One Size DOES NOT Fit All

How to Tailor Individualized Treatment Regimens in Epilepsy Care

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Video Interviews with Epilepsy Experts

Tobias Loddenkemper, MD speaks about the importance of correct analysis and interpretation in pediatric EEG, as part of Practical Neurology’s video coverage of the 2016 American Academy of Neurology (AAN) Annual Meeting. In particular, he addresses maturational variance, pediatric epilepsy syndromes, and how to select candidates for surgery.

Christina Bergqvist, MD discusses the emerging role of dietary therapies for epilepsy, featured in Practical Neurology’s video coverage of the 2015 American Epilepsy Society (AES) Annual Meeting. Dr. Bergqvist shares insights on how the central nervous system changes during status and why dietary therapies may help to stop the process.

Visit the video section of PracticalNeurology.com to see more videos. Stay tuned for our video coverage of the upcoming 2016 AES Annual Meeting in December. Go to www.practicalneurology.com and sign up to receive email updates.
Hyperphosphorylated Tau Correlates to Cognitive Decline in Refractory Epilepsy

An epilepsy-related tauopathy appears to contribute to accelerated cognitive decline and has diagnostic and treatment implications in patients with refractory epilepsy. In a new study, researchers performed pathological examination on tissue from 33 patients who had undergone temporal lobe resection between ages 50 and 65 years to treat drug-refractory temporal lobe epilepsy. They identified hyperphosphorylated tau protein using AT8 immunohistochemistry and compared this distribution to Braak patterns of Alzheimer’s disease and patterns of chronic traumatic encephalopathy. They found that 94 percent of patients showed hyperphosphorylated tau pathology in the form of neuropil threads and neurofibrillary tangles and pre-tangles, while Braak stage analysis showed 12 percent of the epilepsy cohort had a Braak staging III-IV compared to an age-matched non-epilepsy control group from the literature (eight percent).

The researchers also identified a mixture of tau pathology patterns characteristic of Alzheimer’s disease and chronic traumatic encephalopathy. Further, they noted that more extensive the tau pathologies translated to greater decline in verbal learning, recall, and graded naming test scores over one-year post-temporal lobe resection.

—Brain. 2016 Sep; 139(Pt 9): 2441-55

Increased Comorbidities Linked to Pediatric Epilepsy

The frequency of comorbid diseases is high in children with epilepsy, even in presumably uncomplicated cases, according to new findings. Using registry data over a five-year period to describe frequencies of medical, neurologic, developmental, and psychiatric conditions occurring before and after children are diagnosed with childhood epilepsy, researchers compared children with epilepsy to the general child population (adjusting for sex and age), as well as children with complicated epilepsies to those with uncomplicated epilepsies. They found that nearly 80 percent of children with epilepsy had greater than one comorbid disorder. Moreover, all types of disorders were more frequent in children with epilepsy—55 percent had additional medical disorders, 41 percent had additional neurologic disorders, and 43 percent had developmental/psychiatric disorders. The data also indicated that children with complicated epilepsies had the highest overall levels of comorbidity, but the risk of medical and psychiatric comorbidities was also substantial among children with uncomplicated epilepsies.

—Pediatrics. 2016 Sept

Higher Density of Neurons Discovered in White Matter in Patients with Temporal Lobe Epilepsy

Patients with temporal lobe epilepsy have a higher density of neurons in the arch- and neocortical white matter, according to a new study. In an effort to characterize these neurons and investigate their distribution in mesial temporal sclerosis, researchers found that both excitatory and inhibitory cells were present among these neurons and that a subset of neurons in the white matter was Tbr-1-immunoreactive; these neurons coexpressed NeuN and neurofilament marker SMI311R. No co-localization of Tbr1 was observed with the inhibitory neuronal markers, calcium-binding proteins. “We suggest that a large population of white matter neurons comprises remnants of the subplate,” the investigators wrote. “Furthermore, we propose that a subset of white matter neurons was arrested during migration, highlighting the role of cortical maldevelopment in epilepsy associated with mesial temporal sclerosis.”