

Epilepsy and Brain Tumors: Implications for Treatment

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Summary: Primary intraparenchymal tumors of the brain are important etiologic factors in partial or focal epilepsy. Indolent low-grade gliomas may be associated with a long-standing seizure disorder refractory to medical treatment. Surgical resection of the neoplasm and the epileptogenic area may render patients seizure-free. Removal of the tumor alone may also be associated with an excellent survival rate and surgical outcome. Conventional neurosurgical procedures are restricted in patients with

tumors that are deep-seated lesions or involve functional cerebral cortex. Computer-assisted stereotactic surgical procedures have been developed for biopsy and resection of intra-axial brain-mass lesions. Stereotactic tumor resection may allow pathological determination of intracranial lesions and produce a worthwhile reduction in seizure activity in some patients with intractable partial epilepsy. **Key Words:** Epilepsy—Seizures—Brain neoplasm—Gliomas—Neurological surgery—Stereotaxic technics.

The occurrence of seizures and epilepsy in the presence of primary brain tumors has been observed for over one century (Jackson, 1882,1958; Ketz, 1974). Hughlings Jackson determined that the ictal phenomena, including seizure type, may indicate the localization of the neoplastic lesion (Jackson, 1882,1958). He emphasized that a chronic seizure disorder could be symptomatic of a glioma in a patient with partial and secondarily generalized seizures (Jackson, 1882). Jackson made the important observation that the epilepsy could be the initial and only clinical manifestation of a brain tumor (Jackson, 1882,1958). The severity of the seizure disorder and the type of seizure, however, could not predict the neuropathology associated with the epilepsy (Jackson, 1882).

The advent of neurosurgery in treating epilepsy and central nervous system tumors has confirmed these early observations. Seizures occur in approximately 50% of patients with intracerebral neoplasms (Rasmussen, 1975). Neuroectodermal tumors are the most common primary parenchymal tumors of the central nervous system, and the majority are gliomas (Shapiro and Shapiro, 1986; Kornblith et al., 1987). The incidence of seizures and epilepsy associated with primary brain tumors is related to the tumor pathology and cortical localiza-

tion (Lund, 1952; Penfield et al., 1940; Ketz, 1974, Le Blanc and Rasmussen, 1974). "Slow-growing" low-grade or well-differentiated gliomas (Kernohan and Sayre grades I and II)—astrocytoma, oligodendroglioma, and oligoastrocytoma—are the most epileptogenic lesions (Penfield et al., 1940; Lund, 1952; Rasmussen and Blundell, 1959; Ketz, 1974; Le Blanc and Rasmussen, 1974; Rasmussen, 1975). The ganglioglioma, a neural-series tumor with an astrocytic component, and the dysembryoplastic neuroepithelial tumor, composed of glial cells and neurons, are also important neoplastic determinants of a chronic seizure disorder (Kornblith et al., 1987; Daumas-Duport et al., 1988). Tumors located proximal to the centroparietal region more frequently are associated with epilepsy (Penfield et al., 1940; Ketz, 1974; Le Blanc and Rasmussen, 1974).

Seizures are the most common presenting symptom in patients with low-grade tumors. There is a direct correlation between the chronicity of the growth of the neoplasm and the incidence of seizures. In the Montreal series of 230 patients with gliomas, seizures occurred in 70% of those with astrocytomas, in 92% of those with oligodendrogliomas, and in 37% of those with glioblastomas (Penfield et al., 1940). These figures are slightly higher than those in other reported studies, a difference reflecting the referral of patients with seizure disorders (Ketz, 1974). The incidence of epilepsy is lower in patients with glioblastoma multiforme, perhaps because of the shorter duration of the disease

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(Penfield et al., 1940; Ketz, 1974). Patients with malignant gliomas, i.e., anaplastic astrocytoma and glioblastoma multiforme, are more likely to present with focal or generalized cerebral symptoms related to a space-occupying lesion (Shapiro and Shapiro, 1986).

The occurrence of brain tumors in patients with epilepsy has varied with the age of the patients, type(s) of seizures, and the nature of the patient population studied (Ketz, 1974). The most commonly identified neoplasm in patients with intractable epilepsy is the low-grade glioma (Le Blanc and Rasmussen, 1974). In patients with partial epilepsy refractory to medical treatment who undergo surgical ablative procedures, the frequency of tumors is approximately 10–20% (Penfield et al., 1940; Le Blanc and Rasmussen, 1974; Babb and Brown, 1987). Computed tomographic (CT) studies in patients with epilepsy have demonstrated a neoplasm in 10–25% of patients with onset of seizures after the age of 40 years and in fewer than 5% of children (Shorvon, 1987). During presurgical evaluation of 190 patients in one study of intractable partial epilepsy, 15% were found to have an unsuspected intracranial mass lesion and 10% a neoplasm (Spencer et al., 1984). In the Rochester series of 516 patients with epilepsy, which was not restricted to patients with partial seizure disorders, the incidence of neoplastic lesions was 4.1% (Hauser and Kurland, 1975). Tumors occur more frequently in patients with an onset of epilepsy after the age of 20 years; however, in children and in adolescents, brain tumors remain an important cause of intractable partial epilepsy (Blume et al., 1982; Suarez et al., 1986; Drake et al., 1987).

The mechanism(s) of epileptogenesis associated with brain tumors is unknown (Ketz, 1974). Hughlings Jackson observed that the pathogenesis of epilepsy in patients with tumors was related to the presence of a slow-growing tumor in the gray matter and the "sudden excessive and temporary discharge of nerve cells," producing seizure activity (Jackson, 1882, 1958; Ketz, 1974). Penfield suggested that impaired vascularization of the surrounding cerebral cortex may produce hypoxic-ischemic neuronal changes (Ketz, 1974). Direct "irritation" of the cortex by a tumor has also been proposed as the etiology of seizure activity. In the symptomatic partial epilepsies, a focal structural abnormality is intimately associated with the epileptogenic area, the site of seizure onset (Gloor, 1987; Engel, 1989). Specific morphologic neuronal alterations related to a focal brain lesion, e.g., tumors, have been observed that are potentially epileptogenic (Engel, 1989). Changes in γ -aminobutyric acid (GABA)-ergic syn-

apses, the *N*-methyl-D-aspartic acid (NMDA) receptor-cation channel, or axonal calcium or chloride channels may be associated with the enhanced excitatory and/or attenuated inhibitory influences that may facilitate epileptogenesis (Engel, 1989). Brain tumors may produce "denervation hypersensitivity" related to a partial isolation and transection of a region of cerebral cortex (Echlin, 1959; Engel, 1989). There is conflicting evidence concerning the possible importance of hereditary factors in the development of epilepsy in patients with tumors (Ketz, 1974).

A long-standing partial or localization-related seizure disorder may be related to an indolent low-grade glioma with a clinical course indistinguishable from that of a non-neoplastic process (Rasmussen and Blundel, 1959; Le Blanc and Rasmussen, 1974; Rasmussen, 1975; Goldring et al., 1986). Epilepsy occurring in the presence of a brain tumor may be associated with a simple partial seizure activity, "changing" neurological examinations, or persistent focal nonepileptiform changes in the electroencephalogram (EEG) (Blume et al., 1982; Spencer et al., 1984; Dam et al., 1985). However, available evidence conflicts regarding the importance of these variables in identifying a neoplasm-related seizure disorder (Le Blanc and Rasmussen, 1974; Blume et al., 1982; Spencer et al., 1984). The most important parameter in the diagnostic evaluation to differentiate a neoplastic from a non-neoplastic etiology in patients with partial epilepsy is the neuroimaging study, CT scan, or magnetic resonance imaging (MRI) (Goldring et al., 1986; Sperling et al., 1987). MRI is particularly sensitive in the detection of "foreign-tissue" lesions, e.g., tumors and vascular malformations (Sperling et al., 1987). Electrophysiologic studies are necessary to demonstrate an association between the neuroimaging lesion and the epileptogenic area (Engel, 1987a). MRI is superior to CT in imaging gliomas and therefore is the procedure of choice (Sperling et al., 1987).

SURGICAL THERAPY FOR EPILEPSY ASSOCIATED WITH BRAIN TUMORS

Surgical therapy is performed in the management of patients with epilepsy and low-grade gliomas to resect the tumor and to reduce seizure activity without associated neurologic morbidity. The efficacy of surgical treatment of partial epilepsy associated with neoplastic lesions has previously been documented (Rasmussen, 1975; Goldring et al., 1986; Drake et al., 1987). Successful treatment of the seizure disorder may not only render the patients seizure-free but also permit a reduction in antiepileptic

drug toxicity and improve psychological and cognitive performance. The treatment of patients with these lesions and without epilepsy will not be considered. Surgical management of malignant gliomas is performed to establish a tissue diagnosis and to reduce the bulk of the tumor and will also not be included in this discussion.

Penfield et al. (1940) observed that the surgical result, i.e., seizure outcome, in patients with tumors may be as good as the outcome in those with focal atrophic lesions if the tumor is encapsulated and can be extirpated. The recurrence of the neoplasm may be associated with partial seizure activity after a seizure-free interval. Surgical procedures have included resection of the neoplastic lesion and the surrounding epileptogenic area. Penfield and Jasper (1954) suggested that surgical removal of a structural abnormality alone may not produce a reduction in seizure activity and that a second procedure may be required to resect the epileptogenic cortex. However, it has been demonstrated that excision of a low-grade glioma without resection of the epileptogenic area may have a favorable effect on seizure activity (Goldring et al., 1986; Hirsch et al., 1989). Conventional neurosurgical procedures may be restricted in patients who have tumors that are deep-seated lesions or involve functional cerebral cortex (Apuzzo and Sabshin, 1983; Kelly, 1986, 1988; Kelly et al., 1987). Computer-assisted stereotactic surgical resection of intra-axial brain-mass lesions has been developed (Apuzzo and Sabshin, 1983; Kelly, 1986, 1988; Kelly et al., 1987; Wilden and Kelly, 1987). The stereotactic technique is superior to conventional neurosurgical procedures for the precise localization of intra-axial abnormalities (Kelly, 1988). Stereotactic surgical resections can be used to biopsy and resect intracranial lesions that involve functional cerebral cortex with a low morbidity (Kelly, 1988). Initial observations after stereotactic resection of primary brain tumors in patients with epilepsy suggested a reduction in seizure activity (Kelly, 1988).

Volumetric stereotactic surgical resection of intra-axial brain-mass lesions has been performed in more than 300 patients at the Mayo Clinic. A review of 226 consecutive resections between 1984 and 1987 showed the 226 lesions to include 112 glial tumors and 70 nonglial tumors; 36 of the 226 patients had medically refractory partial seizure disorders; all were seizure-free at 3 months postoperatively (Kelly, 1988). The methodology for the volumetric stereotactic computer-assisted procedures has been reported previously (Kelly, 1986, 1988; Kelly et al., 1987). The database acquisition and the actual surgical procedure are performed on two separate

days. An MRI- and CT-compatible headframe is used, and a CT scan, MRI, and a digital subtraction angiogram are performed on all patients. The initial data acquisition is used to determine a surgical avascular trajectory for resection of the lesion. The archival data tapes from the CT scan, MRI, and angiogram are then run into an operating-room computer system. At the time of the surgical procedure, the patient's head is again placed in the stereotactic headframe. The surgical technique depends on whether the lesion is deep-seated or is a superficial cortical abnormality. Deep intracranial lesions are approached through a 3.8-cm cranial trephine. A stereotactically directed, cylindrical retractor 2 cm in diameter is used to maintain the exposure to the lesion. The position of the circular trephine is set by the stereotactic headframe. The computer displays the tumor-slice image within a circle that represents the position and configuration of the cranial trephine. The configuration of CT- and MRI-defined tumor slices is also superimposed on the surgical field to indicate the location, configuration, and extent of the subcortical lesion at successive depth levels. Display information is shown on a video monitor in the operating room. A carbon dioxide laser is used to separate the lesion from the surrounding brain tissue. The tumor is then removed by laser vaporization.

A retrospective study of 45 consecutive stereotactic resections of intra-axial lesions in patients with partial epilepsy was performed (Cascino et al., 1989; Cascino et al., 1990). Thirty patients had medically refractory seizure disorders. The most common pathologic alterations observed were vascular malformations (20 lesions) and glial neoplasms (16 lesions). The lesion localization included the postcentral gyrus ($N = 9$), precentral gyrus ($N = 8$), deep-seated left posterior temporal region ($N = 6$), left fronto-operculum ($N = 1$), and occipital cortex ($N = 2$), sites that may have precluded the use of standard craniotomy with cortical resections. Seizure outcome postoperatively was classified as class I (seizure-free or auras only without loss of consciousness or atypical seizures during antiepileptic drug discontinuation), class II [rare seizures (1–2 per year) since surgery or initially seizure-free and now rare seizure activity], class III (continued partial seizures with an 80% reduction in seizure activity), and class IV (no improvement, although some reduction in seizures may have occurred) (Engel, 1987b). Forty-two of the 45 patients and 27 of the 30 refractory to medical treatment had at least an 80% reduction in seizure activity during a mean of 20 months postoperatively. In the medically refractory patients, 18 of 20 patients and 13 of 15 pa-

tients, who were followed for at least 1 year (mean of 30 months) and 2 years (mean of 34 months), respectively, had favorable surgical outcomes (i.e., class I, II, or III). Thirteen of the 20 patients and 9 of the 15 patients followed for at least 1 year and 2 years, respectively, had a class I surgical outcome. The brain tumors included grade I astrocytoma ($N = 5$), grade I oligodendroglioma ($N = 4$), grade II oligodendroglioma ($N = 4$), grade I oligoastrocytoma ($N = 1$), and grade I ganglioglioma ($N = 2$). The mean age of the 16 patients with neoplastic lesions was 20 years (range of 3–43 years). In the 16 patients with glial neoplasms, 11 had medically refractory seizure disorders. All five patients whose seizures had been medically controlled preoperatively were seizure-free after lesionectomy for a mean follow-up period of 19 months postoperatively. The results of the stereotactic tumor resections in the 11 patients at a mean follow-up period of 25 months (range of 6–46 months) were as follows: class I, 9 patients; class III, 1 patient; and class IV, 1 patient. The nine patients in class I included five who were seizure-free while receiving medication, three who had a single generalized seizure associated with discontinuation of medication, and one who had auras only. The mean duration of follow-up for the two patients who were in class III and class IV was 10 months and 24 months, respectively.

Among the entire group of 45 patients, the operative morbidity included a language or motor deficit in three patients. One of these patients, after resection of a left mesial posterior temporal vascular malformation, developed an anomia that gradually improved, with a nearly complete recovery noted at 1 year postoperatively; a second acquired a transient right hemiparesis in the immediate postoperative period related to a left parietal vascular malformation resection; and a third underwent a surgical procedure that resected a structural lesion from the right motor cortex. No diagnostic abnormality was noted in the third patient. A left monoparesis involving the upper extremity occurred; the only persistent deficit was a reduction in rapid altering movements in the left hand. No morbidity was identified in the biopsy and resection of the 16 gliomas. No surgically related infections, intracranial hemorrhages, or deaths occurred.

ILLUSTRATIVE CASES

Case I

A 24-year-old right-handed man had a partial seizure disorder of 10 years' duration. The patient initially had secondarily generalized seizures. Four

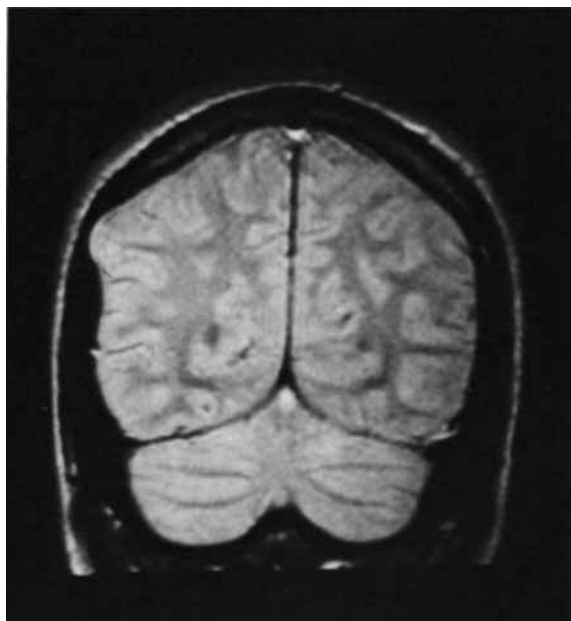


FIG. 1. An MRI in the coronal plane obtained with use of a long repetition time/echo time pulse sequence revealing an abnormality in the right parietal region. (Note that the right cerebral hemisphere is on the left side of the photograph.)

years before evaluation, the patient began having "petit mal" seizures, which included verbal automatisms in which he would speak fluently but in a confused manner. The patient's condition was refractory to maximally tolerated monotherapy and combination therapy with antiepileptic drugs. Prolonged video EEG monitoring revealed intermittent right frontocentrottemporal spike discharges with spike and slow-wave complexes. Multiple clinical and electrographic seizures were recorded associated with an ictal onset in the right frontoparietal region. MRI revealed a "small bulge" in the right parietal lobe consistent with a neoplasm (Fig. 1). A computer-assisted stereotactic craniotomy was performed with the resection of the right parietal lesion. A grade I pleomorphic xanthoastrocytoma was demonstrated. The preresection electrocorticogram revealed active spike activity anterior and posterior to the lesion, with the anterior spiking tending to lead the posterior spiking. An EEG performed 4 months after resection recorded rare right temporal spike activity during sleep. Prior to surgery, the patient was having four to five complex partial seizures per month. No neurologic morbidity was associated with the patient's surgical procedure. Currently, approximately 6 months postoperatively, the patient is seizure-free and asymptomatic while receiving a reduced program of antiepileptic medication.

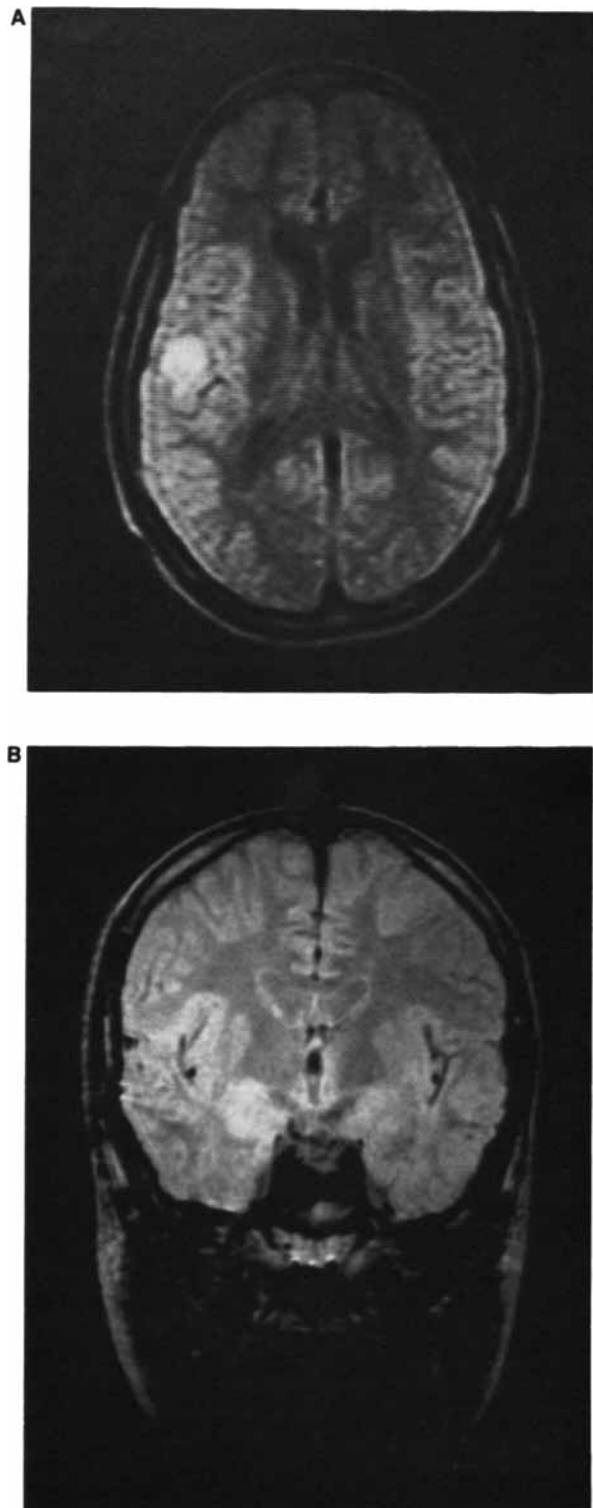


FIG. 2. (A) An MRI in the axial plane revealing an area of increased intensity in the right fronto-opercular region. (B) An MRI in the coronal plane. (Note that the right cerebral hemisphere is on the left side of the photograph.) Both images were obtained with the use of a long repetition time/echo time pulse sequence.

Case II

A 12-year-old left-handed boy had a medically refractory partial seizure disorder of 5 years' duration. The patient had generalized tonic-clonic seizures and complex partial seizures. Secondly generalized events began with focal tonic-clonic activity on the left side. The patient was also having episodes of amnesic automatisms every 3 days. Prolonged video EEG monitoring noted prominent right frontocentral spike activity. MRI revealed a right posterior frontal opercular region lesion (Fig. 2). Computer-assisted stereotactic resection was performed and revealed a grade I oligoastrocytoma. The preresection electrocorticogram revealed superior temporal and inferior central spike discharges. The postresection electrocorticogram noted reduced superior temporal suprasylvian spikes. Sleep and awake EEGs revealed no epileptiform activity 4 months after surgery. The patient is seizure-free and asymptomatic 3 years after surgery. The MRI has not revealed evidence of recurrence of the neoplasm. The patient is still receiving maintenance monotherapy with phenytoin. No morbidity was associated with the surgical procedure.

Case III

A 6-year-old child with a seizure disorder of 18 months' duration was observed to have frequent focal tonic-clonic seizures involving the right lower extremity. No definite impairment of consciousness was noted. The patient was demonstrated to have a medically refractory seizure disorder. Neurologic examination disclosed no definite abnormality. The ictal EEG onset was in the left centroparietal region. MRI revealed a lesion in the left frontoparietal parasagittal region (Fig. 3). A computer-assisted stereotactic biopsy of the left motor cortex lesion was performed. Pathologic examination revealed a grade I pilocytic astrocytoma. No neurologic morbidity was noted postoperatively. At 1 year after surgery, the patient is seizure-free with no obvious recurrence of the tumor but is observed to have a developmental delay. Sleep and awake EEGs were unremarkable at this time. Antiepileptic medication has been continued.

DISCUSSION

Computer-assisted stereotactic resection of low-grade gliomas in patients with epilepsy may be preferred to a standard operative resection of the tumor and epileptogenic area in select circumstances, e.g., on the basis of the results of a sodium amobarbital study or on the localization of the lesion in functional cerebral cortex. The data acquisition prior to the stereotactic procedure may allow for the least

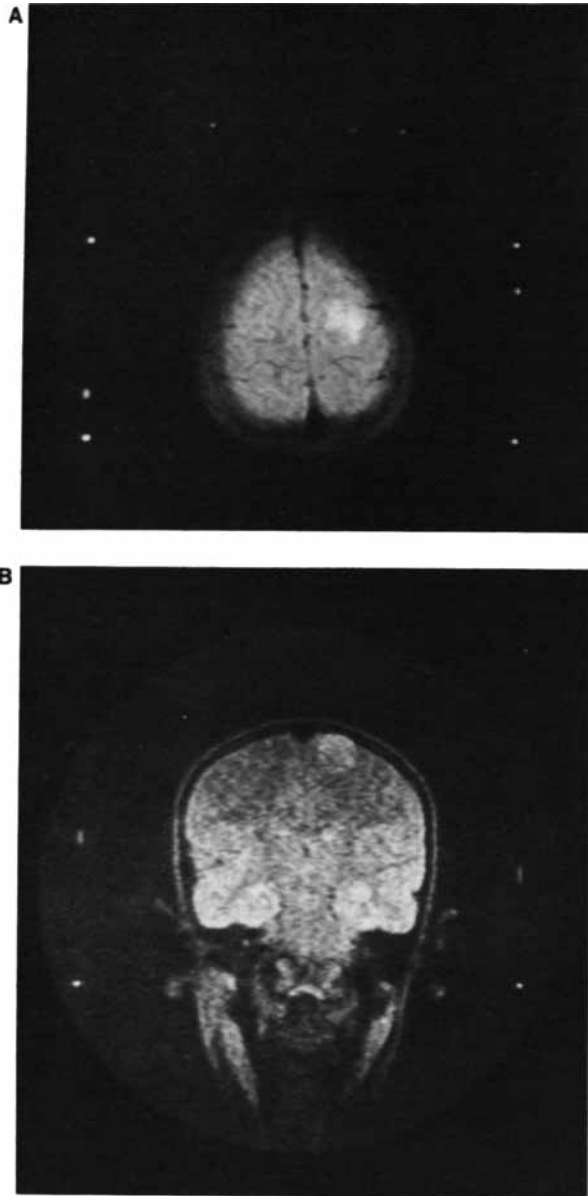


FIG. 3. (A) An MRI in the axial plane revealing an area of increased intensity in the left frontoparietal parasagittal region. (B) An MRI in the coronal plane. (Note that the left cerebral hemisphere is on the right side of the photograph.) Both images were obtained with the use of a long repetition time/echo time pulse sequence.

possible disruption of normal brain tissue. The computer display images may provide information regarding the boundaries of the structural abnormality and normal brain tissue that may not be apparent at the time of gross resection (Kelly, 1988). Potentially, stereotactic resection may result in less neurologic morbidity than that associated with the standard neurosurgical procedures.

Excision of a low-grade glioma alone, without resection of the epileptogenic cortex, may be associated with a favorable seizure outcome (Goldring et al., 1986; Hirsch et al., 1989). Goldring et al. (1986) evaluated the surgical outcome after temporal lobe tumor excision, usually a low-grade astrocytoma, in 20 patients with epilepsy. Only one patient continued to have postoperative seizures, although two patients had seizures early in the postoperative period. The mean follow-up period was 2.5 years (range of 3 months to 10 years). The authors emphasized the presence of a chronic seizure disorder (the mean interval from seizure onset to surgery was 11 years) and an isolated lesion on CT examination in these patients. The type of seizure and the lack of response to medication did not seem to identify the nature of the epileptogenic lesion. Patients who had a simple excision of a temporal lobe tumor were compared with patients who had an anterior temporal lobectomy that included the tumor and hippocampus. During a comparable follow-up period. (> 1.5 years; range of 1.5–10 years), 1 of 12 patients with the simple excision and 2 of 7 patients with a lobectomy continued to have seizures after surgery. Hirsch et al. (1989) evaluated the response to tumor resection alone in 42 children with low-grade gliomas. The incidence of epilepsy preoperatively was 78%. Tumor removal alone was performed without resection of the epileptogenic cortex. The incidence of epilepsy was 19% after surgery. The follow-up period was variable (range of 6 months to 17 years).

Resection or alteration of the epileptogenic cortex surrounding the lesion is a potential mechanism whereby stereotactic tumor excision may reduce seizure activity. Disruption of the cerebral cortex will occur inevitably with the stereotactic procedure. Even incomplete resection of the epileptogenic area may be associated with a significant reduction in seizure activity (Ojemann, 1985). Enhancement of the pharmacologic responsiveness of medically refractory seizures after tumor removal cannot be excluded. It has been proposed that lesionectomy "restores a mechanism that limits the spread of seizures and, thus, the clinical manifestations of epilepsy" (Hirsch et al., 1989). However, tumor excision alone clearly does not abate seizure activity in all patients (Cascino et al., 1989; Cascino et al., 1990). The failure of this procedure may relate to the remoteness of the epileptogenic area or to its noncontiguity with the tumor. The stereotactic procedure may also be unable to resect a diffuse or infiltrating glioma. The possibility of dual pathology, e.g., coexistent hippocampal sclerosis, cannot be excluded (Babb and Brown, 1987; Drake et al.,

1987). However, the most likely explanation for the continued partial seizure activity postoperatively is the inadequate resection of the epileptogenic area (Gloor, 1987).

This discussion represents an initial observation regarding stereotactic lesionectomy in patients with partial epilepsy and neoplastic lesions. An ideal study would have included a prospective analysis of all patients who have had a long-term follow-up period, e.g., a minimum of 2 years (Engel, 1987*b*). The possibility of false reporting, i.e., of increasing favorable outcome regarding seizure activity, is difficult to exclude in a retrospective analysis (Engel, 1987*b*). The follow-up period of the patient group is variable, and long-term control of seizures cannot be assumed. Certainly the most meaningful data for the patients with intractable seizure disorders are the identification of patients who are seizure-free or nearly seizure-free who have been followed for at least 1 or 2 years postoperatively (Engel, 1987*b*). In the 16 patients with neoplastic lesions, 14 had class I surgical outcomes after stereotactic lesionectomy during a short-term period of observation. Importantly, stereotactic lesionectomy may be associated with reduced seizure activity without associated neurologic morbidity even for the resection of lesions involving functional cerebral cortex.

Potentially, the surgical outcome may have been more favorable after resection of the tumor and the epileptogenic area. In our series, 2 of the 11 patients with medically refractory seizure disorders and brain tumors had persistent partial seizure activity after lesionectomy. Four of the class I patients had atypical single seizure episodes or persistent auras. Only 5 of the 11 patients with neoplastic lesions were seizure-free, and all of the patients continued to receive antiepileptic medication. It is anticipated that the long-term follow-up of these patients would demonstrate an increased number with recurrent seizure activity. One patient, after a stereotactic resection of a lateral temporal oligodendroglioma, was seizure-free for 9 months before partial seizure activity recurred. An anterior temporal lobectomy was subsequently performed; intraoperative depth recordings revealed active hippocampal spiking ipsilateral to the lesion. A comparison of surgical techniques is difficult because the location of the lesion in some instances may have precluded a standard operative resection.

Potentially, the stereotactic lesionectomy may complement other surgical techniques in the management of patients with epilepsy. The ultimate aim for the surgical treatment of epilepsy is not only to reduce seizure activity but also to avoid potential morbidity associated with a therapeutic intervention

(Ojemann and Dodrill, 1985). Functional extraoperative and intraoperative cortical stimulation studies, however, may also have allowed resection of these tumors and the epileptogenic area with a favorable seizure outcome and without neurologic morbidity (Ojemann, 1983; Goldring and Gregorie, 1984; Lesser et al., 1984). The evaluation of the surgical outcome must include assessment of acquired postoperative neurologic deficits and the effect on seizure activity (Ojemann and Dodrill, 1985).

There are important limitations regarding the role of stereotactic neurosurgery in the management of partial epilepsy. Neuroimaging studies may identify a potentially epileptogenic abnormality in the minority of patients with partial epilepsy. The most commonly identified pathologies associated with intractable partial epilepsy, i.e., mesial temporal sclerosis and cortical gliosis, may not be associated with a neuroimaging abnormality (Sperling et al., 1987). Presurgical evaluations, including EEG studies, are essential to determine the location of the epileptogenic cortical tissue in patients with partial epilepsy and neuroimaging alterations (Engel, 1987*a*). The instrumentation necessary for the computer-assisted stereotactic resection is expensive and requires trained personnel (Kelly, 1988). Conventional microneurosurgical techniques are not readily extrapolated for this technique (Kelly, 1988). Finally, not all patients appear to experience a worthwhile reduction in seizures after stereotactic lesionectomy (Cascino et al., 1989, 1990). A second procedure involving resection of the epileptogenic area may be necessary in these patients to produce a beneficial effect on seizure outcome and/or a reduction in the need for antiepileptic drugs.

Computer-assisted volumetric stereotactic lesionectomy allows pathologic determination of low-grade tumors and other intra-axial brain-mass lesions and may reduce seizure activity in some patients with intractable partial seizure disorders. This procedure can be performed with a low associated morbidity, even for lesions that are relatively inaccessible to standard operative procedures.

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