

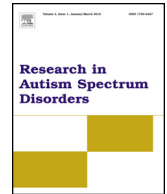


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Review

Electroencephalographic studies in children with autism spectrum disorders



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ABSTRACT

An important factor in the diagnosis and treatment of Autism spectrum disorder (ASD) is prescribed Electroencephalography (EEG). EEG changes may show the following: slowing, asymmetry, sharp waves or spikes, sharp and slow waves, generalized sharp and slow waves, or generalized polyspikes in a distributed or general area, multifocal or focal, unilateral or bilateral, and they may be located in many different areas of the brain. There is a need to look for a EEG phenotype typical of patients with ASD. The importance of gamma waves, rhythm mu, mirror neurons, and their role in patients with ASD was discussed. Epilepsy is reported to occur in one third of ASD patients. In ASD, seizures and EEG paroxysmal abnormalities could represent an epiphenomenon of a cerebral dysfunction independent of apparent lesions. This article reviews ASD and EEG abnormalities and discusses the interaction between epileptiform abnormalities and cognitive dysfunction.

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1. Introduction

Autism spectrum disorder (ASD) is a complex neurological disorder that has been observed, defined, and diagnosed for many years (Griffin & Westbury, 2011). The prevalence of ASD in the general population is around 1 in 88. (Bagasra, Golkar,

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Garcia, Rice, & Pace, 2013). It is a disorder of the brain and is manifested in three areas: impaired social interaction, difficulties with verbal and nonverbal communication, and a limited number of interests and activities. Symptoms usually appear during the first three years of life and can persist throughout a patient's lifetime. Recent researches on autism have led to the development of the concept of ASD, which allows diagnoses to include individuals with varying degrees of impairment and levels of functioning (Bluestone, 2005). Ancillary symptoms may encompass obsessive-compulsive disorder, sleep disturbances, hyperactivity, attentional problems, mood disturbances, gastrointestinal symptoms, self-injurious behavior, ritualistic behavior, and sensory integration disorders. ASD is generally considered a lifelong disability of yet undetermined etiology without an established confirmatory laboratory test and, to date, without universally established, curative pharmacological or behavioral therapies (Duffy & Als, 2012). Many studies suggest that ASD is a connectivity disorder (Assaf, Jagannathan, Calhoun, et al., 2010; Belmonte, Allen, Beckel-Mitchener, et al., 2004). Furthermore, changes in brain development are known in at least some cases to precede observable changes in behavior. It is thus reasonable to conjecture that electroencephalography (EEG) signals may demonstrate discernible patterns, reflecting information about the underlying neural networks that precede changes in behavior (Bosl, Tierney, Tager-Flusberg, & Nelson, 2011a, 2011b). These data, appropriately, have spawned much research into the exploration of potential etiologies as well as the development of diagnostic tests, particularly in terms of neuroimaging and EEG (Bluestone, 2005; Duffy & Als, 2012).

Early detection of abnormalities in EEG signals may be an early marker for the development of cognitive impairment. The differences seem to be the greatest between the ages of 9–12 months. Using several machine-learning algorithms with assessment of the size of the entropy as a feature vector, infants were classified with over 80 percent accuracy with the control group and high risk of autism at the age of 9 months. Classification accuracy for boys was close to 100 percent at the age of 9 months and remained high (70–90 percent) at ages 12 and 18 months. For girls, the classification accuracy was highest at the age of 6 months but declined in subsequent years (Coben, Clarke, Hudspeth, & Barry, 2008; Linden, 2006; Sohal, Zhang, Yizhar, & Deisseroth, 2009).

2. The phenotype of autism

Endophenotypes are biological markers associated with a given disorder and provide insight to its origins. One characteristic of endophenotypes is that they are often present in the first-degree relatives of affected individuals (Tierney, Gabard-Durnam, Vogel-Farley, et al., 2012). Endophenotypes have been identified in family members of individuals with a variety of neuropsychiatric disorders such as depression, schizophrenia (Turetsky, Calkins, Light, Olincy, et al., 2007), bipolar disorder, and Attention Deficit Hyperactivity Disorder (ADHD) (Castellanos & Tannock, 2002). Inheritance of cognitive disorders such as schizophrenia, ADHD, and autism ranges from 50 to 80 percent. It is difficult to identify the actual genetic variants responsible for this inheritance. In the case of schizophrenia and autism, few genetic variants have been identified.

The first endophenotype found in ASD is paroxysmal EEG epileptiform activity. This endophenotype has an incidence of approximately 35–40 percent. With this subtype, the abnormality often appears on the left temporal lobe where speech and language occur.

The second type is characterized by the presence of the phenotype of the mu rhythm. The EEG pattern in the form visible in the central area is neurologically normal. This pattern is normally seen only when the frontal lobes' mirror neuron system is not engaged and disappears when the mirror neuron system is engaged (Neubrandner, Linden, Gunkelman, & Kerson, 2011).

The subtype of the third pattern is a high beta, which can be observed in the EEG results of patients with ASD (Johnstone, Gunkelman & Lunt, 2005). This subtype is characterized by easily ignited mood change or irritability. This may be associated with sensory hypersensitivity, in regard to the sensory areas of the brain, but it can also be associated with impulsivity and explosiveness looking frontally, in particular to the right.

The fourth endophenotype is a coherence dysregulation (Johnstone et al., 2005; Neubrandner et al., 2011; Pop-Jordanova, Zorcec, Demerdzieva, & Gucev, 2010).

It is known that there are no brain tasks that happen in a single part of the brain, and a larger percentage of the brain is needed for individual tasks. Identified patterns of hyperconnectivity in the bilateral frontotemporal regions and between the left and right side of the hemisphere include hypercoherence (too many connections), which often relates to obsessiveness, and hypo-coherence (too little connectivity), which is related to inattention and cognitive difficulties (Thatcher, 2001).

The fifth autism subtype is very high delta activity, which represents significant cortical slowing and often corresponds to extreme activity (hyperactivity), impulsive behaviors, and inattention (Linden, 2006). High delta can activate overlaps or occur in combination with theta activity (which represents inattention, impulsivity, and hyperactivity).

The sixth pattern is characterized by very low-voltage EEG and dominated by slower wave activity (Linden, 2006; Neubrandner et al., 2011). This low-voltage, slow EEG is identified in diffuse encephalopathies and specifically suggests that toxic or metabolic etiologies should be ruled out.

EEG endophenotypes can help us understand where in the brain, in which stage, and during what type of information-processing these genetic variants play a role. With increased understanding of how genes affect the brain, combinations of genetic risk scores and brain endophenotypes may become part of the future classification of psychiatric disorders and ASD (Moskvina, Craddock, Holmans, et al., 2009).

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