J Neurosurg 110:1170–1178, 2009

Surgical treatment of parietal lobe epilepsy

Clinical article

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Object. Parietal lobe epilepsy (PLE) accounts for a small percentage of extratemporal epilepsies, and only a few and mostly smaller series have been reported. Preoperative findings, surgical strategies, pathological bases, and postoperative outcomes for PLE remain to be elucidated.

Methods. Patients with PLE were identified by screening a prospective epilepsy surgery database established in 1989 at the University of Bonn. Charts, preoperative imaging studies, surgical reports, and neuropathological findings were reviewed. Seizure outcome was classified according to Engel class (I–IV).

Results. Forty patients (23 females and 17 males) with PLE were identified and had a mean age of 25.0 years and a mean preoperative epilepsy duration of 13.7 years. Nine patients had a significant medical history (for example, trauma, meningitis/encephalitis, or perinatal hypoxia). Preoperative MR imaging abnormalities were identified in 38 (95%) of 40 patients; 26 patients (65%) underwent invasive electroencephalography evaluation. After lesionectomy of the dominant (in 20 patients) or nondominant (in 20 patients) parietal lobe and additional multiple subpial transections (in 11 patients), 2 patients suffered from surgical and 12 from neurological complications, including temporary partial Gerstmann syndrome. There were no deaths. Histopathological analysis revealed 16 low-grade tumors, 11 cortical dysplasias, 9 gliotic scars, 2 cavernous vascular malformations, and 1 granulomatous inflammation. In 1 case, no histopathological diagnosis could be made. After a mean follow-up of 45 months, 27 patients (67.5%) became seizure free or had rare seizures (57.5% Engel Class I; 10% Engel Class II; 27.5% Engel Class III; and 5% Engel Class IV).

Conclusions. Parietal lobe epilepsy is an infrequent cause of extratemporal epilepsy. Satisfactory results (Engel Classes I and II) were obtained in 67.5% of patients in our series. A temporary partial hemisensory or Gerstmann syndrome occurs in a significant number of patients. *(DOI: 10.3171/2008.2.17665)*

Key Words • dysplasia • epilepsy • ganglioglioma • Gerstmann syndrome • gliosis • parietal tumor • visual field

Parietal lobe epilepsy accounts for a small percentage of extratemporal epilepsies. At the Second International Palm Desert Conference on the Surgical Treatment of the Epilepsies (1992), extratemporal epilepsy surgery accounted for 18% of all epilepsy surgeries performed at 91 centers.[5] In epilepsy surgery studies performed in Bonn, 17% were reported to have been extratemporal operations, compared with 58% temporal, 9% disconnective procedures, and 13% vagus nerve stimulations. Few large surgical series of patients with PLE have been reported. For example, of 60 patients undergoing extratemporal epilepsy surgery reported by Zentner et al.[42] in 1996, only 7 (12%) underwent parietal resection. Of 717 focal resections for epilepsy performed in Warsaw between 1957 and 1996, only 25 (3.6%) were parietal.[11] The single largest reported series of patients with PLE encompassed 82 nontumor and 34 parietal tumor resections performed between 1929 and 1988 at the Montreal Neurological Institute.[31,32] However, these patients were treated prior to the advent of MR imaging. In the modern MR imaging era, preoperative diagnostic findings, surgical strategies, pathological basis, and postoperative outcome for PLE remain to be further elucidated.

Compared with results of surgical treatment for temporal lobe epilepsy, success rates after epilepsy surgery in the parietal region have been less promising. Many authors have grouped parietal, occipital, and occipitotemporal epilepsies together as posterior cortex epilepsies,[2,3,8,9] whereas others have separately analyzed parietal lobe lesional epilepsies.[4,11,16,20,21,38] No matter the grouping, seizure freedom has been reported in ~ 40–60% of patients; however, the diversity of pathological findings, inclusion criteria, and outcome classification scales makes comparisons difficult.

Parietal lobe epilepsy poses a challenge for diagnosis

Abbreviations used in this paper: DNT = dysembryoplastic neuroepithelial tumor; ECoG = electrocorticography; EEG = electroencephalography; MST = multiple subpial transection; PLE = parietal lobe epilepsy.

and treatment. Although somatosensory auras are commonly found in patients with PLE, clinical diagnosis is still difficult because of nonspecific patterns in the early or late course of a seizure, as well as fast ictal spread to distant brain areas.[3,16,19,36] Scalp EEG recordings may also be misleading. For the definite diagnosis of PLE, invasive video-EEG monitoring with subdural and/or depth electrodes is often mandatory,[6,24,27] ideally coregistered to a distinct parietal lesion on MR imaging.

Even once PLE is diagnosed, treatment options are complicated by the eloquence of the parietal lobe. Postoperative outcomes in terms of visual fields, hemisensory syndromes, and Gerstmann syndrome have not been well described. In addition, little is known about the distribution and types of lesions causing PLE in the MR imaging era. In this report, we describe a large series of consecutive patients undergoing parietal resections for lesional PLE in the era of modern MR imaging and video-EEG monitoring. We analyze preoperative findings, surgical strategies, pathological bases, and postoperative outcomes in this infrequent type of extratemporal epilepsy.

Methods

Patient Population

We identified patients undergoing surgery for PLE between January 1990 and December 2004 from a prospective epilepsy surgery database established at the University of Bonn in 1989. Minimal requirements for inclusion in the study were as follows: clinical history of medically intractable epilepsy; parietal lobe involvement on preoperative MR imaging; exclusion of involvement of other lobes of the brain (frontal, temporal, or occipital); complete clinical and electrophysiological data sets; and follow-up seizure data for outcome. Forty patients with exclusively parietal lobe involvement were identified in the database. To determine the exact localization and characteristics of each lesion, MR imaging, operative reports, and pathology data were reviewed.

Preoperative Evaluation

All patients had suffered well-documented chronic and medically intractable epilepsy for > 1 year and had undergone adequate trials of at least 2 first-line antiepileptic drugs before they were referred for preoperative evaluation. All patients underwent continuous, noninvasive, and/or invasive scalp video-EEG monitoring to determine ictal and interictal focal activity. Invasive EEG monitoring via chronically implanted electrodes was performed in patients with the following: inconclusive or discordant results from noninvasive procedures, especially from interictal and ictal EEG; nonlesional high-resolution MR imaging or questionable lesions not clearly distinguishable from normal tissue; or localization of assumed epileptogenic lesions close to or overlapping eloquent areas, thus requiring electrical stimulation for cortical mapping. The details of preoperative workup for epilepsy surgery candidates at our institution have been previously described in detail.[24]

Demographic, Clinical, and Imaging Data

Demographic and clinical data used for this analysis included the following: age at epilepsy manifestation; age at operation; duration of epilepsy; seizure type(s); seizure frequency; presence/absence of other medical history; preoperative neurological status; preoperative imaging results (MR imaging, PET, and SPECT); electrophysiological data (see below); type of surgery; surgical and neurological (permanent or temporary) complications; histological results of the resected specimen; and follow-up and seizure outcome (Engel class).

All patients underwent standard preoperative MR imaging using a 1.5-T unit (Gyroscan model S15; Philips Medical Systems). Axial and sagittal T1-weighted images (TR 500–600 msec, TE 15–25 msec, slice thickness 4–8 mm) and axial and coronal T2-weighted images (TR 2–2.5 msec, TE 80–120 msec, slice thickness 4–8 mm) were obtained routinely. Spin echo sequences were also usually performed. If a tumor was suspected, additional axial and coronal T1-weighted images with and without Gd were acquired. Radiological data were classified based on the neuroradiologist's original MR imaging reading into the following categories: dysplasia, tumor, scar/cyst, vascular malformation, or other lesion. When PET (1 case) and SPECT (5 cases) studies were performed, results were noted.

Electrophysiological Monitoring

Preoperative invasive diagnostic monitoring was performed in 26 (65%) of the 40 patients and included various combinations of depth, strip, and grid electrodes. The type and location of grid and strip electrodes were noted. Intraoperative ECoG was performed in 9 patients (22%).

Operative Data

Operative details recorded and analyzed in the database included the following: date, side, location, and type of operation. Patients with MR imaging–visible lesions underwent lesionectomies usually with a small cortical rim, and 11 patients (27.5%) received additional MSTs in adjacent epileptogenic areas.

Histopathological Findings

Resected specimens were examined histopathologically using previously described methods.[39] Tumors were classified according to the revised WHO classification scheme.[23] Different subtypes of malformations of cortical development were grouped together as dysplasia. For purposes of overall evaluation and correlations, histological diagnoses were categorized into the following: dysplasia, ganglioglioma, low-grade astrocytoma, DNT, vascular malformation, scar/gliosis, and other.

Outcome Data

For neurological outcome, follow-up information was obtained from the last regular annual outpatient visit and/or telephone interviews. Care was taken to search for evidence of hemisensory deficits, hemiparesis, hemineglect, or components of Gerstmann syndrome (agraphia, acal-

culia, finger agnosia, and right-left disorientation).[12,13] For seizure outcome, patients were assigned to 1 of 4 outcome classes according to the Engel scheme[10] as follows: Class I, seizure free or only auras since surgery; Class II, rare seizures (no more than 2 per year or only nondisabling nocturnal seizures); Class III, > 75% reduction in seizure frequency; and Class IV, unchanged (< 75% reduction of seizure frequency). For further analysis, Class I and II outcomes were grouped as satisfactory seizure control, whereas Classes III and IV were grouped as unsatisfactory seizure control.

Statistical Analysis

We analyzed the following potential prognostic factors with respect to their prediction of good seizure outcome: demographic data, preoperative data (including seizure types, seizure frequency, MR imaging results, ictal EEG findings, number of ictal foci, and invasive vs noninvasive EEG), type of surgery, and histopathological findings. In addition, we studied potential interesting interactions among nonoutcome variables in this patient group (pathological findings vs seizure characteristics and pathology vs age of onset). Each factor was analyzed by chi-square or Fisher exact tests for Engel class (I–IV) and satisfactory (Engel Class I and II) versus unsatisfactory (Engel Class III and IV) seizure outcome. Continuous variables were tested using the Student t-test. For nonparametric testing, the Mann-Whitney U test was applied. For multifactorial analysis, a stepwise logistic regression model was applied. Backward stepwise logistic regression was performed with critical probability levels of 0.05 for inclusion and 0.1 for exclusion of factors from the model.

Results

Demographic and Clinical Findings

The overall population of 40 patients with PLE comprised 23 females and 17 males with a mean age of 25.0 years (range 6–48 years) (Table 1). These 40 patients comprised 14% of the total population of patients (292) undergoing extratemporal epilepsy surgery at the University of Bonn over this period. The mean preoperative epilepsy duration was 13.7 years (range 1–41 years). Nine patients had a positive medical history including meningitis/encephalitis (in 3 patients), perinatal hypoxia (in 2), febrile seizures (in 2), preterm birth (in 1), and brain trauma (in 1). Due to the parietal pathology, 7 patients suffered from an incomplete hemisensory syndrome and 2 patients from Gerstmann syndrome.

Simple partial seizures, complex partial seizures, and generalized seizures were frequently found in all patients. Simple partial seizures were found in 31 patients (77.5%) with a mean frequency of 93/month; complex partial seizures were found in 36 patients (90%) with a mean frequency of 59/month; and generalized seizures were found in 26 patients (65%) with a mean frequency of 23/month. The most common combination of seizure types was simple partial/complex partial/generalized seizures in 12 patients (30%) (Table 1). Seizure frequencies varied over a wide range and generalized seizures were mostly secondary to complex partial seizures. In our series, only 6 patients had a history of aura.

TABLE 1: Demographic and clinical characteristics in 40 patients with PLE*

Factor	No. of Patients (%)†
sex	
male	17 (42)
female	23 (58)
mean age in yrs (range)	
at op	25 (6–48)
at manifestation‡	
all patients	11 (0–42)
dysplasia	8 (0–26)
low-grade tumor	17 (1–39)
other	8 (1–42)
other medical history	
meningitis/encephalitis	3 (7.5)
perinatal hypoxia	2 (5)
preterm birth	1 (2.5)
brain trauma	1 (2.5)
febrile seizures	2 (5)
neurological deficit	
hemisensory syndrome	7 (17.5)
Gerstmann syndrome	2 (5)
seizure type	
SPS	1 (2.5)
CPS & GS	9 (22.5)
SPS & GS	3 (7.5)
SPS & CPS	9 (22.5)
SPS, CPS, & GS	12 (30)
aura, SPS, CPS, & GS	2 (5)
aura, SPS, & CPS	4 (10)

* CPS = complex partial seizures; GS = generalized seizures; SPS = simple partial seizures.
† Unless stated otherwise.
‡ Eleven patients (27.5%) had dysplasia, 16 (40%) had low-grade tumors, and 12 (30%) had other entities.

Electrophysiological Findings

With surface EEG, adequate results of interictal recordings were available for analysis in 26 patients (65%). Of these 26, 7 had evidence of 1 ipsilateral focus, 11 had multiple ipsilateral foci, and 8 patients had additional contralateral foci. Ictal activity was recorded in 21 patients (52%). Of these 21, only 3 patients had evidence of 1 ipsilateral focus, 14 had multiple ipsilateral foci, and 4 patients additional contralateral foci. The often inconclusive results from surface EEG analysis were a common reason for invasive evaluation. Of the 26 patients with invasive EEG evaluation, 25 had grid electrodes, 11 had additional strip electrodes on the cortical convexity, and 2 had additional depth electrodes. One patient had depth electrodes and interhemispheric strip electrodes. Intraoperative ECoG was used in 9 patients after resection to

check the surrounding cortical area for residual epileptiform activity.

Neuroimaging Findings

In 38 (95%) of 40 patients, a structural lesion was detected on preoperative MR imaging. Radiological diagnoses included developmental tumors/gangliogliomas (in 11 patients), focal cortical dysplasia (in 9), other low-grade tumors (in 6), vascular malformations (in 3), and lesions without definitive classification (for example, scar or cyst in 9). Examples of lesions are shown in Figs. 1–4. Two patients did not have findings of lesions on preoperative MR imaging. Positron emission tomography was performed in 1 patient and revealed decreased glucose utilization ipsilateral to the parietal lesion. Five patients underwent SPECT, which revealed hypoperfusion ipsilateral to the parietal lesion in all cases.

Histopathological Findings

A definitive histopathological diagnosis was obtained in 39 cases (97.5%) (Table 2). Lesions included focal cortical dysplasia (in 11 patients), ganglioglioma (in 9), scar/gliosis (in 9), DNT (in 3), low-grade astrocytoma (in 4), vascular malformation (in 2), and granulomatous inflammation (in 1). In 1 case, no histopathological diagnosis could be made.

The overall MR imaging accuracy for detecting distinct histopathological lesions was 69%; that is, 27 of 39 lesions were correctly diagnosed preoperatively. All low-grade astrocytomas, DNTs, and vascular malformations were correctly identified preoperatively. For focal cortical dysplasia MR imaging accuracy was 64%, and for gangliogliomas it was 67%. The lowest diagnostic accuracy was for scar/gliosis (5 [56%] of 9 cases) and for our 1 case of granulomatous inflammation.

Procedures and Complications

All 40 patients underwent lesionectomy (typically including a small rim of noneloquent cortex) restricted to the parietal lobe (dominant and nondominant parietal lobes in 20 patients each). Eleven patients (27.5%) underwent additional multiple subpial transections of eloquent nonlesional but ictal cortex. In cases not diagnosed using MR imaging, topectomy was performed guided by intracranial EEG recordings.

Postoperative surgical complications were seen in 2 patients (5%). One had a pulmonary embolism and the other a cerebrospinal fluid fistula; neither caused permanent morbidity. Twelve patients (30%) suffered from transient neurological deterioration, which usually consisted of incomplete Gerstmann syndrome (in 7), hemisensory syndrome (in 4), or hemiparesis (in 1) that subsequently resolved at latest follow-up. A permanent neurological deficit was observed in 3 patients (7.5%): complete Gerstmann syndrome (in 1), incomplete hemisensory syndrome (in 1), and visual field deficit (in 1, consisting of an incomplete hemianopia). There was no case of hemineglect, and there were no deaths.

Seizure Outcome

The mean follow-up was 45 months (range 5–32 months). Twenty-three patients (57.5%) were classified as completely seizure free (Engel Class I) and 4 patients (10%) had rare nondisabling seizures (Engel Class II). Five patients in this subgroup with satisfactory seizure outcome were completely seizure free without auras since surgery.

Eleven patients (27.5%) were categorized as Engel Class III (reduction of seizure frequency > 75%) and 2 patients (5%) as Engel Class IV (< 75% reduction of seizure frequency). Thus, an overall satisfactory outcome (Engel Classes I and II) was seen in 27 patients (67.5%) and unsatisfactory seizure outcome (Engel Classes III and IV) was seen in 13 patients (32.5%).

To formally analyze multiple clinical and surgical factors simultaneously in the prediction of outcome, we performed a multifactorial univariate logistic regression analysis. Sex, age at epilepsy manifestation, duration of epilepsy, EEG characteristics (including interictal and

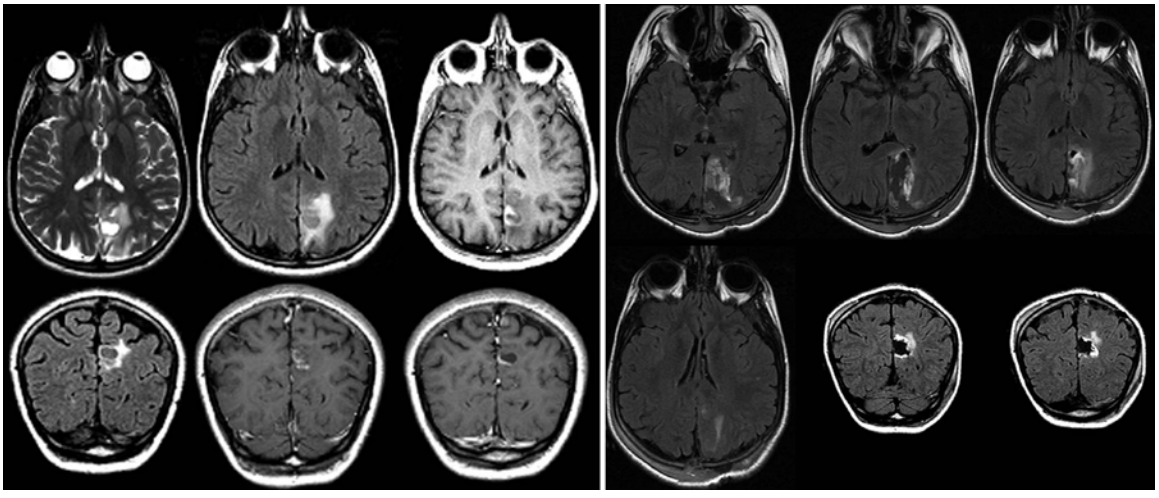


FIG. 1. Examples of a parietal low-grade tumor. Preoperative *(left)* and postoperative *(right)* axial and coronal T1-weighted, T2-weighted, and FLAIR MR images obtained in a 22-year-old woman with a 5-year history of medically intractable complex partial seizures. The patient became seizure free after lesionectomy. Pathological analysis revealed ganglioglioma.

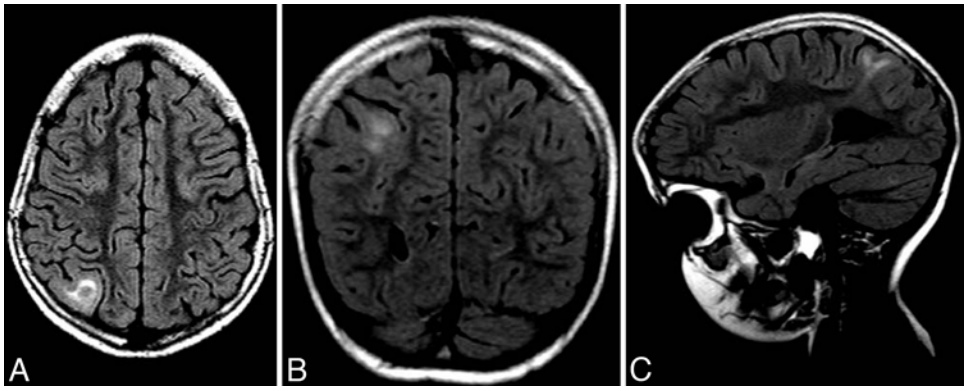


FIG. 2. Example of a parietal dysplastic lesion. Preoperative axial (A), coronal (B), and sagittal (C) FLAIR MR images of a hyperintense lesion in the right parietal lobe. The resected specimen was classified as a focal cortical dysplasia (Type IIb). The patient was seizure free after lesionectomy.

ictal EEG classifications), type of surgery, histological findings, presence/absence of invasive EEG, ECoG, MST, and postoperative neurological deficit were tested in the model with satisfactory (Engel Classes I and II) versus unsatisfactory (Engel Classes III and IV) seizure outcome as the dependent variable. A backward calculation was performed with inclusion at a probability value of 0.05 and exclusion at a probability value of 0.1. The result of this analysis was that no single factor tested was significant in predicting seizure outcome.

Discussion

Previous Series of PLE Surgery

By far the largest series of operations for PLE were reported by authors from the Montreal Neurological Institute (Table 3).[28,31,32] In 1991, Rasmussen[28] summarized the entire Montreal Neurological Institute experience up to 1980 for parietal, central, and occipital epilepsy resections. In 82 patients treated using nontumoral parietal resections between 1929 and 1988, 65% became seizure free or had rare seizures, and patients without postresection EEG epileptiform discharges had a more favorable outcome.[31] In 34 patients treated for parietal tumors between 1934 and 1988, 75% became seizure free or had rare seizures.[32]

Other smaller series of patients with PLE have been published over the past 15 years (Table 3).[34,35] A retrospective evaluation of 10 patients with PLE treated between 1983 and 1988 revealed that 90% were reported to have had "excellent" results after surgery.[38] In 1993, Cascino et al.[4] reported a retrospective study of 10 circumscribed parietal resections (lesionectomies) performed at the Mayo Clinic between 1985 and 1991 with a follow-up of 1–5 years. Nine patients had a mass lesion (tumor or arteriovenous malformation) and the other patient had gliosis. Interestingly, all patients except the patient with gliosis became seizure free (90%) following resection.

In 1998, Gawel and Marchel[11] reported 25 parietal resections performed between 1957 and 1996 at the Medical University of Warsaw. The most frequent pathologies were glial scars and tumor. Fourteen patients were assessed with 1–6 years of follow-up, and 6 (43%) were seizure free. In 2000, Olivier and Boling[26] reported their series of parietal and occipital resections. They reported 59 parietal resections in 39 patients with a seizure-free (Engel Class I) outcome of 52%, and 39 occipital resections in 30 patients with a seizure-free outcome of 71%. In each location, pathological findings in decreasing order of incidence were tumors, gliosis, dysplasias, and arteriovenous malformations. In 2003, Kasowski et al.[19] summarized a series of 28 patients with PLE treated at Yale between 1990 and 2003. Lesions included gliosis (in 12 patients); low-grade tumors (in 8); dysplasias (in 6); and vascular malformation, viral infection, and laminar necrosis (in 1 patient each). With a median follow-up of 6.2 years, 16 patients (55%) were free of seizure and auras.

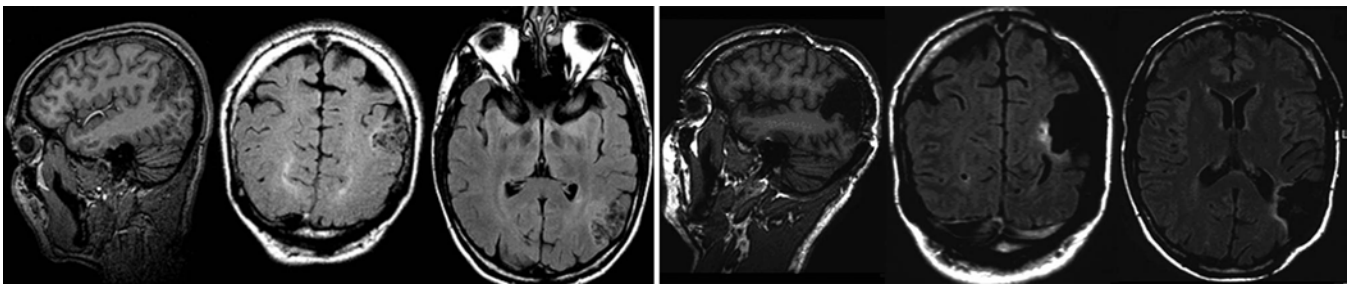


FIG. 3. Example of a parietal DNT. Preoperative *(left)* and postoperative *(right)* sagittal, coronal, and axial MR images demonstrating a hypointense lesion of the left parietal lobe. The resected specimen was classified as a DNT. The patient was seizure free after lesionectomy.

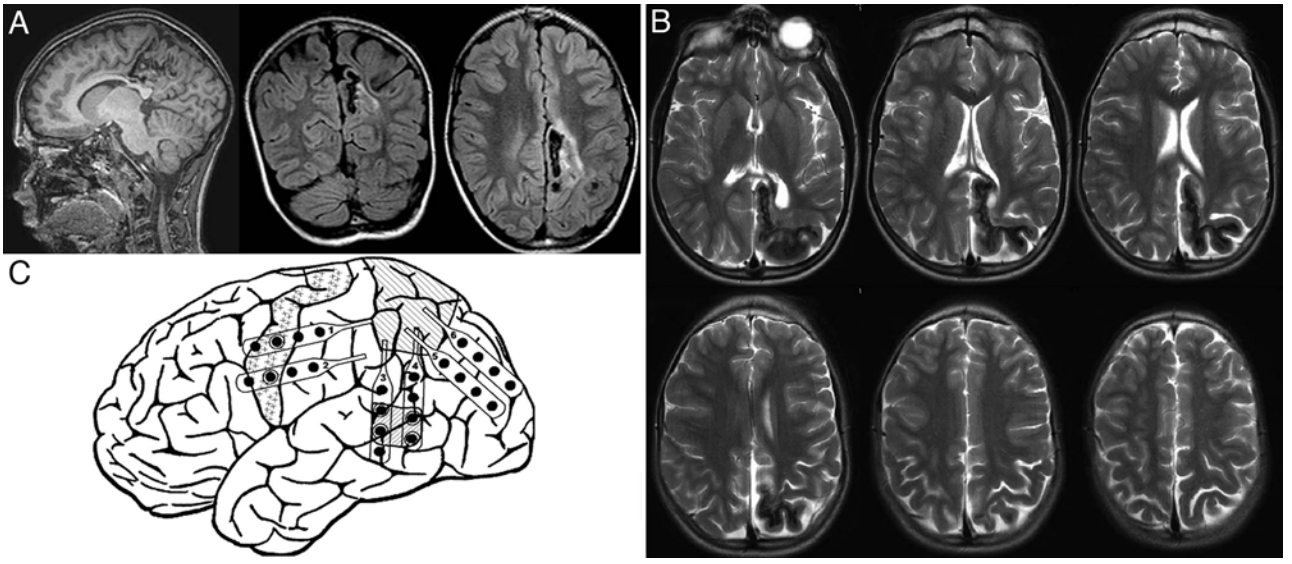


Fig. 4. Example of a parietal dysplastic lesion. Preoperative sagittal, coronal, and axial FLAIR (A) and axial T2-weighted (B) MR images demonstrating a left parietal lobe lesion in an 8-year-old boy with medically intractable seizures. After postcentral lesionectomy supported by neuronavigation (C, *dashed area*), intraoperative ECoG identified epileptiform activity of the motor cortex (gyrus with *crosses*, *circled* contacts of strips 1 and 2) and multiple subpial transections were performed. Temporodorsal epileptiform discharges disappeared after cortical resection (*circled* contacts of strips 3 and 4). The specimen was classified as focal cortical dysplasia (Palmini Type IIb), and postoperatively the patient became seizure free.

One of the largest modern series of PLE involved 38 patients with PLE who were surgically treated between 1994 and 2001 in Seoul.[20,21] Nearly all (37 [97%] of 38 patients) underwent invasive intracranial monitoring to determine the epileptogenic zone. In this series, there was a higher proportion of cortical dysplasia (94%) than in other studies. Fifteen patients (40%) became seizure free (Engel Class I).

Parietal Lobe Epilepsy: Clinical Characteristics

Because of the relatively small number of patients who have undergone surgery so far for PLE, experience is still limited compared with, for example, temporal lobe epilepsy. In our series, surgery for PLE accounted for only 40 (14%) of 292 of all extratemporal epilepsy surgeries during this period at our institution. The literature indicates that there is a strong selection bias in extratemporal epilepsy series, depending on referral, inclusion criteria, and consideration of potential surgical candidates.

Semiological features of PLE have been reviewed in detail elsewhere.[36] Lateralized somatosensory auras may be found, but these are not universal.[3,19] In addition, ictal semiology of parietal lobe seizures may mimic temporal lobe seizures, with staring, immobility, and automatisms.[16] Known parietal lobe lesions can also result in seizures with frontal lobe or supplementary motor area–like semiologies.[17] Therefore, seizure semiology has little definitive diagnostic value for PLE.

Given that neither semiological nor EEG findings provide reliably specific findings in PLE, diagnosis and selection of patients for surgery is difficult. In the present study, all patients had medically refractory epilepsy; however, MR imaging findings played a major role in nearly all patients to suggest parietal seizure localization. This lesion-based hypothesis was then deepened by analysis of clinical and EEG findings.

Interictal and ictal surface EEG recordings may contribute to the generation of an adequate seizure focus hypothesis in some cases if they can lateralize and/or localize the ictal onset zone. To clarify potentially misleading clinical and electrophysiological results, evaluation with the aid of chronically implanted electrodes may be necessary.[24] This appears to be particularly the case with PLE; in our series, 26 (65%) of 40 patients required invasive monitoring with implanted electrodes to define the epileptogenic area and to reliably differentiate seizure spread from parietal origin versus nonparietal seizure origin. Another recent series of patients with PLE used invasive monitoring in 37 (97%) of 38 patients.[20]

Imaging Modalities

Several modern studies of PLE included exclusively patients who had undergone MR imaging prior to surgery.[19,20] As in these modern studies, the vast majority of patients in our study (38 [95%] of 40) showed MR imaging–detectable lesions. The presence of a lesion on MR imaging is generally accepted to portend a better prognosis for seizure freedom.[7,41] Thus, we took a "lesion-directed" approach; however, identification of a lesion alone is not sufficient to offer epilepsy surgery. Magnetic resonance imaging proved to be very sensitive, but specificity interestingly depended on final diagnosis: it was highest for tumors and vascular malformations and lowest for dysplasia and scar/gliosis (Table 2). The clinical impact of the lower specificities may be of limited importance, however, as surgical planning is not dependent on final histological diagnosis.

In some nonlesional extratemporal epilepsies, FDG-

TABLE 2: Histopathological diagnoses and MR imaging sensitivity in 39 patients*

Histopathology	No. of Cases (%)	No. Of Cases Diagnosed Preoperatively on MRI (%)
focal cortical dysplasia	11 (28.2)	7 (64)
ganglioglioma	9 (23.1)	6 (67)
scar/gliosis	9 (23.1)	5 (56)
LGA	4 (10.3)	4 (100)
DNT	3 (7.7)	3 (100)
vascular malformation	2 (5.1)	2 (100)
granulomatous inflammation	1 (2.6)	0
total	39 (100)	27 (69)

* In 1 case, the histopathology was unknown. Therefore, this case is not included in the total. Abbreviation: LGA = low-grade astrocytoma.

TABLE 3: Results of a literature review on previous series of PLE surgery

Authors & Year	Patient Population	Outcome
Kim et al., 2004[21]	27/40 patients w/ PLE underwent op; follow-up >12 mos	54% seizure free
Kim et al., 2004[20]	38 patients w/ parietal resections	40% seizure free
Kasowski et al., 2003	28 patients w/ resections or pure MST (2 patients); 74 mos follow-up	55% seizure and aura free
Boesebeck et al., 2002	42 patients w/ parietooccipital resections; 24 mos follow-up	45% seizure free
Olivier & Boling, 2000	59 parietal resections in 39 patients	52% seizure free
Gawel & Marchel, 1998	25 patients w/ parietal resections; 12–72 mos follow-up for 14 patients	6/14 patients (43%) seizure free
Salanova et al., 1995[31]	82 patients w/ parietal resections btwn 1929 & 1988; follow-up 2–50 yrs (79 patients); only nontumor lesions	65% seizure free or rare seizures; more favorable outcome seen in patients w/o postresection ECoG abnormalities
Salanova et al., 1995[32]	34 patients w/ parietal resections btwn 1934 & 1988; follow-up 1–40 yrs; only tumor lesions	75% seizure free or rare seizures
Cascino et al., 1993	10 patients w/ parietal resections; 12–60 mos follow-up	90% seizure free; all seizure free except 1 patient w/ gliosis
Williamson et al., 1992	10 patients w/ parietal lobe resections	90% w/ "excellent" results

PET and SPECT have been especially helpful in defining the epileptogenic zone.[16,22,25,41] Ho et al.[16] used ictal SPECT to demonstrate parietal hyperperfusion in each of 14 cases of lesional PLE. In our series, we made use of FDG-PET in 1 case to support the clinical and/or imaging hypothesis. Interictal SPECT was performed in 5 patients in our series, and in all cases revealed hypoperfusion ipsilateral to the parietal lesion. In a recent series of patients with occipital lobe epilepsy, Kim et al.[22] found ictal SPECT to be sufficiently lateralizing in 76%, but the correct localization was only possible in 29%. In their study, FDG-PET was superior in localizing epileptogenic zones, with 93% correct lateralization and 60% correct localization. Thus, PET and SPECT can be regarded as diagnostic adjuncts, but their findings must be interpreted carefully. Further study is necessary to determine the specific role of each modality.[25]

Surgery and Complications

Our practice is to tailor the lesionectomies to include the lesion and the surrounding presumed epileptogenic zones as derived from noninvasive or invasive EEG monitoring. In a minority of cases, results from PET and SPECT are also considered for resection planning. Proximity to eloquent areas is the main reason that we perform lesionectomy rather than lobectomy. In 11 patients (27.5%) the epileptogenic zone overlapped eloquent cortex; these patients underwent additional MST in addition to lesionectomy. Seizure outcome was not significantly different in this subgroup from that undergoing lesionectomy alone. Other studies have previously shown that MST may be a helpful surgical adjunct in eloquent areas, as seizure outcome can be superior when combined with a resective approach.[33,40]

A concern during parietal lobe surgery is aggravation of existing or creation of new neurological deficits. In this report, we predominantly focused on visual fields, hemisensory syndromes and hemiparesis, and Gerstmann syndrome. Gerstmann syndrome, consisting of agraphia, acalculia, finger agnosia, and right-left disorientation, is classically thought to result from damage to the dominant angular gyrus,[12,13] although intraoperative stimulation studies have also shown functional sites in other parietal areas such as the supramarginal gyrus and intraparietal sulcus.[29] We found a significant proportion of patients with transient postoperative deficits (30%), largely from incomplete Gerstmann syndrome or hemisensory syndrome. However, a permanent deficit was observed in only 3 patients (7.5%). The lack of any evidence of postoperative hemineglect syndrome in this patient population is notable and is consistent with the findings of Russell et al.[30] who found no hemineglect following resection of nondominant parietal gliomas in 8 patients.

Histological Findings

The most common histopathological finding was low-grade tumors (in 16 patients) followed by focal cortical dysplasia (in 11), glial scars/gliosis (in 9), vascular malformation (in 2), and granulomatous inflammation (in 1). The spectrum of these parietal lesions is different from that in other areas of the brain and has not been well described. Others have found a predominance of malfor-

mations of cortical development[20] or tumors and vascular malformations.[4] The most notable result was that we, like Kasowski et al.,[19] found a large proportion of cases of scar/gliosis. There was variable evidence of other pathological features such as cortical and subcortical astrocytosis, hemosiderin deposition, microglial activation, and cyst formation. These findings emphasize the importance of glial scars not only in the "classic" location of mesial temporal sclerosis but also in contributing to parietal epileptogenesis.[1] Indeed, gliotic lesions can be associated with continuous epileptiform discharges.[15]

Seizure Outcome

Twenty-three patients (57.5%) were seizure free and another 4 patients (10%) had rare nondisabling seizures. Thus, overall satisfactory seizure outcome was achieved in 27 patients (67.5%). Unsatisfactory seizure outcome (Engel Classes III and IV) was seen in 13 patients (32.5%). Our outcome results compare favorably with other modern series with MR imaging–based diagnoses and Engel-based outcome measures. Direct comparison with older studies is difficult due to the absence of MR images, differences in patient selection, and use of different classification schemes. Overall, it seems that the introduction of modern MR imaging, video-EEG monitoring, and individualized use of invasive monitoring may have improved the seizure-free outcome of epilepsy surgery in the parietal lobe by 10–20%. Similar improvements over time have been noted for temporal lobe epilepsy surgery.[7,37]

Using multivariate analysis, we did not find any significant predictors for seizure outcome in our group of patients. Specifically, age at onset, clinical or EEG factors, histopathological diagnosis, and type of operation (lesionectomy vs lesionectomy plus MST) did not correlate significantly with seizure-free outcome. Other studies have reported that duration of epilepsy may affect outcome, although this is not consistently reported to be significant.[14,18] In a recent study of 44 patients with posterior cortex epilepsies, Dalmagro et al.[8] found that a favorable outcome was associated with shorter epilepsy duration.

Conclusions

Parietal lobe epilepsy is a rare but significant cause of extratemporal epilepsy. Satisfactory results (Engel Class I or II) were obtained in 67.5% of patients in our series with a mean follow-up of 45 months. Our study supports the improvement of seizure outcomes with high-resolution MR imaging and careful video-EEG monitoring and other appropriate tests. Postoperative parietal neurological deficits occur in a significant proportion of patients, although most of these are transient. The preoperative epilepsy duration of 13.7 years in our series should be regarded as a challenge to accelerate patient selection and presurgical evaluation in the future.

Disclosure

Dr. Binder was supported by a Van Wagenen Fellowship from the American Association of Neurological Surgeons. Drs. Schramm and Kral are supported by the Deutsche Forschungsgemeinschaft, Transregio Sonderforschungsbereich TR3.

Acknowledgments

The authors thank Prof. Dr. T. Pietsch and PD Dr. A. Becker for providing histological findings and diagnoses. They also thank Prof. Dr. C. E. Elger for continuous support and helpful discussions.

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Manuscript submitted November 16, 2007.
Accepted February 8, 2008.
Please include this information when citing this paper: published online February 6, 2009; DOI: 10.3171/2008.2.17665.

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