'Treat the patient, not the EEG'?

Editorial

It would be difficult to find a pediatrician or neurologist who has not heard this statement when discussing abnormalities in children's EEG results. Like all generalizations, it oversimplifies a complex situation; in this case the relationship between clinical seizures and epileptiform EEGs. Epileptic seizures are the neurological signs and symptoms resulting from abnormal, synchronous discharge of cerebral neurons. The recognition of epilepsy is traditionally based on the presence of current paroxysmal clinical events. Epileptiform EEG activity, on the other hand, is the recorded correlate of pathological neuronal discharge which occurs in those with epilepsy but also in a surprising number of children without seizures (for example, in a considerable number of children with cerebral palsy). Thus, the admonition is to rely on the patient's symptoms rather than the presence of EEG abnormalities for treatment decisions.

So where is the complexity? As the study by Deonna et al.[1] in this month's journal shows, complexity may lie in recognizing seizures and their subtle effects. Based on this and related research, there is reason to believe that prominent epileptiform discharges may correlate with poorly recognized, but developmentally important, neuropsychological deficits in some children.

This relationship was convincingly demonstrated by Aarts et al.[2] in the mid-1980s when simultaneous EEG monitoring and detailed neuropsychological testing documented transitory cognitive impairments (TCIs) during generalized and focal epileptiform discharges. Their study was also the first to show that TCIs result from disruption of isolated cognitive functions localized at the region of epileptiform discharge, rather than from generalized effects on consciousness. Subsequent research has confirmed the occurrence of isolated memory and language deficits when children attempt to perform specific tasks during periods of 'subclinical' epileptiform discharge[3]. Consequently, these observations have expanded our view of the clinical symptoms of epilepsy to include functionally-specific cognitive processing deficits which are identifiable only with specialized testing.

Whether TCIs actually impact on children's daily functioning or have cumulative effects on cognitive, linguistic, or psychosocial development is not yet clear. Nonetheless the occurrence of TCIs has raised questions about the relationships among epileptiform EEGs, seizures, and developmental impairments. These issues are particularly important in two groups of children: those with known epilepsy associated with cognitive dysfunction and those without recognized seizures who have primary developmental deficits and severely-epileptiform EEGs.

It is well known that persistent and prolonged epileptiform discharge can be associated with developmental regression[4], although most children with 'silent' epileptiform activity do not appear to be adversely affected. It now appears that there are also children with epileptiform EEGs who develop limited and often fluctuating impairments of linguistic, oral–motor, and other developmental skills[5]. As the deficits may not be readily apparent in these cases, and although we do not 'treat' the EEG as such, it is the EEG that nonetheless indicates the need for specialized testing and further evaluation.

With the exception of children with autism and Landau-Kleffner syndrome, the relationship between cognitive symptoms and childhood epilepsy has received relatively little attention from North American researchers. The time has come for collaboration with our European collegues and increased sensitivity to the potential developmental impact of prominent epileptiform discharges. At the very least, we should consider increasing the use of simultaneous video-EEGs and neuropsychological testing in children with severely epileptiform EEGs, including those with benign partial epilepsy.

Without question, further research is needed. Critical issues include the prevalence of cognitive effects related to epileptiform activity in population-based studies, the role of age at onset and localization of epileptiform discharges in producing symptoms, the scope and long-term outcome of these cognitive impairments, and the potential benefits of therapeutic intervention. Such studies will begin to answer the important questions raised by Deonna and co-authors. Progress is often made by challenging truisms: to paraphrase Claude Bernard, the great physiologist of the mid-nineteenth century, 'It is what we think we already know that often prevents us from learning'[6].

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