Congenital porencephaly and hippocampal sclerosis
Clinical features and epileptic spectrum

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Article abstract—We studied clinical features and seizure localization in 14 patients with porencephaly and intractable seizures. Perinatal complications were present in nine patients, childhood febrile convulsions in two, congenital hemiparesis in 12, and intellectual impairment in seven. Ten patients had psychomotoric simple partial seizures, and one had generalized tonic-clonic seizures. Surface EEG showed temporal lobe onset in nine patients (one bitemporal) and extratemporal onset in four. MRI showed porencephaly in the distribution of the middle cerebral artery in eight patients, posterior cerebral artery in three, internal carotid in one, and multiple vessels in two. MR-based volumetry revealed hippocampal formation atrophy in 13 patients (eight unilateral and five bilateral) and amygdalar atrophy in 10 patients (nine unilateral and one bilateral). Hippocampal formation atrophy was concordant with CPS semiology in 10 patients (71%) and with EEG temporal localization in nine patients. Two patients had pathologic confirmation of mesial temporal sclerosis and were seizure free after temporal lobectomy. We conclude that mesial temporal sclerosis often coexists with porencephaly and is the likely seizure focus in the presence of concordant electroclinical data. This recognition implies that effective surgical intervention can be offered to certain patients with porencephaly-related seizure disorders. The dual pathology and association with perinatal cerebral vascular occlusion suggest a common ischemic pathogenesis.

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Considerable heterogeneity exists in the literature concerning the clinical and radiologic findings of porencephaly.1-7 Porencephaly occurs as a component of vascular cerebral infarction during the prenatal or perinatal period and commonly manifests as congenital hemiplegia, intellectual impairment, and epilepsy. Despite the well-recognized association of porencephaly with seizures, the literature on seizure semiology in this condition is scarce, and even less well studied is the origin of the epileptogenic zone. It is generally assumed that the seizures originate in proximity to the porencephalic lesion, and surgical intervention is often discouraged because of difficulty in accurate localization of the seizure foci. Two reports in the literature suggest the possibility of seizure origin in the temporal lobe distant from the porencephalic lesion.8,9 Remillard et al.9 reported temporal lobe epilepsy in a small series of patients with porencephaly and perinatal occlusion of the posterior cerebral artery. These patients had electroclinical evidence of temporal lobe seizures but pathologic findings were not provided by the authors. Cendes et al.9 described the occasional coexistence of mesial temporal sclerosis with extrahippocampal pathology, including porencephaly. However, the clinical characteristics of seizures in the presence...
of porencephaly and hippocampal sclerosis have not been systematically addressed. Resolving this question would help determine whether certain porencephaly-related seizure disorders are amenable to temporal lobe surgery.

MR-based volumetry is the most reliable method for the presurgical detection of mesial temporal sclerosis.10-12 We therefore used this technique to determine the presence of hippocampal formation (HF) atrophy in patients with porencephaly and intractable seizures. Since the amygdala (AM) may also be involved in temporal lobe epilepsy,10-13,16 we included this structure in our volumetric analysis. We correlated MRI findings with electroclinical data to assess further the site of seizure localization when mesial temporal sclerosis coexists with porencephaly.

**Methods.** Patients were recruited from the University of Alabama at Birmingham (UAB) Epilepsy Center. In all patients, porencephaly was detected during routine MRI investigation of intractable seizures. Patients with acquired cystic lesions of known cause (e.g., following trauma, tumor resection, or infection) and cystic developmental malformations (e.g., schizencephaly) were excluded from the study. The medical records of all patients were reviewed with specific attention to perinatal history, developmental milestones, previous head trauma, encephalitis, childhood febrile convulsions, status epilepticus, family history of epilepsy, seizure description, neuropsychological assessment, and physical examination. The electroclinical data of all patients were reviewed retrospectively. All patients had routine EEG studies using the 10-20 system with additional anterior temporal electrodes, and the majority had long-term video EEG monitoring with scalp electrodes. One patient underwent intracranial EEG monitoring with subdural electrodes. The seizures were classified according to the International League Against Epilepsy.17,18

**MR imaging.** All subjects were scanned on a 1.5-T Gyroscan (Philips Medical System, Best, The Netherlands) using a standardized protocol. Sagittal T1- and coronal T2-weighted spin-echo, fluid attenuation inversion recovery (FLAIR), and inversion recovery (IR) sequences were acquired. For volumetric studies, a three-dimensional sequence (TR, 20 ms; TE, 6.2 ms; number of signal average, 1; field of view, 23 cm; flip angle, 28 deg; matrix size, 218 × 256) was acquired in the coronal plane perpendicular to the long axis of the hippocampus and formatted to 1.5-mm contiguous coronal slices.

**MRI visual analysis.** Inversion recovery and FLAIR sequences were evaluated qualitatively for the presence of hippocampal signal alterations indicative of mesial temporal sclerosis (signal hypointensity on T1-weighted IR images and signal hyperintensity on T2-weighted images).19 The location of the porencephalic lesion was determined according to arterial supply territory as identified by Pat-<ref>ton.20

**MRI volume analysis.** The images were transferred to a Silicon Graphics Indigo workstation (Mountain View, CA) for volumetric measurement using a software program developed in the Computer Assisted Neurosurgery laboratory of the UAB. The regions of interest were outlined by the same rater (SSH) with the manual contouring func-

tion, and slice volume was calculated automatically by the computer program. Anatomic guidelines for outlining the AM and HF were according to the protocol described by Watson et al.21 Using this protocol, the rater (SSH) has an intraobserver test-retest the coefficient of variation under 2%. The interrater variability in our laboratory is 5.1%.

Volumes of the AM and HF were obtained on each patient and compared with values from a normal control group of 10 healthy volunteers (seven men and three women; age range, 25 to 38 years). The HF and AM volumes were normalized to minimize variability in volumes due to different sizes of the patients. We used the method of volume normalization described by Jack et al.22 as follows:

\[ NV = OV - Grad \times (TCVi - TCVmean) \]

where NV is the normalized HF or AM volume, Grad is the regression gradient or slope of the regression line of HF or AM volume regressed on total intracranial volume for the control group, TCVi is the cranial volume for the individual subject, and TCVmean is the mean intracranial volume for a group of control subjects.

The cranial volume was estimated from area measurements on 14 sagittal slices of 5-mm thickness at equal spacings throughout the cerebrum, using the same volume analysis program as that for the HF and AM volume measures. Abnormal volumes were defined as 2 SDs below the mean for the control subjects for normalized data. The degree of asymmetry between sides was determined by subtracting left-sided (L) from right-sided (R) volume for each structure using corrected values (e.g., R – L AM, R – L HF). The R – L volumes were also obtained from the normal control subjects for comparison.

**Surgery and pathology.** Temporal lobe resections were performed according to a standardized protocol at our institution. The resection included the uncus, AM, and anterior 2 cm of the hippocampus with minimal neocortical resection. Tissue was processed according to validated techniques. The diagnosis of mesial temporal sclerosis was based on qualitative severe neuronal cell loss and astroglia-<ref>sis in the hippocampus.21

**Statistical analysis.** We used Fisher's exact test to compare categorical data, and an unpaired t-test to compare the means of two groups. The significance level was set at \( p < 0.05 \).

**Results.** Fourteen patients, eight men and six women with mean age at time of evaluation of 37.6 years (range, 15 to 54 years), were studied (table 1). A history of perinatal ischemic insult was present in nine patients, febrile convulsions in two, and childhood status epilepticus in one. Seven patients had intellectual impairment and 12 had congenital hemiparesis associated with hemiatrophy.

All patients had intractable epilepsy at the time of investigation. The mean age of seizure onset was 10.7 years (range, 1 to 48 years), mean duration of epilepsy at the time of evaluation was 26.9 years (range, 4 to 51 years), and mean frequency of seizures was 20.3 per month. Ten patients had complex partial seizures (CPS) of psychopa-<ref>retic type (comprising behavioral arrest and unresponsive-ness), three had simple partial seizures (SPS) of sensorimotor type, and one had generalized tonic-clonic seizures that ceased with medication after many years of poor control.

Surface EEG showed an abnormal background with fo-
### Table 1 Clinical features and ictal and interictal EEG findings

| Patient no. | Age (y)/ Sex | Age at seizure onset (y) | IQ       | Focal signs            | Seizures   | Interictal surface EEG focus | Ictal surface EEG focus |
|------------|--------------|--------------------------|----------|------------------------|------------|-----------------------------|-------------------------|
| 1          | 34/M         | 10                       | Low average | R. hemiatrophy        | CPS*       | L. temporal                  | Nonlocalizing           |
| 2          | 30/F         | 10                       | Average   | R. hemiatrophy        | CPS*       | L. temporal                  | Nonlocalizing           |
| 3          | 15/M         | 9                        | Average   | L. hemiparesis        | CPS*       | R. temporal                  | Nonlocalizing           |
| 4          | 39/M         | 1                        | Average   | L. hemiatrophy        | CPS*       | L. centroparietal            | Not performed            |
| 5          | 42/M         | 2                        | Low average | L. hemiatrophy       | GTCS       | L. centroparietal            | Not performed            |
| 6          | 52/F         | 48                       | Average   | None                  | SPS†       | R. occipital                 | Not performed            |
| 7          | 32/M         | 22                       | Average   | None                  | CPS*       | R. occipital                 | R. temporal              |
| 8          | 52/M         | 4                        | Retarded  | L. hemiatrophy        | CPS*       | R. frontal                  | Not performed            |
| 9          | 31/M         | 5                        | Low average | L. hemiatrophy       | CPS*       | R. temporal                  | Nonlocalizing           |
| 10         | 49/F         | 10                       | Average   | L. arm weakness       | CPS*       | R. temporal                  | Nonlocalizing           |
| 11         | 39/F         | 15                       | Retarded  | R. arm weakness       | SPS†       | L. temporal                  | Nonlocalizing           |
| 12         | 40/F         | 2                        | Average   | R. hemiatrophy        | SPS†       | L. frontal                  | Nonlocalizing           |
| 13         | 24/M         | 9                        | Retarded  | L. hemiatrophy        | CPS*       | R. occipital                 | R. occipital             |
| 14         | 54/M         | 3                        | Retarded  | L. hemiparesis        | CPS*       | Bitemporal                  | Not performed            |

* Complex partial seizure (CPS) comprised of an early period of staring, relative immobility, and minimal automatisms.
† Simple partial seizure (SPS) with sensorimotor manifestation.

M = male; F = female; R. = right; L. = left; GTCS = generalized tonic-clonic seizure.

### Table 2 MRI findings and visual analysis of hippocampus

| Patient no. | Side   | Location                                 | Arterial territory | Hippocampus                     |
|------------|--------|------------------------------------------|--------------------|---------------------------------|
| 1          | Left   | Frontoparietotemporal and basal ganglia  | MCA (distal)       | L. HF atrophy                   |
| 2          | Left   | Temperoparietio-occipital                | PCA                | L. HF atrophy                   |
| 3          | Right  | Parieto-occipital, inferotemporal, and thalamus | PCA               | R. HF atrophy and sclerosis     |
| 4          | Right  | Frontoparietal, superior temporal         | MCA (distal)       | R. HF atrophy and sclerosis     |
| 5          | Right  | Parieto-occipital                         | PCA                | Normal                          |
| 6          | Bilateral | Parieto-occipital, posteroinferotemporal | Distal multivessel | R. HF atrophy and sclerosis     |
| 7          | Right  | Basal ganglia                             | MCA (deep branch)  | L. HF atrophy                   |
| 8          | Right  | Anterofrontal, parietotemporal            | ICA                | R. HF atrophy                   |
| 9          | Right  | Frontoparietal, superior temporal         | MCA (distal)       | R. HF atrophy                   |
| 10         | Right  | Parietotemporal                           | MCA (distal)       | R. HF atrophy and sclerosis     |
| 11         | Left   | Basal ganglia, centrum semiovale          | MCA (deep branch)  | L. HF atrophy and sclerosis     |
| 12         | Left   | Parietotemporal, basal ganglia            | MCA (deep branch)  | L. HF atrophy and sclerosis     |
| 13         | Right  | Frontoparietotemporal                     | MCA (proximal)     | Normal                          |
| 14         | Bilateral | Frontoparieto, superior temporal         | Distal multivessel | Normal                          |

MCA = middle cerebral artery; PCA = posterior cerebral artery; ICA = internal carotid artery; L. = left; R. = right; HF = hippocampal formation.
Figure 1. Volumetric TI-weighted MRI in the coronal angulated plane shows porencephaly in the right posterior cerebral artery territory and coexisting right hippocampal atrophy in a patient with intractable seizures of right temporal lobe origin (Patient 3).

patients was the bilateral hippocampal abnormality visually identified.

Quantitative MRI-based volumetric analysis. For the 10 control subjects, the mean (2 SD) normalized HF and AM volumes, using the same analysis procedure and the same rater are left HF, 3.63 cm$^3$ (0.42); right HF, 3.96 cm$^3$ (0.54); left AM, 2.32 cm$^3$ (0.40); right AM, 2.44 cm$^3$ (0.40). The normalized HF and AM volumes for the patient group are summarized in figure 3. Based on the criterion that values less than 2 SD below the mean for the control group are abnormal, 13 patients had HF atrophy (eight unilateral and five bilateral) and 10 had AM atrophy (nine unilateral and one bilateral). Only one of the 14 patients had normal HF and AM volumes. In all patients, unilateral HF or AM atrophy was ipsilateral to the porencephalic lesion.

Data analysis. Unilateral or bilateral HF atrophy corresponded with the clinical manifestation of psychoparetic CPS in 10 of 13 patients (77%), while the other three patients had sensorimotor SPS as the only seizure manifestation. The one patient with normal HF volumes had nocturnal generalized seizures that were subsequently controlled with antiepileptic medication. This patient did not have evidence of temporal lobe seizure onset. In the eight patients with unilateral HF atrophy, six (75%) had surface EEG concordant with ipsilateral temporal localization and two had extratemporal epileptiform abnormalities. In the five patients with bilateral HF atrophy, one had bilateral independent temporal foci (Patient 14), two had ictal temporal discharges lateralized to the side with more severe HF volume loss, and two had extratemporal epileptiform abnormalities.

Unilateral HF atrophy was ipsilateral to the side of the porencephaly in all patients. Bilateral porencephalic lesions correlated with bilateral HF atrophy in two patients. In the other three patients, the more severe HF atrophy was ipsilateral to the porencephalic lesion. There was a trend for patients with bilateral HF atrophy to have more severe volume loss than patients with unilateral HF atrophy (mean HF volume, 2.2 cm$^3$ on more severe side for the bilateral group compared with the mean HF volume, 2.79 cm$^3$, in the unilateral group; $p = 0.053$; unpaired t-test).

For the whole patient group there was no significant correlation between age at seizure onset and the presence of HF atrophy. However, when the unilateral and bilateral groups were compared, the mean age at seizure onset was significantly higher for the bilateral group (18.2 years versus 7.1 years, $p = 0.0020$; unpaired t-test). There was no

Figure 2. Patient 4. (A) Volumetric T1-weighted MRI in the coronal plane shows porencephaly in the distribution of the right middle cerebral artery and right hippocampal atrophy in a patient with intractable partial seizures of right temporal lobe origin. (B) Inversion recovery MRI shows signal intensity change in the right hippocampus in the same patient.
correlation between presence of HF atrophy and the duration of epilepsy at the time of evaluation, nor was there a correlation between HF atrophy and frequency of seizures. Fisher's exact test showed no statistical difference between the unilateral and the bilateral groups for duration of epilepsy and frequency of seizures.

Surgical results. Two patients (Patients 1 and 2) underwent temporal lobectomy, both with pathologic confirmation of mesial temporal sclerosis. At the 18-month follow-up, both have remained seizure free apart from occasional auras in one patient (Patient 1). Two other patients are currently awaiting temporal lobectomy.

Discussion. Seizure disorders associated with porencephaly have not been well studied and previous reports have tended to include cystic lesions of diverse etiologies. The electroclinical profile of seizures in porencephaly have occasionally been mentioned in the literature. Naef reported focal motor, generalized, adversive, and psychomotor seizures in 13 of 32 patients, including patients with developmental malformation. Half of the patients had EEG indicative of focal activity, and of these some corresponded to the location of the cyst but others were localized over the contralateral hemisphere. Gou- tieres et al. also reported a variety of seizure types in porencephaly including psychomotor, partial motor, and generalized seizures. However, this study was based on patients with congenital hemiplegia including some patients with no radiologic abnormalities, and therefore the findings may not be representative of a homogeneous population with congenital porencephaly. In contrast to previous studies, psychomotor CPS was the most common seizure manifestation in our series, occurring in 10 of the 14 patients (71%). This seizure type is recognized by the International Classification of Epilepsies and Epileptic Syndromes to be suggestive of temporal lobe origin, but the majority of patients in our study had porencephalic lesions distant from the temporal region. In addition, we found ictal or interictal EEG localization to the temporal region in nine of 14 patients (64%), and these patients accorded with CPS manifestation. Only three patients in our study had sensorimotor SPS, and in two of these patients there was an associated extratemporal epileptic focus on EEG.

Our study demonstrates a very high frequency of HF and AM atrophy in a population with porencephaly and intractable seizures. Dual pathology was previously reported by Cendes et al. in a heterogeneous group of patients with lesional epilepsy, including porencephaly. In contrast to the study of Cendes et al., we found a much higher frequency of coexisting HF atrophy (93% versus 31%). This discrepancy may be partially explained by our selection bias toward porencephaly patients with intractable seizures, and thus the true incidence of dual pathology cannot be determined from this study. Nevertheless, our results suggest that patients with porencephaly and intractable seizures represent a different population, and this has practical therapeutic implications. Our study showed that, overall, the most common origin of epileptic discharges was the temporal lobe, and there was concordance between HF atrophy and electroclinical localization to the ipsilateral temporal region. In three cases of HF atrophy, the patients had sensorimotor SPS and extratemporal EEG localization, suggesting that the HF atrophy was asymptomatic in these patients. The origin of seizures when two potentially epileptogenic lesions coexist has not been previously determined in patients with HF atrophy and porencephaly. We have shown that HF atrophy is the more likely epileptogenic lesion in these patients, and therefore effective surgical intervention should be facilitated for intractable porencephaly-related CPS.

Concurrent AM atrophy and bilateral HF atrophy were common in our study (57% and 36% of total patients respectively). The bilateral HF volume loss was detected only by volumetric analysis with normalization of data, as bilateral hippocampal signal

![Figure 3. (A) MRI-based volumetry of the hippocampal formations in the 14 patients. Values for control mean less 2 SD are indicated by the solid lines. Solid bars indicate left hippocampus; shaded bars indicate right hippocampus. Diamonds indicate left hippocampus (c. mean - 2 SD); circles indicate right hippocampus (c. mean - 2 SD). (B) MRI-based volumetry of the amygdala in the same patients. Solid bars indicate left amygdala; shaded bars indicate right amygdala. Diamonds indicate left amygdala (c. mean - 2 SD); circles indicate right amygdala (c. mean - 2 SD).](image-url)
abnormalities were not visually identified in any of the patients. None of the patients with bilateral volume loss had surgery, so it is difficult to draw conclusions about benefits of temporal lobectomy in this group. However, two of the five patients had well-lateralized ictal temporal localization, and a recent study suggests that good surgical outcome is possible in certain bitemporal cases.26

The explanation for the relatively high prevalence of bilateral HF atrophy is unclear. The mean age at seizure onset was significantly higher for the bilateral group compared with the unilateral group (18.2 years versus 7.1 years). However, there was no difference between the unilateral and the bilateral groups for duration of epilepsy and frequency of seizures, suggesting that the bilateral damage is unlikely to be a result of ongoing seizure activity. There was no previous history of status epilepticus or head injury in the bilateral group. The one patient with childhood status epilepticus had very severe unilateral HF volume atrophy (Patient 8).

Finally, the high frequency of coexistence of AM-HF atrophy with porencephaly in our series is striking and unique, raising questions about the pathogenesis. There is still much controversy regarding the pathogenesis of mesial temporal sclerosis. Hippocampal neurons are vulnerable to a variety of stresses in animal experiments, and various early CNS insults including febrile convulsions, birth trauma, and status epilepticus have been associated with the pathologic entity of mesial temporal sclerosis.23,27-29 However, neither childhood febrile convulsions nor status epilepticus were common in this study population. In contrast, the majority of the patients had a history of pre- or perinatal injury in association with the porencephaly. The occurrence of dual pathology has been proposed to indicate kindling and secondary epileptogenesis in the hippocampus.30,31 Our study suggests that this is a less likely mechanism since there was no correlation between the presence of mesial temporal sclerosis and the frequency of seizures, nor was there a correlation with the duration of epilepsy.

Our study suggests that a more likely mechanism for the dual pathology is a common pathogenic mechanism in early life. Porencephaly is known to be related to pre- and perinatal cerebral vascular occlusion.7,22,32 Furthermore, perinatal occlusion of the posterior cerebral artery has been previously reported in a small series of patients with temporal lobe epilepsy and porencephaly.8 In our study only three patients had porencephaly in the posterior cerebral artery territory compared with nine patients who had it in the middle cerebral artery territory. The posterior cerebral artery supplies the occipital and part of the parietal lobe, and the inferomesial and lateral areas of the temporal lobe, as well as Ammon’s horn and part of the parahippocampal gyrus.34 The hippocampus and temporal lobe lie in a watershed area supplied mainly by the posterior cerebral and anterior choroidal arteries.34,35 Either an arterial thrombosis or a drop in BP in the boundary zones between the two areas of arterial supply may result in tissue necrosis and cavitation, causing a porencephalic cyst. The same ischemic mechanism can contribute to mesial temporal sclerosis. Coccchi and Glooo36 emphasized that any drop in BP can render the hippocampus susceptible to ischemic damage, since the hippocampus lies in a watershed area and the blood flow would cease earliest at the branches furthest from the blood supply. Although this hypothesis is attractive, we could not assess whether watershed ischemia was present, due to the presence of extensive ipsilateral porencephaly in all patients.

Diaschisis or deafferentation associated with cerebral infarction can also explain the ipsilateral as well as bilateral AM-HF damage. The most likely mechanism underlying diaschisis is the interruption of hemispheric connections by the infarct, causing deafferentation and transneural metabolic depression or impaired resting blood flow in the remote ipsilateral or contralateral cerebral hemisphere.36-38 In humans, cerebral blood flow has been reported to be decreased contralateral to the infarction, 1 to 2 weeks after onset of an ischemic lesion.39 Thus, diaschisis following pre- or perinatal cerebral vascular occlusion may lead to remote damage in the contralateral AM-HF.

The results of this study have implications for clinical management. Seizure foci secondary to porencephaly may be difficult to localize electroclinically and should not be assumed to arise from around the cyst. Patients with congenital porencephaly and uncontrolled seizures should have optimal MRI protocols performed, including temporal lobe sequences such as IR and FLAIR to evaluate the hippocampal structures. With the recognition of coexisting HF atrophy, temporal lobectomy can now be offered to suitable patients with porencephaly-related seizure disorders, if MRI evidence of mesial temporal sclerosis is present and if the clinical and EEG features indicate temporal lobe seizure onset.

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