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EEG Biofeedback for Autism Spectrum Disorder: A Commentary on Kouijzer et al. (2013)

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Abstract Research conducted by Kouijzer et al. (Appl Psychophysiol Biofeedback 38(1):17–28, 2013) compared the effects of skin conductance biofeedback and EEG-biofeedback on patients with autistic spectrum disorders to determine their relative efficacy. While they found a difference between treatment and control groups, there was no significant difference on many variables between the two treatment groups. From this, the increase in symptom alleviation from autistic spectrum disorder was attributed to non-specific factors surrounding the study. We now offer alternative explanations for their findings and propose different options for future studies. We hypothesize that the location and type of neurofeedback used adversely impacted the findings. We speculate that had they used a form of EEG-biofeedback that can combat deficiencies in connectivity and also trained the areas of the brain most affected by autism, there may have then been a significant difference between the effectiveness of EEG-biofeedback versus skin conductance biofeedback.

Keywords EEG-biofeedback · Autism spectrum disorders · Neurofeedback · Coherence · Connectivity

Introduction

There have been many studies to assess the positive effects EEG-biofeedback has on individuals with autistic spectrum disorders (ASD). Researchers have examined and shown improvements in children with ASD including aspects of their social interactions, executive functioning and in both verbal and non-verbal communication after undergoing EEG-biofeedback (Coben and Padolsky 2007; Jarusiewicz 2002; Kouijzer et al. 2009, 2010; Scolnick 2005; Sichel et al. 1995; Thompson et al. 2010). Not only does EEG-biofeedback effectively influence power frequencies (Scolnick 2005; Sichel et al. 1995; Kouijzer et al. 2009), but it can also change coherence and connectivity patterns across specific regions of the brain (Coben and Padolsky 2007).

With most experiments, methodological issues decrease the validity of results and the above referenced EEG-biofeedback studies are not without similar struggles. One of the main critiques of the effectiveness of EEG-biofeedback on autism is the notion that positive increases in performance could be attributed to non-specific factors surrounding treatment and not the treatment itself. Heinrich et al. (2007) speculate that improvements are from the patient's routine contact with the therapist and/or other non-specific factors such as treatment expectancy.

The aforementioned study by Kouijzer et al. (2013) attempted to control for these shortcomings and sought to investigate if it was in fact, the EEG-biofeedback that caused improvements in children with autism or other non-specific factors. They studied 35 children confirmed to have autism based on the autism diagnostic interview-revised (ADI-R; Lord et al. 1994) and randomly divided them into three groups: an EEG-biofeedback group, a skin conductance biofeedback group, and a wait list group that received no treatment. She judged the improvements of the participants based on the Clinical Global Impression 'improvement scale'. Cognitive flexibility was assessed with the trail making test (TMT; Reitan 1956), inhibition was assessed through the Stroop Task (Stroop 1935) and planning through the Tower of London test (TOL; Kovacs

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2005a). Attention was evaluated through the Test of Sustained Selective Attention (TOSSA; Kovacs 2005b) and working memory through the subtest Digit Span that was adopted from the Wechsler Intelligence Scale for Children, 3rd version, Dutch version (WISC-III-NL; Kort et al. 2002). Lastly, a Mitsar EEG 201 system recorded and digitized their 19-channel EEG with a full measurement of physiological brain functioning. Kouijzer et al. (2013) performed and gathered the EEG biofeedback with an electrode attached to the participant's scalp at either Cz or FCz depending on where the largest deviation from normality was, according to the Neuroguide database (Thatcher et al. 2003). Skin conductance biofeedback was measured via electrodes attached to participants' index and ring fingers. Both scalp and finger electrodes were hooked up in both EEG and SC conditions, in order to protect the experimenter from knowing which intervention they were administering. Researchers used a monopolar form of EEG-biofeedback, with the referencing site behind the ears. After analyzing all the data, Kouijzer showed that, with the exception of cognitive flexibility, both EEG and SC biofeedback had similar improvements in all tested measures when compared to the wait list group. Participants showed significant increases on the TMT after undergoing EEG-biofeedback. Similarly, symptoms of ASD changed the same amount between the EEG and SC groups based on questionnaire responses.

Discussion

Again, like most studies, Kouijzer et al.'s was not without limitations. Kouijzer et al. (2013) trained at Cz or FCz, but many other studies have shown that area to be minimally affected by autism. A study conducted by Coben et al. (2013) examined variable resolution electromagnetic tomography (VARETA) images to determine the specific areas of the brain impacted adversely in children with autism. When examining the VARETA images of participants with ASD, they found consistent trends among the children. These data showed increased activity in the cerebellum, thalamus, hippocampus, parahippocampal, cuneus, cingulate, and lingual gyrus and in temporal, precentral, postcentral, parietal, and occipital cortical regions. According to VARETA images, there were consistent neuroanatomical structures that showed abnormalities across all participants with ASD and these areas may represent the structures that show dysfunction in ASD. These findings support the theory that a characteristic of ASD is bilateral brain dysfunction impacting both anterior and posterior axes. The amygdala, superior temporal sulcus region, and fusiform gyrus were also found to function differently in ASD (McAlonan et al. 2005). Travers and Alexander (2013) found differences in the level of functioning of the frontal gyrus as well. Another multimodal imaging study conducted by Travers and Alexander (2013) on a 63-year old man showed the corpus callosum, the amygdala, hippocampus, caudate nucleus, and frontal and occipital lobe to be areas of interest. They discovered that fiber tract bundles were larger in the medial temporal area of the right hemisphere rather than the left. Other diffusion tensor imaging (DTI) studies have found differences between patients with ASD and control groups in the white matter (WM) around the amygdala as well as other areas (Barnea-Goraly et al. 2004; Noriuchi et al. 2010).

Coben (2013) also revealed increased frontal coherence and decreased anterior and posterior/temporal coherence between the two hemispheres in patients with ASD, leading to the conclusion that children with ASD often have bilateral dysfunction that affects both anterior and posterior axes as well. Consistent with previous literature, their researched showed how people with autism have disruptions in brain connectivity. Other work done by Li et al. (2012) and Wass (2011) discovered that patients with autism exhibit hyperconnectivity in frontal and local short neuronal paths. They also exhibit hypoconnectivity in posterior to anterior or posterior to temporal long-distance pathways.

Pelphrey et al. (2004) and Welchew et al. (2005) have shown links between dysfunctions in social cognition and language deficits in autism to neural substrates. After completing a sentence comprehension test, the arcuate fasiculus between Broca's area and Wernicke's area was shown to have less functional connectivity in participants with autism than the control group (Just et al. 2004; Travers and Alexander 2013). This test was administered to evaluate information organization and neural synchronization during language tasks and showed the correlation between language deficiencies and ASD. Not only does Autism affect the left side of the brain hindering language, but it also influences the right side affecting social interactions. Travers and Alexander (2013) discovered that the right hippocampal fusiform pathway in patients with ASD had decreased diffusion perpendicular to the fiber tract and that this was associated with decreased performance on the Benton facial recognition task (BTOFR; Benton et al. 1983).

The thalamus is another area where alteration in connectivity in autism is found. Microstructural differences in radiation of the anterior thalamus have also been discovered in ages 12–24 months, 11, 12.8, and 39 years (Travers and Alexander 2013). Multiple studies of patients with ASD have decreased leftward lateralization in ASD in the arcuate fasciculus (Fletcher et al. 2010; Lo et al. 2011), and the uncinate fasciculus (Lo et al. 2011), and in the WM of the superior temporal gyrus (Lange et al. 2010).

A study conducted by Catani et al. (2008) attempted to analyze short intercerebellar fiber connections and long afferent and efferent cerebellar tracts. They discovered decreased fractional anisotropy in the short fiber connection compartment and in the long efferent fibers. These deficiencies have the potential to impair the development of motor movements, motor learning, and other non-motor functions (Catani et al. 2008). Based on these studies, we see that autism is primarily a disorder of neural connectivity and therefore needs to be treated as such.

We believe results in the study conducted by Kouijzer et al. (2013) could have been different had they applied the EEG-biofeedback differently. Because they used a monopolar form of training, they were unable to train communication across brain regions. From the previous research, we see that ASD affects many areas, such as posterior to anterior connections, connections to the temporal lobes and connections in deep brain structures. Rather than focusing on the more critical areas, Kouijzer et al. (2013) instead trained areas Cz and FCz, which are more commonly unaffected by ASD. In order for treatment to be maximally effective, we theorize that it is imperative that the researcher train the most impacted areas of the brain that are at the root of the symptoms of autism. We speculate that the difference between EEG-biofeedback and SC-biofeedback would have been more significant if such regions were chosen and trained in a fashion to emphasize impairments in coherence or connectivity. We hypothesize that an effective way to treat people with ASD is by EEG-biofeedback, focusing on coherence and connectivity in the proper location of the brain. This is not a proven best method to treat autism, but based on prior research, is a viable option for future studies. We seek to discover evidence to develop the most effective treatment for those with ASD and recommend further research be conducted that could validate or dispute our theories.

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