Hippocampal sclerosis (HS) and atrophy (HA): Cause or effect of epilepsy?

Three papers correlate MRI study of the relationship of HS and HA to epilepsy. Fuerst et al. present evidence based on studies of 46 adults with temporal lobe epilepsy that HS is an acquired lesion caused by recurrent seizures. Similarly, Schulz and Ebner report that one identical twin who had prolonged febrile seizures at 8 months subsequently had severe epilepsy and when evaluated at age 30 had evident temporal lobe atrophy. Briellmann et al. document progressive HA over a period of <3 years in a patient with brief but recurrent seizures.

see pages 184, 315, and 318

The accompanying editorial by Sutula and Pitkänen notes the growing evidence that seizures cause HA/HS and may beget further seizures—thus, HA/HS are both the result of seizures and a cause of intractable seizures. They note that treatment is all the more urgent, and that understanding the genetic factors underlying epilepsy may lead to new targets for treatment.

see page 169

Are the brains of people who stutter different?

Zebra finch

Foundas et al. reported that the planum temporale, a region important for auditory processing, was more symmetric and larger in adults with persistent developmental stuttering. Atypical gyral configurations were also seen in these individuals. Thus, atypical anatomy may put an individual at risk for the development of stuttering.

see page 207

The accompanying editorial by Rosenfield reviews the history of stuttering and the distinction between developmental and acquired stuttering (and related conditions). He laments that stuttering provokes amusement, as illustrated by Biran and Steiner's note on "Doc" in "Snow White" (see page 363). His zebra finch (illustrated) model holds promise for explaining the genetics and pathophysiology of stuttering.

see page 171

Separating AD and semantic dementia by neuropsychological and MRI testing

Cross-sectional area of the brain at the level of the splenium

Galton et al. compared volumes of temporal lobe subregions, including the hippocampus, in 26 patients with AD and 18 matched patients with semantic dementia (SD). Hippocampal atrophy was found in both groups but SD had a distinct pattern of nonhippocampal involvement, and the semantic deficit correlated with left fusiform involvement.

see page 216

The accompanying editorial by Morris and Balota considers the relationship of SD to other variants of frontotemporal dementia and notes how MRI, neuropsychological tests, and (still essential) histopathology are defining the dementias. They note that the Galton et al. study points to frontal lobe dysfunction as a likely contributor to memory loss in AD.

see page 173

Diagnosis of neurocysticercosis

The diagnosis of neurocysticercosis is a challenge owing to widely varying clinical and imaging findings. Del Brutto et al. present stringent diagnostic criteria for neurocysticercosis that should permit objective evaluation of clinical, imaging, immunologic, and epidemiologic data of patients with suspected disease.

see page 177

continued on page 168

Early seizure and status epilepticus after stroke

In a population-based stroke incidence cohort, Labovitz et al. note that lesion location and stroke subtype are strong risk factors for seizure and status epilepticus within 1 week of stroke. However, early seizure does not predict 30-day mortality after adjusting for stroke severity.

see page 200

Corpus callosum (CC) abnormalities in prenatal alcohol exposure

Callosal outline differences between prenatal alcohol exposed subjects and controls

Sowell et al. show that the CC is both displaced and reduced in size within the brain of subjects with prenatal alcohol exposure. Abnormalities are more prominent in posterior brain regions, and the amount of CC displacement is related to the degree of cognitive impairment in these subjects. Notably, CC displacement is also found in subjects with less severe alcohol-related findings.

see page 235

Aberrant cerebral and cerebellar growth in early life in autism

Courchesne et al. studied boys 2 to 16 years of age using MRI: 60 boys with autism (30 under 5 years) and 52 normal controls. Overgrowth of cerebral gray and cerebral and cerebellar white matter was present early in life in autism (by 2 to 3 years of age), and this was followed by abnormally slowed growth through adolescence. Results suggest abnormal regulation of brain growth in autism.

see page 245

Does methamphetamine cause neurotoxicity in brain development?

Methamphetamine, a popular drug of abuse, has been suspected to affect brain development. Smith et al. used ^1H MRS to evaluate brain chemistry of children exposed to methamphetamine in utero, and found increased creatine compounds in the striatum, but normal brain structure and normal peaks for the neuronal marker *N*-acetylaspartate. These findings suggest an abnormality in energy metabolism in the brains of these children.

see page 255

Carbamazepine treatment in pregnancy

Diav-Citrin et al. prospectively followed up 210 first trimester carbamazepine-exposed pregnancies and found a twofold increase in the rate of major congenital anomalies and reduction of birth weight when compared to a control group. Cardiovascular anomalies were notable.

see page 321

Normal phenotype in a female carrier of a *DAX* mutation

Demelas et al. describe a novel missense mutation in the *DAX* gene in a lissencephaly family where a female, nonmosaic carrier of the mutation with random X-inactivation was normal by examination and cranial MRI. This report is important because the carrier of a *DAX* mutation was asymptomatic.

see page 327

Acute, chronic, and recurrent varicella zoster virus (VZV) neuropathy without zoster rash

Fox et al. describe VZV-associated acute, chronic, and recurrent neuropathy without zoster rash. All three patients received antiviral therapy and improved. Antibody to VZV in CSF appears to confirm nervous system infection even without evidence of VZV DNA. This paper was controversial during the review process. The presumption that these three cases have zoster is based on IgG antibody to VZV in CSF and on the uncontrolled response to treatment.

see page 351

Neurology[®]

July 24 Highlights

Neurology 2001;57;167-168

DOI 10.1212/WNL.57.2.167

This information is current as of July 24, 2001

Updated Information & Services

including high resolution figures, can be found at:
<http://n.neurology.org/content/57/2/167.full>

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
http://www.neurology.org/about/about_the_journal#permissions

Reprints

Information about ordering reprints can be found online:
<http://n.neurology.org/subscribers/advertise>

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright . All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.