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Optimizing Performance in Psychology Students

Neurofeedback as a Performance Enhancing Tool

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Abstract

Neurofeedback has been shown to be successful in treating epilepsy and ADHD and in enhancing performance in musicians and dancers. The objective of the current study was to examine the effect of a neurofeedback beta1/theta protocol as a tool for optimizing performance in healthy psychology students. To achieve this, 19-channel EEG was recorded during a visual Go/NoGo task at two time points, both prior to and following either ten sessions of neurofeedback training (10 individuals) or ten sessions of computerized working memory training (5 individuals), with the hypothesis that neurofeedback but not computerized working memory training would lead to statistically significant gains in sustained attention. The pre- and posttest results of the two groups were compared on measures of reaction time, reaction time variability, errors, and P300 latency and amplitude for both Go and NoGo conditions. The results showed no statistically significant differences between the experiment group and the control group at either time points. The experiment group showed statistically significant ($p<0.005$) changes in reaction time at posttest, also displaying a high practical significance (Cohen's $d = 1.16$). Results showing that only the experiment group displayed statistically significant improvements on variables of sustained attention suggest that neurofeedback has merits in the field of peak performance, with practical implications for everyday life.

Keywords: Neurofeedback, beta1/theta, sustained attention, peak performance

Preface

This study was established when my class mate and I were working as student assistants in a mandatory course in applied EEG and neurofeedback for psychology students who attended the Clinical Psychology Program at NTNU in the autumn of 2013. The data presented in this study were collected by psychology student Tora Thorsrud and me, after rigorous training and under the supervision of Associate Professor Stig Arvid Hollup. I would like to thank them both for a very fruitful and educational teamwork throughout the data collection period and also all the participants who provided the data for this research project.

I would also like to extend my gratitude to my supervisor, Stig for his guidance and help in constructing the research design, and for allowing and enabling me to perform all the necessary statistical analysis on my own. To have been able to be such a big part of planning and carrying out this research project from start to finish has been both demanding and extremely rewarding, both in terms of performing the practical aspects of the data collection, recording EEG signals and administering neurofeedback training and computerized working memory training, and in terms of writing the scientific article that constitutes my graduate thesis in clinical psychology. This would not have been possible without the enthusiastic guidance from my supervisor, Stig Hollup.

I would also like to thank my partner, Bjørn for his never-ending patience and support throughout the process of writing this graduate thesis. Also, I want to thank my amazing mom for all her positive feedback and her continuous efforts to motivate me throughout this process.

Abbreviations

AAPB	Association for Applied Psychophysiology and Biofeedback
ADD	Attention Deficit Disorder
ADHD	Attention-Deficit/Hyperactivity Disorder
APA	American Psychological Association
CPT	Continuous Performance Test
dB	Decibel
EEG	Electroencephalography
EPSP	Excitatory Postsynaptic Potential
ERP	Event Related Potential
Hz	Hertz
ICA	Independent Component Analysis
IPSP	Inhibitory Postsynaptic Potential
ISNR	International Society for Neurofeedback and Research
ms	Milliseconds
NTNU	Norwegian University of Science and Technology
PTSD	Posttraumatic Stress Disorder
QEEG	Quantitative Electroencephalography
RP	Relaxation Percentage
RT	Reaction Time
RTV	Reaction Time Variability
SMR	Sensory Motor Rhythm
SPL	Sound Pressure Level
TP	Training Percentage
VCPT	Visual Continuous Performance Task
μV	Microvolts

"The human brain is estimated to have about a hundred billion nerve cells, two million miles of axons, and a million billion synapses, making it the most complex structure, natural or artificial, on earth" (Green, Heinemann, & Gusella, 1998, p. 427).

Adding to this extraordinary complexity is the fact that the brain can change both its structure and function as a result of experience, a phenomena commonly known as plasticity (Kolb & Whishaw, 1998). Such alterations can occur when we acquire a new skill or when the brain is damaged, but it is also a natural part of brain development (Duffau, 2006; Takeuchi et al., 2010).

A vast range of therapeutic interventions build on the principle of plasticity to achieve betterment and positive results, as is the case with behavioral therapy, errorless learning in rehabilitation of brain injury, and neurotherapies such as transcranial magnetic stimulation and EEG-neurofeedback (Duffau, 2006; Hammond, 2007). Neurofeedback, also known as EEG biofeedback, is a technique that aims to alter brain wave patterns by providing real-time feedback about a person's ongoing brain activity (Chapin & Russell-Chapin, 2014; Hammond, 2007). Before explaining this advancing form of neurotherapy it is important to understand the basic principles of electroencephalography.

The Electroencephalogram

Electroencephalography (EEG) is a method for measuring and recording electric activity in the brain and was first documented and recorded in humans by Hans Berger, a German psychiatrist in the late 1920s (Kaiser, 2005). EEG signals can be obtained by placing small recording electrodes on the scalp where they pick up the electric potentials created by neurons found in the cortex of the brain. These signals are registered and stored to a computer, often during different tasks and conditions, such as resting with eyes open or eyes closed and provide us with an EEG chart showing the measured changes in voltage over time.

Cortical pyramidal cells provide probably the main contribution to the EEG signal. These neurons have a very distinctive shape, with the soma shaped as a triangle or pyramid and a long apical dendrite that is positioned perpendicular to the cortical surface. This structure makes the pyramidal cells act as electric dipoles where presynaptic neurons create postsynaptic potentials in the membrane of the pyramidal cells. These can be either excitatory (EPSPs) or inhibitory (IPSPs) in nature. As the pyramidal cells are highly interconnected with each other and with other kinds of neurons, one single pyramidal cell receives input from a large number of nerve cells (Bressler & Ding, 2006). The IPSPs and EPSPs are integrated

through spatial and temporal summation for each pyramidal cell, and an action potential will be produced when the pyramidal cell is sufficiently depolarized. On the EEG the upward deflections are caused by either superficial excitatory input or by deep inhibitory input to the pyramidal cells. A downward deflection will be produced by either superficial inhibitory input or deep excitatory input. An EEG wave is the product of the postsynaptic potentials from the recordings of the simultaneous EPSPs and IPSPs from a large number of neurons that fire within the same time frame. The more synchronized the postsynaptic potentials in a pyramidal cell are and the more synchronized all the neurons in the same time frame are, the greater the amplitude on the EEG will become (Freberg, 2006; Kirschstein & Köhling, 2009; Pascual-Marqui, Esslen, Kochi, & Lehmann, 2002).

EEG signals vary in their characteristics and can be non-periodic, periodic and non-sinusoidal or periodic and sinusoidal. The latter signals are usually labeled and classified according to their frequency bands, although the precise bandwidths are somewhat debatable (Kaiser, 2000, 2001). Also, the different frequency bands are associated with different functional correlates (Neuper & Pfurtscheller, 2001). Waves with a frequency between 0.5 and 4 cycles per second (Hertz) are called delta waves which are slow, high amplitude waves most pronounced during deep sleep. Theta waves (4-8 Hz) are commonly found in the deeply relaxed brain and are according to Hammond (2007) the hallmark of daydreaming and drowsy states. Alpha waves, occurring within the range of 8-12 Hz are high amplitude waves and this activity is associated with relaxed wakeful states, as in the eyes closed condition. Beta waves belong in the range from 13 to 30 Hz and are small amplitude faster waves, found in the alert and concentrated brain. Brain waves above 30 Hz are called gamma (Hammond, 2007; Yucha & Montgomery, 2008) and are also related to alert information processing. An important detail to point out is that all the different frequency bands can be found in the brain at the same time, reflecting the diverse functional states of different brain areas. It is theorized that a main function of the different frequency ranges might be related to transfer and synchronization of information across brain areas (Buehlmann & Deco, 2010).

Event related potentials. In addition to the many frequency bands there are several other EEG components that can yield useful information about brain activity and brain functioning. Among these the event related brain potentials (ERPs) have been found particularly informative and they can be used to assess the neural basis of sensory, motor and higher-order cognitive processes (Bressler & Ding, 2006). Insight into these processes has traditionally been achieved by looking at ERP components spanning over short time periods

immediately prior to or following the occurrence of a stimulus or a measurable event
(Bressler & Ding, 2006).

The ERP waveform itself consists of different components, labeled according to their
electric polarity and time of occurrence. Hence, the P300 is the positive sinusoidal deflection
that occur approximately 300 milliseconds after an event, and it has received a lot of attention
in research. The component is measured by way of its amplitude and latency. The amplitude
is defined as the difference between mean baseline voltage prior to stimulus-onset and the
peak of the highest positive deflection, in microvolts (µV), and it is affected by variables such
as task modality and the age of the subject (Polich, 2007). The latency of the P300 is
measured in milliseconds (ms) and is defined as the time from the onset of a stimulus to the
point of maximal positive amplitude, within a predefined time period (Polich & Kok, 1995).
Thus, P300 latency reflects the time needed to discover and evaluate a stimulus and is also
found to be a predictor of cognitive performance where shorter latency is associated with
better cognitive performance and mental efficiency compared to a longer latency period
(Polich, 2007).

According to Polich (2007) the P300, also known as the P3 is thought to reflect a
cascade of information processes, involving mechanisms of attention and memory. Though
we have limited knowledge concerning how and why the brain produces this ERP component,
what we do know is that the P3 can be subject to habituation and dishabituation and that it is
affected by several factors such as genetics and an individual's overall level of arousal
(Polich, 2007; Polich & Kok, 1995). The signal strength is also affected by the amount of
attentional resources required for solving the task at hand, and the time interval between two
stimuli/events. The P300 component has been found to be a marker for the updating of the
working memory, and it is a reliable assessment variable according to Polich (2007)
displaying a long term test-retest reliability of 0.96 for latency and >0.8 for amplitude
(Brunner et al., 2013).

Research has demonstrated that both the composition of brain wave oscillations and
ERPs are important indicators of neurological functioning and mental health and disorders.
Normative EEG databases have been developed for a variety of tasks and conditions
(Thatcher, 1998; Thatcher, Walker, Biver, North, & Curtin, 2003), enabling EEG measures to
be of importance in both clinical diagnosis and in the evaluation of therapy. EEG has a wide
area of application and has given us considerable information about brain functioning. It has
been used to discover and localize epileptic seizures (Acharya, Vinitha Sree, Swapna, Martis,
& Suri, 2013) and to differentiate clinical populations from each other and from healthy

norms. Statistically significant differences in the EEG have been found for ADHD, schizophrenia, agoraphobia, obsessive-compulsive disorder, bipolar disorder, major depression, migraine, different types of dementia, alcoholism and PTSD among others (Kam, Bolbecker, O'Donnell, Hetrick, & Brenner, 2013; Lubar, 1991; Saletu, Anderer, Saletu-Zyhlarz, & Pascual-Marqui, 2005; Smyth & Winter, 1964; Snyder et al., 2008). These findings have earned EEG its status as a brain imaging tool (Michel & Murray, 2012).

QEEG. The quantification of the EEG (qEEG) has been made possible by way of computers and programs that can perform statistical analysis on the raw EEG material (Kaiser, 2000), and provides us with what has been termed a "brain map" (Arns & Lyle, 2011). The procedures of qEEG can also be used to calculate what Prichep and John (1992) called an abnormality vector in a brain signal space, represented as standard deviations from the normative mean value and can be used to classify patients according to overall severity of brain dysfunction. In a review of the field by Coburn et al. (2006) the authors concluded that the clinical use of qEEG can aid in the detection and differential diagnosis of "both disorders of childhood, such as learning disabilities and attention-deficit disorders, and those occurring primarily during adulthood, such as depressive, bipolar, and dementing disorders" (p. 495). The discovery that a number of clinical populations are characterized by certain brain wave patterns and qEEG profiles which deviate predictably from the healthy population have implications for neurotherapies. Neurotherapy is a term that encompasses many different interventions (Duffau, 2006; Hammond et al., 2011) with the common purpose of altering one or more aspects of neuronal network functioning (Chapin & Russell-Chapin, 2014) by way of stimulation or feedback procedures.

Neurofeedback

Neurofeedback training is a procedure in which one works to alter specific brain wave patterns to obtain a more optimal activity (Chapin & Russell-Chapin, 2014) by using a small number of electrodes on the region of interest. The number (usually 3 to 6) and placement of the electrodes depends on the specific aim of the neurofeedback training (Yucha & Montgomery, 2008). The EEG signals detected by the electrodes are amplified and digitized by specialized hardware, then sent to a computer where certain aspects of the signal are mapped to some form of feedback (Chapin & Russell-Chapin, 2014). This is also known as a brain-computer interface, and the feedback can be either visual, auditory or both. Most common are graphs and digits but also changes in color or patterns or even animations that change as a direct result of variations in the participant's EEG are possible (Yucha &

Montgomery, 2008). The real-time feedback represents the client's underlying EEG activity and allows for monitoring and manipulation of this activity by the client. The goal of this training is the voluntary production and control of EEG signals to achieve a specific result (Vallabhaneni, Wang, & He, 2005).

An important specification when it comes to the definition of neurofeedback is that it is a form of self-regulation training based on operant learning mechanisms of brain activity (Sherlin et al., 2011). This means that there are certain principles that must be followed for the training to be effective. The paper put forth by Sherlin et al. (2011) emphasizes that to achieve the desired training effect, feedback must not be delayed by more than 250-350 milliseconds, and the setup must be discrete and uncomplicated so as to not overshadow the response-reinforcement association. To ensure specificity of the training the instructor's knowledge of the frequency bands to be trained and of the EEG physiology is critical, as is the minimization of produced artefacts which can contaminate the EEG signals (Hammond et al., 2011; Hammond, Stockdale, Hoffman, Ayers, & Nash, 2001). Secondary reinforcement, if provided must be linked exclusively to the learning process, and transfer trials should be used to ensure generalization of the neurofeedback training beyond the clinical task and setting.

Neurofeedback training protocols. Given that there are many different frequency bands and that they exist simultaneously in different brain areas, there are a great many possible protocols to choose from for neurofeedback training. Which protocol to use depends on the presenting symptoms of the client and whether the aim is to increase or decrease the amplitude of a specific brain wave target or a combination of the two. Neurofeedback training in patients with Attention-Deficit/Hyperactivity Disorder (ADHD) provides a good example of how neurofeedback protocols may be implemented, with the typical ADHD profile showing excess slow wave activity, often in combination with reduced beta activity (Arns, Heinrich, & Strehl, 2013; Yucha & Montgomery, 2008). Different protocols have focused on the down-regulation of delta, theta and sometimes alpha activity, the up-regulation of beta activity or a change in the ratio between low frequency and high frequency activity (Arns et al., 2013; Yucha & Montgomery, 2008).

Neurofeedback is not a "one size fits all" treatment, so in order to reach the goal of regulation and normalization of brain function one needs to tailor the training to the individual's brain wave patterns. Such individualization is best accomplished when based on a thorough clinical assessment that may include the client's behavioral symptoms, clinical history, neuropsychological or psychological tests and EEG or qEEG (Hammond, 2007;

Hammond et al., 2011). In the field of neurofeedback research this has presented researchers with the dilemma of weighing the use of standardized protocols against the use of individualized training protocols. Standardized protocols are required for the comparison of results across studies to evaluate efficacy, but individualized protocols may be required for neurofeedback to become a more efficient treatment for the individual client and thus necessary to yield significant improvements (Yucha & Montgomery, 2008).

Applied neurofeedback. Neurofeedback has been practiced since the 1960s and early on much of the focus revolved around facilitating relaxation by way of increasing activity in the alpha frequency band and around reducing intractable epilepsy (Hammond, 2007; Tan et al., 2009). Research on neurofeedback for epilepsy included cats, monkeys and humans and was associated with clinical improvement across studies in terms of reduction in seizure frequency, severity and duration. These improvements have been reported across several different types of seizures, including patients not responding to anticonvulsant medications (Lubar & Bahler, 1976; Tan et al., 2009). Neurofeedback has produced significant clinical results concerning the treatment of epilepsy, the most common neurological disease in the world (Acharya et al., 2013) and has been rated as level 4 out of 5 as an evidence based treatment for this condition. The rating was part of a standardized assessment of the efficacy and effectiveness of neurofeedback, conducted by Yucha and Montgomery (2008) and was performed in accordance with the guidelines accepted by the International Society for Neurofeedback and Research (ISNR) and the Association for Applied Psychophysiology and Biofeedback (AAPB). These guidelines are similar to those put forward by the American Psychological Association (APA) (for details, see Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Yucha & Montgomery, 2008).

The clinical effect of neurofeedback for Attention-Deficit/Hyperactivity Disorder was first demonstrated by Lubar and Shouse in the 1970s who found improvement in variables of hyperactivity and distractibility (Arns et al., 2013). Since then the interest in neurofeedback for ADHD-like symptoms has increased dramatically, resulting in empirical evidence for the clinical effect of a number of different protocols, including regulation of the relationship between the amount of beta- and theta-activity and regulation of the sensory motor rhythm (SMR) (Arns et al., 2013; Gani, 2009; Gevensleben et al., 2010; Lansbergen, van Dongen-Boomsma, Buitelaar, & Slaats-Willemse, 2011; Liechti et al., 2012). Today, neurofeedback is used by many clinicians in the treatment of ADHD, although there has been uncertainty regarding the evidence-based level of this treatment. As a result of this uncertainty Arns et al.

(2009) conducted a meta-analysis of the field, examining the empirical evidence for the effect of neurofeedback and of stimulant medication on what is commonly described as the core symptoms in ADHD: Impulsivity, inattention and hyperactivity.

This meta-analysis included 15 studies, counting 1194 participants. Among these, there were six randomized studies where three compared neurofeedback to stimulant medication, which is seen as the "gold standard" for treating ADHD. The studies included used either passive or active control groups in order to control for nonspecific effects, such as cognitive training and therapist contact. The conclusion drawn from this analysis was that neurofeedback could be considered as clinically meaningful in the treatment of ADHD. Effect sizes were large for impulsivity and inattention and medium for hyperactivity. In accordance with guidelines for the assessment of evidence-based clinical efficacy and effectiveness mentioned previously neurofeedback as a treatment for ADHD was considered "Efficacious and specific" (level 5), the same level as stimulant medication (Arns et al., 2009). An important difference however, is that while medications lose their effect when discontinued neurofeedback has been demonstrated to show long term effects and even continued improvement in some cases on a two year follow-up (Gani, 2009). In addition, neurofeedback is only on very rare occasions associated with negative side effects and when found they are transient, easily reversible and often due to administration of neurofeedback without the proper competence or monitoring (Hammond et al., 2011; Hammond et al., 2001). Stimulant medication on the other hand is regularly associated with adverse side effects, including insomnia, reduced appetite, nervousness, headaches, abdominal pain and hypertension among the most common ones (Arns et al., 2009; Efron, Jarman, & Barker, 1997; Ritalin "Novartis", 2014).

Over the last decades neurofeedback has been found to be of clinical significance in treatment and normalization of pathologic brain activity in a range of different psychological and neurological disorders. Findings include but are not limited to ADHD (Arns et al., 2013; Gani, 2009; Gevensleben et al., 2010; Lansbergen et al., 2011), ADD (Arns et al., 2009; Thompson & Thompson, 1998), epilepsy (Lubar & Bahler, 1976; Tan et al., 2009), generalized anxiety disorder, PTSD, insomnia, traumatic brain injury and substance- and alcohol abuse (Yucha & Montgomery, 2008). It has also been demonstrated to improve mood compared to mock feedback (Raymond, Varney, Parkinson, & Gruzelier, 2005). As the evidence base for this method has expanded, researchers have started to explore the use of neurofeedback to optimize performance in healthy subjects and recent studies have reported

results that show a correlation between neurofeedback learning and positive outcome measures (Gruzelier, 2013a).

This new emerging field of optimization, also called peak performance has thus far demonstrated that a wide range of neurofeedback protocols have led to improvement in skills ranging from enhanced microsurgical skills and reduced anxiety in surgeons (Ros et al., 2009) to improved visuospatial rotation (Doppelmayr & Weber, 2011) and enhanced mood (Raymond et al., 2005) in healthy subjects. In a review article of this field, Gruzelier (2013a) refers to a variety of studies which have reported that neurofeedback can lead to improvement in sustained attention, orienting skills and executive attention, reaction time, implicit procedural memory, higher IQ score as well as to enhancement of different aspects of mood and well-being. Neurofeedback has also been found to significantly improve performance in both novice and elite musicians in children as well as adults and to enhance creativity and performance in dancers and actors with differing levels of experience (Gruzelier, 2013b; Gruzelier et al., 2013).

In two studies with healthy participants, Egner and Gruzelier (2001, 2004) demonstrated that neurofeedback has frequency-specific effects on attention and on event related brain potentials. In the latter study they achieved this by randomizing 25 students to one of three conditions: Increase the amount of beta activity in the range of 12-15 Hz also known as the sensorimotor rhythm (SMR), increase the amount of beta activity in the range of 15-18 Hz also known as beta1 or to an active control group. The participants were tested on two different measures of sustained attention before and after the intervention. Those who received neurofeedback were also measured on target P300 amplitude during an oddball task. The results from this study showed that SMR training led to better attention through heightened perceptual sensitivity, a reduction in the number of omission errors on the oddball task and a reduction in reaction time variability. Beta1 training led to an increase in arousal associated with faster reaction time and an increase in the target P300 amplitudes. Such changes were not found in the active control group, and were thus attributable to the neurofeedback training.

Ghaziri et al. (2013) have also demonstrated significant enhancements in both auditory and visual sustained attention performance in healthy students following neurofeedback training enhancing the beta1 frequency band. They also found that the protocol lead to modifications in both white matter pathways implicated in sustained attention and in grey matter volume in brain regions associated with the same type of attention and thus affecting brain plasticity.

Concerning the amount of neurofeedback training and the number of sessions necessary to invoke neurofeedback learning in healthy individuals there are varying findings. Egner and Gruzelier (2001) found that ten sessions of training was sufficient to ensure successful learning of the beta1/theta protocol, whereas Doppelmayr and Weber (2011) found successful learning of an SMR protocol after 30 training sessions, but no such learning after 30 training sessions on the beta1/theta protocol.

Sustained Attention

The attention system of the brain is like other sensory and motor systems in terms of it being anatomically separate from the data processing systems that perform operations on specific inputs (Posner & Petersen, 1990). Attention is carried out by a number of different brain areas and can be divided into different subsystems with differing but interrelated functions.

Sustained attention is a psychological construct pertaining to the ability to perform monitoring tasks and has predominantly been investigated and assessed in watch-keeping tasks (Sarter, Givens, & Bruno, 2001; Staub, Doignon-Camus, Després, & Bonnefond, 2013) such as the continuous performance test (CPT). In this test paradigm sustained attention is precisely defined as a state of readiness to detect and respond to certain changes in the environment occurring at random time intervals over prolonged periods of time (Staub et al., 2013) and it is characterized by the overall ability to detect signals and a decrement in performance over time. The cause of this vigilance decrement is uncertain but it has been hypothesized that attentional resources become depleted over time on the task or that attentional resources are directed away from the assignment as subjects get bored from the monotonous task (Staub et al., 2013). One variant of the CPT is the Visual continuous Performance Task (VCPT), where the outcome measure of reaction time is a combined result of both the ability to sustain attention and of hand-eye coordination. Hand-eye coordination is an important skill for many everyday tasks, such as driving a car, and it has been found to be associated with cognitive and social skills in children (Yu & Smith, 2013).

The ability to sustain attention, also termed vigilance has been found to represent a basic attentional function on which the capacities for both "higher" aspects of attention and of cognitive capacity in general are dependent (Sarter et al., 2001). This ability also plays a critical role for goal directed behavior (Staub et al., 2013) and possibly even consciousness. According to Sarter et al. (2001) psychological research on sustained attention has largely focused on parametric, construct-specific issues in neuropsychiatric populations or in sleep

deprived individuals (Staub et al., 2013) rather than on its implications for higher order cognitive functions such as learning and memory. Consequentially, data on normal awake subjects are scarce. The data that exist for both normal awake subjects and for clinical and sleep deprived subjects show that this cognitive ability is essential for functioning in everyday life, with impacts on modern living skills such as driving a car and on cognitive abilities such as detecting social cues or learning novel contingencies.

The Present Study

The aforementioned studies, together with the conclusion that "There is now sufficient evidence validating the role of EEG-neurofeedback in enhancing function to dispel the lingering vestige of prejudice against the value of this EEG methodology" (Gruzelier, 2013a, p. 15) have been essential to the construction of the present study. Our design was especially inspired by the studies of Egner and Gruzelier (2004) and Gevensleben et al. (2010) with the use of a healthy student population and the inclusion of an active control group. An active control group design was chosen in order to control for differences in mental effort as a possible explanation for any outcome differences between the two groups at posttest. Learning curves were calculated and inspected to ensure that both groups had indeed tried to master the task they were to perform, and that learning had in fact taken place. Any changes in favor of the experimental group at posttest would thus be attributable to the effects of neurofeedback and not to the fact that the groups had exerted differing levels of mental effort.

The main objective of this study was to investigate neurofeedback as a performance-enhancing tool in a healthy Norwegian student population. This was achieved by contrasting a beta1/theta protocol with computerized working memory training on outcome measures with reaction time (rt), reaction time variability (rtv), errors of omission and commission and P300 components on a sustained attention task (VCPT) performed during EEG recording. The hypothesis was that only neurofeedback would lead to significantly improved sustained attention.

Methods

Subjects

Data were collected from fifteen healthy adult students (five males, ten females, mean age: 23.5 years, standard deviation: 3.7) from the Clinical Psychology Program at the Norwegian University of Science and Technology (NTNU). The students were randomly selected from a group of 48 volunteers in a class who attended a mandatory course in applied EEG and neurofeedback. The subjects were randomized to participate in either ten sessions of

neurofeedback training (ten participants) or ten sessions of computerized working memory training (five participants). The participants received no monetary rewards, and the study was approved by The Regional Committee for Medical Research Ethics. All participants gave their written consent to partake in the study (see Appendix).

EEG Recordings

EEG recordings were conducted for all participants both prior to and following the ten sessions of neurofeedback or working memory training. The recordings were carried out using a 21-channel EEG system, produced by Mitsar Ltd.(http://www.mitsarmedical.com), with a cap containing 19 tin electrodes. The electrode cap included Fz, Cz, Pz, Fp1/2, F3/4, F7/8, T3/4, T5/6, C3/4, P3/4 and O1/2, in accordance with the 10-20 system (Jurcak, Tsuzuki, & Dan, 2007). Reference electrodes were placed on each earlobe. Data were sampled at 250 Hz. Participants were sitting up-right in a comfortable chair approximately 1.5 meters from a 22 inch computer screen during recordings so that the presented images were perceived at a visual angle of 9 degrees. The auditory system was calibrated for a 60 dB SPL. Recordings were performed by two student assistants, after rigorous training and under the supervision of a qualified EEG instructor. EEG recordings were performed during three conditions: Resting with eyes open for three minutes, resting with eyes closed for three minutes, and during a behavioral sustained attention task (VCPT).

Visual Continuous Performance Task

The behavioral task was a visual Go/NoGo task, consisting of 400 pairs of images presented on a computer screen. The task was divided into four separate sequences where each sequence lasted five minutes. The participants were given a two-minute break following each sequence. One sequence included 100 trials, and each trial consisted of two sequentially presented images, each displayed for 100 milliseconds, with an inter-stimulus interval of 1000 milliseconds. The inter trial intervals were 3500 ms.

Three categories of visual stimuli were used in this task: 1) 20 different images of animals, 2) 20 different images of plants, and 3) 20 different images of humans as well as an auditory stimulus. There were four different experimental conditions: animal-animal (Go), animal-plant (NoGo), plant-plant (Ignore) and plant-human (Novel) where a sound was presented together with the second image (see figure 1). Each sequence consisted of a pseudo - random presentation of 100 pairs of images, with equal probability for each category and for each experimental condition.

Animal-animal pairs (prime and target) were defined as the Go condition. Here the participants were instructed to press the left button on a computer mouse as fast as they could with their dominant hand. Animal-plant pairs represented the NoGo condition, where participants were to refrain from responding. For the plant-plant and plant-human conditions they were to refrain from responding. To ensure that all participants understood the task they were given a trial-run before recording was started.

All stimuli were presented at the center of the computer screen, had a visual angle of 9° and equal brightness. The instruction to respond as quickly as possible to the Go condition but making as few errors as possible was explicit for every participant. Recent research has demonstrated that results vary depending on the instructions given and on whether speed or accuracy is emphasized (Aasen, 2013; Band, Ridderinkhof, & Van der Molen, 2003). Care was especially taken to deliver the exact same instructions to all participants at both pretest and posttest to avoid such effects.
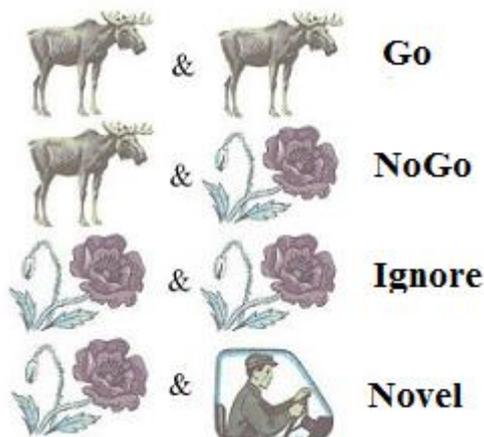
Figure 1: *VCPT conditions*

QEEG Measures and Feature Extraction

Individual mean reaction time (rt) and reaction time variability (rtv) was calculated post-experimentally based on valid Go trials with the WinEEG software. A response was considered correct if it occurred within a time window of 150-1000 milliseconds after the second stimulus presentation in a trial. Omission errors (not responding to the animal-animal conditions) and commission errors (pressing the button in NoGo conditions) were also calculated for each individual.

Target P300 amplitude (µV) and latency (ms) was measured by using the conventional peak measurement method described by Polich and Kok (1995) and Polich (2007). Peaks were detected and registered manually within a specified time window following the second stimulus (200-600 ms) and was obtained from the electrode site showing the greatest response

for each individual in the Go and NoGo conditions. Amplitude baseline was set at the 50 ms time period preceding the second stimulus onset. Differences on these variables between pre- and posttest were calculated separately for the experimental group and the control group, using a two-tailed paired-samples t-test.

Artefact Correction

Eye blink related artefacts were isolated by applying ICA (Independent Component Analysis) to the raw EEG material. ICA is a method for separating an assumed composite signal into subcomponents based on the assumption that there is a statistical independence between the components. This is also known as blind source separation. The ICA method may identify a unique spatiotemporal signal generator, like an eye blink and provide a template for its morphology. This template is thereby subtracted from the raw EEG resulting in a relevant artifact removal. ICA is also used for source localization of brain rhythms (Grin-Yatsenko, Baas, Ponomarev, & Kropotov, 2010; Tereshchenko, Ponomarev, & Kropotov, 2008).

Neurofeedback: Beta1/Theta Ratio

Ten participants performed ten neurofeedback training sessions, with an average of two sessions per week, for five consecutive weeks, using the program "Braintuner" Ltd. (http://www.mitsarmedical.com). Neurofeedback was performed using a cap with three silver electrodes, two of which were located along the central midline of the scalp, above the area of the anterior cingulate cortex. The third was located over the right temporal lobe, and was used as a reference electrode. The signals were transmitted to a computer via the Brain Tuner amplifier, and visual feedback consisted of a vertical blue pillar. The height of the pillar changed as a direct result of the participant's brain activity; it increased when there was a high beta1 to theta ratio, and decreased when there was a low beta1 to theta ratio.

All neurofeedback sessions started with a two-minute recording to calculate the baseline beta1/theta ratio, with the participant seated in a comfortable chair about 1.5 meters from a 22 inch computer screen. Each session consisted of five training blocks, each of five minutes duration. Following each block was a one minute break, in which the pillar was in a fixed center position. The word "relaxation" or "training" was displayed near the top of the screen to indicate the current condition.

A horizontal line divided the screen plain in half, and the objective for the participant was to keep the pillar above the midline as much of the time as possible during training sessions. Relaxation sessions with duration of one minute were interleaved where the participants were instructed to not concentrate on anything in particular. Figures displaying

the percentage of time that the column had been above the midline were presented in the top right corner of the computer screen for each training and relaxation period.

The workings of the feedback-loop were explained to each participant, and they were instructed to let themselves be guided by the feedback in order to learn how to affect the height of the pillar in the desired direction. The goal of the training sessions was for the students to learn to regulate the amount of beta1 activity relative to the amount of theta activity, visually illustrated as keeping the blue pillar above the midline during the training condition. They were also instructed to lower beta1 activity relative to theta activity during the relaxation condition. Between sessions the students were encouraged to practice the state of elevated beta1 activity as a homework assignment, in order to maximize transfer effects.

At the end of each session the amount of beta1 activity relative to the amount of theta activity for each training block and each relaxation period was displayed graphically on the computer screen. Beta1 percentage for all five training blocks (T) and all relaxation periods (R) were also presented numerically (see figure 2).
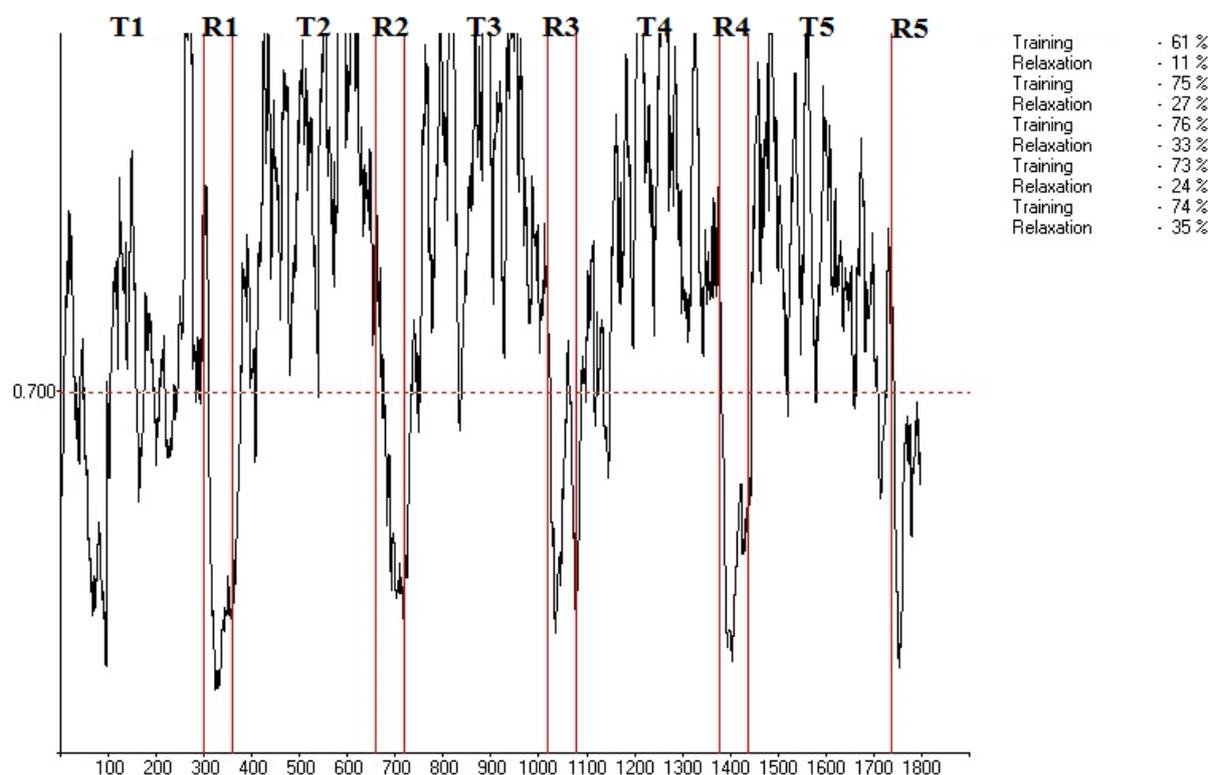

Figure 2: *Graphical and numerical presentation following a neurofeedback session*

Computerized Working Memory Training

The control group consisted of five students, performing ten sessions of computerized working memory training over a period of five weeks. Each session lasted approximately 25 minutes. The task performed was an n-back test, where the subjects were instructed to report

the location of a square stimulus after a given number (n) of subsequent stimuli. The computer screen showed a 3x3 matrix, and the location to remember was presented visually at the same time as a letter was presented auditorily. The participant had to couple the letter and the location, and as the participant improved the task was made progressively more difficult by increasing the n. This sort of working memory task has been found to increase the capacity of both verbal and visuospatial working memory in healthy individuals (Alloway, Bibile, & Lau, 2013), as well as to reduce core features of learning disabilities in adolescents born at an extremely low birth weight, partly through increased verbal learning (Løhaugen et al., 2011). Takeuchi et al. (2010) reported that working memory training led to increased working memory capacity, as well as to increased structural integrity of white matter in brain areas closely related to working memory functions.

Statistical Methods

The Student *T*-test was used to test for differences between the two groups with equal variances assumed, unless Levene's test showed significant deviation from equal variances. All behavioral data from the VCPTs were analyzed using IBM SPSS 21.0 (SPSS Inc; Chicago, Illinois). Mean and standard deviation was computed for each parameter/variable, and the significance level was set at p = .05 (two-tailed). Independent samples t-tests were conducted to examine success in matching the two groups (age, gender, rt, rtv, errors, P3Go latency (ms), P3NoGo latency (ms), P3Go amplitude (μV), P3NoGo amplitude (μV)). Paired samples t-tests were performed to calculate intragroup pre-posttest differences in the experimental group and in the control group, and effect size was calculated as Cohen's *d* for paired samples t-test ($d = \frac{\bar{X}_D}{S_D}$). Independent samples t-tests were used for intergroup comparison of the posttest results. A difference score (Diff) for each of the VCPT variables was also calculated by subtracting the pretest value from the posttest value for each individual.

The average beta1 training percentage (TP) and the average beta1 relaxation percentage (RP) during each of the ten neurofeedback sessions were calculated for all individuals in the experimental group to assess if, and how well each participant had learned to regulate their beta1/theta ratio. The difference between average beta1 TP and RP for each session was also calculated in order to assess whether the participants became better at both increasing beta1 activity during training as well as increasing theta/reducing beta1 activity during relaxation, so as to achieve better control of the production of brain rhythms. The data were collected manually from the Braintuner software, and the necessary calculations were

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performed in Microsoft Excel (Microsoft Corp.). A learning curve was also computed for each participant.

Two outcome variables, indicating the degree of successful learning of the neurofeedback protocol, were calculated from the neurofeedback data: NF1) the change in average TP over time, and NF2) the change in average TP-RP over time. This was accomplished by averaging the relevant scores achieved from the first two sessions and from the last two sessions of neurofeedback training. The NF1 variable was calculated by averaging the TP scores (T1 through T5, see figure 2) for session one and two, making up a single "TP-early score". The same procedure was performed for session nine and ten, constituting a single "TP-late score". The TP-early score was then subtracted from the TP-late score to calculate any change that might have occurred during the neurofeedback training period. A positive value on this outcome variable would indicate heightened beta1 activity at the end of the neurofeedback training relative to the early stages of training.

The NF2 variable was calculated by averaging the RP for all five relaxation periods for both session one and two (RP-early), and for session nine and ten (RP-late). The average difference between TP and RP was calculated for early sessions (one and two) and late sessions (nine and ten), and the early score was then subtracted from the late score. A positive value on this outcome variable would indicate a greater difference between TP and RP at the end of the neurofeedback training relative to the early stages of training, and thus successful neurofeedback learning.

The Pearson's product-moment correlation coefficient was calculated in SPSS for the experiment group to assess the relationship between differences in the pre- and posttest outcome variables (rt, rtv, errors, P3go and P3nogo (latency and amplitude)) and the two neurofeedback learning variables (NF1, and NF2).

Results

Demographic and Clinical Characteristics

The two groups showed similar characteristics on the demographic variables, with age marginally shifted downwards in the control group. There were no statistically significant differences between the two groups at pretest, and so the null hypothesis was not rejected. The criterion for homoscedasticity was fulfilled, with Levene's test for equality of variances showing $p > 0.05$ for all variables. Demographic and pretest data for both groups are presented in table 1.

Table 1: *Demographic and pretest variables*

	Neurofeedback (N = 10)			Control (N = 5)			T-test for equality of means		
	Cases (%)	Mean	SD	Cases (%)	Mean	SD	t-verdi	df	p-verdi
Sex							-0.36	13	0.72
Female	7 (70)			3 (60)					
Male	3 (30)			2 (40)					
Age (yrs)		24.7	4.0		22.2	2.3	1.27	13	0.23
RT (ms)		264.8	19.1		254.6	27.2	0.85	13	0.41
RTV		5.3	1.1		5.1	1.8	0.23	13	0.82
Error		1	1.3		0.8	0.8	0.32	13	0.75
P3Go (ms)		314	25.0		304	16.0	0.81	13	0.43
P3NoGo (ms)		314	26.3		312.8	22.9	0.09	13	0.93
P3Go (µV)		12.1	3.1		12.7	2.0	-0.38	13	0.71
P3NoGo (µV)		15.9	3.6		16.4	2.9	-0.27	13	0.79

Note: df = degrees of freedom; ms = milliseconds; µV = millivolt; RT = reaction time; RTV = reaction time variability;

SD = standard deviation

Table 2: *Posttest variables*

	Neurofeedback (N = 10)		Control (N = 5)		T-test for equality of means		
	Mean	SD	Mean	SD	t-verdi	df	p-verdi
RT (ms)	240.0	15.4	243.6	18.7	-0.40	13	0.70
RTV	4.4	1.4	3.9	1.0	0.69	13	0.50
Error	0.7	0.8	1.8	1.8	-1.31	4.87	0.25
P3Go (ms)	308.8	35.5	311.2	11.1	-0.15	13	0.89
P3NoGo (ms)	307.2	13.8	308.0	20.2	-0.09	13	0.93
P3Go (µV)	10.7	2.3	11.7	1.8	-0.84	13	0.41
P3NoGo (µV)	16.4	4.3	18.1	3.7	-0.76	13	0.46

Note: df = degrees of freedom; ms = milliseconds; µV = millivolt; RT = reaction time; RTV = reaction time variability;

SD = standard deviation

Intragroup Changes

Neurofeedback. A paired-samples t-test was conducted to compare the results of the neurofeedback group at pretest and posttest. There was a statistically significant difference between the scores for reaction time; rt_1(M = 264.8, SD = 19.1) and rt_2(M = 240, SD = 15.4); t(9) = 3.7, p = 0.005. These results suggest that neurofeedback really does have an effect on how fast we react. Specifically, our results suggest that when healthy psychology students learn neurofeedback their reaction time decreases. Cohen's effect size value (d = 1.16) suggests a high practical significance of this improvement in reaction time.

A Pearson product-moment correlation coefficient was calculated to assess the relationship between the VCPT difference variables and the two neurofeedback variables. There was a strong positive correlation between the variables P3NoGo(µV)diff and NF1, r = 0.82, n = 10, p = 0.004. This result suggests that the more a participant was able to heighten his/her average training percentage (TP) over the neurofeedback sessions the greater increase the participant showed on P3NoGo amplitude from pre- to posttest.

There was a moderate positive correlation between the two variables P3Go(µV)diff and P3NoGo(µV)diff, r = 0.64, n = 10, p = 0.048. This result suggests that those who showed increased P3NoGo amplitude at posttest also showed increased P3Go amplitude.

There was also a moderate positive correlation between the two variables NF1 and NF2, r = 0.64, n = 10, p = 0.048. This result suggests that those who became better at increasing their beta1 activity during the neurofeedback training conditions also became better at enhancing theta/reducing beta1 activity during the relaxation conditions, and thus achieved a better overall conscious control over the production of brain rhythms.

Control group. A paired-samples t-test was conducted to compare the results of the control group at pretest and posttest. There were no statistically significant differences.

Intergroup Changes

An independent samples t-test was conducted to compare the experiment group and the control group at posttest. Posttest data for both groups are presented in table 2. Levene's test for equality of variances shows p<0.05 for the error variable, thus equal variances were not assumed for this variable. There were no statistically significant differences between the two groups at posttest, and thus the null hypothesis could not be rejected.

<div align="center">

Discussion

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The main aim of this study was to examine the legitimacy of neurofeedback as a performance enhancing intervention in healthy psychology students. This was done by

exploring the differential effects of neurofeedback and computerized working memory training on sustained attention, using a visual continuous performance test as a pretest-posttest metric. To reiterate, we had hypothesized that only neurofeedback would lead to enhanced sustained attention. In accordance with our hypothesis we found that only the experiment group showed a statistically significant decrease from pre- to posttest in reaction time ($p = 0.005$) which suggests that neurofeedback really does have an effect on how fast we react, as opposed to working memory training where no such improvement was found ($p = 0.29$).

Specifically, our results suggest that normal healthy individuals can learn to increase a specific component of their EEG activity by way of neurofeedback, which in turn may facilitate attention processes and lead to a significant improvement in reaction time. This is in line with existing research from the peak performance field (Egner & Gruzelier, 2001, 2004; Ros et al., 2009; Vernon et al., 2003) and parallels results reported by Egner and Gruzelier (2004) when using the same beta1 protocol. The effect size value ($d=1.16$) suggests a high practical significance of this improvement, pointing towards the encouraging possibility that a relatively modest amount of sessions of neurofeedback training can lead to significant everyday gains in sustained attention and reaction time in highly functioning individuals.

In regards to the degree of successful learning of the neurofeedback protocol, our data show that the more voluntary control participants were able to achieve over beta1 activity during training, the more voluntary control they were able to assert in the relaxation conditions, as indexed by increased beta/theta ratio. This in turn confirms that the beta1 protocol does not just lead to an overall increase in beta1 activity, but to a more flexible and dynamic regulation of the targeted brain rhythms, and thus better overall conscious control of the targeted EEG activity.

It has previously been reported that healthy participants show increased P3 target amplitudes following neurofeedback learning, using both SMR protocols and beta1 protocols (Egner & Gruzelier, 2001, 2004). These results have been interpreted to indicate that learning of both protocols is associated with improved integration of relevant environmental stimuli. The results from the current study replicate such findings, as our data also suggest that participants who were able to learn neurofeedback, as indexed by the increase in beta1/theta ratio over the ten sessions of training, also show a stronger inhibition response in NoGo tasks, as evidenced by increased power of the P3 NoGo component at posttest ($r = 0.82$). Increase in P3 NoGo power was also associated with an increase in P3 Go power ($r = 0.64$) in the experiment group. In light of the research by Brunner et al. (2013) on the test-retest reliability of the P3 NoGo amplitude it seems safe to conclude that the effect on the P3 amplitude moves

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beyond mere test-retest effect, and can thus be attributable to the successful learning of neurofeedback.

Previous research has yielded some diverging results concerning the frequency specific effects of the beta1 protocol on behavioral measures. In 2001, Egner and Gruzelier reported that beta1 learning was associated with outcomes indicative of enhanced cortical arousal or excitation, which in healthy participants lead to arousal levels beyond those required for optimal performance. They interpreted the increase in P3 amplitude to reflect a higher general cortical background excitation rather than an enhancement of the specific neural processes associated with the P3, such as processing capacity or resource allocation (Polich, 2007; Polich & Kok, 1995). In Egner and Gruzelier (2001) a negative correlation between beta1 learning and a decrease in commission errors was reported, but this result was not replicated by Egner and Gruzelier (2004), though they did replicate the association between beta1 learning and P3 increments.

In accordance with Egner and Gruzelier (2001, 2004) the present study did show statistically significant reductions in reaction time following beta1 learning. No negative effect of beta1 learning on the error variable was found in the present study however, which is in accordance with the findings of Egner and Gruzelier (2004), but not those reported by Egner and Gruzelier (2001). Thus, our results lend support to the use of the neurofeedback beta1 protocol as a means to optimize performance in healthy individuals, possibly by way of enhancing resource allocation and/or processing capacity. There is also the possibility that performance was enhanced due to an optimal increase of cortical arousal following neurofeedback. As described by Polich (2007) and Polich and Kok (1995) the P3 amplitude is affected by many factors, both cognitive and biological, and increased arousal has been shown to lead to increments in the P3 amplitude. This interpretation of the results could also explain the positive effects of the beta1 protocol in attentional disorders, and would be in accordance with the proposal that beta1 training may serve to increase cortical excitation in under-aroused ADD/ADHD samples (Lubar, 1991).

Contrary to what we hypothesized, our data show no statistically significant differences between the experiment group and the control group on any of the outcome variables at posttest. These results could suggest that there is no advantage to neurofeedback over computerized working memory training on improving variables of attention. Another possible explanation is that there are in fact differences between the two interventions, but that such differences are masked by either the small sample size used in this study or by the relatively modest number of neurofeedback sessions. This explanation is substantiated by the

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fact that the experiment group, but not the control group, showed a statistically significant reduction in reaction time at posttest (p = 0.005), which is a surprisingly high significance level in a sample consisting of only ten participants.

Caution is necessary however, when interpreting the improved reaction time as solely caused by neurofeedback learning. Another factor that may well be at least partially responsible for this reduction is the relatively short time period between pre- and posttest, ranging from 6 to 7 weeks. Such a short test-retest time interval can increase the risk of carry over effects, and statistically significant decreases in reaction time have been reported without any intervention with test-retest time intervals ranging from 6 to 18 months (Brunner et al., 2013).

The study by Brunner et al. (2013) used a sample more than twice the size of that in the present study. They found average reductions in reaction time of 7 ms and a decrease in SD from 17 to 14 from time1 to time2 using the same Go/NoGo paradigm as the present study. In our study, the average reduction in reaction time was 24.8 ms for the experiment group and the SD was greater at both pre- and posttest (19.1, and 15.4 respectively) compared to the sample of Brunner et al. These data show that even though the sample size in the study by Brunner et al. is more than twice the size of that in the present study, the variance between participants is greater in our study, especially at pretest. This is probably to some extent caused by our limited sample size, which allows for potential outliers to have a greater effect on the mean and SD. However, Brunner et al. does not present the effect size for the statistically significant change in mean reaction time, making it difficult to compare the practical significance of this improvement with the improvements found in our study. What is clear, just from looking at the numerical change, is that the improvements we found following neurofeedback by far exceeds those found as a mere test-retest effect by Brunner et al. The fact that the active control group in the present study does not show any statistically significant improvements further supports that at least a part of the positive effect on reaction time and sustained attention can be ascribed to the neurofeedback training.

When further examining the data, another explanation for the lack of statistically significant outcome differences between the control group and the experiment group emerges. Although the control group show decreases in reaction time, they also show an increase in errors made on the behavioral task (VCPT). This could suggest that the control group in fact has not improved, but rather that they at posttest exhibit a higher level of arousal and that they have changed their response criteria in such a way that they decrease their reaction time at the expense of making more errors. This kind of fast but inaccurate response tendency has been

associated with increased arousal in a noradrenergic alertness/vigilance attention network (Egner & Gruzelier, 2001; Posner & Petersen, 1990). In comparison, the experiment group shows a trend indicating greater accuracy following the ten sessions of neurofeedback. This interpretation supports the hypothesis of there being beneficial effects of neurofeedback over computerized working memory training, with a statistically significant and practically meaningful improvement in reaction time and a nonsignificant decrease in the mean number of errors made, including a decrease in standard deviations from the mean, as was hypothesized in this study.

Limitations

The restricted sample size offers a strong limitation to this study, concerning both the results and their interpretation, and their generalizability. The limited number of participants in the study was mainly due to the limited time and resources offered by the course that created the framework for this research project. The fact that the sample was selected from a single class of psychology students may also limit the generalizability of the results to other populations.

Another limitation to this study is the fact that the control group is smaller than the experimental group. Ideally, the groups should be matched for number of participants, but due to restricted resources this was not feasible. The inclusion of a passive control group could also have contributed to the strength of this study, by allowing for comparison between the neurofeedback intervention and possible test-retest effects on the VCPT. Furthermore, the modest number of neurofeedback sessions could explain the lack of statistically significant intergroup differences at posttest. This is supported by Doppelmayr and Weber (2011) who found that 30 sessions was not enough to ensure beta1/theta protocol learning in their study of healthy individuals. On the other hand, Egner and Gruzelier (2001) reported learning effects after ten neurofeedback sessions, although this was with a larger number of participants than what was used in the present study.

The restricted amount of time separating the pretest and the posttest can also be seen as a limitation, as this can increase the risk of carry over effects and thus contaminate the results. Despite the short test-retest interval the control group in this study did not show any statistically significant changes at posttest, suggesting minimal carry over effects.

Conclusions

The beta1/theta ratio protocol demonstrated statistically significant and practically meaningful improvements on reaction time in a healthy student population. The improvement

in reaction time is a result of improved sustained attention and enhanced hand-eye coordination, skills that play a significant role in everyday life. Hand-eye coordination is important when we pick up a book, when we interact with other people, and when we are driving a car or a motor cycle, and has also been demonstrated to be associated with cognitive and social skills in children (Yu & Smith, 2013). Improved hand-eye coordination can thus have practical implications for all these tasks and abilities, for instance it has important implications for athletic performance, such as in a variety of ball games.

The ability to react to stimuli in a fast and precise manner can be especially important in unexpected situations to avoid accidents. For instance can a decrease in reaction time affect the distance needed to stop your car if a child suddenly runs into the street. Sustained attention also plays a significant role in a wide variety of cognitive abilities, such as detecting social cues, which in turn is necessary for effective communication. Sustained attention is also paramount when learning new skills, especially when the complexity of the skill increases. As such, neurofeedback can indeed be said to have a significant role in optimizing performance in healthy individuals, with important implications for everyday life.

Implications for Future Research and Clinical Practice

To be able to further investigate the positive contribution of neurofeedback in optimizing performance in healthy individuals more studies are needed, with larger samples and a more diverse population than what was used in the present study. Larger samples would offer greater statistical power and more reliable results, as would randomized controlled studies, being the gold standard in scientific research.

It may also be fruitful to investigate the impact of a greater number of neurofeedback sessions on variables of sustained attention. Studies that compare groups receiving differing amounts of neurofeedback training should be completed in order to try to establish how many training sessions are needed to yield optimal results regarding peak performance.

Concerning implications for clinical practice, neurofeedback can be used to optimize performance in healthy individuals, with benefits for both the individual and the larger society. Neurofeedback can be utilized as a means to optimize athletic performance, both in novices and in elite performers, and could also be of value in formal learning facilities, both as a means to achieve peak performance but also as a tool for aiding pupils who perform below par. The significance of the ability to sustain attention for modern living skills such as driving a car, and for cognitive abilities such as learning novel contingencies and detecting

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social cues, deems neurofeedback an important instrument for optimizing performance, and possibly as a means to restore loss of function due to injury or aging.

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USE OF NEUROFEEDBACK TO OPTIMIZE PERFORMANCE

Appendix

Informasjon og forespørsel om deltakelse i studiet:

"Effekter av nevrofeedback trening i normalpopulasjon"

Bakgrunn og hensikt med studien

Dette er en forespørsel om å delta i et forskningsprosjekt for å kartlegge effekter etter nevrofeedback trening, målt med kvantitativ elektroencefalografi (qEEG) og en oppmerksomhetstest. Prosjektet er godkjent av Regional komité for medisinsk og helsefaglig forskningsetikk.

Hva studien innebærer for deg

Hvis du sier ja til å delta i studien, innebærer det at du frigjør data fra labøvelsen som du deltok i på kurset psypro4412. Dvs, det er kun ditt resultat fra nevrofeedback trening og data fra qEEG-opptaket som vi ber om tilgang til. Ditt navn vil ikke knyttes til disse data eller benyttes i noen annen sammenheng.

Det er ingen risiko forbundet med disse undersøkelsene.

Slik ivaretas ditt datamateriale og personopplysninger

Forskningsdata lagres elektronisk ved laben, og kun personer med relevant tilgang til studien har tilgang til disse. Ditt navn vil ikke kunne knyttes til datamaterialet som inngår i prosjektet. Vedkommende som behandler data er underlagt taushetsplikt og er kjent med forvaltningsloven § 13 sammenholdt med § 13a, 13b, 13c og 13f. Alle opplysninger vil bli behandlet konfidensielt og i henhold til Helsinki- deklarasjonen.

Dine rettigheter

Du har rett til å få innsyn i hvilke opplysninger som er lagret og registrert om deg. Du har videre rett til å få korrigert evt. feil i de opplysningene vi har registrert. Hvis du senere trekker deg fra studien, kan du kreve materialet slettet. Du kan også kreve å få slettet alle opplysninger vi har registrert. Ved henvendelse til prosjektansvarlig kan du få nærmere opplysninger om dette. Du kan ikke få slettet opplysninger eller destruert materiale dersom de er anonymisert, er viderebehandlet eller dersom opplysningene allerede er inngått i vitenskapelig arbeid.

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Du bestemmer selv

Det er frivillig å delta i studien. Dersom du velger å ikke delta, er det ikke nødvendig å oppgi grunn. Du kan til enhver tid trekke deg fra studiet. Om du skulle bestemme deg for å ikke delta, får dette ingen konsekvenser for deg nå eller i fremtiden.

På grunn av arbeid med å etablere en qEEG-database er det av interesse å ta vare på EEG-data etter opptak. Dine data er anonymisert og kan ikke knyttes til deg på noen måte. Dersom du likevel ønsker å reservere deg fra at dine qEEG-data lagres etter prosjektslutt har du mulighet til dette når du fyller ut samtykkeskjema.

Prosjektansvarlig / mer informasjon

Prosjektleder: Førsteamanuens Stig Hollup, Psykologisk Institutt, NTNU

Hvis du har spørsmål om studien eller trenger informasjon utover den i kapittel A og kapittel B, kan du ta kontakt med Stig Hollup: stig.hollup@svt.ntnu.no eller tlf 97044042

Samtykkeskjema for studien

" Effekter av nevrofeedback trening i normalpopulasjon"

Deltakelsen i studien er basert på ditt frivillige, informerte samtykke. Dersom du ønsker informasjon utover det som fremkommer i informasjonsskrivet, har du fullstendig anledning til å be om det. Dersom du etter å ha fått den informasjon du synes er nødvendig, sier ja til å delta i prosjektet, bes du signere samtykkeerklæringen. Du kan når som helst, og uten begrunnelse be om at alle data innhentet fra deg, slettes.

Jeg, _____ (navn med blokkbokstaver), bekrefter at jeg har mottatt skriftlig informasjon om studien, har fått anledning til å innhente den informasjon jeg har hatt behov for, og er villig til å delta i prosjektet.

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Jeg samtykker i at data fra mitt qEEG-opptak og nevrofeedback trening kan tas vare på etter prosjektslutt: ja ☐   nei ☐

Sted / Dato   Signatur prosjektdeltaker:

Trondheim, 05.06.2014