The purpose of this review is to discuss how new advances in neuroimaging and functional network analyses are applied to electroencephalography (EEG) biofeedback or neurofeedback. Clinical efficacy of one or a few scalp EEG recordings used in the treatment of attention-deficit hyperactivity disorder (ADHD) has been repeatedly demonstrated over the past 34 years. However, a problem is that improved clinical outcome often requires 40 to 80 sessions, which is expensive and difficult for patient compliance. This review cites the scientific literature of direct measures of the nodes and connections between nodes in the attention and default mode networks that are correlated with ADHD using functional magnetic resonance imaging, positron emission tomography, and EEG inverse solutions such as low-resolution electromagnetic tomography. Three-dimensional EEG biofeedback that targets dysregulation in Brodmann areas of the attention and default networks provides increased specificity and can result in improved clinical outcome in fewer sessions.

Introduction

The scientific literature on EEG biofeedback for attention-deficit hyperactivity disorder (ADHD) reports good clinical outcome after about 40 to 80 sessions for about 80% of patients. This includes evidence-based medicine studies using sham controls and/or blind designs that typically require 40 sessions or more to achieve a good clinical outcome (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Wangler et al., 2011). However, with advancements in modern neuroscience in which functional disorders can be linked to brain networks, it is possible to achieve clinical improvement in fewer than 20 sessions. For instance, real-time functional magnetic resonance imaging (fMRI) biofeedback can achieve modification of blood flow in specific brain regions in one 20-min session (e.g., anterior cingulate gyrus; Sulzer et al., 2013; see Dr. Niels Bierbaumer, who explains why only 20 min is required, at http://www.gocognitive.net/interviews/neurofeedback-psychiatric-disorders).

The reason that a single 20-min session achieves clinical change is because of the anatomical specificity of the fMRI method in which one part of the brain linked to the patient’s symptoms is targeted for biofeedback or neurofeedback (NFB). In contrast, surface electroencephalography (EEG) is diffuse and less specific; for example, the EEG recorded from a single scalp location (e.g., Cz) senses sources from widespread regions of the brain and is a mixture of many different frequencies. Surface Z scores improve specificity by isolating dysregulated locations and rhythms, especially when using the Laplacian transform and low-resolution electromagnetic tomography (LORETA) Z scores (i.e., real-time or “live” comparisons to a normative database). For example, LORETA Z score biofeedback also often produces results in one 20-min session because EEG source localization has accuracies similar to fMRI of about 1 cm to 3 cm and therefore is much more specific than is surface EEG (see Koberda, 2011; Koberda, Hiller, Jones, Moses, & Koberda, 2012; Koberda, Moses, Koberda, & Koberda, 2012; Thatcher, 2010, 2012, 2013).

As mentioned previously, the use of a normative database and Z scores is one method to achieve improved clinical outcome in fewer sessions because a more precise and less ambiguous approach is undertaken (Thatcher, 2010, 2011, 2013). A Z score is a standardized score, which indicates the standard deviations a single data point is from the mean of a group of healthy, age-matched subjects. The higher the Z score, the more abnormal the data point. The Z score NFB approach targets specific brain areas and trains toward zero, which indicates normalization (Thatcher et al., 2005; Thatcher & Lubar, 2008). The Z score approach also provides seamless integration with an initial quantitative EEG (QEEG) assessment in which a patient’s symptoms and complaints are linked to functional systems in the brain to
identify the weak systems as defined by Luria (1973) followed immediately by Z score NFB. Increased specificity also includes not directly modifying compensatory systems to improve the efficiency of EEG biofeedback (i.e., fewer sessions with equal or better clinical outcome in comparison with non–Z score biofeedback). Since 2006, there have been a series of publications of Z score NFB showing good clinical outcome with 10 or fewer sessions (Collura, Guan, Tarrent, Bailey, & Starr, 2010; Collura, Thatcher, Smith, Lambos, & Stark, 2008; Hammer, Colbert, Brown, & Illoj, 2011; Stark, 2008; Thatcher, 2010, 2013). Figure 1 illustrates the difference between conventional EEG biofeedback and Z score biofeedback.

**Brain Networks and ADHD**

Modern neuroscience using different measurement modalities, including fMRI, positron emission tomography, EEG/magnetoencephalography, diffusion tensor imaging, and neuropsychological, genetic, and neurochemical studies, has implicated the attention network, default mode network (DMN) and fronto-striatal network abnormalities as contributing to ADHD (Bush, Valera, & Seidman, 2005; Castellanos & Proal, 2012; Durston, 2003; Giedd, Blumenthal, Molloy, & Castellanos, 2001; Kelly, Margulies, & Castellanos, 2007; McCarthy et al., 2013; Schneider, Retz, Coogan, Thome, & Rosler, 2006; Sokunbi et al., 2013; Tamm, Barnea-Goraly, & Reiss, 2012; Vaidya et al., 2005; Vaidya & Stollstorff, 2008; Zametkin & Liotta, 1998). Functional imaging studies on ADHD, in particular, have increased almost exponentially over the past decade. A current PubMed search combining the terms ADHD and imaging returned 1,259 results, with more than 100 papers published in the past year alone. Given this vast body of research on neuroimaging of ADHD, this article cannot be comprehensive, nor will it critique individual articles. Instead, it will focus on new advances in understanding of brain networks and connections between nodes of networks within the ADHD literature. It will then seek to place this new information in the context of recent advances in EEG biofeedback in which dysregulation in nodes and connec-

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**Figure 1.** Top row is conventional or standard electroencephalography (EEG) biofeedback in which different units of measurement are used in an EEG analysis (e.g., uV for amplitude, theta/beta ratios, relative power 0% to 100%, coherence 0 to 1, phase in degrees or radians, etc.), and the clinician must “guess” at a threshold for a particular electrode location and frequency and age to reinforce or inhibit a give measure. The bottom row is Z score biofeedback, in which different metrics are represented by a single and common metric (i.e., the metric of a Z score) and the guess work is removed because all measures are reinforced to move Z scores toward $Z = 0$, which is the approximate center of an average healthy brain state based on a reference age-matched normative database in real time. The goal is to reinforce increased stability in brain networks linked to symptoms.
tions in relevant brain systems can be made the target of real-time biofeedback with the goal of reinforcing stability and increased information processing in these networks.

**Attention Network**

Attention is a crucial aspect of consciousness and involves selecting a small subset of stimuli from a larger universe for entry into consciousness and the creation of new memories and new actions. Petersen and Posner (2012) reviewed years of neuroimaging research on attention and iterated their earlier models of hierarchical attention starting with (a) alerting responses, (b) orientation responses, and, (c) executive attention. The alerting network is based in the brainstem with emphasis on the locus coeruleus, cerebellum, basal ganglia, and regions of the frontal and parietal cortex. The orienting network consists of a dorsal and ventral branch, where the dorsal network consists of the frontal eye fields and the intraparietal sulcus and superior parietal lobe. The ventral attention network consists of parts of the temporal-parietal junction and the ventral frontal cortex. The executive attention network consists of the cingulate cortex-opercular frontal cortex and the fronto-parietal system.

Real-time LORETA Z score EEG biofeedback is used to target current source density or the amount of current produced by a synchronized group of neurons in the attention networks depending of the indicators of dysregulation based on age-matched Z scores. The goal is to reinforce increased stability within the various aspects of the human attention network depending on the patient’s symptoms. Figure 2 is a Talairach atlas representation of the Brodmann areas (nodes) and connections between Brodmann areas of the dorsal attention network that is used in LORETA Z score NFB (Koberda, 2011; Koberda, Hiller, et al., 2012; Koberda, Moses, et al., 2012; Thatcher, 2012, 2013).

Figure 3 is a Talairach atlas representation of the Brodmann areas (nodes) and connections between Brod-
mann areas of the ventral attention network that is used in LORETA Z score NFB.

Figure 4 is a Talairach atlas representation of the Brodmann areas (nodes) and connections between Brodmann areas of the executive attention network that is used in LORETA Z score NFB.

Default Mode Network
During reflective moments in which one is at rest and not engaged in a task and instead ruminating about the past and future (e.g., self-narrative), there are changes in the synaptic synchrony of millions of neurons connected in a network called the “default mode network” (DMN). The DMN is suppressed when the attention network is active or anticorrelated with the attention network and is constituted primarily by the posterior cingulate gyrus, hippocampus, medial frontal lobes, temporal lobes, and parietal lobes with approximately five times the number of synaptic connections than any other cortical network (Buckner, Andrews-Hanna, & Schacter, 2008; Hagmann et al., 2008). Reciprocal inhibitory and excitatory dynamics occur in the subsecond time domain that only QEEG is capable of detecting in contrast to eyeball visual examination of EEG traces.

The DMN is what one’s brain does when not engaged in specific tasks. It is the busy or active part of our brains when we are mentally passive. According to Bressler and Menon (2010), the “DMN is seen to collectively comprise an integrated system for autobiographical, self-monitoring and social cognitive functions” (p. 285). It has also been considered as responsible for rapid episodic spontaneous thinking. That is, the spontaneous mind wandering and internal self-talk and thinking that one engages in when not working on a specific task or when completing a task that is so automatized (e.g., driving a car) that our mind starts to wander and generate spontaneous thoughts. It is likely that people differ in the amount of spontaneous mind wandering (which can be both positive creative thinking or distracting thoughts), with some having a very unquiet mind that is hard to turn off and others being able to turn off the inner thought generation and self-talk and display tremendous
self-focus or controlled attention to perform a cognitively or motorically demanding task. The anterior insula and anterior cingulate gyrus are referred to as the salience network, which is involved in switching the DMN and executive attention network on and off in a reciprocal manner (Bressler & Menon, 2010; Lerner et al., 2009; Sridharan, Levitin, & Menon, 2009).

In evaluating and treating ADHD, the link between the attention network and the DMN is important. For example, imagine a child sitting in a classroom attending to a lecture, when suddenly a thought from the past intrudes into consciousness. It could be something a parent said or someone else in the past said, and there is an attention shift in mood and conscious content away from the lecture to a self-narrative mediated by the DMN. After a few moments, the child shifts attention back to the lecture, but now some of the lecture is missed, and it may have been an important part of the lecture. Excessive intrusion of self-narrative memories and thoughts is an example of why both the attention network and DMN as well as the insular cortex involved in switching between the two systems may need to be included in an NFB protocol. It is important for the clinician to ask questions about the nature of the problems the client has with sustaining or shifting attention to better predict which aspects of the attention dynamic may be dysregulated in order to select an optimal protocol for LORETA Z score NFB.

Figure 5 is a Talairach atlas representation of the Brodmann areas (nodes) and connections between Brodmann areas of the DMN that is used in LORETA Z score NFB.

Standard surface EEG measures of connectivity such as coherence, phase differences, comodulation, and phase reset have also been applied to Brodmann areas and three-dimensional networks using LORETA (Hochstetter et al., 2004; Langer et al., 2012; Langer, von Bastian, Wirz, Oberauer, & Jäncke, 2013; Lehman, Faber, Gianotti, Kochi, & Pascual-Marqui, 2006; Pascual-Marqui, Koukkou, Lehmann, & Kochi, 2001; Thatcher, 1995, 2013; Thatcher, Biver, & North, 2007; Thatcher, Wang, Toro, & Hallett,
As mentioned previously, direct measurement of network connectivity has provided increased specificity and improved clinical outcome in fewer sessions using three-dimensional LORETA Z score NFB (Koberda, 2011; Koberda, Hiller, et al., 2012; Koberda, Moses, et al., 2012; Thatcher, 2010, 2013). Recently, the ability to use fundamental network metrics such as phase lock duration and phase shift duration between Brodmann areas comprising the attention and default networks has been successfully used for NFB. Figure 6 is an example of phase shift duration between some of the Brodmann areas that comprise the DMN and emphasizes the increased temporal and anatomical specificity when using three-dimensional EEG source analyses such as LORETA.

EEG phase lock duration has a correlation of approximately .86 with EEG coherence; however, the three-dimensional measurement of phase lock duration between Brodmann areas produces more discrete and precise ranges of phase lock than is observed at the scalp surface (Thatcher, 2012; Thatcher, North, & Biver, 2008, 2009). Figure 7 is an example of the increased temporal discreteness and temporal precision of phase locking observed with LORETA analyses of time series from different Brodmann areas that comprise the DMN.

Figure 8 shows a further example of increased spatial and temporal specificity of EEG phase shift and phase lock when applied to three-dimensional imaging of the default and attention networks. That is, there is a systematic relationship between the duration of phase shift and phase lock and the distance between Brodmann areas as nodes of networks (Thatcher, 2012). As Donald Hebb conceived, neurons that “fire together wire together,” and dysregulation in networks is indicated by either too much or too little functional connectivity involved in neural resource recruitment and resource allocation for brief periods of time (Hebb & Penfield, 1940; Hebb, 1949, p. 62). Optimal information processing in networks can now be reinforced using NFB at spatial and temporal resolutions that are much greater than in studies that use only one or two scalp electrodes.
Conclusions
The goal of this brief review was to introduce the reader to new advances in EEG network analyses as applied to EEG biofeedback of the attention and default networks in patients with ADHD. Improved clinical outcome in fewer sessions can be achieved by thorough clinical assessment that links symptoms to networks of the brain related to the symptoms followed by reinforcement of stability and increased information processing between nodes of the relevant dysregulated networks. The nearly 60 years of science in support of the mechanisms of operant conditioning and the application of operant conditioning to treat ADHD have reached a level of maturity at which clinicians can use affordable EEG technology to treat patients and achieve improved clinical outcome in fewer sessions than was the case in the past. It is important to continue to use
Assessment and Treatment of ADHD Using Network Analyses

modern neuroscience of brain networks for both assessment and treatment while at the same time adhering to the fundamental principles of operant conditioning (Thatcher, 2012).

References


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