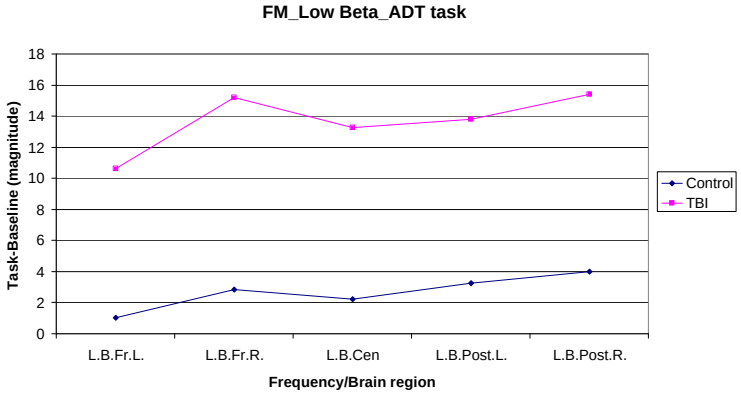


Figure 21. FM_ADT_Low Beta

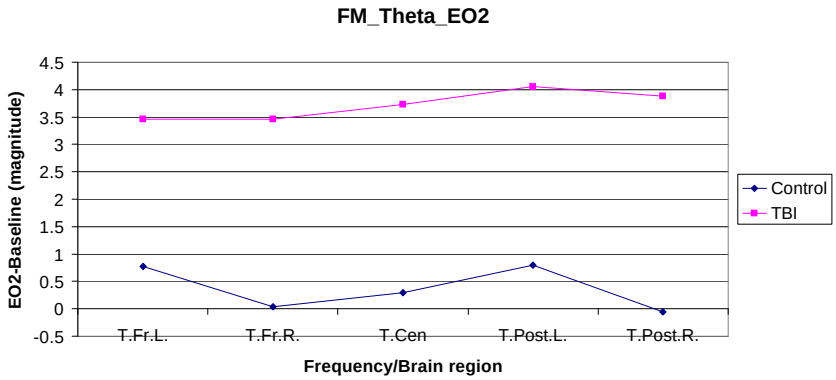


Figure 22. FM_EO2_Theta

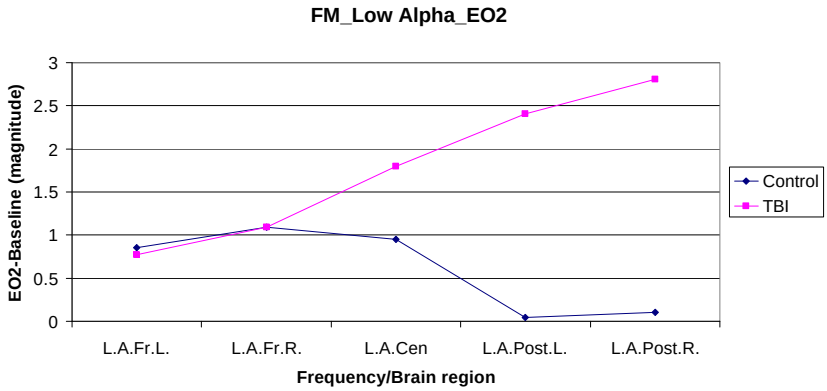


Figure 23. FM_EO2_Low Alpha

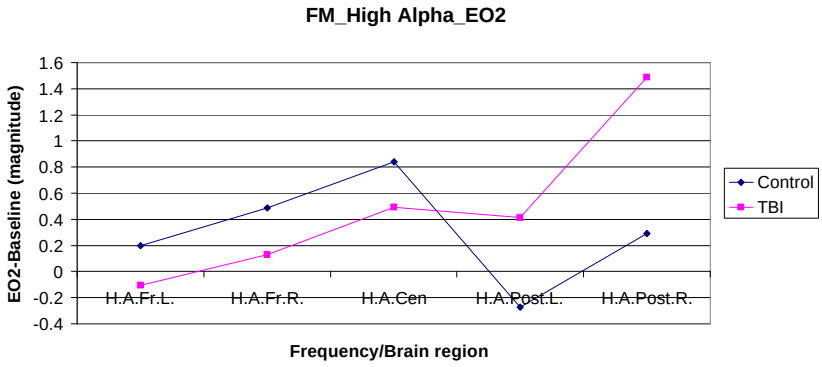


Figure 24. FM_EO2_High Alpha

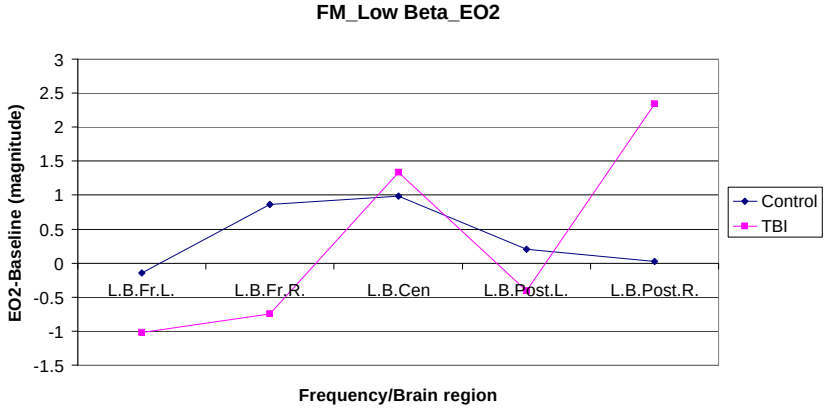


Figure 25. FM_EO2_Low Beta

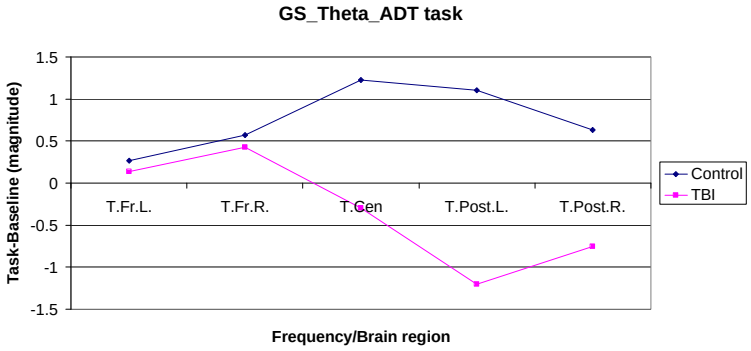


Figure 26. GS_ADT_Theta

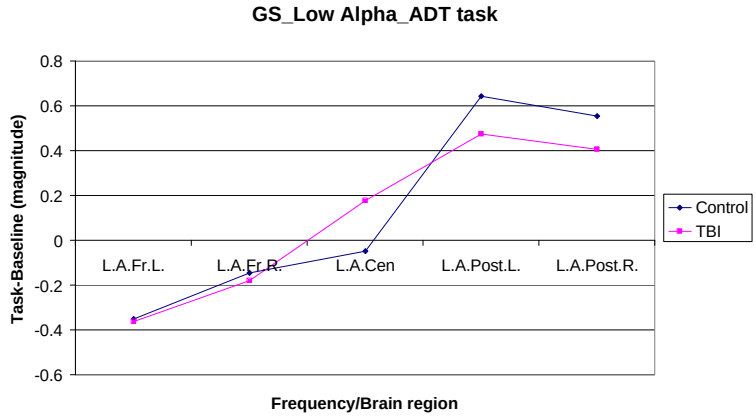


Figure 27. GS_ADT_Low Alpha

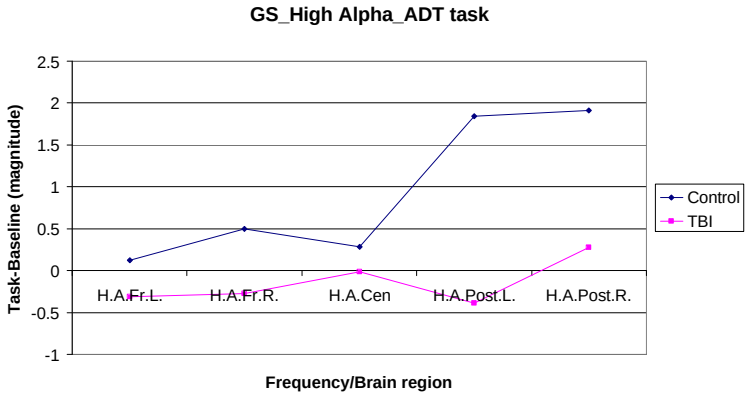


Figure 28. GS_ ADT_High Alpha

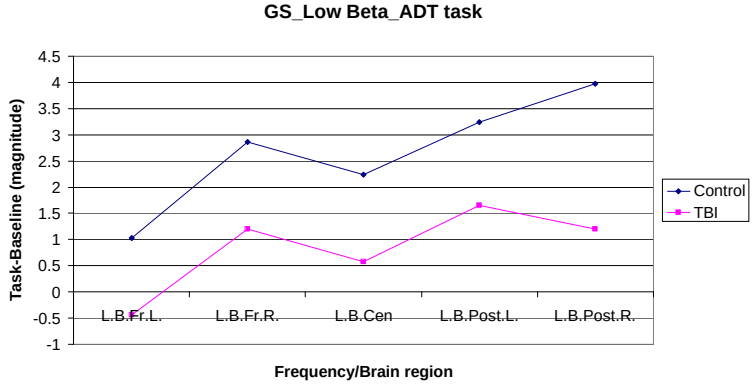


Figure 29. GS_ ADT_Low Beta

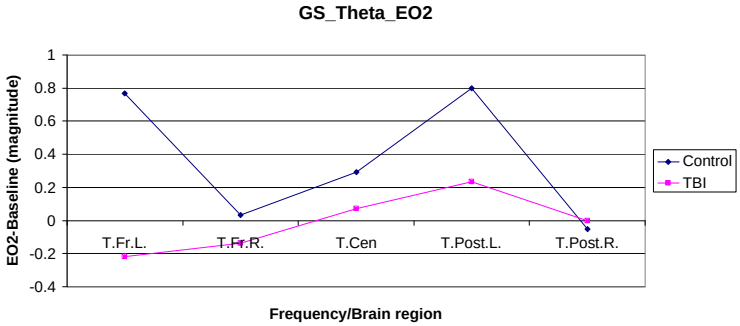


Figure 30. GS_EO2_Theta

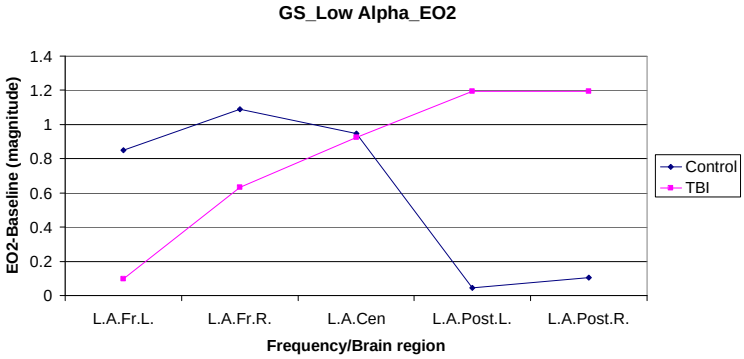


Figure 31. GS_EO2_Low Alpha

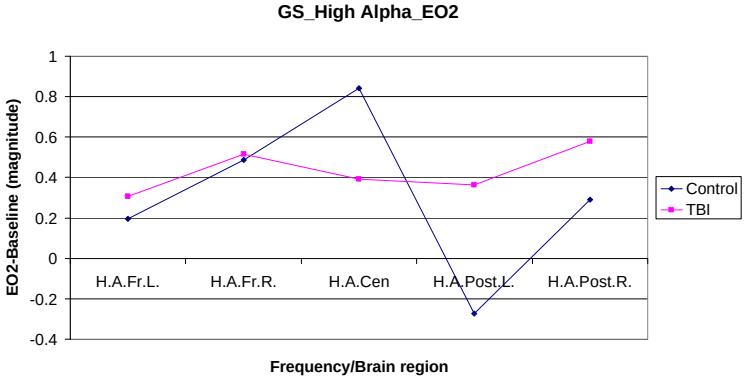


Figure 32. GS_EO2_High Alpha

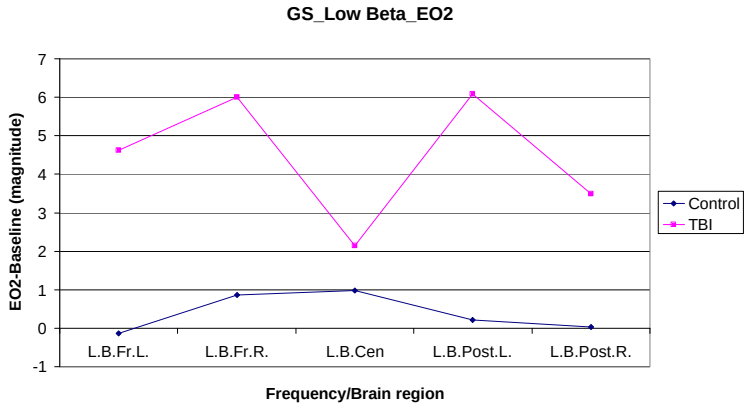


Figure 33. GS_EO2_Low Beta

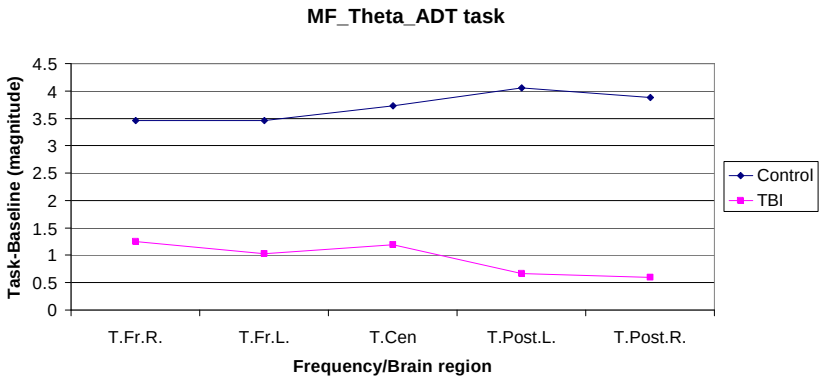


Figure 34. MF_ADT_Theta

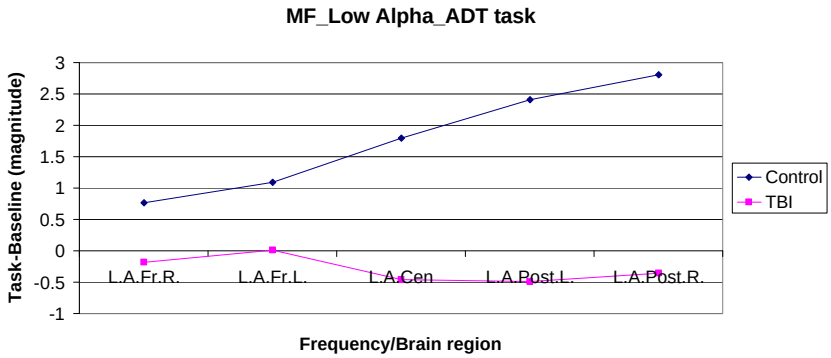


Figure 35. MF_ADT_Low Alpha

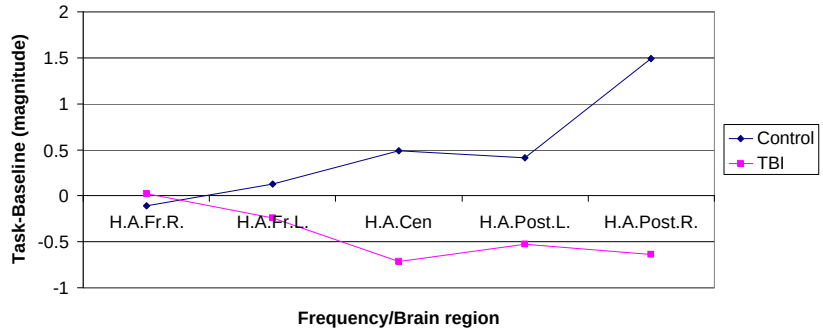


Figure 36. MF_ADT_High Alpha

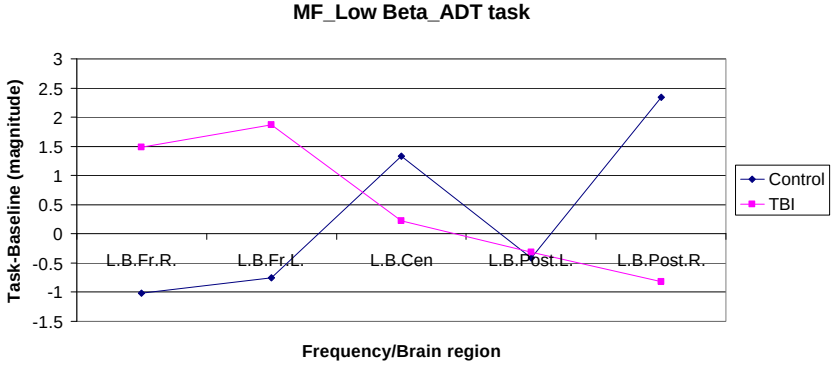


Figure 37. MF_ADT_Low Beta

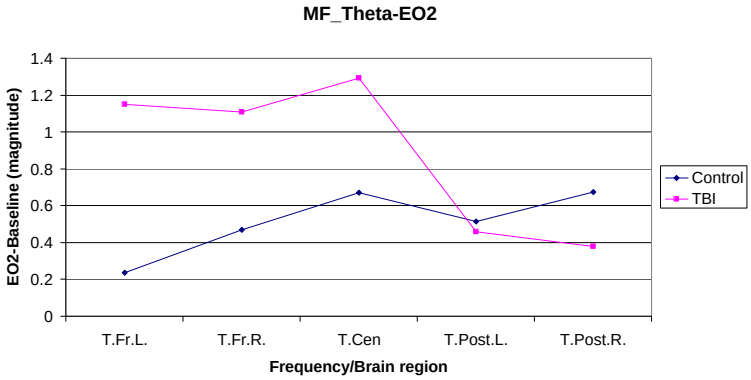


Figure 38. MF_EO2_Theta

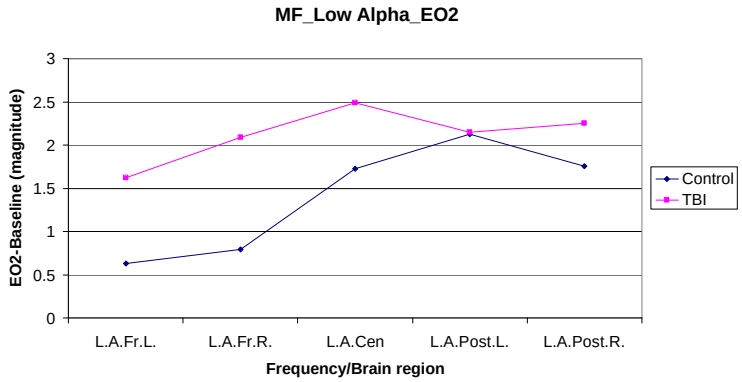


Figure 39. MF_EO2_Low Alpha

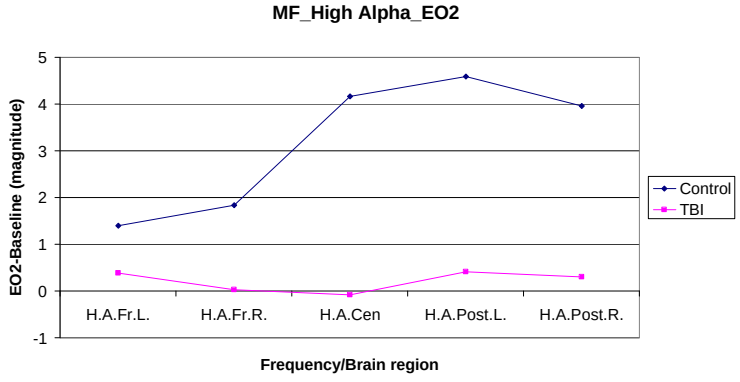


Figure 40. MF_EO2_High Alpha

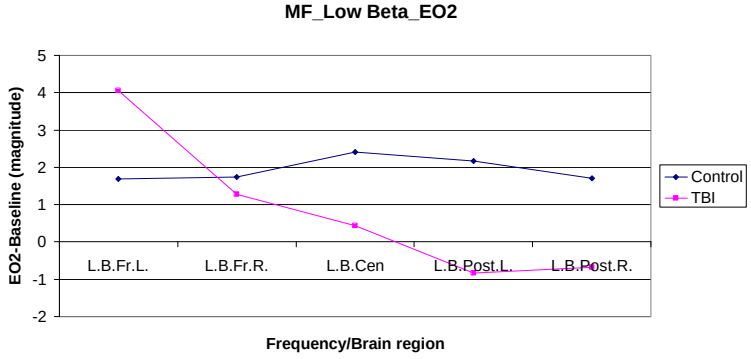


Figure 41. MF_EO2_Low Beta

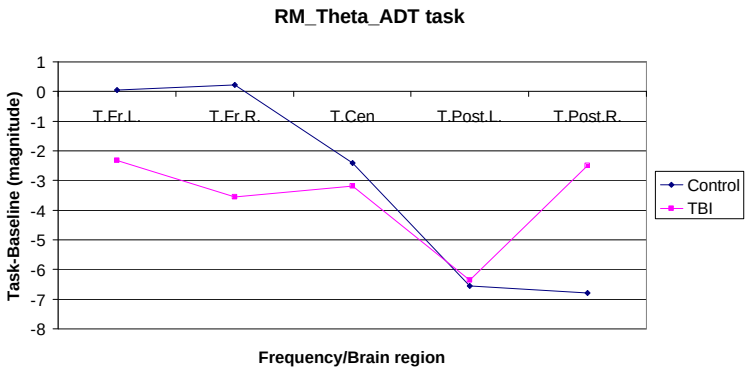


Figure 42. RM_ADT_Theta

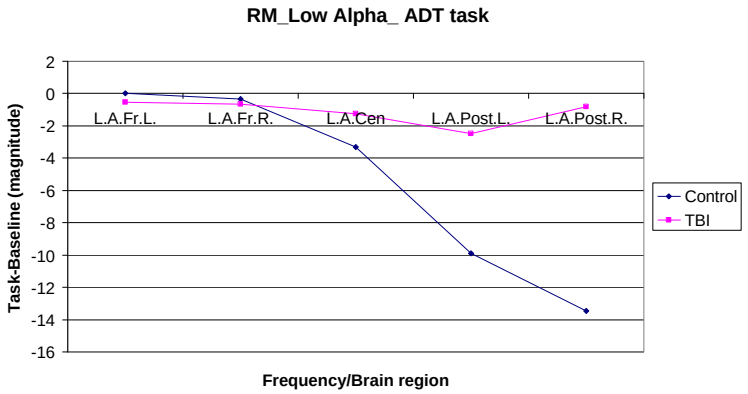


Figure 43. RM_ADT_Low Alpha

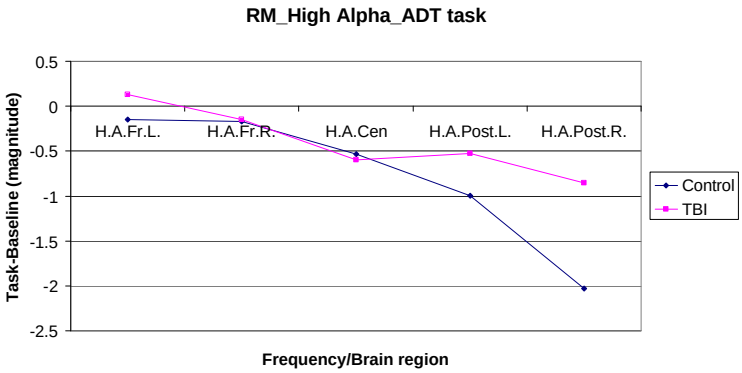


Figure 44. RM_ADT_High Alpha

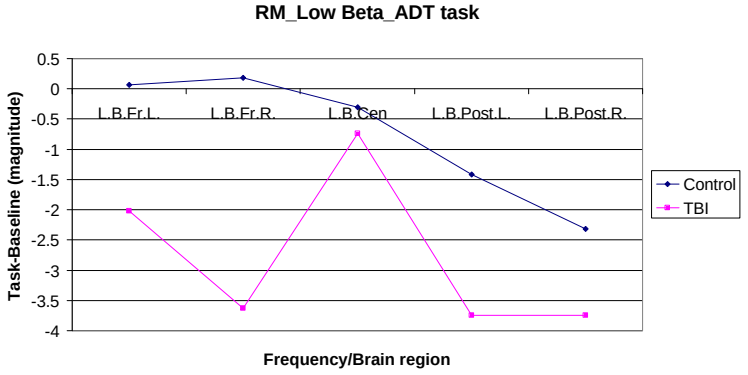


Figure 45. RM_ADT_Low Beta

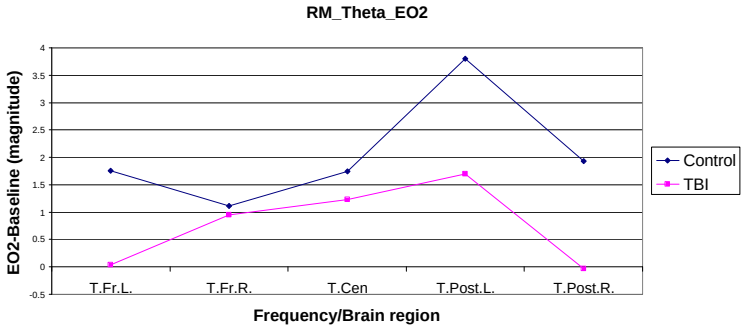


Figure 46. RM_EO2_Theta

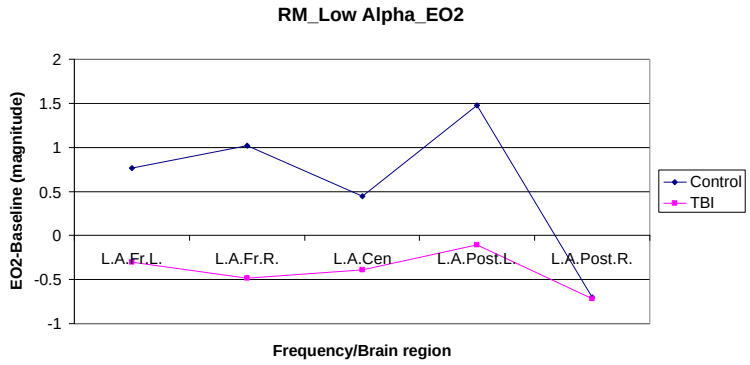


Figure 47. RM_EO2_Low Alpha

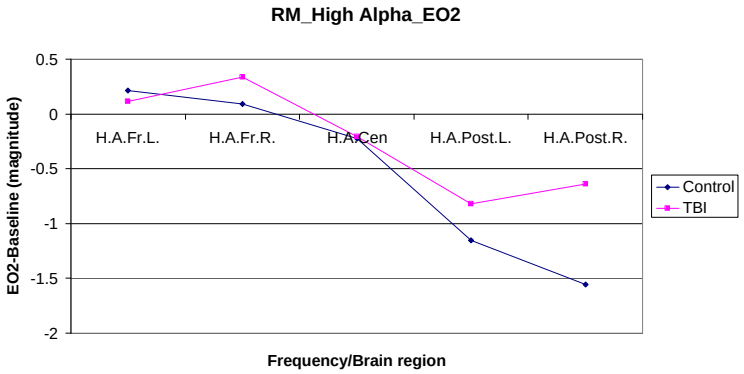


Figure 48. RM_EO2_High Alpha

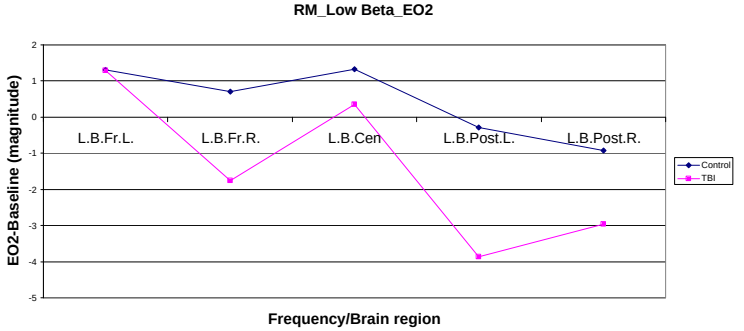


Figure 49. RM_EO2_Low Beta

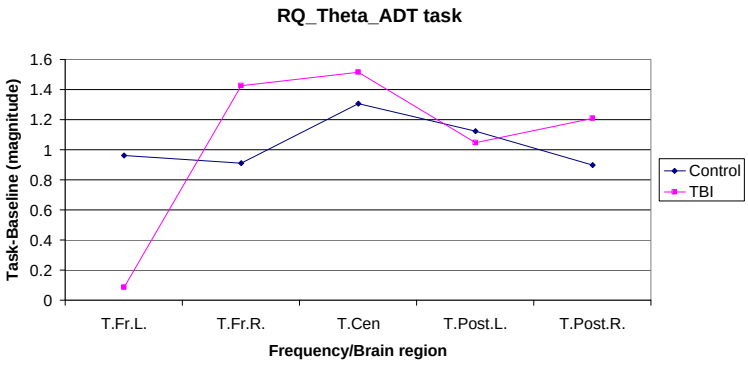


Figure 50. RQ_ADT_Theta

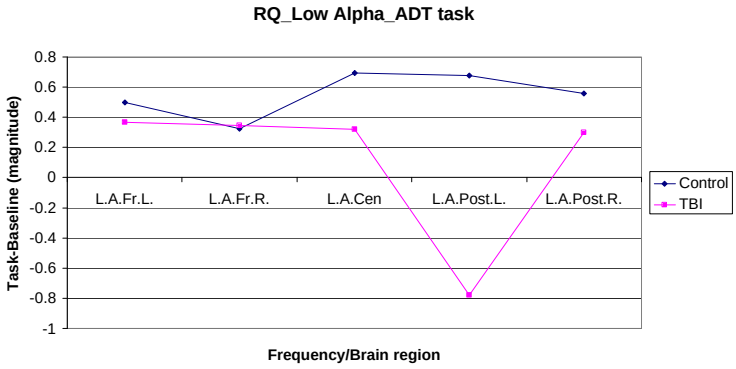


Figure 51. RQ_ADT_Low Alpha

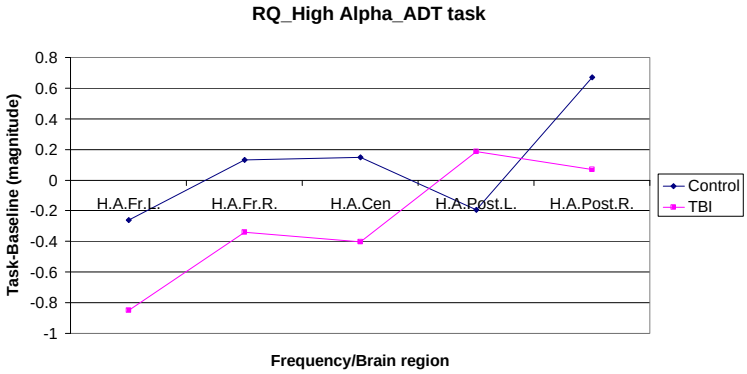


Figure 52. RQ_ADT_High Alpha

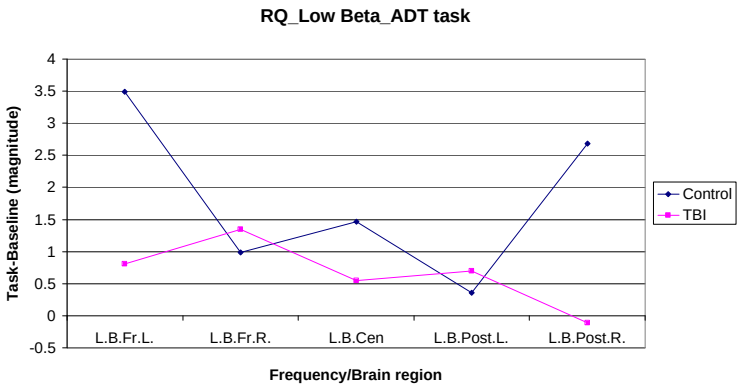


Figure 53. RQ_ADT_Low Beta

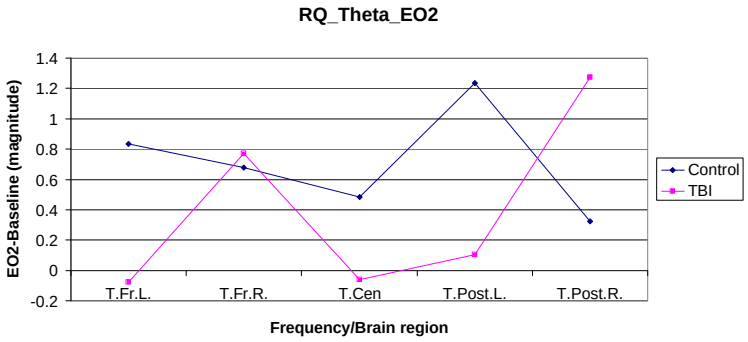


Figure 54. RQ_EO2_Theta

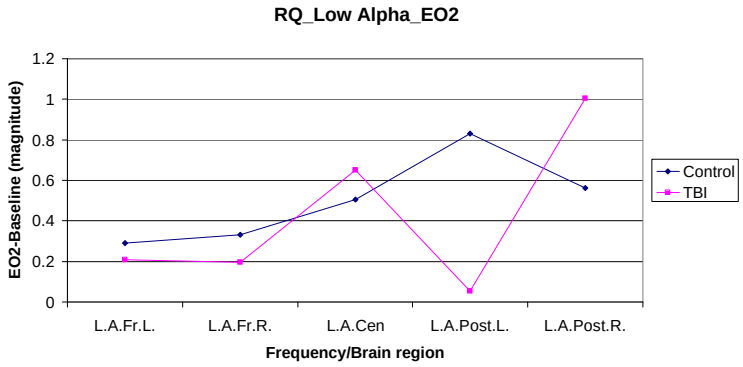


Figure 55. RQ_EO2_Low Alpha

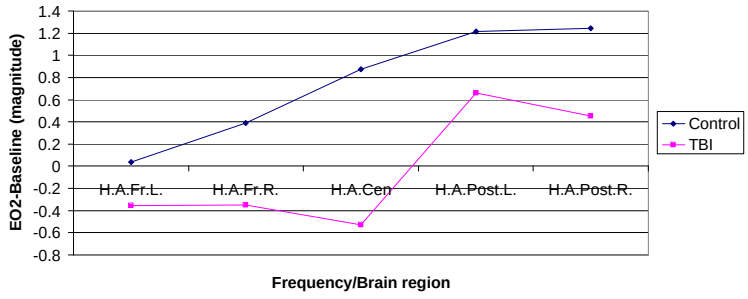


Figure 56. RQ_EO2_High Alpha

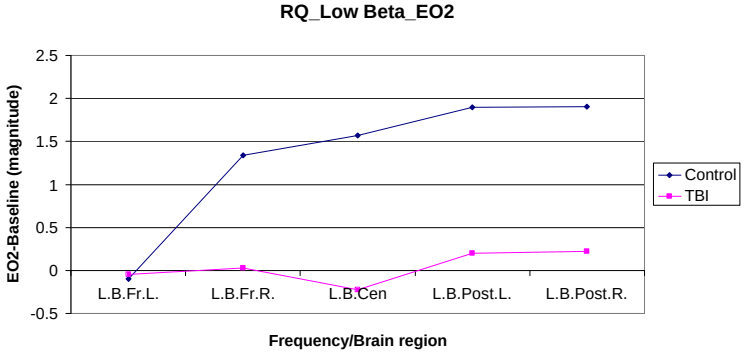


Figure 57. RQ_EO2_Low Beta

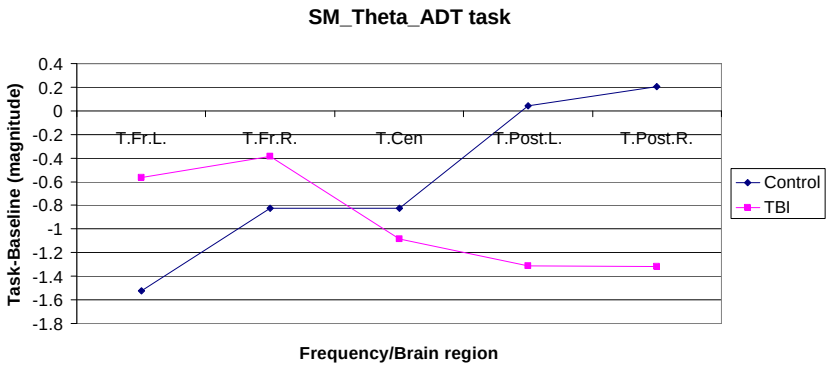


Figure 58. SM_ADT_Theta

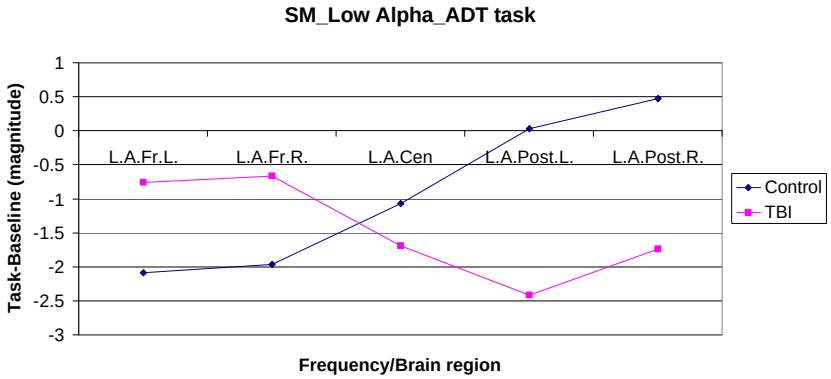


Figure 59. SM_ADT_Low Alpha

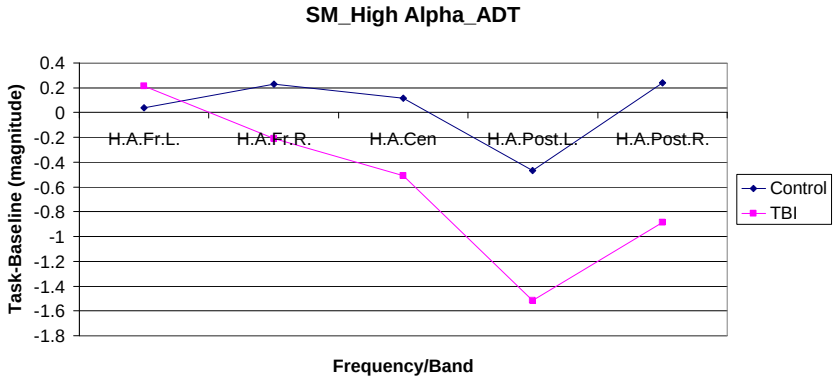


Figure 60. SM_ADT_High Alpha

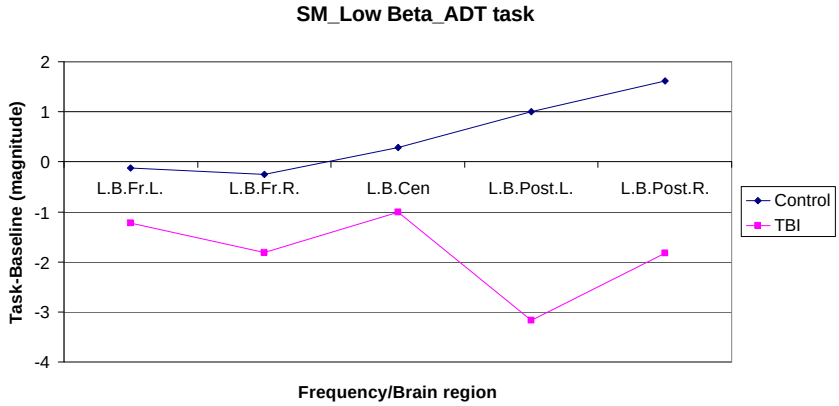


Figure 61. SM_ADT_Low Beta

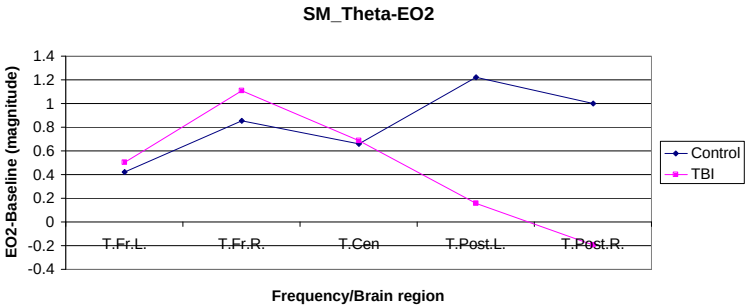


Figure 62. SM_EO2_Theta

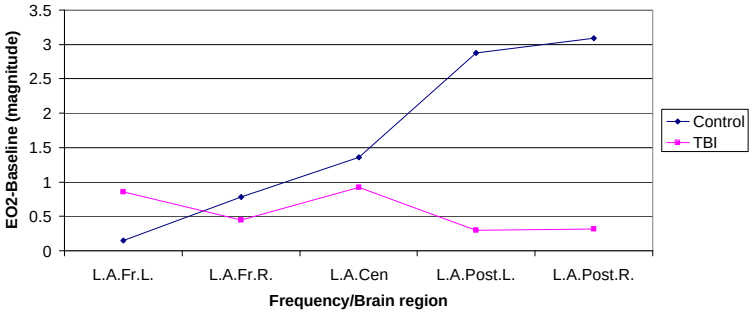


Figure 63. SM_EO2_Low Alpha

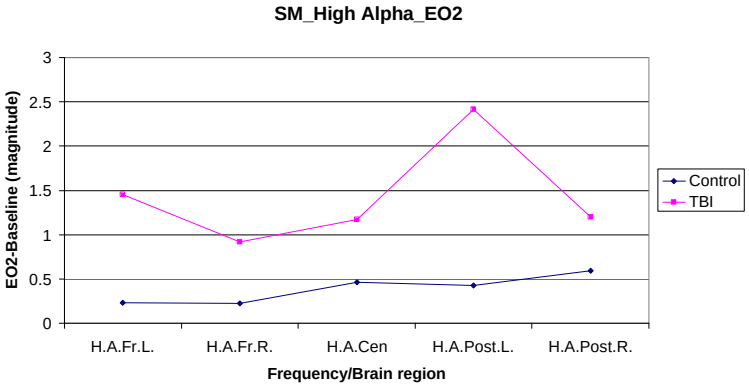


Figure 64. SM_EO2_High Alpha

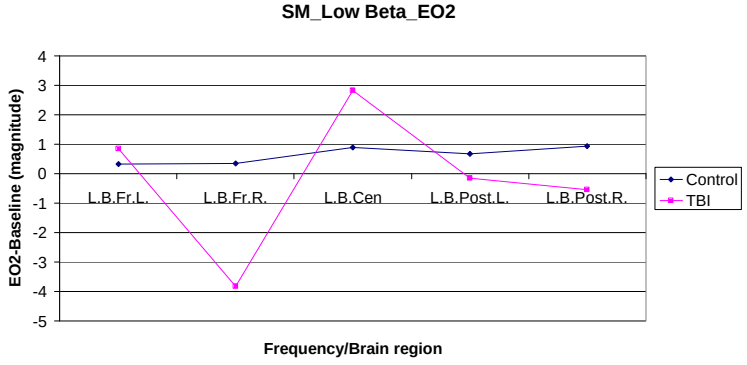


Figure 65. SM_EO2_Low Beta

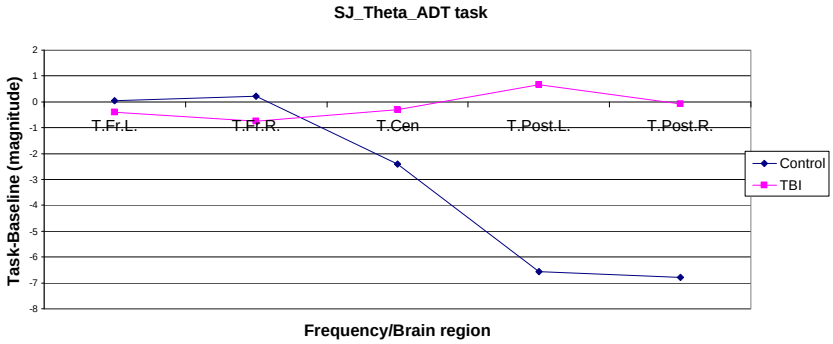


Figure 66. SJ_ADT_Theta

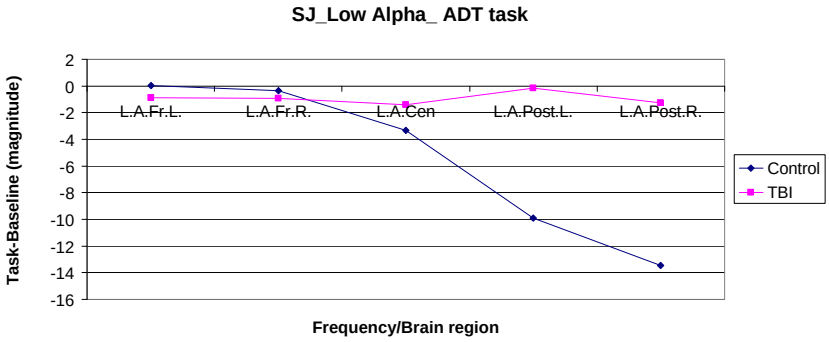


Figure 67. SJ_ADT_Low Alpha

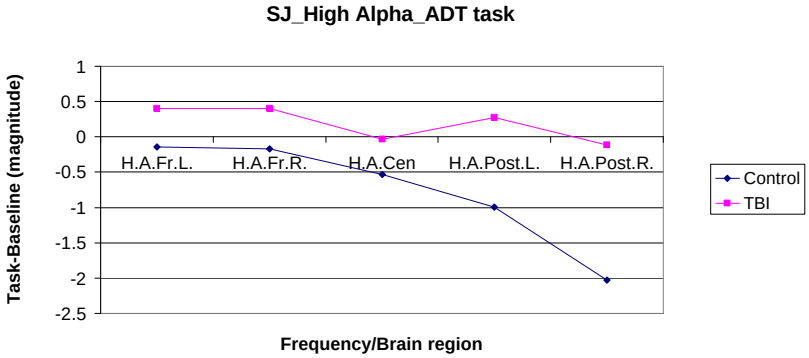


Figure 68. SJ_ADT_High Alpha

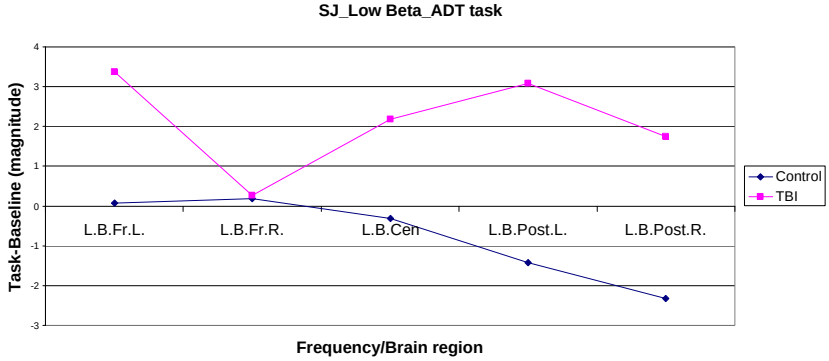


Figure 69. SJ_ADT_Low Beta

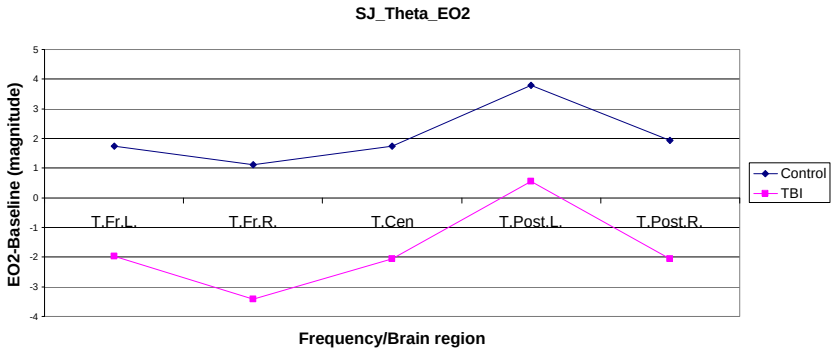


Figure 70. SJ_EO2_Theta

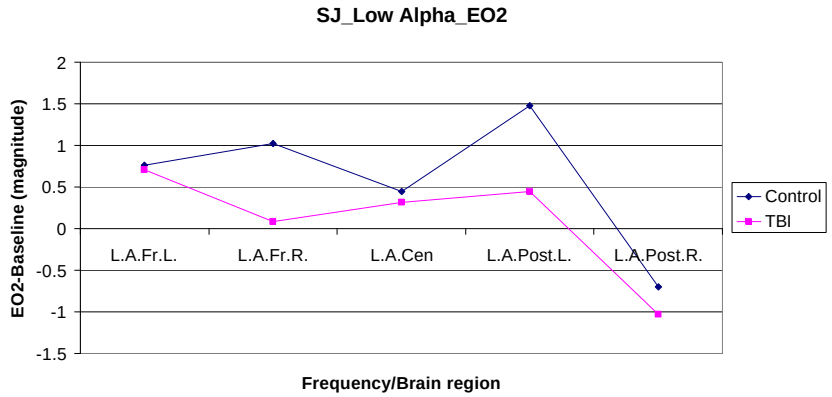


Figure 71. SJ_EO2_Low Alpha

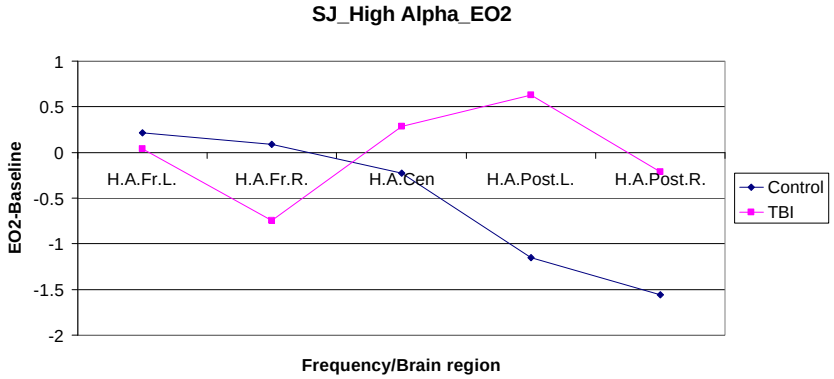


Figure 72. SJ_EO2_High Alpha

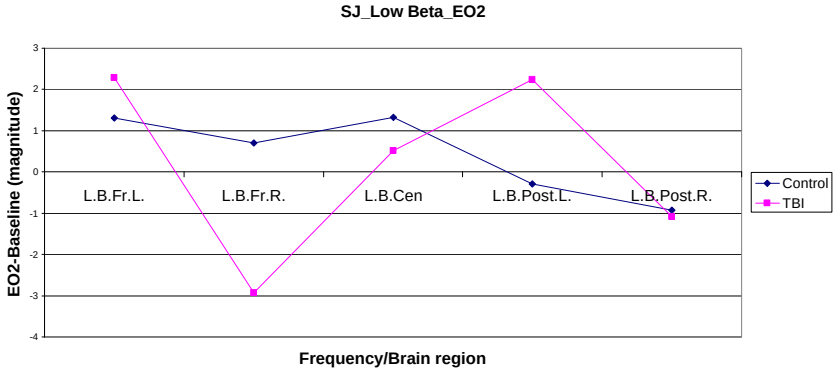


Figure 73. SJ_EO2_Low Beta

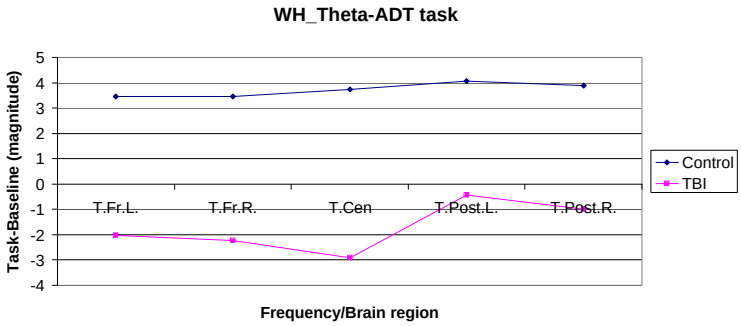


Figure 74. WH_ADT_Theta

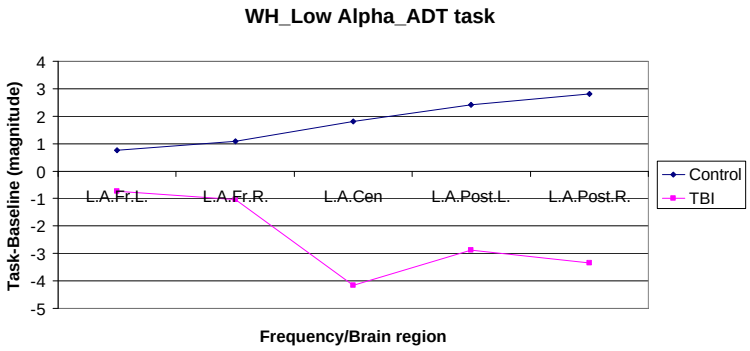


Figure 75. WH_ADT_Low Alpha

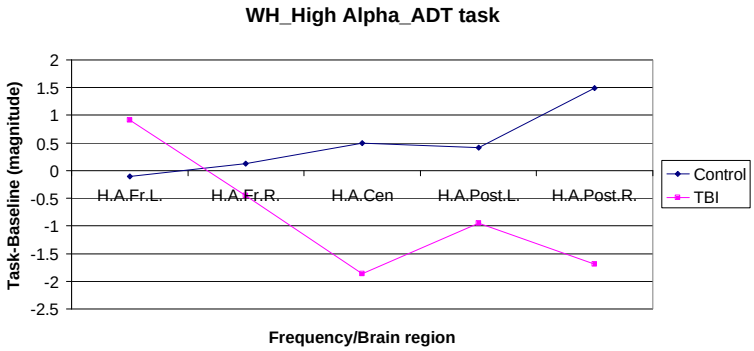


Figure 76. WH_ADT_High Alpha

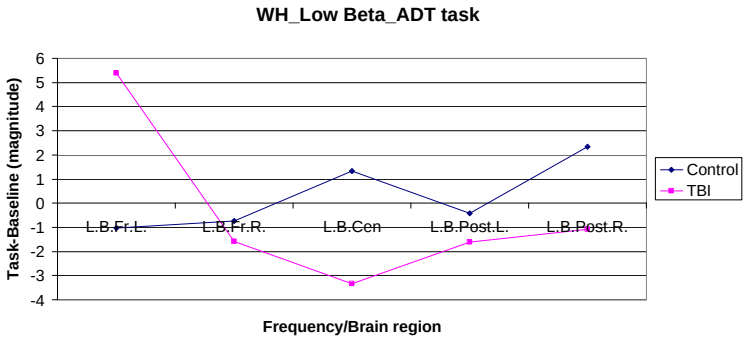


Figure 77. WH_ADT_Low Beta

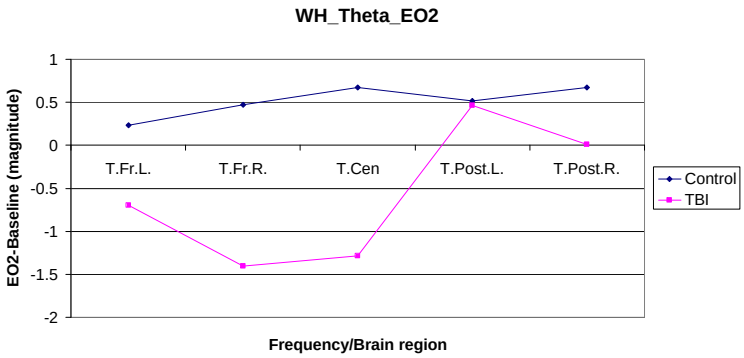


Figure 78. WH_EO2_Theta

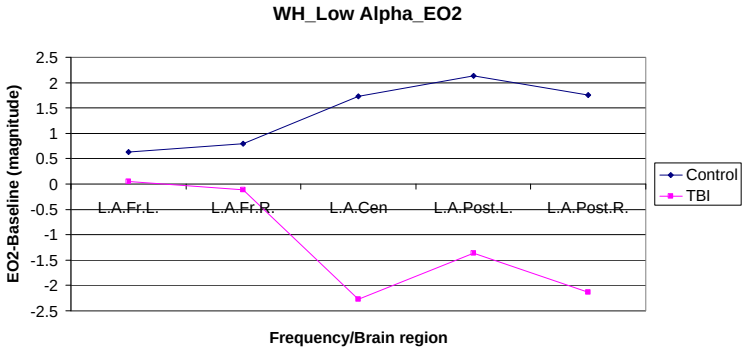


Figure 79. WH_EO2_Low Alpha

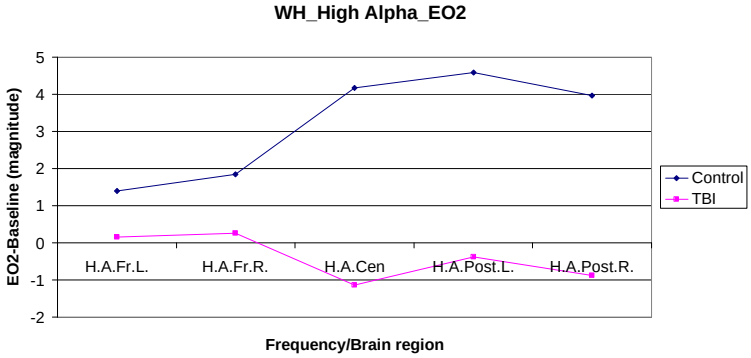


Figure 80. WH_EO2_High Alpha

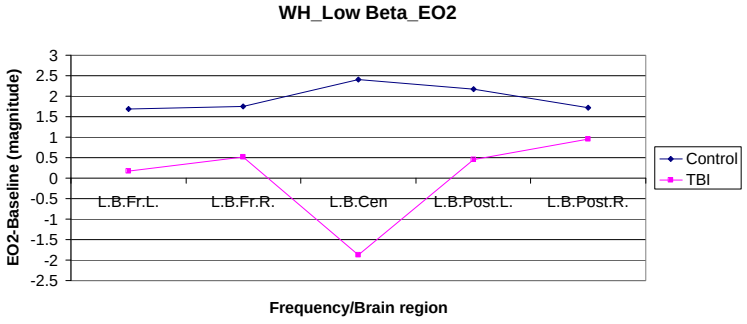


Figure 81. WH_EO2_Low Beta

Sustained Attentional Deficit_ADT

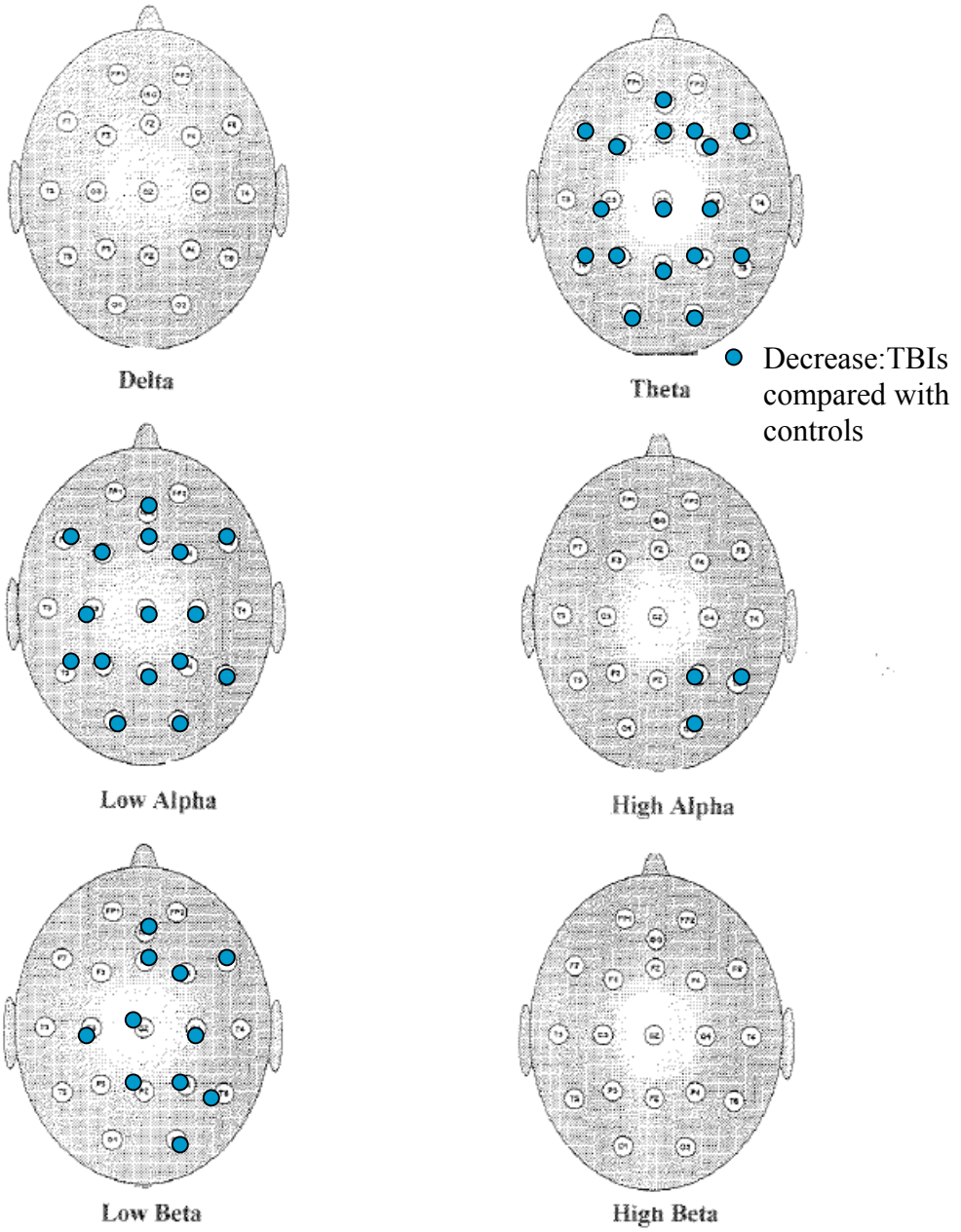
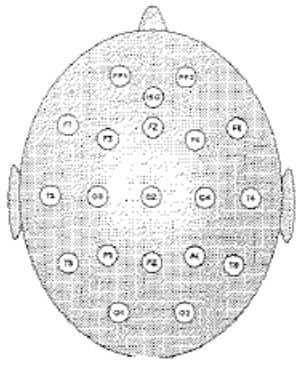
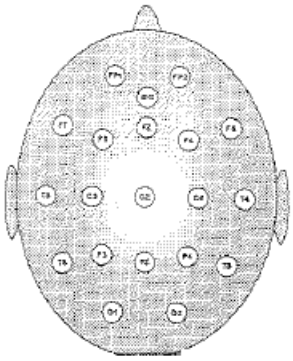


Figure 82: Sustained Attentional Deficit-ADT

Sustained Attentional Deficit_EO2

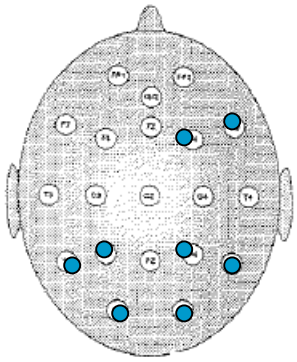


Delta

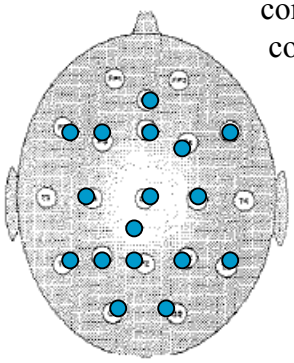


Theta

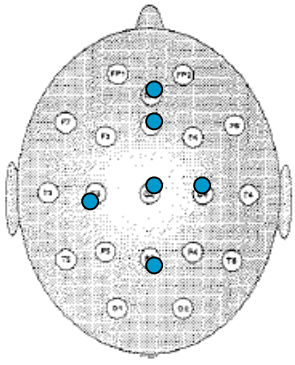
Decrease:TBIs compared with controls



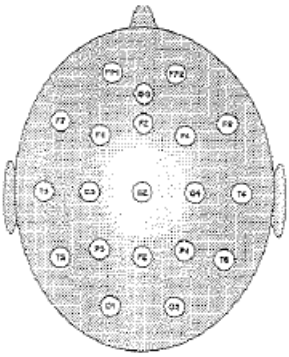
Low Alpha



High Alpha



Low Beta



High Beta

Figure 83: Sustained Attentional Deficit-EO2

Selective Attentional Deficit _ADT

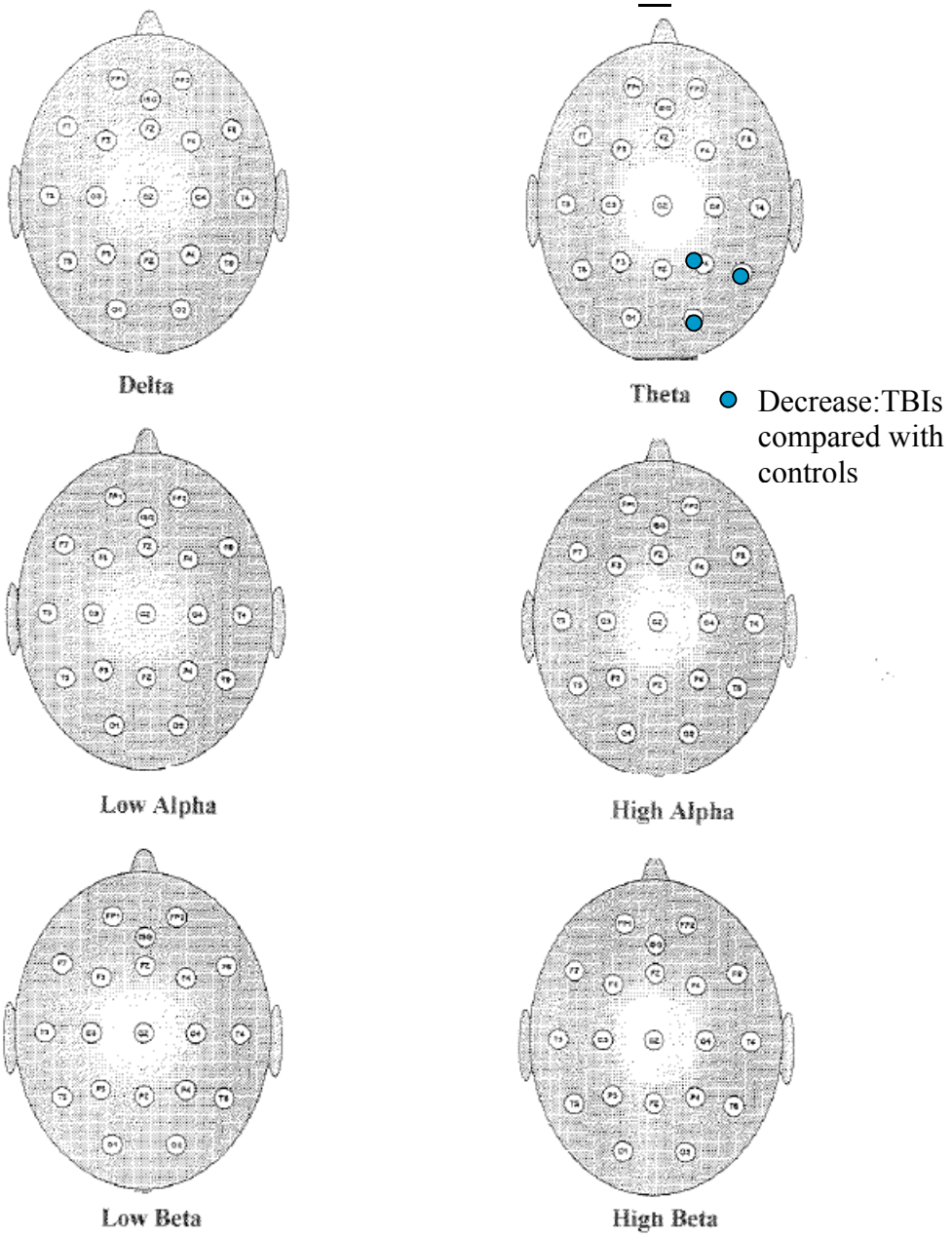
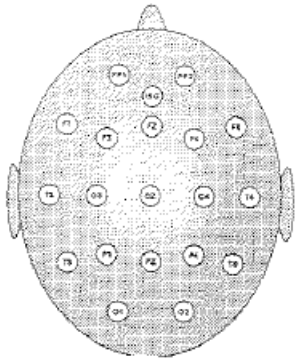
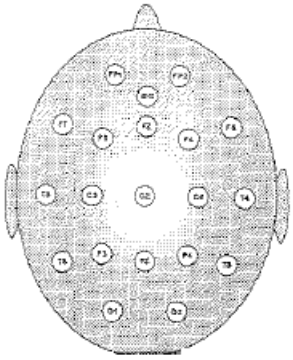


Figure 84: Selective Attentional Deficit-ADT

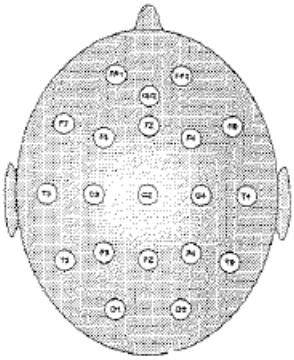
Selective Attentional Deficit _EO2



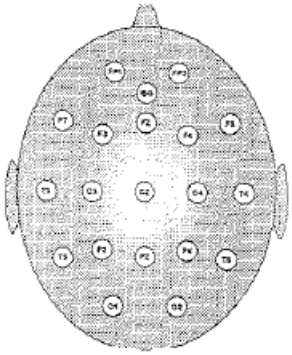
Delta



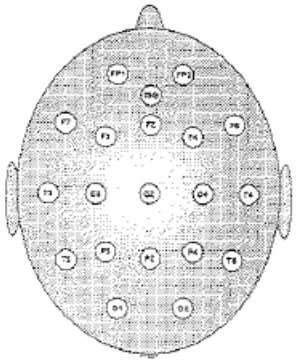
Theta



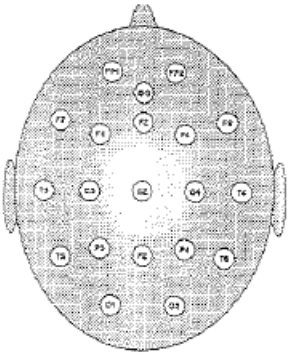
Low Alpha



High Alpha



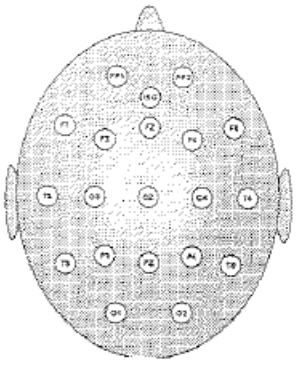
Low Beta



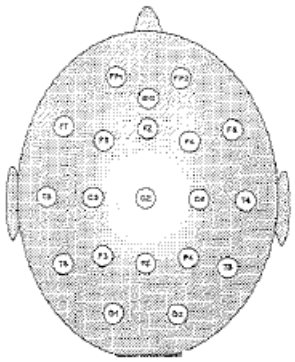
High Beta

Figure 85: Selective Attentional Deficit-EO2

Divided Attentional Deficit-ADT

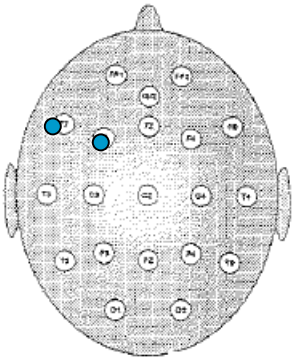


Delta

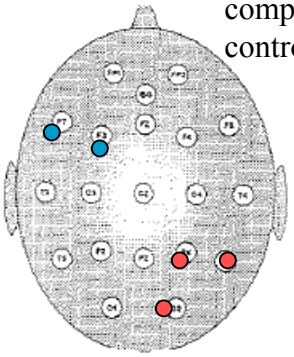


Theta

Decrease:TBIs compared with controls

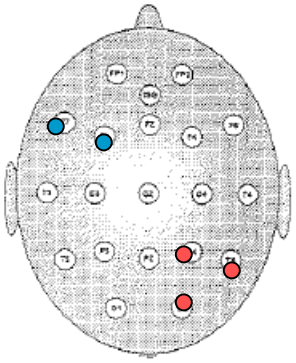


Low Alpha

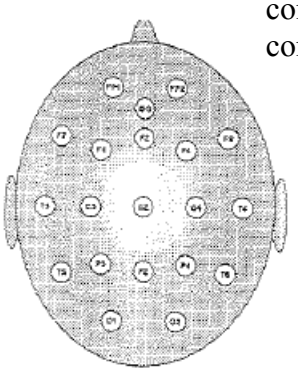


High Alpha

Increase: TBIs compared with controls



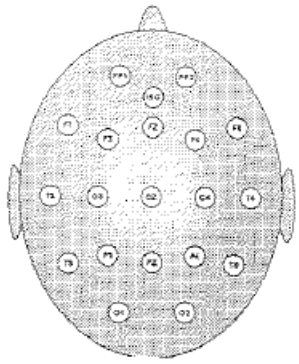
Low Beta



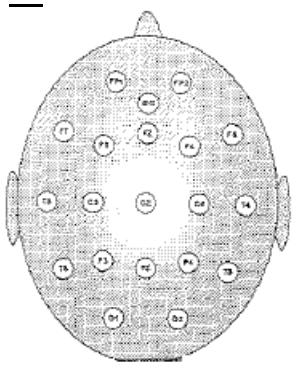
High Beta

Figure 86: Divided Attentional Deficit-ADT

Divided Attentional Deficit_EO2

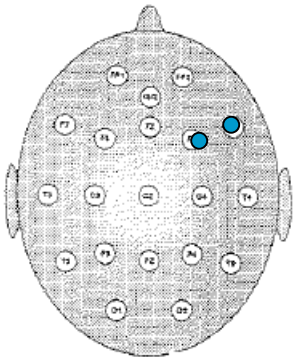


Delta

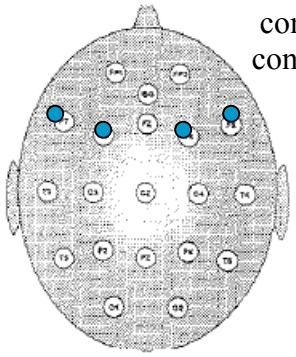


Theta

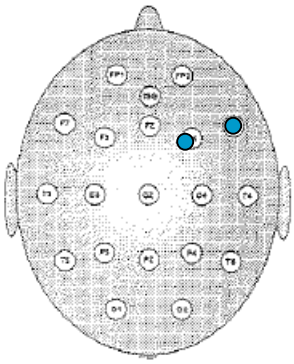
Decrease: TBIs compared with controls



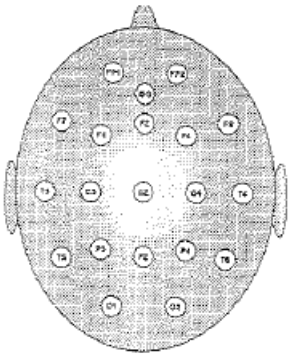
Low Alpha



High Alpha



Low Beta



High Beta

Figure 87: Divided Attentional Deficit-EO2

VITA

Tina Stathopoulou was born in Athens, Greece in 1972. She has a Master's in Experimental Psychology from the University of Tennessee, Knoxville, under the supervision of Dr. Joel F. Lubar. She made her undergraduate studies in the American College of Greece, major Psychology. For four years she had worked with children with different neurological disorders, in rehabilitation centers in Greece. She has also studied French Literature (Licence) in the University of Sorbonne, Paris IV. She has finally obtained a Piano Diploma from the National Conservatory of Greece.

In August the 8th, 2002, she defended her doctoral dissertation in the University of Tennessee, Knoxville, in the area of Experimental Psychology. She has presented in five conferences, in all of which she has taken student scholarships. She has also published all the abstracts of the conferences she has presented as well as a paper in a referee journal. Moreover, she is about to send her second publication based on the results of her thesis. She will work for two years as a postdoctoral student in the University of Pennsylvania, Philadelphia. She is finally interested in obtaining an advanced degree in either Clinical or Counseling Psychology.