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Reading the mind: a critical evaluation of neurofeedback as a tool in clinical neuroscience

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Trying to resolve the enigmas of the brain has been one of the many attempts in cognitive neuroscience the past century. The crux is to find out what is happening in the brain without intervening in the numerous processes that occur simultaneously. One of these non-invasive techniques is electroencephalography (EEG) and it is based on the notion that neurons and synapses communicate by electrical activity. This happens as a result of the constant ionic current fluctuations that occur when a neuron reaches firing potential threshold. The combined activity, both spontaneous and evoked, can be recorded by electrodes placed on the scalp. The recorded activity can then be displayed on a time or frequency spectrum to visually distinguish the enormous amount of neuronal activity.

While this method is nearly a century old it is certainly not outdated but, instead, it has become one of the foundations for brain measurement research in cognitive neuroscience and the original drug research that led to the discovery of human EEG signals by German physiologist Hans Berger in 1928 is still being replicated in modern times (Herning et al., 1985; Herning et al., 1994). The original experiments displayed just the recordings of a single electrode but nowadays continuous recording is usually performed with 32 to even 256 channels simultaneously in order to receive a complete recording that receives neuronal activity from most cortices. The raw recordings are often quite weak and need to be amplified to gain a better signal-to-noise ratio (Jackson & Bolger, 2014). The rhythmic activities in the amplified recordings then can be transformed from a time spectrum to a frequency spectrum by means of a fast Fourier transformation (FFT) to analyze which band of frequencies are present in the multi-channel signal (Kaiser, 2005).

Generally, the frequency spectrum of EEG measurements is divided into different bands based on the topographic origin on the scalp or the biomarkers associated with them. The Delta band (1-4 Hz) is associated with dreamless periods of sleep. The Theta band (4-7

Hz) is associated with drowsiness and meditation, while the Alpha band (8-14 Hz) portrays relaxation and mental control. Furthermore, the fastest frequency band, the Beta band (15-30 Hz) occurs during active movement and deep concentration. The specific frequency borders, band representations and functions, however, are subject to a continuing discussion that probably will not be resolved for decades (Klimesch, 1996; Başar et al., 2001; Palva & Palva, 2007).

First line of research

Along with this extensive line of research another branch of research sparked when the brain behavior link was established. This second branch was established in the in the sixties of the previous century when Kamiya (1969) performed operant condition on participants that were asked to evaluate their current level of Alpha band activity. The participants were rewarded when they were correct and therefore learned to implicitly control their brain activity. This type of research was the first to use neuronal feedback to condition participants to exert control over their Alpha band activity, establishing a link between conscious thought and neuronal activity.

After the first experiments neurofeedback research has seen a massive spike and has found a role in clinical therapy as well (Butnik, 2005). But the foundation has remained the same. The whole concept revolves around providing information back to the participant about the current brain state. This can be done in the form of visual and auditory feedback, or in the form of a game (Dongen-Boomsma et al., 2013; Butnik, 2005). Similar to classical conditioning experiments, positive reinforcement is provided for desired brain activity and negative reinforcement for undesired brain activity. While the fundamentals of the paradigm are the same in every study, applications range from childhood attention deficit hyperactivity disorder (ADHD) therapy (Fuchs et al., 2003) to memory enhanced training for the elderly (Lecomte & Juhel, 2011). However, not every experiment has been able to yield significant

results and criticism on the experimental design and methodology is voiced as well (Rossiter, 2004). Furthermore, clear methodological protocols in the neurofeedback field are absent and methodological differences between studies make it difficult to maintain a clear perspective of the current state of neurofeedback as a clinical tool.This essay will therefore try to unravel the methodological tangles and attempt to establish what is currently possible and point out the current issues surrounding the field. For example, since neurofeedback therapy embraces a vast variety of methods and a practitioner does not need a special neurofeedback license, numerous therapies are offered throughout the world without clear scientific proof and background. Therefore, a clear distinction must be made between empirically based studies on the therapeutic effectiveness and the therapeutic claims that are made without substantial evidence.

The literature in this essay will mostly originate from ADHD research since this is the field where most studies are conducted due to the neural symptoms of ADHD. For example, one of the consistent finding in the neurological symptoms of ADHD is an increased activity in the theta EEG band and a decreased acitivity in the beta EEG band (Arns, Conner, & Kraemer, 2011). But it must be noted that the nature of neurofeedback paradigms allows it to be easily transferred to other therapeutic research for conditions that demonstrate similar sets of neurological symptoms, such as Tourette syndrome which is characterized by excessive theta band activity (Messerotti Benvenuti et al., 2011).

First, this essay will dissect what has been empirically established with therapeutic neurofeedback. It will discuss a set of papers which can be regarded as exemplary for the field. Then the author will attempt to validate the claims that are made by various commercially available therapies. After that, the author will continue to point out the gap between the claims and the literature. Finally, the topic will move away from the therapeutic applications and discuss the alternative utilizations of neurofeedback.

Neurofeedback as ADHD treatment

The relation between mental states and brain waves is most apparent within children and adolescents that suffer from ADHD. It is often found that the brain produces more slow frequency band activity, which is associated with low alertness, and less high frequency band activity, associated with mental arousal (Monastra et al., 1999; Butnik, 2005). Moreover, Lubar et al. (1995) raised great expectations by developing a theta-alpha ratio based on QEEG measurements, quantitative measurements that displays the summation of activity as opposed to the localization of specific activity, where participants with ADHD showed a higher ratio than their healthy counterparts. Chabot & Serfontein (1996) continued this line of work by creating profiles based on QEEG meaurements, they were able to correctly identify 93% of the participating ADHD subjects based on the brain wave activity alone.

Monastra et al. (1999) further substantiated the classification by correctly assessing 86% of the participants with ADHD based on QEEG alone. This assessment was performed by calculating age group dependent critical standard deviation values based on the normal EEG output. If a ratio exceeded this critical value, a participant was classified in the ADHD group. The authors, however, have not provided details for the filtering and FFT techniques used, making it difficult to critically evaluate the quality of their recorded EEG data which was a mere 6 min per participant, divided over the four tasks that had to be performed during the measurement; reading, listening, drawing and sitting idle. Furthermore, in the younger age groups, the amount of participants in the ADHD groups vastly outnumbers the control group (213 compared to 30 in the youngest group and 91 compared to 34in the second cohort) which is remarkable since the whole classification is based on healthy participant data, so random fluctuations during measurement would hamper the calculated critical ratio values.

These findings formed the foundation for further neurofeedback therapy where the goal is to train the individual to normalize EEG patterns and recognize the mental state

associated with these patterns. For ADHD therapy, since most patient groups involve children and adolescents, the rewards used for the correct behavior is often given during a game (Butnik, 2005). Clients are trained to reduce the amount of slow wave activity and increase the amount of fast wave activity. In order to assess a significant contribution to the reduction of ADHD symptoms, Fuchs et al. (2003) compared behavioral results between children that were treated with the commonly used methylphenidate and children that received three months of neurofeedback training. The training consisted of 30 to 60 minute sessions, three times per week, during which the children played a game similar to the classical Pacman maze game. Four different frequency bands were determined, with the theta (4-7 Hz) and beta2 (22-30 Hz) bands as unwanted, and the sensimotor rhythm (SMR, 12-15 Hz) and beta1 (15-18 Hz) as wanted bands. Threshold power levels were determined at 0.5 and 1μV lower or higher from baseline activity, depending on the wanted frequency band. The participant received positive visual reinforcement when the power in the preferred band exceeded the threshold 60% of the samples in a 500ms epoch of EEG measurement. When this reward was obtained, the Pacman-like icon gained brightness and speed in the game. But when the threshold was exceeded in the unwanted bands the icon stopped moving and turned black. Further description of the reward classification and calculation is unfortunately omitted from the paper, making it difficult to evaluate how the desired frequency band is transferred on screen into the game.

After three months of therapy the authors report increased scores on the test of variables of attention (TOVA) tasks, both the group that was treated pharmacologically and the group that received neurofeedback training displayed similar results. Furthermore, parents indicated a lower symptom score after treatment. Although the authors point out the possibility of learning effects that attributed to the score difference after treatment, they do not refute that possibility. Furthermore, no EEG measurements from the neurofeedback training

are provided within the paper to help the reader to analyze the results. This makes it difficult to compare the power spectrum before and after training. When such an analysis can be performed, the link between understanding brain wave patterns and behavior will be strengthened, but for now it is difficult to rule out other causes that can attribute to the effect.

An alternative explanation for the reported effect could be a placebo effect due to the methodological design of the study. Parents of the children that participated could choose between a pharmacological treatment and neurofeedback treatment, crossing the golden standard in clinical experiments of randomized control studies. Therefore, the participant's interaction with the researcher and their parents could have influenced the outcome. Moreover, no follow-up study was performed so there is no indication of lasting effects as a result of the neurofeedback training. These, methodological flaws seem to be the underlying cause of critique on neurofeedback as a treatment.

Methodological shortcomings

The studies that have been conducted can be divided into two types of semi-randomization; probably not blinded assessment, often made by a parent or caretaker, and probably blinded assessment, often made by a teacher that is unaware of the treatment (Arns et al., 2009; Micoulaud-Franchi et al., 2014). According to a meta-analysis merely five out of twelve clinical experiments used a double-blind randomized control set-up (Micoulaud-Franchi et al., 2014). Out of these five studies, four used the theta-beta ratio as the learning protocol. Furthermore, none of the five studies used the same electrode configuration and three different control conditions were used. Only one study (van Dongen-Boomsma et al., 2013) used an identical setup between the experimental and the control condition, providing the participant with sham neurofeedback, therefore diminishing confounding variables such as the amount of interaction with the experimenter and the presence of electrical devices as a placebo effect the most.

Moreover, three out of five studies used different ADHD symptom measurements, and the percentage of pharmacologically treated participants differed from 0% to 50% between the studies, while it has been found that methylphenidate influences baseline theta-beta ratios (Loo, Teale, & Reite, 1999). Children that respond positively to methylphenidate treatment show a decreased theta and alpha wave amplitude and an increased beta activity, resulting in a decreased theta-beta ratio. On the contrary, children that do not respond to pharmacological treatment show the opposite effect, increasing their baseline theta-beta ratio. This implies that experimenters should not only take into account if the child has had methylphenidate treatment, but also whether the treatment was successful and when the last dose has been administered.Furthermore, it becomes unclear which part of the behavioral improvements is due to the successful or unsuccessful methylphenidate treatment, the neurofeedback session or a placebo effect due to the mere attention for the child. This huge variation in method combined with the low amount of studies makes it difficult to perform a convincing meta-analysis.

One of the few studies that adhered to the golden standard of double-blind, randomized, placebo-controlled setups was conducted by van Dongen-Boomsma et al. (2013). In this study, forty-one children diagnosed with ADHD were randomly assigned to EEG-neurofeedback therapy or a sham-neurofeedback therapy training. The sham condition could not be identified above chance level by parents or children. Futhermore, none of the participating children had participated in a neurofeedback study before and one of the inclusion criteria was a deviating EEG-signal typical for ADHD symptoms (Monastra et al., 1999). Despite the careful selection of participants and methodologically sound experiment design no difference in treatment group was found. Both groups showed equal improvement on the overall ADHD symptoms scores, suggesting that the mere attention and focus on the child can attribute the improvements. The lack of significant condition differences is in line

with earlier placebo-controlled neurofeedback experiments (Perreau-Linck et al., 2010; Arnold et al., 2013). Both studies did find a significant improvement for ADHD symptoms over time, again suggesting that motivation and expectancy of both children and parents may play a bigger role then the neurofeedback therapy. Furthermore, a consistent dilemma is the lack of participants willing to complete multiple neurofeedback session for months (van Dongen-Boomsma et al., 2013). On top of that, every child that partook in one of the studies was on some form of psychostimulant medication, including the widely used methylphenidate, possibly influencing the baseline theta-beta ratio prior to feedback treatment. This causes another dilemma for the field since discontinuation of medication treatment is ethically questionable and it would be difficult to find adults that would permit discontinuation for several months. As a result, participating children received both methylphenidate and neurofeedback treatment, convoluting the ability to detect behavioral improvemet due to neurofeedback. Unfortunately, as van Dongen-Boomsma et al. (2013) remark, psychostimulants as ADHD treatment are so widely used that the inclusion of children without medication would hamper the generalizability of the results.

To summarize, the issue for the field of ADHD research is not only the lack of a consistent therapy protocol and sound methodological design but also the lack of proof that links neurofeedback therapy with lasting cortical changes which exclusively can be the result of neurofeedback therapy (Moriyama et al., 2012). Therefore, proof must be found outside the ADHD field, with an alternative population sample, in order to evaluate the merit for ADHD therapy.

For example, Cannon & Lubar (2011) found significant EEG baseline changes in terms of absolute power of the beta frequency band between measurements directly afterneurofeedback training and a follow-up measurement several months later, along with improvements on working memory and processing speed scores. EEG source localization

suggested that the core of these lasting cortical changes occurred in the anterior cingulate cortex. Additionally, it has been found that neurofeedback enhances cognitive control by means of gamma band activity training (36-44 Hz; Keizer, Verment, & Hommel, 2010). According to the authors, the base of the enhancement lies in local neural synchrony, the synchronous firing of neurons within a specific brain area and it is reflected in EEG measures by an increased power of a frequency band at a specific location. Keizer, Verment & Hommel provided participants auditory feedback on two electrode recordings in the form of a tone that occurred whenever the power on the gamma band activity exceeded the threshold. The power was updated every 0.125 seconds and combined into a moving average. The recordings, however, were only conducted on two electrode sites (Oz and Fz). Participants were, quite ambiguously, instructed to increase the tone occurrence. It was found that the power increased not only between sessions but also within a single session, suggesting a gradual but lasting increase in power. Furthermore, the neural synchronicity in the gamma frequency band also transferred into a longer-range synchronicity between the two electrode sites. But again, the measurements were only performed at two locations, so other source localizations cannot be ruled out. The outcomes of the behavioral results suggest that increased gamma band activation due to neurofeedback leads to enhanced top-down control.

This study shows that even with a straightforward paradigm and a plain neurofeedback protocol, there is a clear connection between frequency power and behavioral performance. Other accounts substantiate this finding by training the upper alpha frequency band (Zoefel, Huster, & Herrmann, 2011) and the beta frequency band (Staufenbiel et al., 2014), albeit limited to an experimental setting. All authors mention a limited applicability for neurofeedback into real-life situations. The cause for the limited transferability could be due to the fact that the participant is sitting idle while recordings are performed, this does not reveal anything about the power spectrum during day to day tasks and if it is possible to alter

these with neurofeedback training. More practically, measuring brain activity while participants are moving and performing day to day tasks will prove to be difficult due to low signal-to-noise ratios Moreover, the motivation of the participants during these idle tasks is questionable, possibly resulting in shortened attention span, heightened drowsiness and therefore influencing baseline frequency bands.

Neurofeedback in a clinical setting

Taking these arguable characteristics of neurofeedback into account, one must be cautious when it comes to the available neurofeedback treatments. A concise investigation through the websites of the available treatment groups in the Netherlands, e.g. Neurofeedback Instituut Nederland (NIN) and Brainclinics, reveals that the same literature previously discussed in this essay is used to substantiate the treatments efficacy. For example the results of Fuchs et al. (2003) are used to support the clinical treatment on the NIN website, while this paper is published without detailed reward classification and EEG measurements. Furthermore, no follow-up study was conducted to reveal any long lasting effects and the possibility of a placebo effect has not been ruled out. Sufficient additional scientific evidence is not provided on the website.

Contrary to the NIN website, the Brainclinics website cites an impressive amount of neurofeedback research, including a detailed account of the diverging alpha peak frequency typical for ADHD symptoms. Furthermore, it is postulated that individual differences in the alpha peak frequencies in ADHD patients might account for the lack of findings in contemporary ADHD neurofeedback research. This difference, the website states, explains the conflicting results in some neurofeedback treatment studies that on hand found a lack of brain wave response after treatment, and on the other hand found an increased response. Curiously, the reference for the individual difference in alpha wave power is not cited in the text or in the

reference list and the study that is cited does not mention individual differences in alpha peak waves (Lansbergen et al., 2011).

While the amount of literature provided on the Brainclinics website as evidence for neurofeedback is impressive and sufficient, they also honestly state that neurofeedback for ADHD is by no means a magical solution for ADHD symptoms and the treatment is still considered highly experimental. Potential clients are then directed to an overview of the literature (Arns, 2008; Arns et al., 2009; Arns, Drinkenburg, & Kenemans, 2012; Arns, Conners, & Kraemer, 2013) and a "recently" published handbook for neurofeedback in ADHD treatment (Arns, 2012).

Unfortunately, the founder and director of the institute that offers the treatment is also the first author in a major part of that overview. Although all the literature listed provides some sort of evidence towards the efficacy of neurofeedback, there is still no convincing large scale study published with a flawless methodology, conducted by researchers not related to the institute. Meanwhile the treatment sessions are offered for a substantial fee and improvement will take on average thirty sessions. One can only hope that they inform their clients thoroughly about the lack of evidence before they start their sessions.

In all fairness, the previously discussed findings are not mentioned to undermine the efficiency of neurofeedback treatment; some studies did find decreased ADHD symptoms after treatment (Arns et al., 2009). The point that is being converted is that more objective research is needed, as well as standardized protocols, both in a methodological and therapeutic sense, to critically evaluate the efficacy of neurofeedback therapy for ADHD. That being said, if the clients and caretakers report a decrease of symptoms and increased quality of life, there should be no need to undermine those results. Every reduced symptom counts in that matter. But this is merely from a clinical perspective. From a scientific

perspective the therapy cannot be regarded as efficient as long as a placebo effect cannot be ruled out (Lansbergen et al., 2011).

Conclusion

To conclude, this essay has discussed the much anticipated upcoming of neurofeedback for ADHD therapy. Expectations were high when the initial neurofeedback results were published and it was discovered that brain waves are subject to operant conditioning (Kamiya, 1969). Lubar et al. (1995) laid down the foundation of a clinical purpose for neurofeedback by developing a tool to calculate and alter the theta-beta frequency power ratio. This framework raised great expectancies in the following decade and sparked a multitude of research lines to explore the efficacy of neurofeedback as a therapy for ADHD treatment (Fuchs et al., 2003; Butnik, 2005).

However, the lack of studies that adhere to the golden standard of randomized double-blind assignment to conditions, suggest that the working mechanisms of neurofeedback are not yet fully revealed (Micoulaud-Franchi et al., 2014). The studies that adhered to the golden standard could not significantly rule out the placebo effect or fully attribute the decrease of symptoms to the neurofeedback treatment (Arnold et al., 2013). Moreover, the widespread use of psychostimulants as treatment for ADHD and the ethical problems of discontinuing this treatment undermine the results from neurofeedback as well. Especially when the effect of these psychostimulants on brain oscillations is not yet fully understood (van Dongen-Boomsma et al., 2013).

In order to strengthen the claims of neurofeedback therapy, more studies have to be conducted to investigate the connection between the manipulation of certain frequency bands and behavioral outcomes. Keizer, Verment, & Hommel (2010) have already showed a direct link between the two in a non-therapeutic setting. More research in this line must be performed to transfer the obtained link between brain waves and behavior to neurofeedback as

a treatment tool for ADHD. Before that, claims from this research field must be taken lightly from an empiric perspective. Moreover, in order to establish itself as a serious treatment option, the field must refrain from all possible appearances of conflict of interest.

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