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Neurofeedback as a Treatment for ADHD: A Methodological Review with Implications for Future Research

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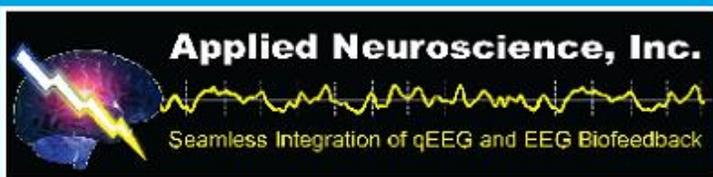
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Neurofeedback as a Treatment for ADHD: A Methodological Review with Implications for Future Research

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ABSTRACT. Attention deficit/hyperactivity disorder (ADHD) represents one of the most common psychiatric disorders in childhood, resulting in serious impairment across a variety of domains. Research showing that a high proportion of children with ADHD exhibit a dysfunctional electroencephalogram (EEG), relative to aged matched peers, provides a rationale for the use of neurofeedback as an intervention. The aim of neurofeedback training is to redress any EEG abnormality, resulting in a concomitant improvement in the behaviour and/or cognitive performance of these children. This review focused on studies using neurofeedback to treat children with ADHD, with particular emphasis on the methodological aspects of neurofeedback training. Specifically, the review examined the modality of feedback provided, the different training parameters and their underlying rationale, and the particular montages used. In addition, the review also focused on the duration, frequency and total number of training sessions required to obtain a positive effect in

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terms of a change in the individual's EEG, behaviour and/or cognitive performance. Finally, the long-term effects of neurofeedback and the potential negative side effects were reviewed. Throughout, the review provides a number of directions for future research.

KEYWORDS. ADHD, EEG, quantitative EEG, neurofeedback, methodology

INTRODUCTION

EEG-biofeedback, or neurofeedback, represents a plausible treatment for children with attention-deficit/hyperactivity disorder (ADHD). However, there is continued debate concerning the efficacy of neurofeedback as an intervention for such a disorder. For the past three decades Lubar and colleagues have championed the cause of neurofeedback, producing some positive results (Lubar & Shouse, 1976; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Lubar, Swartwood, Swartwood, & Timmerman, 1995; Shouse & Lubar, 1979). However, others remain skeptical stating that, "there is not enough evidence from well controlled scientific studies at this time to support the effectiveness of EEG biofeedback for AD/HD children" (Barkley, 1990, p. 10) and that it represents "... an experimental treatment, the validity of which has not yet been determined" (Baydala & Wikman, 2001, p. 454).

In an effort to improve our understanding of the efficacy of neurofeedback as a treatment for children with ADHD, we conducted a review of the literature. Others have already provided summaries of the main clinical studies and reviewed the efficacy of neurofeedback in the management of children with ADHD (Baydala & Wikman, 2001; Nash, 2000). We intend to complement those analyses with an examination of the rationale for the use of neurofeedback and a summary of the main methodological issues yet to be resolved. In this way we hope not only to stimulate those in the area to think how best they could address such methodological issues, but also to provide possible directions for future research. Thus, this methodological review begins by summarising the nature of ADHD. This is followed by an examination of the literature suggesting that children with ADHD exhibit a dysfunctional electroencephalogram (EEG), which provides a rationale for the use of neurofeedback. Third, we focus on peer reviewed outcome studies that have utilised neurofeedback as an intervention for children with ADHD. In particular we specifically examine the neurofeedback treatment process, asking a number of questions concerning the methodology of such a process.

THE NATURE OF ADHD

Prevalence

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common mental health disorders of childhood (Hoza & Pelham, 1993). Estimates of the prevalence of ADHD range from a low of one percent up to a high of 14 percent (Wolraich, 1999). The seeming disparity of these rates has been influenced by alterations in diagnostic criteria and differences in definitions used (see Swanson et al., 1998). During childhood the disorder is more common among boys, with the ratio of boys to girls estimated at approximately 4:1 for all three of the DSM-IV subtypes (Clarke, Barry, McCarthy, & Selikowitz, 2001a). It has been suggested, however, that this ratio may decrease with age (Swanson et al., 1998). Furthermore, boys are more prone to display behavioural disturbances, whilst girls predominantly exhibit inattentive behaviour (Pary, Lewis, Arnp, Matuschka, & Lippmann, 2002).

Attention-deficit/hyperactivity disorder was originally thought to occur only in children. However, it is now recognised as a condition with symptoms persisting into adulthood for between 40 to 70% of individuals (Bellak & Black, 1992). Longitudinal studies also suggest that difficulties with hyperactive-impulsive behaviour may emerge first, followed by symptoms of attention (Murphy, Barkley, & Bush, 2002). The hyperactive-impulsive subtype appears to exist primarily among pre-school age children and is substantially less likely to occur in older children that are referred to a clinic. In contrast, the inattentive subtype appears to emerge at a later age of onset than either the hyperactive-impulsive or combined subtypes. Follow up studies of children diagnosed with ADHD have shown a decrease in their hyperactive/impulsive symptoms with increasing age; however, the symptoms of inattention tend to remain (Swanson et al., 1998).

Diagnosis

There are no pathognomonic measures to diagnose children with ADHD. The diagnosis remains dependent on the observations of those adults most familiar with the children, such as parents and teachers. Clinical diagnosis of ADHD is invariably achieved using either the latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, American Psychiatric Association [APA], 1994) or the International Statistical Classification of Mental Disorders (ICD-10, World Health Organisation [WHO], 1992). However, there may be some variation between the DSM and ICD taxonomies (for a review see Swanson et al., 1998). Both require the individual to exhibit a range of symptoms for at least six months to a degree that is maladaptive and inconsistent with developmental level in two or more settings, leading to clinical impairment in social, academic or occupational functioning and evident

prior to the age of seven years (APA, 1994; WHO, 1992). However, according to DSM-IV there are three main subtypes of ADHD: inattentive (ADHD_{in}), hyperactive (ADHD_{hyp}) and combined (ADHD_{com}). In contrast, the ICD-10 classifies children as having a 'hyperkinetic disorder' as opposed to attention-deficit/hyperactivity disorder. Nevertheless, this also contains the three key components of inattention, hyperactivity and impulsivity.

Clinicians can supplement the diagnostic information provided by the DSM-IV and the ICD-10 by using a range of tests specifically constructed to identify characteristics of ADHD. These include the Test of Variables of Attention (TOVA; Greenberg, 1987), which is a computer administered continuous performance test widely used as an adjunct for the diagnosis of ADHD. The TOVA provides an assessment of an individual's performance on a task that requires the tracking of visual stimuli with a differential response/non-response to target and non-target stimuli. A failure to respond to a target stimulus provides a measure of omission errors, denoting inattention. The response to non-target stimuli provides a measure of commission errors, which relates to impulsivity/hyperactivity. Response rate and consistency of response (i.e., variability) are also measured to assess the ability of the individual to attend and process visual information. In addition to this, a number of questionnaires have been developed to measure the hyperactive/impulsive and inattentive aspects of behaviour. These include the Child Behaviour Checklist (CBCL; Achenbach, 1999), the Connors' Parent Rating Scale-Revised (CPRS-R; Connors, Sitarenios, Parker, & Epstein, 1998a), the Connors' Teacher Rating Scale-Revised (CTRS-R; Connors, Sitarenios, Parker, & Epstein, 1998b), the Attention Deficit Disorders Evaluation Scales (ADDES; McCarney, 1995) and the Strength and Difficulties Questionnaire (SDQ; Goodman, 1997). This is by no means a comprehensive list and merely highlights the more common questionnaires that are widely used, and are considered to be useful in determining if the child under consideration exhibits any ADHD symptoms.

An added consideration in diagnosing ADHD is the identification of comorbidities that can, and frequently do occur. A variety of studies have examined the prevalence of comorbid conditions for children with ADHD (for a review see Jensen, Martin, & Cantwell, 1997). Some suggest that up to 50% of children with ADHD also meet the criteria for socially disruptive conduct disorder (CD) or oppositional defiant disorder (ODD; Pliszka, 1998), with affective and anxiety disorders coexisting in approximately 11% of children with ADHD (Pary et al., 2002). Research has also shown that an early age of onset (i.e., less than 6 years old) is associated with higher comorbidity rates as well as poorer outcome (McGee, Williams, & Feehan, 1992).

In summary, ADHD represents a common childhood disorder occurring more frequently in boys than girls. Its primary symptoms of inattention, hyperactivity, and impulsiveness present in different combinations in different subtypes. In addition, children diagnosed with ADHD often meet the criteria for a number of other affective and behavioural disorders.

DYSFUNCTIONAL EEG

There is an emerging consensus that examination of the structural and/or functional differences in children with ADHD compared to non-clinical controls may provide the clarification needed to understand the abnormalities in the brain of individuals with ADHD (for recent reviews see Barry, Clarke, & Johnstone, 2003; Farone & Beiderman, 1998; Heilman, Voeller, & Nadeau, 1991; Shaywitz, Fletcher, Pugh, Klorman, & Shaywitz, 1999). Increasingly, researchers have focused on measured cortical activity, or the electroencephalograph (EEG), of the individual. For instance, the frequency composition of an individual's EEG is thought to reflect the age and the functional status of his/her brain. With normal maturation, EEG frequencies increase as a function of age, with slow wave activity being replaced by faster waveforms (Benninger, Matthis, & Scheffner, 1984; Gasser, Verleger, Bacher, & Sroka, 1988; John et al., 1980). Furthermore, John et al. (1980) have suggested that such changes are consistent across different socio-economic groups. These continuous changes in the power spectrum of the EEG may relate to unspecific maturational processes such as increasing thickness of myelination. However, children with ADHD exhibit an abnormal pattern of EEG activity. For instance, EEG analysis has revealed that up to 80% of children with ADHD exhibit abnormalities, particularly in the frontal/polar regions (Chabot, di Michele, Prichep, & John, 2001). Indeed, it has been suggested that the greater the level of EEG abnormalities, the more the individual exhibits behavioural problems (Clarke et al., 2001a).

Examination of the EEG of children with ADHD found that they exhibited an increase in absolute amplitude in the theta band (4-7.75 Hz) during a resting condition, predominantly in the frontal regions (Mann, Lubar, Zimmerman, Miller & Muenchen, 1992). On tasks requiring sustained attention these children also showed a greater increase in theta activity in frontal and central regions, and a decrease in beta1 (12.75-21 Hz) activity in posterior and temporal regions. One suggestion is that the excess theta may result from increased thalamic alpha generator output, with theta resulting from a slowed alpha rhythm, or a disinhibition of hippocampal theta generators (Steriade, Gloor, Llinas, Lopes da Silva, & Mesulam, 1990). The finding of an excess in the slow wave activity of ADHD children, particularly in the frontal midline regions, is particularly robust and has been replicated a number of times (e.g., Chabot et al., 2001; Chabot & Serfontein, 1996; Clarke et al., 1998; Monastra et al., 1999). In addition, Clarke et al. (1998) have shown that ADHD children exhibit less posterior beta (13.5-20.5 Hz) and alpha (7.5-13.5 Hz) relative to age-matched controls. Furthermore, Chabot and Serfontein (1996) reported that 30 percent of children with attentional problems (i.e., ADHD and ADD) exhibited an interhemispheric asymmetry identified as an excess of right hemisphere power. This abnormal right hemisphere function was suggested to be the result of a dysfunction in interhemispheric communication.

It should be noted, however, that whilst the findings indicating abnormalities in the EEG are robust, there is some disparity in the literature regarding the specific frequency ranges of the different EEG bands. For example, the theta band has been used to define a frequency range of between 2.5-7.7 Hz (Clarke, Barry, McCarthy, & Selikowitz, 1998), 4-7.75 Hz (Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992) and 4-8 Hz (Monastra et al., 1999). It is not clear why many researchers choose to define the frequency range of a specific EEG band in such different ways. As such, when interpreting EEG data one should be clear about the specific frequency ranges that form each band (for an interesting discussion of this point see Kaiser, 2001).

Recent research has addressed the question of whether the inattentive, hyperactive, and combined subtypes present distinct EEG profiles. Clarke et al. (1998) recorded the EEG of sixty children aged 8 to 12 years, in an eyes-closed resting paradigm. They found that the ADHDcom group exhibited greater levels of absolute delta (0.5-2.5 Hz) and theta (2.5-7.5 Hz), and lower levels of absolute beta (13.5-20.5 Hz) compared to the ADHDin group. These differences were reflected in terms of the degree of severity of a difference from the norm, rather than in the nature of the EEG abnormalities. As a result, Clarke et al. (1998) suggested that the two subgroups of ADHD may not be neurologically independent. They later replicated this pattern of findings using a larger sample (Clarke, Barry, McCarthy, & Selikowitz, 2001b). However, they did find that measures from the frontal region showed qualitative differences between the two ADHD groups, with greater absolute and relative theta and theta/alpha ratios found in the frontal region compared to the central region for the ADHDcom group relative to the ADHDin group. These findings, they suggest, may be the result of a frontal lobe dysfunction in the ADHDcom group. In contrast, the ADHDin group may have another form of central nervous system (CNS) dysfunction not primarily associated with the frontal lobes. Furthermore, a study comparing individuals classified as either ADHDin or ADHDcom to a non-clinical control group in an eyes-open resting paradigm found that both ADHD groups exhibited greater activity in the 12-15 Hz range (Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996).

Research has also shown that over time the EEG activity of individuals classified as ADHDcom reduces to a level similar to that exhibited by those classified as ADHDin. However, the difference between the ADHDin and non-clinical controls remains constant, even with changes in age (Clarke et al., 2001a). This led to the suggestion that there are two distinct components in ADHD that are quantifiable using EEG. The first is a hyperactive/impulsive component that appears to normalise with increasing age and may be the result of a maturational lag. The second is an inattentive component that does not appear to normalise with age. This led Clarke et al. (2001a) to suggest that inattention may be associated with a more permanent developmental deviation in CNS functioning.

It should be noted, however, that not all researchers have found clear differences in the EEG profile of children with ADHD when compared to non-clinical groups (e.g., Cox et al., 1998; Diamond, 1997). Cox et al. (1998) compared a group of ADHD boys with age-matched controls using an eyes-open paradigm and found no differences in terms of average percentage power for theta (4-8 Hz), alpha (8-13 Hz) and beta (13-22 Hz). However, there were a number of limitations to this study, including the use of a small sample size and the fact that participants selected their own preferred activity during the EEG recording as opposed to being provided with a single standard activity.

That particular patient groups have been shown to exhibit distinct patterns of cortical activity has resulted in researchers increasingly using the EEG as part of the classification process. For instance, early research used the difference in EEG activity between children diagnosed with a psychiatric disorder and non-clinical controls in an effort to improve diagnostic accuracy (e.g., Capute, Niedermeyer, & Richardson, 1968). More recently, quantitative analysis of the electroencephalogram (QEEG) has been included as part of the diagnostic process for children with ADHD (Monastra, Monastra, & George, 2002). Although this technique offers the possibility of improving the classification process, it should be noted that there is some diversity in the literature regarding the specific task of the participant and the exact number of recording channels utilised during a QEEG analysis of children with ADHD. This ranges from recordings taken whilst participants complete reading, listening and drawing tasks, using a single recording channel at CZ (Monastra, Lubar, & Linden, 2001; Monastra et al., 1999), or when participants are in an eyes-closed resting state using either 19 channels (Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Chabot & Serfontein, 1996), or 21 channels (Clarke et al., 1998, 2001b; Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002). As yet no clear consensus has emerged amongst researchers as to the most efficient and effective means of identifying possible EEG abnormalities, and as such one needs to be aware of the different techniques used when making possible comparisons across study groups.

Monastra et al. (1999) examined the QEEG of 482 individuals aged 6 to 30 years, recorded from a single channel at the vertex (i.e., CZ) whilst the participants completed reading, listening and drawing tasks. They found that all those classified as ADHD exhibited higher ratios of theta (4-8 Hz) to beta (13-21 Hz) power, and that the ratios were even greater for the younger participants. Furthermore, the theta-beta power ratios were able to serve as a basis for differentiating between participants with ADHD and non-clinical control participants. The predictive power of the QEEG was high, with greater than 85% consistency between classifications derived from the QEEG index and measures of behaviour and academic performance. More recently this was replicated with a group of 129 individuals aged 6-20 years (Monastra et al., 2001). In addition, Monastra et al. (2001) conducted a direct comparison of the classification agreement of the QEEG with both behavioural and continuous perfor-

mance task (CPT) measures by examining 285 individuals aged 6-20 years. They found high classification agreement between QEEG scans and the ADDES (McCarney, 1995) behavioural measure (83%), and between QEEG and the TOVA (Greenberg, 1987; 70%). These findings led Monastra and colleagues (Monastra et al., 2001; Monastra et al., 1999) to suggest that values derived from the QEEG analysis can serve as the basis for accurate classification of participants as having ADHD, with a level of accuracy comparable to the existing behavioural and CPT tests used to identify ADHD.

In summary, a large number of children with ADHD exhibit a distinct EEG profile consisting of excess slow wave activity and a deficit in the fast wave activity. These differences have been used to accurately identify those with ADHD. This suggests that future research could benefit from including a QEEG examination, which provides an objective psychophysiological measure that can be used as part of the diagnostic process and which may be compared to a normative database to ascertain possible differences in the cortical profile of children with ADHD (e.g., Thatcher, 1998, 1999). A key point is that the abnormalities seen in the EEG of children with ADHD provide a strong scientific rationale for the use of neurofeedback as an intervention process that may be able to redress these EEG abnormalities. However, it should be noted that the majority of research to date using neurofeedback as an intervention for children with ADHD has not used information gained from a QEEG, either as part of the diagnostic process or to inform specific parameters of the neurofeedback training. For instance, of the fourteen studies reviewed in Table 1 only three incorporated QEEG as part of a pre-treatment screening process to aid in the diagnosis, and in one case support the training of specific frequencies (see, e.g., Alhambra, Fowler, & Alhambra, 1995; Boyd & Campbell, 1998; Monastra et al., 2002). Thus, although the research outlined above provides strong evidence for differences found in the EEG of children with ADHD, whether neurofeedback training that is informed by such information would result in a more efficacious treatment has yet to be resolved, and remains the domain of future research. Nevertheless, it is imperative that clinicians and researchers that use neurofeedback to treat children with ADHD monitor the EEG of these children over time for any changes as a function of the treatment process. Otherwise it will be unclear how much of any improvement is due to neurofeedback and how much is due to other non-specific factors.

NEUROFEEDBACK

Neurofeedback is a sophisticated form of biofeedback based on specific aspects of cortical activity. It refers to an operant conditioning paradigm in which an individual learns to modify the amplitude, frequency and/or coherence of the electrophysiological aspects of his/her own brain. The goal of

neurofeedback training is to teach the individual what specific states of cortical arousal feel like and how to activate such states voluntarily. For example, during neurofeedback training the EEG is recorded and the relevant frequency components are extracted and fed back to the individual using an online feedback loop in the form of audio-visual information. Such a format is able to represent each of the frequency components separately: for example, as a bar with the amplitude of the frequency represented by the size of the bar. The individual's task is to increase the size of the training-frequency bar and simultaneously decrease the size of the bars representing the inhibitory-frequencies. On meeting this goal a tone may sound and a symbol appear to indicate a point scored, with the aim to score as many points as possible.

Based on the research outlined in the Dysfunctional EEG section above showing that children with ADHD exhibit abnormal EEG patterns, neurofeedback provides a mechanism for the child with ADHD to 'normalise' his/her cortical profile by decreasing the excess slow activity and increasing the fast wave activity. By redressing this EEG abnormality the child is expected to exhibit increased focused attention and more appropriate levels of arousal which, in turn, might improve academic performance. Although medication may influence the EEG of children with ADHD for a short time period (e.g., Chabot, Orgill, Crawford, Harris, & Serfontein, 1999; Clarke et al., 2003), it has been suggested that the use of neurofeedback may lead to a long-term normalisation of the patients' EEG and result in long-term alleviation of symptoms (e.g., Tansey, 1993).

The exact physiological processes involved in changing the pattern of cortical activity are not well understood; nevertheless, learning to alter one's EEG activity is not thought to be difficult. We are unaware of any research directly addressing whether it is possible for everyone to learn to alter their EEG via neurofeedback. However, research has shown that healthy individuals (Egner & Gruzelier, 2001; Vernon et al., 2003), those suffering brain injury (Thornton, 2000; Tinius & Tinius, 2000), epileptics (Serman & Macdonald, 1978; Serman, Macdonald, & Stone, 1974; Uhlmann & Froscher, 2001) and schizophrenics (Gruzelier, Hardman, Wild, & Zaman, 1999) have been able to exhibit changes in their cortical activity following neurofeedback training. (For a comprehensive bibliography see Hammond, 2001b, available in an updated version at www.isnr.org). At a very simple level it may be that following neurofeedback training the individual becomes aware of the different EEG states and is capable of subsequently producing them when required. However, many individuals report that whilst they can produce the different EEG patterns when required, they are not entirely sure how this is done. This suggests that neurofeedback may involve implicit or non-conscious learning.

If neurofeedback training can improve cognitive and behavioural performance, it represents a highly desirable intervention for children with ADHD. However, such training can be a labour intensive treatment and thus, for anyone considering undergoing or providing such a treatment it is especially im-

TABLE 1. Summarising aspects of the neurofeedback training process with ADHD children from some of the main clinical studies. Including the type of group trained (LD = Learning Disorder; H = Hyperkinetic; ADD = Attention Deficit Disorder; ADHD = Attention/Deficit Hyperactivity Disorder), the specific feedback modality, the neurofeedback protocol (R = Reward Frequency; I = Inhibitory Frequency; SMR = Sensorimotor Rhythm; β = Beta; θ = Theta) with frequency ranges in parenthesis. The montage used, the duration, frequency and total number of training sessions conducted, and whether positive effects were seen in patients' EEG, behaviour and cognitive performance.

Reference	Neurofeedback Protocol						Sessions			Changes in	
	Group	Feedback	Parameters	Montage	Length	Frequency	Total	EEG	Beh	Cog	
Lubar & Shouse (1976)	H	Visual & Auditory	R-SMR (12-14) I- θ (4-7)	Bipolar (C13 + C14)	40 mins	3 per week	Mean 47.3	Yes	Yes		
Shouse & Lubar (1979)	H	Visual & Auditory	R-SMR (12-14) I- θ (4-7)	Bipolar (C13 + C14)	40 mins		Mean 66.5	Yes	Yes		
Lubar & Lubar (1984)	LD & H	Visual & Auditory	R-SMR (12-15) I- θ (4-7) Then switched to R-B1 (16-20)	Bipolar (F7 + T5/F8 + T6)	40 mins	2 per week	20-54	Yes		Yes	
Alhambra et al. (1995)	ADD/HD	Visual & Auditory	R-SMR (12-15)- β (15-35) I- θ (4-7)	Monopolar (CZ)	35-40 mins		20-30	Yes	Yes	Yes	
Lubar et al. (1995)	ADHD	Visual & Auditory	R-B1 (16-20) I- θ (4-7)	Bipolar (FCZ + CPZ)	1 hour	1 per day, five times per week	40	Yes	Yes	Yes	
Lubar et al. (1995) (expt 4)	ADHD	Visual & Auditory	R-B1 (16-20) I- θ (4-7)	Bipolar (FCZ + CPZ)	1 hour		30-45	Yes			
Rossiter & LaVaque (1995)	ADHD	Visual & Auditory	*Aged 8-14y I- θ (4-7) # Aged 14-20y R-SMR (12-15) - B1 (16-20) I- θ (4-7) Aged > 20y R-SMR/B1	*Bipolar (FCZ + CPZ) # Monopolar (CZ)	45-50 mins	3-5 per week	20			Yes	

Linden et al. (1996)	ADD/ADHD	Visual & Auditory	R-β1 (16-20)-θ (4-7)	Bipolar (CZ + PZ)	45mins	2 per week	40	Yes	Yes
Rossiter (1998)	ADHDin ADHDcom	Visual & Auditory	R-SMR (12-15) β1 (15-18) I-β2 (22-30) - θ (4-7) - δ (0.5 - 4)	Monopolar (CZ)	40mins	5 per week	60	Yes	Yes
Boyd et al. (1998)	ADHD	Visual & Auditory	R-SMR (12-15 Hz)	Monopolar (CZ)	30mins	1 per week	20	Yes	Yes
Thompson & Thompson (1998)	ADD *ADHD	Visual & Auditory	R-β1 (15-18)-θ (4-8) * R-SMR (13-15)	Monopolar (CZ) *Bipolar (FCZ + CPZ)	50mins	2 per week	40	Yes	Yes
Carmody et al. (2001)	ADHD	Auditory only	R-beta1 (16-18Hz) I-delta/theta (2-7Hz) then R-SMR (13-15Hz)	Monopolar (C3) Monopolar (CZ)	30mins	3-4 per week	36-48	No	Yes
Monastra et al. (2002)	ADHDin ADHDcom	Visual & Auditory	R-β1 (16-20) I-θ (4-7)	Bipolar (FCZ + CPZ)	30-40 mins	1 per week	34-50 (43)	Yes	Yes
Fuchs et al. (2003)	*ADHDin # ADHDhyp ^ ADHDcom	Visual & Auditory	* R-β1 (15-18) I-β2 (22-30) + θ (4-7) #R-SMR (12-15) I-β2 (22-30) + θ (4-7) ^R-SMR- β1 I-β2 (22-30) + θ (4-7)	Monopolar * at C3 # at C4 ^ at C4 then C3	30-60 mins	3 per week	36	Yes	Yes

portant that a number of questions be addressed. These include, what type of feedback is most efficient, what specific frequency parameters should be reinforced/inhibited and why, and what sort of recording/monitoring set-up of the EEG is most effective. Additional questions relate to the neurofeedback sessions themselves. For example, how long should each session be? How often do sessions need to be completed and how many sessions are required before a positive effect can be identified in the EEG profile, the behaviour and/or cognitive performance? Furthermore, an important question to ask of any treatment is whether the effects are long lasting or not. Also, what if any are the negative effects of neurofeedback? We address each of these questions below, reviewing what is known about each of the issues and highlighting what is not. In addition, we provide some possible directions for future research. Table 1 provides a summary of some of these aspects of neurofeedback training from some of the major neurofeedback studies focusing on children with ADHD.

Modality of Feedback

Information recorded from the EEG can be fed back to the individual in a number of different formats such as auditory, visual, or combined auditory and visual format. We are unaware of any research directly examining the potential effects of modality on neurofeedback. Nevertheless, the type of feedback needs to be considered as it must be capable of providing sufficient information for the feedback loop to operate effectively. In this regard, traditional biofeedback research has shown that attention demands placed on the individual by the feedback signal can affect performance (Qualls & Sheehan, 1981).

Presenting two simultaneous signals (i.e., auditory and visual) may enhance attention to the task. As attention to one signal wanders the remaining signal may be capable of re-directing attention back to the task. This idea is supported by research showing enhanced attention, as indexed by faster reaction time, to multimodal information (Giray & Ulrich, 1993).

Traditional biofeedback research has shown that the mode of presenting sensory feedback can influence the response (Lal et al., 1998). Lal et al. compared the use of visual, auditory or a combination of visual and auditory biofeedback to lower blood pressure in a group of thirty-six participants. They found that using visual or combined visual and auditory feedback was more effective than using auditory feedback alone. There was no difference between the combined visual and auditory approach and the visual protocol alone, suggesting that adding auditory feedback provides no advantage for lowering blood pressure. However, it may be that the visual feedback provides more information, which can be more easily coded than auditory information (Pollard & Ashton, 1982).

Regarding neurofeedback, as can be seen from Table 1, the majority of researchers use a combination of both visual and auditory feedback. However,

Carmody, Radvanski, Wadhvani, Sabo, and Vergara (2001) utilised auditory feedback in the form of high, medium and low-pitched tones to represent distinct aspects of the EEG as well as EMG. They examined the behaviour, academic performance and QEEG of children with ADHD aged 8 to 10 years and found that after 36 to 48 sessions of neurofeedback, children with ADHD exhibited a reduction in commission errors, suggestive of a reduction in impulsivity, and a slight improvement in teacher ratings of attention. However, there was no clear pattern of change in their EEG profile as evidenced from the QEEG. It may be possible that the lack of a clear and positive effect for behaviour and for the QEEG is the result of using auditory feedback only. For example, those research groups that monitored EEG changes (see Table 1) and utilised a combination of auditory and visual feedback, report clear changes in the QEEG profile of the participants (e.g., Alhambra et al., 1995; Lubar & Shouse, 1976; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Lubar, Swartwood, Swartwood, & Timmerman, 1995; Lubar & Lubar, 1984; Monastra et al., 2002; Shouse & Lubar, 1979; Thompson & Thompson, 1998).

In general, there seems to be a consensus of opinion that providing both auditory and visual feedback may be the most efficacious way of informing the participant about the state of his/her EEG. Nevertheless, this area would benefit from a comprehensive assessment of the effect of the modality of feedback on outcome measures.

Neurofeedback Protocols

In neurofeedback the term protocol may be used to refer to a wide range of details that form a part of the overall training paradigm. However, here we focus on two of the most important aspects of the training: (a) the specific selection of the reinforcement and inhibitory parameters and their frequencies, and (b) the particular montage used to deliver this training.

Parameters. Here parameter refers to the specific EEG frequency, or frequencies, that the individual is trained to alter. Given the range of EEG frequencies, what is the optimal training protocol for children with ADHD? Moreover, do different parameters have differential effects on cognition and behaviour for different groups? Overall, neurofeedback research on children with ADHD has focused on three frequency parameters: theta (4-8 Hz), SMR (12-15 Hz) and beta (15-20 Hz).

The majority of research groups utilising neurofeedback as an intervention for children with ADHD train the inhibition of slow theta activity (see Table 1). Furthermore, this type of training has been utilised for all subtypes of ADHD (e.g., Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; Monastra et al., 2002; Rossiter, 1998) as well as individuals classified as ADD (Thompson & Thompson, 1998). The rationale for theta inhibition training stems from the excess theta activity shown by children with ADHD at both rest and during cognitive performance, relative to age matched peers. (See the section on Dys-

functional EEG above.) However, there is still some debate concerning the precise cause of this EEG abnormality. An early suggestion was that children with ADHD are under aroused, or hypoaroused, making it difficult for them to produce the faster wave activity, resulting in an excess of slow wave activity (Lubar & Shouse, 1976; Shouse & Lubar, 1979). More recent alternatives include the suggestion that excess theta activity represents either a maturational delay in cognitive processing ability (Clarke et al., 1998; Mann et al., 1992), or a deviation from normal development (Chabot & Serfontein, 1996). Nevertheless, all seem to agree that the excess in slow theta activity is problematic and can usefully be addressed by neurofeedback training.

When children with ADHD are trained to enhance a particular component frequency of their EEG, it is most commonly either the sensorimotor rhythm (SMR; 12-15 Hz) or low beta (also known as beta1; 15-20 Hz). Early research reported an association between the production of a 12-14 Hz rhythm from the Rolandic cortex during periods of movement suppression (Serman et al., 1974). This, combined with its localisation to the sensorimotor cortex resulted in it being labelled the sensorimotor rhythm (SMR). Evidence of the inhibitory effect of the SMR rhythm on motor activity comes from both quadriplegics and paraplegics who exhibit excessive SMR production (Serman et al., 1974). These findings led to the suggestion that immobility is the most characteristic behavioural correlate of SMR oscillations (Serman & Wyrwicka, 1967). Based on the studies of Serman and colleagues indicating a functional relationship between SMR activity and motor inhibition, it seemed appropriate to consider the application of increasing SMR activity via neurofeedback for children with hyperactive/impulsive behaviour, in which excessive activity or poor motor control is a central feature. Thus, early neurofeedback research focused on increasing SMR activity in children with ADHD in an attempt to reduce the symptoms of their hyperkinetic behaviour, with positive results (Lubar & Shouse, 1976; Shouse & Lubar, 1979). Additional research has shown that voluntary production of the SMR rhythm requires the individual to stabilise and/or suppress motor activity while remaining attentive. This has the effect of reducing the negative hyperactive/impulsive behaviours whilst simultaneously improving attentional capabilities (e.g., Fuchs et al., 2003; Lubar & Lubar, 1984; Thompson & Thompson, 1998). Thus, it appears that enhancing SMR activity may be most beneficial for those children in whom the hyperactive/impulsive behaviours are most prominent.

The rationale for training children with ADHD to enhance their beta activity is based on two findings. First, research examining the QEEG profile of children with ADHD has revealed that they exhibit less beta than age-matched peers (Clarke et al., 1998). In addition to this is the suggested association between beta activity and attention (Linden, Habib, & Radojevic, 1996). Linden et al. (1996) have suggested that higher beta amplitude may be associated with states of high alertness, concentration and focused attention. In addition, research examining the spectral EEG components of individuals during a vi-

sual-spatial selective attention task found increased beta band activity when they attended to stimuli compared to when they did not (Gomez, Vazquez, Vaquero, Lopez-Mendoza, & Cardoso, 1998). Furthermore, research has shown that requiring individuals to divide their attention between two tasks results in a concomitant decrease in beta activity (Kristeva-Feige, Fritsch, Timmer, & Lucking, 2002). These findings are consistent with the suggestion that beta activity may represent a psychophysiological correlate of attentional processing (Vazquez-Marrufo, Vaquero, Cardoso, & Gomez, 2001). Therefore, the low levels of beta produced by children with ADHD are thought to have a detrimental effect on their ability to focus and concentrate. This suggests that training beta activity may benefit those children suffering predominantly from problems of inattention and/or low arousal.

All neurofeedback training to date has utilised the traditional method of training fixed frequency bands. However, it has been suggested that frequency ranges may vary from individual to individual and change as a function of age (Klimesch, 1999). As such the analysis and training of the traditional fixed frequency bands may represent a limitation in the use of neurofeedback. Klimesch and colleagues (Klimesch, 1999; Klimesch, Schimke, & Pfurtscheller, 1993; Klimesch, Schimke, & Schwaiger, 1994) have suggested that when examining the EEG, the frequency bands should be defined individually for each participant. This is particularly important, as different components within the same frequency band have been shown to reflect distinct aspects of cognitive processing (e.g., Klimesch, Doppelmayr, Pachinger, & Russegger, 1997; Klimesch, Doppelmayr, Russegger, Pachinger, & Schwaiger, 1998). Thus, neurofeedback training may prove to be more effective if the training is tailored to the frequency bands of each individual participant.

In summary, there are three main neurofeedback parameters utilised for children with ADHD. These include inhibiting theta, and enhancing either SMR or low beta activity. The theta protocol has been used for all subtypes of ADHD. However, training SMR may be more beneficial for treating the hyperactive/impulsive component whilst training low beta may be more useful in addressing the deficits in attention. Future research could directly address this question by comparing the effect of SMR training to that of low beta training for children with ADHD. Based on the research outlined above, the expectation would be that SMR training should directly affect the hyperactive/impulsive component of ADHD but have little or no effect on the inattentive component. In contrast, low beta training would be expected to reduce levels of inattentiveness but have little or no effect on the hyperactive/impulsive behaviours. Furthermore, recent research suggests that defining each individual's frequency components and training them accordingly may prove to be a more effective method than focusing on traditional fixed frequency bands. Future research could elucidate this by comparing the effectiveness of neurofeedback training using protocols based on traditional fixed frequency

bands versus individual frequency ranges, and then ascertain which is the most efficacious in relieving the symptoms of ADHD.

Neurofeedback Montage. All electric potentials recorded from the scalp are referential, in the sense that potentials at one location are measured with respect to those at another location. The precise number and configuration of electrodes used to record this information is known as a montage. Technical and practical clinical considerations have meant that, traditionally, neurofeedback training is conducted with single-channel recordings using either a referential (monopolar) or sequential (bipolar) montage. This raises the question: which montage is the more effective and why?

It is widely believed that the difference between the referential and sequential montages is the degree to which they measure activity at a given location. Yet this is not necessarily the case. Recorded potentials reflect the integration of the electric field along any path between two recording points. As such, any potential measured at a point on the scalp is not necessarily a characteristic of that point but is, in fact, a characteristic of the path to that point (Katznelson, 1981). Hence, the potentials recorded can be interpreted as activity below the active electrode only if you assume one stable generator, located just below the skull and no additional dipoles at other locations and orientations. However, it has been suggested that this scenario is unlikely (for a more comprehensive discussion of this point, see Nunez, 1981).

Nevertheless, if you accept the limiting assumptions concerning the size, number, and distribution of the neural generators, then referential training can be said to provide a measure of the absolute amplitude at a single 'active' site (e.g., CZ) compared to a 'neutral' reference. Thus, the only way one can learn to alter the relevant EEG component using this montage is by changing the level of activity at or near the electrode site. The main advantage of the referential montage under these conditions is that it allows you to identify activity at a particular location. However, the information is accurate only to the degree that the underlying assumptions are valid.

In contrast, the sequential montage does not depend on such restrictive assumptions for data interpretation. However, the data that result are more ambiguous. For instance, a sequential montage provides a picture of the relationship between cortical activation at two sites (e.g., FCZ and CPZ) but reveals nothing about what's happening at each individual site. As is the case for a referential montage, what is observed during sequential training is what remains in the EEG following rejection of the 'common mode.' Thus, the potentials that are recorded are a measure of how the activity differs between the two sites. As measured in a sequential montage, an increase in amplitude in a given frequency might be accomplished by (a) increasing synchronous synaptic potentials at site A and decreasing them at site B, (b) decreasing the potentials at site A and increasing them at site B, or (c) altering the phase relationship between the two sites (for an interesting discussion of these points, see Putman, 2001). In contrast to the referential montage that is said to provide information on activ-

ity in a given cortical location, the sequential montage may result in feedback that more directly reflects relationships between various parts of the cortex as well as sub-cortical areas. This has led to the suggestion that neurofeedback using a sequential montage might result in “coherence training in disguise” (Putman, 2001).

Each montage has its uses and limitations, the referential montage is purported to provide information about the magnitude of activity in a given area while the sequential montage provides added information about the relationship between cortical activity in two areas. In this regard, Lubar (2001) has suggested that a sequential montage provides a greater opportunity to learn by providing more options for change. In addition, there is evidence of a stronger association between EEG power and cerebral perfusion, as measured by positron emission topography, under electrodes recorded with a sequential montage compared with a referential one (e.g., Cook, O’Hara, Uijtdehaage, Mandelkern, & Leuchter, 1998). Furthermore, a sequential montage may be more robust to movement artifacts since these are more effectively cancelled out in the common mode rejection between two closely spaced electrodes. This might make the sequential montage more attractive to clinicians treating hyperactive children. As a ‘rule of thumb,’ Lubar (2001) recommends that the specific montage used should be the one in which there is the greatest spread between the reward and inhibit frequencies. Utilising the montage that provides the larger spread of power should make it easier to learn the neurofeedback contingencies.

The studies summarised in Table 1 are fairly evenly divided between the uses of either a referential or sequential montage. Nevertheless, all report positive results. As such, it remains an empirical question as to whether one montage is more effective than the other, or even whether the feedback is operating via the same mechanisms. For example, it may be the case that the brain is only able to make global changes, not specific localised ones. In that event, the resulting neural changes might be similar regardless of the type of montage used. In that instance, however, the information from one montage might be utilised more efficiently than the other.

In summary, neurofeedback training may be conducted using either a referential or a sequential montage. An examination of the peer-reviewed outcome studies failed to elicit any evidence of research directly comparing the efficacy of these two configurations during neurofeedback training, although it has been claimed that both montages yield similar results clinically (Putman, 2001). If the two montages are supposed to provide different sorts of information about brain function, it is imperative that claims for neurofeedback as a specific intervention address these issues. As such, it remains the domain of future research to identify which montage is most effective and why.

Neurofeedback Training Sessions. As mentioned above, neurofeedback training is labour intensive. Those considering undergoing or providing the treatment should know what the ideal duration and frequency of a training ses-

sion is and how many sessions may be required before a positive change in EEG profile, behaviour and/or cognitive performance can be objectively identified and sustained.

Although a number of studies have attempted to examine the efficacy of neurofeedback as an intervention for children with ADHD, none have directly compared the duration, frequency and total number of neurofeedback sessions required to elicit a positive outcome for such children. Nevertheless, there is some consensus regarding the duration of each training session. The studies summarised in Table 1 each conducted neurofeedback training sessions that lasted 30 to 60 minutes. However, this may have more to do with the ability of the individual to remain focused for short periods of time, rather than represent a time limit on the duration of neurofeedback training per se.

The frequency of the neurofeedback sessions summarised in Table 1 ranges from once a day to once a week, with the average falling between two to three sessions per week. However, it is unclear at this stage whether a linear relationship exists between the frequency of neurofeedback training and the speed/level of improvement. Future research could directly address this issue by examining whether increasing the number of neurofeedback sessions per week has a direct and proportional improvement on the EEG profile, behaviour and/or cognitive performance of the individuals. At the very least these measurements should be monitored over time to clarify precisely what effects the neurofeedback intervention may have.

Positive changes in the EEG, behaviour and/or cognitive performance have been seen to occur after a minimum of 20 sessions (e.g., Boyd & Campbell, 1998; Lubar & Lubar, 1984; Rossiter & La Vaque, 1995) and a maximum of more than 40 sessions of neurofeedback training (e.g., Carmody, Radvanski, Wadhvani, Sabo, & Vergara, 2001; Lubar & Shouse, 1976; Monastra et al., 2002; Shouse & Lubar, 1979). However, it is unclear at this stage why there is such variability in the number of sessions. One possibility is that this may be due to individual variability, in terms of learning how to alter one's EEG activity via neurofeedback. Alternatively, it may relate to the severity of the disorder, with the more severe cases taking longer to exhibit any positive effects. Nevertheless, future research could help to elucidate these points by monitoring the EEG, behavioural and cognitive changes over time to identify precisely the duration and number of sessions required to influence the outcome of the individual.

An additional point concerning the total number of sessions required is the location where such training should take place. Traditionally neurofeedback training has utilised a therapist directed model, with the therapist providing the treatment for each session. However, the number of sessions available may be limited by the amount of time the therapist can give to such a time-consuming intervention, or the financial constraints experienced by either therapist or patient. The development of new and user friendly equipment means that this need not be the case. It has been suggested that the use of a patient directed

approach may increase the flexibility of the intervention allowing for an increasing number of sessions to be completed, as well as reducing the costs (Rossiter, 1998). In the patient directed approach, the therapist makes an initial assessment and develops the specific protocols to be used. The implementation of the training is taught to the patient's family who continue treatment at home using a home trainer. Future research might address the efficacy of this approach and whether it might influence the generalisation of the learning to other settings.

A further alternative is to apply the neurofeedback training whilst the child is still in school. Limited research utilising this approach has shown that making the treatment a regular part of the individual's education can increase the flexibility in terms of training schedule as well as reducing the costs (Boyd & Campbell, 1998).

Of the fourteen studies summarised in Table 1, eight reported changes in either mean amplitude or frequency ratios in the EEG of children with ADHD. This resulted in EEG profiles of the ADHD children moving closer to that of age matched peers. Nine reported positive changes in behavioural measures and eleven reported improvements in cognitive performance. Only four of the studies reported data from all three measures, with only three of these showing an overall improvement following neurofeedback training. Carmody et al. (2001) reported that there were no clear changes in slow delta-theta activity or in the faster SMR or beta EEG activity, as would be expected following neurofeedback training. Carmody et al. (2001) suggested that this may be the result of a change in the specific parameter, from beta to SMR enhancement, part way through the training. However, a plausible alternative is that the lack of any clear changes in the QEEG profile of the children may be the result of using only auditory feedback as opposed to a combination of visual and auditory feedback (see Modality of Feedback above). Such a possibility is, at this moment, speculative and requires a direct comparison of the effectiveness of feedback modality on changes following neurofeedback training.

In order to clarify the effect neurofeedback training can have on children with ADHD we would suggest that, as a minimum, future researchers monitor changes in the QEEG profile, behavioural patterns and cognitive performance of the children pre- and post-training. In addition, it would also help to explicitly demonstrate what, if any, is the relationship between possible changes in the EEG and concomitant changes in behaviour and cognition.

Neurofeedback Effects: Temporary or Permanent? An important question regarding any form of treatment is whether the positive effects gained during the treatment are a temporary adaptation or a lasting benefit. One way of addressing this is to conduct a number of long-term follow-up studies.

Monastra et al. (2002) conducted a one-week follow-up study of children and adolescents with ADHD that had received either a comprehensive package of clinical care, including medication, parent counselling and school consultation, or a combination of the comprehensive clinical package and neurofeedback. At

the end of the intervention, following a one-week wash out period, QEEG analysis revealed a significant decrease in the theta/beta ratios for the group that received the clinical package combined with neurofeedback compared to those that received the clinical package alone. This indicated evidence of an electrophysiological change, unrelated to medication effects. In addition, the group that received the clinical package combined with neurofeedback exhibited a sustained level of performance on the TOVA that was within the unimpaired range. In contrast, the group that had received the clinical package alone exhibited a significant decrease in their TOVA performance. This suggests that neurofeedback, combined as part of a comprehensive treatment package, may provide long-term benefits.

One of the most comprehensive follow-up analyses of neurofeedback was conducted by Tansey and colleagues (Tansey, 1993; Tansey & Bruner, 1983). Originally Tansey and Bruner (1983) treated a 10-year-old boy diagnosed with developmental reading disorder and hyperactivity. After 20 neurofeedback sessions aimed at enhancing 14 Hz activity the boy was able to produce more of this activity more often. Concurrent with this change in cortical activity there was a specific remediation in reading and comprehension as well as a reduction in his hyperactive behaviour. Furthermore, an initial follow-up done at 24 months post-treatment revealed that he had maintained his behavioural, attentional and academic progress. Ten years after the termination of his treatment Tansey (1993) again examined his EEG, as well as his social and academic performance, and found that the individual continued to exhibit both academic and personal success as well as a normalised EEG profile. However, it is difficult to generalise such findings from a single case study. Also, the sensors used by Tansey and colleagues (Tansey, 1993; Tansey & Bruner, 1983) were larger than those used by other investigators, in that they were 6.5 cm long by 1.3 cm wide, placed approximately 2.6 cm posterior to CZ. Furthermore, all training was conducted with the participant reclined, eyes closed receiving only auditory feedback. Nevertheless, this does suggest that neurofeedback training may result in long-term stability of cortical activity with concomitant benefits in behavioural and academic performance. Such a possibility is consistent with the suggestion put forward by Lubar (1995) that the long-term benefits of neurofeedback training are a result of a learning process that involves the acquisition of self-regulatory skills through operant conditioning. This led Lubar (1995) to conclude that the gains made during neurofeedback treatment are likely to be permanent.

Not all follow-up studies, however, have shown positive long-term results. Although research examining the use of neurofeedback training in a school setting showed improved scores on national achievement tests in a case study of a 10-year-old boy on methylphenidate, a follow-up examination one year after the training was completed revealed that the gains in performance were not maintained (Wadhvani, Radvanski, & Carmody, 1998).

Thus, at this moment evidence for the long-term efficacy of neurofeedback remains equivocal. This highlights the necessity for future research to ensure that follow-up analyses are conducted in order to ascertain precisely what the long-term benefits of such a treatment are.

Negative Effects of Neurofeedback. For any treatment the potential risks or side effects need to be carefully and systematically considered. For example, theoretically there is always the concern of potentially inducing seizures from neurofeedback training, although to date there is no evidence to suggest that this might occur.

Early research utilising an A-B-A design found that neurofeedback training to enhance SMR (12-14 Hz) activity and inhibit theta (4-7 Hz) activity resulted in an increase in baseline levels of SMR activity and a concurrent improvement in hyperkinetic and inappropriate behaviours (Lubar & Shouse, 1976; Shouse & Lubar, 1979). However, a reversal of the neurofeedback training contingencies, from inhibiting theta and enhancing SMR activity to enhancing theta and inhibiting the production of SMR activity, resulted in a reversal of EEG baseline activity to pre-training levels and a concomitant deterioration in behaviour. This led some to suggest that it may be possible for neurofeedback training to have a negative effect, highlighting the importance of training the correct component of the EEG (Ayers, 2001; Chartier, 2001; Hammond, 2001a; Nash, 2001; Stockdale, 2001).

There are no reported negative side effects following neurofeedback training in any of the studies summarised above, and a search of the peer-reviewed literature failed to produce any evidence of neurofeedback having a negative effect. However, clinicians have voiced some concerns. Hammond (2001a) commented that the potential negative effects could include the possibility that individuals may feel anxious and experience difficulty sleeping following training to increase beta activity. In addition, Stockdale (2001) commented that on several occasions training to increase SMR resulted in increased levels of agitation for one patient. However, it should be noted that such reactions are both transient and rare. This led to the suggestion that every session of neurofeedback may result in some shift in consciousness and without QEEG data to guide the training the reinforcement may be in the wrong direction (Chartier, 2001). These comments would suggest that careful assessment of the QEEG might be useful for specifically tailoring and monitoring neurofeedback training so that it meets individual needs.

Controlled Studies. Despite the seeming consistency of the studies summarised in Table 1 which suggest that neurofeedback training may have a positive effect on children with ADHD, the lack of adequate controls and the introduction of possible confounds, such as, failure to control for treatment bias, combining neurofeedback with other interventions, and the failure to control for the level of therapist-patient interaction, represent significant methodological flaws. As such the beneficial effects attributed to neurofeedback might be due, in part, to placebo factors. A review of scientific arti-

cles on neurofeedback and ADHD published from 1966 through 2000 carried out by Baydala and Wikman (2001) found that these methodological flaws resulted in such studies being considered as invalid.

It is often suggested that the best evidence for a clinical intervention would be from a randomised double blind study that includes a control group of patients with the same set of symptoms (e.g., Guyatt, Sackett, & Cook, 1993). However, the “call for placebo or sham controlled double blind studies violate fundamental ethical principles guiding human research in circumstances in which known standard treatments are available” (La Vaque & Rossiter, 2001, p. 24). This represents a core principle of the Declaration of Helsinki, which La Vaque and Rossiter argue, serves as an international ethical guide. La Vaque and Rossiter point out that, rather than comparing a new treatment (e.g., neurofeedback) to a no-treatment placebo, it should be compared to a protocol of ‘known efficacy’ to determine whether such an intervention would result in an equivalent effect. This type of design is often referred to as an active control study, or treatment equivalent study.

With respect to the treatment of ADHD, the efficacy of stimulant medication (e.g., methylphenidate, dextroamphetamine) is well established (Barkley, 1990). Therefore, sham feedback would deny patients access to a proven treatment. Furthermore, La Vaque and Rossiter (2001) point out that offering the treatment to a placebo group after the study has been completed still fails to meet the requirements of the Declaration of Helsinki, because the known standard treatment (i.e., medication) is withheld during the term of the study. Thus, the only ethical means of examining the efficacy of a new treatment, such as neurofeedback, is to compare it to a known effective treatment, not a placebo control. Furthermore, relying on placebo-controlled studies only entails the risk of approving a treatment that may be superior to a placebo, but inferior to the current treatment already available. Active control studies comparing neurofeedback to the standard treatment (i.e., medication) have shown that neurofeedback is ‘equally effective’ in reducing the symptoms of ADHD in children (Fuchs et al., 2003; Rossiter & La Vaque, 1995). However, both studies failed to randomly allocate participants to either condition, and also failed to take into account the degree of therapist-patient interaction, which could significantly influence those in the neurofeedback group who received a much higher level of patient-therapist contact. This implies that caution should be used when interpreting a finding of no difference between a new treatment and an established treatment using an active control paradigm.

An alternative is that the use of placebo controls may be justifiable provided the participants give full and informed consent (Striefel, 2001). In that case it may be possible to argue that a known effective treatment (i.e., medication) is not being withheld if a competent participant has been fully informed

and still chooses to participate in a study involving placebo controls. This approach has been used in the past to examine the effects of EEG and EMG biofeedback training on male opiate addicts in a randomised double-blind paradigm (Cohen, Graham, Fotopoulos, & Cook, 1977). Cohen et al. provided clear and precise information to the addicts regarding the nature of the study and the fact that neither experimenter nor patient would know under which condition an individual had been allocated. After obtaining informed consent the addicts agreed to have their daily dose of synthetic opiate, methadone, reduced to zero and receive only the EEG and EMG biofeedback training or the placebo training. Following fourteen sessions aimed at increasing alpha amplitude and decreasing EMG they found that the experimenters could not determine the experimental status of the participants at better than chance levels, and that the participants were also unable to determine their group allocation. However, this procedure not only reduced the therapeutic success of the intervention, but also revealed no difference in outcome between the contingent and non-contingent groups, which led Cohen et al. to suggest that placebo factors played a role in the treatment. This would suggest that randomised double-blind designs can be useful in teasing apart the active component of a treatment intervention, provided full and informed consent is obtained. However, not all individuals have the capacity to make independent decisions that are in their own best interests. Such individuals may be incapacitated in some way, such as the mentally ill, or have yet to mature to the point of self-determination, as may be the case for children (for a more comprehensive discussion of these issues, see La Vaque & Rossiter, 2001; Striefel 2001).

In summary, designing interventions for the use of neurofeedback for children with ADHD is fraught with a number of ethical issues to which, as yet, there is no clear consensus on how to respond. Indeed, it has been noted that for neurofeedback to gain acceptance, the skeptics require it to meet higher standards than those set for other treatments (Lubar & Lubar, 1999). However, if such an intervention could possibly benefit children with ADHD then it remains the task of those in the field to attempt to meet those high standards. One suggestion aimed at addressing the clinical validity of studies where the clinician is not 'blind' as to the treatment given, is to 'blind' those that assess the clinical outcomes, which may reduce any possible systematic distortion (Guyatt et al., 1993). In addition, future studies should ensure that individuals are randomised to respective conditions, equal levels of contact are maintained between the therapist/researcher and the patient, objective measures are utilised to examine performance pre- and post-intervention, and most importantly, that psychophysiological measures are taken to give an indication of learning (i.e., changes in specific components of the EEG as a function of neurofeedback) and of pre-versus post-changes.

SUMMARY

ADHD represents one of the most common psychiatric disorders of childhood, affecting more boys than girls. A large number of these children exhibit an abnormal EEG profile suggesting that a quantitative analysis of the EEG may aid clinicians in the diagnostic process, as well as providing a clear rationale for the use of neurofeedback as a treatment. In addition, neurofeedback offers a plausible alternative for children with ADHD whose treatment may be limited by side effects and/or poor medication response. However, a better understanding of the neurofeedback process and its efficacy as a treatment for ADHD would be enhanced by encouraging future research to address a number of issues. These include, identifying whether auditory, visual or a combination of auditory and visual feedback would be most effective. Clarifying which feedback specific training parameter is most efficacious, and whether the parameters remain the same for each of the three ADHD subtypes. In addition, follow up studies need to be conducted to examine the potential long-term effects of neurofeedback training. Overall, the findings from the clinical research speak of interesting possibilities; however, it remains the domain of those in the scientific community, aided by clinicians, to clarify precisely what these possibilities are.

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