EEG for children with ASD: evidential considerations for routine screening

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The discovery of frequent EEG abnormalities in ASD was first reported in 1970 by Gubbay et al.

Estimates for the risk of epilepsy in children with ASD range from appox 1:10 to 1:3. In the general population the prevelence of epilepsy is less than 1%.

The visual inspection of the EEG recording performed by an EEG expert remains the only reliable method capable of detecting paroxysmal or epileptiform activity, particularly when they are infrequent or low in amplitude.

The overall literature mean of the prevalence of E.D.s (epileptiform discharges) was 40% in ASD cases.

Hara concluded in a 10-year retrospetive follow-up study (n=130) of children with ASD without a hx of seizures that childhood E.D.s predicted subsequent onset of epileptic seizures during adolescence. 25% developed either partial or generalized seizures, with onset from 8-26 years of age.

An overnight EEG study (n=889) of children without a dx of epilepsy but with ASD finds a 60% prevalence rate of E.D.s compared to 5% prevalence rate of E.D.s in healthy children.

EEG abnormalities in ASD include generalized and focal slowing, epileptiform activity and seizures. There is growing evidence that E.D.s are not asymptomatic as has once been widely accepted. It is suspected that the location of the E.D's may contribute to clinical symptomatology. Some studies suggest that temporal discharges are more common.

Investigators have established that E.D.s manifest differently depending on where they originate in the brain.

Right hemisphere – impairments of visual-spacial and visual-motor tasks more common

Both Right and Left – memory deficits

Benign rolandic E.D.s – depression and behavioral issues

Limbic system – anxiety and panic attacks

Temporal lobes – episodic behavioral dyscontrol

Frontal lobes – violent acts committed against strangers

A single interictal discharge can produce neuronal, vascular and metabolic changes throughout the brain. Although controversial, Trojaborg asserts that there is an increased incidence of cognitive, behavioral and/or emotional issues associated with paroxysmal activity regardless of the location of the foci.

ASD and epilepsy share etiology. There are shared abnormalities in gene transcription regulation cellular growth, synaptic channel function, and maintenance of synaptic structure. They share underlying brain structure abnormalities. SPECT shows hyper- and hypo-perfusion in the prefrontal lobes, cingulate gyrus, superior temporal gyrus and mesial temporal lobes.

There are two man hypotheses about the relationship between ASD and epilepsy:

1) Shared genetic defects in GABAergic fibers
2) That primary epilepsy may impact synaptic plasticity and predispose the manifestation of ASD

There appears to be 2 peaks of epilepsy onset in ASD: one in early childhood and a second in adolescence. Hormonal influences on neuronal excitability suggest that puberty may be a trigger for epilepsy.

Kanemura, et al, found that EEG paroxysmal abnormalities in childhood predict onset of epileptic seizures in adolescence. Epileptiform discharges may be an indication of underlying brain neurophysiological dysfunction, which may manifest in behavioral aberrations, even if not sufficient to result in observable seizures.

Treating and preventing epileptigenesis may help to both avert seizures and prevent further cognitive impairment with children.

25% of children with ASD who have epileptiform abnormalities go on the develop epilepsy.

For individuals with ASD who are receiving treatment for behavioral concerns, there is a high prevalence of psychotropic prescribing, most frequently including antipsychotics, stimulants and antidepressant drugs. These medications, particularly the antipsychotics, tend to lower the seizure threshold, which creates the potential in turn to exacerbate psychiatric, cognitive and behavioral issues.

The evidence provided in this review suggests that EEG screening is necessary for the selection of psychotropic medications for children with ASD.