ADHD IN PRACTICE 2010; Vol 2 No 1

Beverley Steffert PhD Psychologist, Director, Learning Recovery, Cambridge
Tony Steffert BSc MSc Researcher, Department of Psychology, Goldsmiths, University of London

Neurofeedback and ADHD

Neurofeedback (NF) is based on brain imaging studies, quantitative electroencephalography (qEEG),[1] positron emission tomography[2] and single photon emission computed tomography (SPECT),[3,4,5,6] which all show cortical brainwave slowing in the majority of attention deficit hyperactivity disorder (ADHD) patients, as well as too much or too little coherence across functional areas of the brain. Evoked potentials too, reveal subtypes of ADHD, which are known as endophenotypes and will influence the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (DSM–V), due to be published in 2011. An endophenotype is both a psychiatric concept and a biological marker forming a link between the genotype and the phenotype.[7] Diverse behavioural symptoms of a complex disease, such as ADHD, are grouped into a set of up or down regulated genes that from a cluster – eventually a neural network. For example, impulsivity; the correlation between gene expression and behavioural measures of impulsivity, such as the delay-discounting task and other behavioural measures, suggest an interaction between genes expressing mesolimbic dopamine activiation and impulsivity.

The brain areas mediating attention, impulsivity and hyperactivity have been of particular interest to neuroscientists specialising in ADHD. These are the right prefrontal cortices and anterior cingulate gyrus responsible for attention and the basal ganglia and cerebellum involved in movement control. Abnormalities in EEG results have been reported in children with ADHD for some decades; an extensive review of the literature was published in 1991[8] and a meta-analysis appeared more recently.[9] A review of the efficacy of NF for ADHD was published in 2000.[10]

Typologies of ADHD can be distinguished using EEG and, in particular, qEEG which takes into account the interaction between the frequency (Hz) and amplitude (μV) of the electrophysiology of the brain. The major pattern associated with the inattentive type of ADHD is excessive low frequency (4–7 Hz) combined with too little high frequency (18–21 Hz) in midline and frontal cortices,[11,12] while a smaller proportion of 'hyperaroused' patients will exhibit raised levels of high amplitude and frequency (above 21 Hz) activity.

The brain's rhythms have an organising principle so that each person has an optimum 'setpoint' of oscillation. However, genetic, traumatic or environmental events can deregulate these rhythms leaving individuals in unstable emotional, cognitive or physical states, unable to draw on normal inhibitory and excitatory mechanisms, thus affecting impulsivity, alertness and attention.

Given the consistency of findings that brainwave excesses/deficits are associated with ADHD,[1,13,14] and that these abnormal cortical rhythms can be normalised with NF to reduce symptoms,[15,16] the technique became a viable alternative to medication in the USA, especially in cases where drugs did not decrease symptoms or the side-effects required withdrawal.[17,18] Many Eastern European countries, do not allow the use of stimulants for children and NF has a greater use in these locations.

The mechanics of neurofeedback

The whole idea of abnormally slow or fast brainwaves is built on years of research, first on EEG sleep patterns and, later, on the correlation of the level of arousal to particular frequency bands. It is accepted by all neuropsychologists that there is an inverted U-shape to emotional state, with slow frequencies associated with drowsiness and fast frequencies associated with anxiety, anger or hypervigilance. While all frequencies are appropriate for a given situation, the midrange frequencies (12–21 Hz) are those that most people want to be able to produce at will and benefit from the associated feelings of calm, control and mental alertness.

NF can be described as biofeedback for the brain. Biological sensors are placed on the scalp and these electrodes convey the electrophysiology of the underlying brain to a computer that displays the raw EEG signal. This is then split into frequency bands (see Figure 1): slow – up to 7 Hz; medium – 8–12 Hz; fast – 13–21 Hz.

The higher the frequency the more aroused, mentally and physically, the person is. Frequencies above 21 Hz are associated with excessive autonomic arousal from anxiety to anger, while very fast rhythms, above 40 Hz (gamma), fulfil functions beyond arousal. Conceptualisations range from a 'perceptual binding' of incoming sensory data to aspects of learning and memory.[19,20]

The aim of the NF practitioner is to encourage the production of healthier brainwaves, so that the patient who has an excessive slow wave is helped to produce a faster, higher, frequency. Thus, the patient learns to increase the voltage in that 'rewarded' frequency, at the same time as reducing the voltage of the very slow (less than seven cycles per second) and very fast (more than 22 cycles per second) sets of brainwaves. The

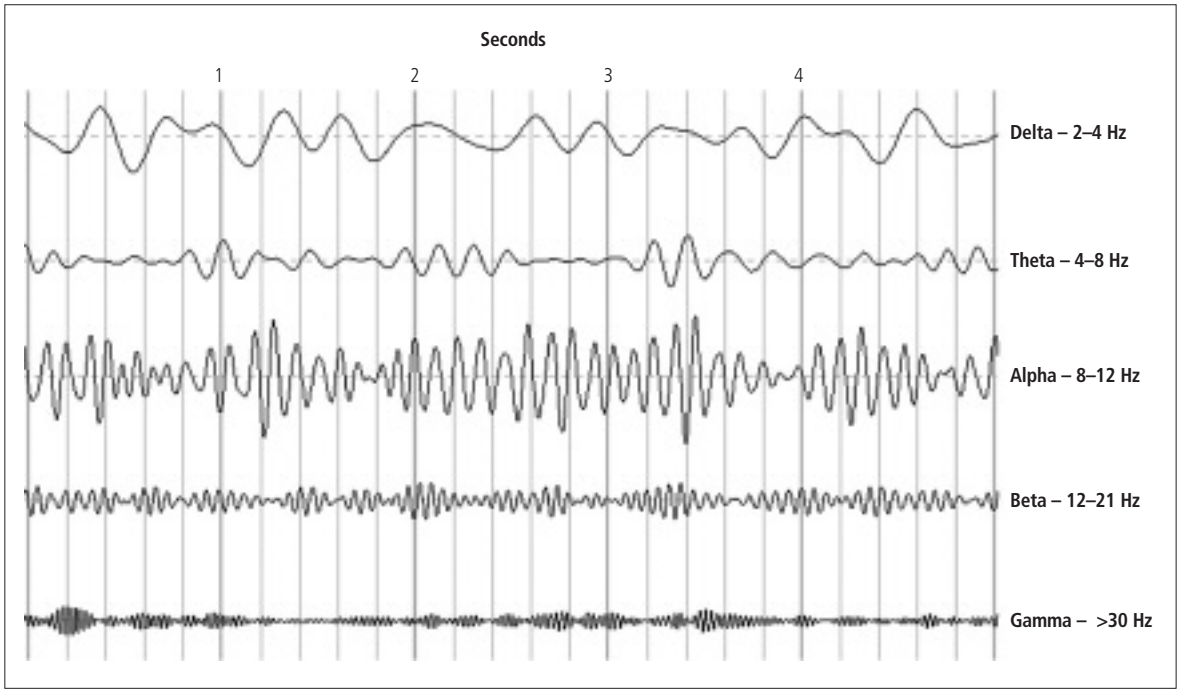

Figure 1. Electroencephalography frequency bands

feedback software gives a signal (auditory, visual or vibratory) when the patient produces more normal brainwaves at least 60–70% of the session.

Typically, at least for children, the signal used in NF will be very much like a computer game, so that points are scored for keeping a car on a road, a monkey climbing a tree, and so on, in exchange for a tangible reward. For adults, the signal used in NF might simply be maintaining music (if the patient drops below the 70% threshold, the music stops) or even watching a graphical interaction of their own brainwaves (the frequency amplitude and interactions of their own brainwaves can be displayed as a sort of dynamic bar chart that they are encouraged to control). The reward is either the calmer or else more alert feeling that the production of these healthier brainwaves can bring and it is held to be a case of operant conditioning of the brain. Making this state conscious, explicit and accurately attributed to a focusing of the brain by the patient is the first step in becoming aware of one's feeling state and typically takes up to six sessions,[21] but once the patient can link their brainwave state to their psychological state the therapeutic process has begun.

Peripheral measures of biofeedback, such as breathing rate, heart rate variability, galvanic skin response or temperature control are frequently used to introduce the patient to the idea of voluntary control of his or her physiology.

Does it last?

It is one thing to be able to steer a car along a tortuous mountain road with one's rewarded brainwave on a biofeedback computer game, but it is quite another to be able to generalise the electrophysiology of attention, alertness or calmness to the classroom or workplace. NF is not electroconvulsive therapy, transcranial magnetic stimulation or anything that forces the brainwaves to alter. It simply provides a signal for the patient who cannot, before they are properly trained, recognise and produce their optimum brainwave state at will. However, after a few sessions of being 'rewarded' the brain resets itself to a new steady state. The new steady state of the brain is the real reward – it feels good, and many patients will recognise this sensation after three or four sessions. Each session strengthens the neural network involved, as the amplitude increases or decreases according to the needs of the individual patient, and any changes are recorded.

While simple success is measured by a small, but steadily increasing, amplitude of the rewarded frequency, the NF practitioner expects to see the EEG normalised after 20 sessions or so. However, amplitude may change ahead of behaviour and vice versa, so some researchers think that the critical variable in the situation is flexibility, in other words, being able to match appropriate psychophysiology to the demands of the situation. There are also individual differences in one's ability to condition onself – that is be aware of one's own internal state and have the motivation to change. If your status depends on being the bad boy of the class (often a preferred label to cover the fear of being thought stupid) then it is a long slog for the NF practioner. Often the patient is given 'homework', such as listening to recordings of the weekly NF session every day, or how to anchor the new feeling of calm to sustain it outside the NF clinic. Pre- and post-treatment

ADHD IN PRACTICE 2010; Vol 2 No 1

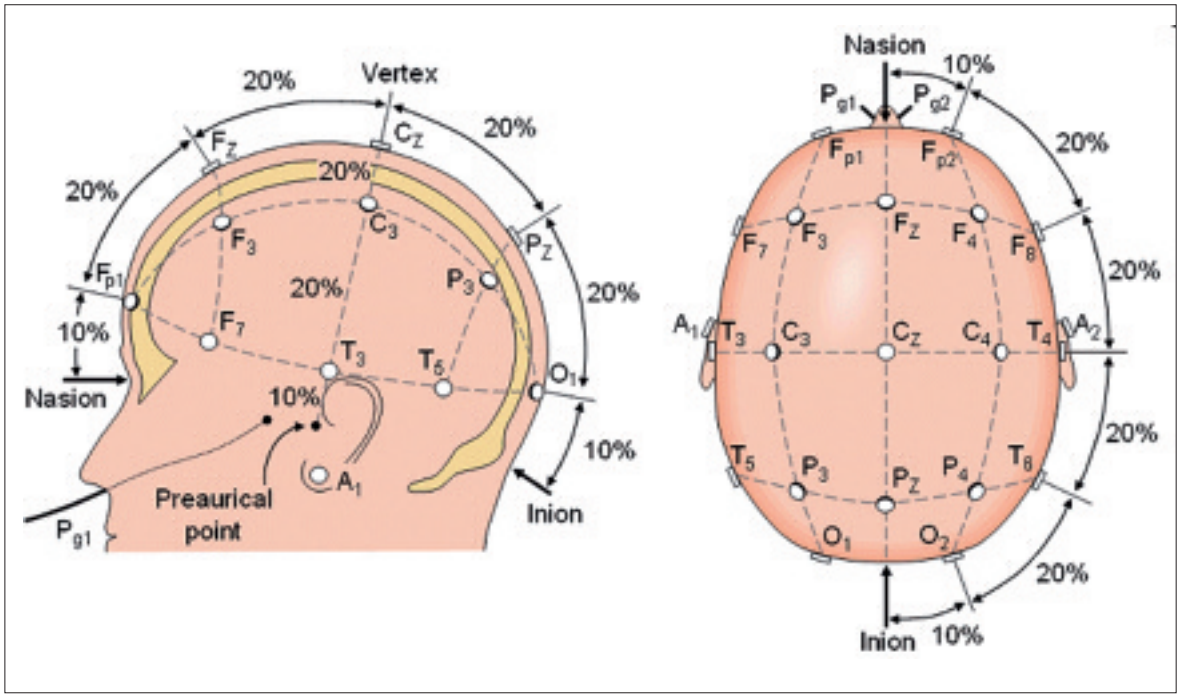

Figure 2. The 10/20 system of electrode placement

measures of attention and impulsivity can be taken with a computerised continuous performance test (available at www.braintrain.com).

Reviews of the efficacy of NF in controlled studies using an 'ABA' design show improvement on standardised intelligence tests (due to increases in working memory), attention and behavioural control.[10] For example, Lubar[22] decreased ADHD symptoms by downtraining (decreasing amplitude) theta but then reproduced the same symptoms by uptraining these low frequencies. Although this is a recognised scientific paradigm, ethical funding committees research no longer allow this on human subjects. Comparisons of the effectiveness of NF versus medication have generally shown NF to be at least as effective, if not better, than the latter option.[10]

Undoubtedly, medication is immediately effective, however, issues regarding non-compliance and long-term side-effects are now being recognised. A recent, large-scale, long-term study[11] showed that, after 14 months, drugs for ADHD were, in many cases, less effective than they had been initially. However, the major drawback of medication is the implication for the patient. A pill implies illness, whereas NF shows the patient how to control his own brain. An eight-year-old child with ADHD may think that he is 'ill' and that a pill will solve his problems, but this is possibly not the best lesson to learn. Empowering a child to see that their difficulty can be overcome with mental effort and practice may help him or her in the long term.

Of course, 'talking' and behavioural therapies can also result in beneficial changes in metabolic activity in the brain over time – but the keyword is time, even long-term cognitive behavioural therapy has been shown[23] to have minimal benefit in terms of the reduction of ADHD symptoms.

Brain-based subtypes of ADHD

Research with qEEG-based diagnosis has led to a more sophisticated understanding of the brain-based mechanisms of ADHD. Kropotov,[1] has shown subtypes of ADHD, as well as more sophisticated ways of measuring ADHD.

The first subtype, mentioned above, is an increase of low frequency oscillations in the frontal lobes – predominantly in the 4–7 Hz range, known as theta rhythms. Sometimes, delta waves (1–4 Hz), are also observed. The presence of excessive low frequencies is characteristic of the largest group of ADHD sufferers – those with typical ADHD combined type.[1]

Secondly, an abnormal increase of frontal midline theta waves in the 5.5–8 Hz range, with its maximum amplitude in the frontal midline (see Figure 2 for the international 10/20 system of electrode placement). This rhythm is produced in long bursts of more than one second that increase with task load. This indicates an abnormal glucose response to task demands and makes allocating mental energy to the task in hand very difficult.[24]

The third pattern is the opposite of the theta rhythm – and this is an abnormal increase in beta frequency, (13–30 Hz) more associated with the overfocused, angry or anxious type of ADHD.[25]

The fourth pattern is an excess of alpha activity (8–12 Hz) at posterior, central or frontal areas of the brain – more associated with the dreamy, inattentive type of ADHD, which is highly distractible, but not hyperactive.[26] Amen suggests two others using SPECT:

- An overactivity in limbic areas combined with decreased activity in the prefrontal cortex.
- An overactivity of high frequency spreading over the whole cerebral cortex, the cingulated gyrus, parietal, temporal and prefrontal lobes. This leads to excessive impulsivity, rapid speech, oversensitivity to sensory stimuli, unpredictability and cyclic mood changes.[27]

Each subtype suggests a different NF protocol, in other words, a different reward frequency and placement for the electrodes. It also suggests the appropriateness of different types of medication for ADHD subtypes and indicates that co-morbidities may limit the success of stimulant therapy. There are also increasing concerns about long-term side-effects of medication on growth, cardiovascular and neurophysiological systems. While NF remains a safe, non-invasive alternative to medication the question of efficacy remains somewhat equivocal. Studies comparing NF with other modes of attention training or behavioural techniques (such as breathing control, nutritional or herbal supplementation, psychotherapy, or cognitive behavioural therapy)[28] usually show a better reduction in ADHD symptomatology, but these tend to be fairly small-scale studies.

Actually, the biggest issue with NF as a treatment is time. NF is a subtle technique, more reliant on individual motivation than, for instance, drugs are. It is not unusual for treatment to take six to ten months if sessions are weekly. In the case of co-morbidities such as obsessive–compulsive disorder, depression and oppositional defiant disorder, which each have their own risk factors, NF treatment can be too long a slog for all involved. Like any therapy there are also non-responders. Non-responders tend to be those patients whose home, school or work situation does not allow them to relax and tune into changes in their brainwaves. It is due to these non-responders that nutritional and psychological strategies are essential back-up tools for most NF practitioners. Exact quantitative figures do not exist, but anecdotal surveys of major practitioners suggest the usual 80:20 ratio of most therapies. However, as knowledge about brain dynamics, such as the coherence, phase and symmetry of brain areas, as well as evoked potentials (which trace the passage of information through the brain in a time-locked fashion), increases more sophisticated NF that normalises these is likely to increase the present success rate of NF in ADHD ■

References

1. Kropotov JD. *Quantitative EEG, Event-Related Potentials and Neurotherapy*. London: Academic, 2009.
2. Lou HC, Henriksen L, Bruhn P. Focal cerebral hypoperfusion in children with dysphasia and/or attention deficit disorder. *Arch Neurol* 1984; **41:** 825–829.
3. Castellanos FX, Giedd JN, Berquin PC *et al*. Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Arch Gen Psychiatry* 1996; **53:** 607–616.
4. Mostofsky SH, Cooper KL, Kates WR, Denckla MB, Kaufmann WE. Smaller prefrontal and premotor volumes in boys with attention-deficit/hyperactivity disorder. *Biol Psychiatry* 2002; **52:** 785–794.
5. Durston S, Hulshoff Pol HE, Schnack HG *et al*. Magnetic resonance imaging of boys with attention-deficit/hyperactivity disorder and their unaffected siblings. *J Am Acad Child Adolesc Psychiatry* 2004; **43:** 332–340.
6. Kim BN, Lee JS, Shin MS, Cho SC, Lee DS. Regional cerebral perfusion abnormalities in attention deficit/hyperactivity disorder. Statistical parametric mapping analysis. *Eur Arch Psychiatry Clin Neurosci* 2002; **252:** 219–225.
7. Ritsner MS (ed.) *The Handbook of Neuropsychiatric Biomarkers, Endophenotypes and Genes*. London: Springer, 2009.
8. Benson DF. The role of frontal dysfunction in attention deficit hyperactivity disorder. *J Child Neurol* 1991; **6** (Suppl): S9-12.
9. Arns M, de Ridder S, Strehl U, Breteler M, Coenen A. Efficacy of neurofeedback treatment in ADHD: the effects on inattention, impulsivity and hyperactivity: a meta-analysis. *Clin EEG Neurosci* 2009; **40:** 180–189.
10. Nash JK. Treatment of attention deficit hyperactivity disorder with neurotherapy. *Clin Electroencephalogr* 2000; **31:** 30–37.
11. Matousek M, Rasmussen P, Gillberg C. EEG frequency analysis in children with so-called minimal brain dysfunction and related disorders. *Adv Biol Psychiatry* 1984; **15:** 102–108.
12. Janzen T, Graap K, Stephanson S, Marshall W, Fitzsimmons G. Differences in baseline EEG measures for ADD and normally achieving preadolescent males. *Biofeedback Self Regul* 1995; **20:** 65-82.
13. Monastra VJ, Lubar JF, Linden M *et al*. Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: an initial validation study. *Neuropsychology* 1999; **13:** 424–433.
14. National Institute for Health and Clinical Excellence (NICE). *NICE implementation uptake report: Attention deficit hyperactivity disorder (ADHD), management of ADHD in children, young people and adults*. London: NICE, 2009. https://www.nice.org.uk/media/032/FA/UptakeReportADHD.pdf (last accessed 12/04/10)
15. Lubar JF. EEG biofeedback and learning disabilities. *Theory into Practice* 1985; **26:** 106–111.
16. Lubar JF, Lubar JO. Neurofeedback Assessment and Treatment for Attention Deficit/Hyperactivity Disorders. In: Evans JR, Abarbanel A (eds). *Introduction to Quantitative EEG and Neurofeedback*. London: Academic Press, 1999.
17. Gadow KD, Sverd J, Sprafkin J, Nolan EE, Grossman S. Long-term methylphenidate therapy in children with comorbid attention-deficit hyperactivity disorder and chronic multiple tic disorder. *Arch Gen Psychiatry* 1999; **56:** 330–336
18. Schachter H, Pham B, King J, Langford S, Moher, D. How efficacious and safe is short-acting methylphenidate for the treatment of attention-deficit disorder in children and adolescents? A meta-analysis. *CMAJ* 2001; **165:** 1475–1488.
19. Tzelepi A, Bezerianos T, Bodis-Wollner I. *Functional properties of subbands of oscillatory brain waves to pattern visual stimulation in man. Clin Neurophysiol* 2000; **111:** 259–269.
20. Tallon-Baudry C, Bertrand O, Delpuech C, Permier J. Oscillatory Gamma Band (30-70 Hz) activity induced by a visual search task in humans. *J Neuroscience* 1997; **17:** 722–734.
21. Kamiya J. Operant control of EEG alpha rhythm and some of it's reported effects on consciousness. In: Tart CT (ed.) *Altered States of Consciousness*. New York: Wiley, 1969
22. Lubar J, Shouse MN. Use of biofeedback in the treatment of seizure disorders and hyperactivity. *Adv Clin Child Psychol* 1976; **1:** 203–265.
23. Safren SA, Otto MW, Sprich S *et al*. Cognitive-behavioral therapy for ADHD in medication-treated adults with continued symptoms. *Behav Res Ther* 2005; **43:** 831–842.
24. Zametkin AJ, Nordahl TE, Gross M *et al*. Cerebral glucose metabolism in adults with hyperactivity of childhood onset. *N Engl J Med* 1990; **323:** 1361–1366.
25. Albrecht B, Brandeis D, Uebel H *et al*. Action monitoring in boys with attention-deficit/hyperactivity disorder, their nonaffected siblings, and normal control subjects: evidence for an endophenotype. *Biol Psychiatry* 2008; **64:** 615–625.
26. Clarke A, Barry R, McCarthy R, Selkowitz M. Electroencephalogram differences in two subtypes of attention-deficit/hyperactivity disorder. *Psychophysiology* 2001; **38:** 212–221.
27. Amen DG, Carmichael BD. High Resolution Brain SPECT imaging in ADHD. *Ann Clin Psychiatry* 1997; **9:** 81–86.
28. Monastra VJ, Lynn S, Linden M. Electroencephalographic biofeedback in the treatment of attention-deficit/hyperactivity disorder. *Appl Psychophysiol Biofeedback* 2005; **30:** 95–114.

Key points

- **Neurofeedback can be described as biofeedback for the brain. Biological sensors convey the electrophysiology of the underlying brain to a computer.**
- **The aim of the neurofeedback practitioner is to encourage the production of a healthier brainwaves.**
- **Many Eastern European countries, do not allow the use of stimulants for children and, therefore, neurofeedback has a greater use in these locations.**