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Material obtained at open surgery for temporal epilepsy in 110 patients was studied morphologically. Spread of neuronal damage was observed in the most cases. Acute swelling was apparent in 59.2%, in connection with karyolysis in 21.4%, tigrolysis, and even development of “shadow-cells.” Shrunken neurons constituted 55.0%. The changes were of focal character: either neuronal groups or separate neural cells were damaged.

Electron microscopic studies of the surgical material confirmed a mosaic cortical distribution of considerably damaged neurons. Shrunken electron-dense cells with deformed nuclei and destroyed or partially changed organelles were scattered in neocortex or concentrated in separate microfoci. Macrophages from the vascular bed, astrocytes, and oligodendroglialcytes often were near them. The surviving neurons contained lysosomes and lipofuscin granules.

The normal structure of multiple interneuronal synaptic contacts was disturbed. Axonal terminal contained either an excessive quantity of synaptic follicles or a small group of agglutinated follicles, indicating disturbed synaptic relations between neurons. The number of axospinous contacts was considerably reduced.

The structure of neurogial cells (oligodendrocytes and astrocytes) was characterized by certain reactive and destructive signs. Multiple structural changes in myelin sheath was noted in the white substance immediately beneath the cortex of epileptic focal area, with separate axons partially or totally demyelinated.

Gliosis and Expression of Small Heat-Shock Proteins in Brain Patients with Childhood Epilepsy. Krystyna Renkawek and Willy O. Renier (Institute of Neurology, University of Nijmegen, Nijmegen, The Netherlands).

Our object was to study the contribution of astrocytic gliosis to epilepsy. The significance of gliosis is unclear and may be related both to degradation and protection of neuronal tissue. Two small heat-shock proteins (hsp) hsp27 and α-crystallin are expressed at increased levels in proliferating astrocytes in neurodegenerative diseases. Their function in the brain is not clear. The small hsp confers in vitro protection against protein aggregation and to a lesser degree, hsp27. The number of immunoreactive cells was much higher in the white matter than in the gray matter. The immunoreaction showed remarkable variations between particular cases but was lower in contrast to the strong and uniform immunoreactivity seen for the presence of glial fibrillary acidic protein, a commonly used marker for fibrillar astrocytes. Our preliminary results showed that the restricted presence of small hsp in glial cells, but not in neurons, makes these proteins excellent targets for the study of glial cell contribution, in response to stress, in human epilepsy.

Abnormal GABAAergic System in Hemimegalencephaly: Immunohistochemical Study. Hiroshi Arai, Toshisaburo Nagai, Hiroshi Kimura, and Shinarto Okada (Department of Pediatrics, Osaka University Medical School, Osaka, and; *Institute of Molecular Neurobiology, Shiga University of Medical Science, Shiga, Japan).

Hemimegalencephaly is often associated with early-onset intractable epilepsy. Our clinical research with single photon emission computed tomography and EEG, showed the affected hemisphere displayed excessive theta activity, which was considered epileptiform. To elucidate the basic mechanisms of such epileptic activity, we performed immunohistochemical study of the surgically resected specimens.

Tissues were obtained from multilobar cortical resection performed for the treatment of medically intractable seizures of a 4-year-old boy. Their histological characteristics were compatible with those of previous reports. They were fixed immediately after excision at 4°C in mixture of 4% paraformaldehyde and 0.3% glutaraldehyde in 0.1 M phosphate-buffered solution. Cryostat and vibratome sections were processed for immunohistochemistry.

Immunostaining for GABA showed much increased positive staining in affected cortex, whereas that for GABAA receptor (a subunit) showed decreased immunoreactivity as compared with control tissues. No decrease in either glutamate or GluR1-positive structures was observed in the serial sections. Other inhibitory neurotransmitters such as taurine and nitric oxide synthase showed immunoreactivity similar to that of control tissues. Results indicated that GABAAergic transmission in the hemimegalencephalic brain is impaired mostly by decreased expression of GABAA receptor, which could be one of the bases of epileptic activity resistant to various antiepileptic drugs.

Three-Dimensional Features of Dendritic Geometry in Epileptogenic Cortex from Therapy-Resistant Partial Epilepsy Patients: A Three-Dimensional Confocal Laser Scanning Microscopy Study. *P. V. Belichenko, +T. C. Nordborg, $B. Rydenhag, K. Malmgren, FA. Hedström, *P. Uvebrant, and F.A. Dahlström (*Brain Research Institute, Russian Academy of Medical Science, Moscow, Russia; and +Neurobiology Section, $Neurosurgery, KNeurology, and Clinical Neurophysiology; and *Pediatrics, The Medical Faculty, Göteborg University, Göteborg, Sweden).

We recently introduced a strategy for studying the three-dimensional dendritic geometry at the microscopic level (Hum Brain Mapping 1994;1:185-93). Using this approach, we investigated biopsy material from the epileptogenic zone of 15 patients with therapy-resistant partial epilepsy (TRPE), undergoing epilepsy surgery. The results of the TRPE patients were compared with autopsy brain material from 3 patients with epileptic Rett’s syndrome and with 6 normal control cases. Different types of dendritic abnormalities were observed in these diseases (Epilepsy Res 1994;18:233-47; Neuro Report 1994;5:1509-13). In the TRPE cases, the results showed different types of dendritic abnormalities on single pyramid cells in the epileptogenic cortex in layers I, II, III, and V and in the subcortical white matter. Many cells had two or three dendrites originating from the apical part, rather than, as in normal cases, a single apical dendrite. In Rett’s syndrome cases, the results demonstrated somewhat different types of dendritic abnormalities as compared with TRPE and control cases. The normally occurring specialization in pyramidal architecture in different cortical areas was absent. Microdysgenesis was not observed, in contrast to TRPE cases, in which microdysgenesis was frequent. The results will be discussed in relation to possible etiology, to different types of epileptic activity, and to different types of possible pharmacological approach and in relation to clinical and neurophysiological data. Supported by Grants No. 14X-2207 and 12V-10808 from the Swedish MRC, from The Royal Swedish Academy of Science in Stockholm, the Royal Society for Arts and Science in Göteborg, and Grant No. 33000 from the International Science Foundation.)


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