Epilepsy Surgery

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39.1 Introduction

Almost 50,000,000 people in the world are estimated to have epilepsy, with a prevalence of 1–2 cases per 1,000 persons in developed countries [20] and 18 per 1,000 persons in developing ones [4]. While most epileptic patients can achieve seizure control with the use of antiepileptic drugs (AEDs), about 25–45 % of them continue to have refractory seizures despite an appropriate clinical treatment [20, 26]. Medical refractoriness should be suspected when two appropriately chosen, well-tolerated, first-line AEDs or one monotherapy and one combination regimen have failed to achieve acceptable seizure control [5, 23].

Surgical treatment may be an important alternative for those patients whose quality of life is significantly impaired by the seizures or by the adverse effects of medication. Surgical candidates must be evaluated by a specialized multidisciplinary team after an extensive presurgical study based on video-electrographic monitoring, neuroimaging, and neuropsychological study. Surgery is best indicated for those whose seizures start in a noneloquent brain area, which can be removed without causing a severe neurological deficit. In some cases, palliative procedures are performed in order to reduce the frequency and severity of the seizures, even when the expectation of a cure is low.

A wide variety of surgical options are available today. The most frequent is temporal lobectomy with amygdalohippocampectomy. Other procedures are lesionectomies, neocortical resections,

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multiple subpial transections (MSTs), hemispherectomy, and corpus callosotomy.

Surgery for the treatment of epilepsy in children and adolescents has been indicated in earlier ages. Factors like neuronal plasticity and the effect of epilepsy on early brain development reinforce this indication [11].

39.2 Presurgical Evaluation

The main goal of presurgical evaluation in patients with intractable epilepsy has been the identification of the epileptogenic zone. This includes not only the actual area that is generating the patient's habitual seizures but also those regions with potential epileptogenicity [7].

Five zones should be identified in the evaluation: (1) the symptomatogenic zone (cortical area that, when activated by the epileptic discharge, reproduces the patient's typical ictal symptoms), (2) the epileptogenic zone (cortical area capable of generating seizures and whose removal or disconnection will result in seizure freedom), (3) the irritative zone (cortical area capable of generating interictal spikes on the electroencephalogram [EEG]), (4) the ictal onset zone (cortical region from which we can objectively demonstrate that seizures are arising), and (5) the functional deficit zone (area that shows abnormal functioning in the interictal period) [46]. These results can be achieved with careful analysis of seizure semiology and electrophysiological studies (video-EEG, VEEG) associated with dates from anatomical and functional neuroimaging. Neuroimaging is particularly important for the evaluation of patients and the identification of focal lesions such as tumors, cortical dysplasia, areas of gliosis, neuronal loss with atrophy, and other developmental abnormalities.

Functional imaging studies using radiotracers, such as positron emission tomography (PET) and single-photon emission computed tomography (SPECT), are performed to identify or confirm the ictal focus in preparation for surgery. In some cases it is necessary to perform the Wada test, when the language and memory function on the same side are tested after sodium amytal infusion into the homolateral carotid artery.

Magnetoencephalography (MEG) is a recent method that is not available at all centers. MEG is a method used to localize epileptic activity and consists of the evaluation of the magnetic fields produced by electrical activity in the brain.

The gold standard for surgical localization in intractable epilepsy is the ictal EEG pattern identified by long-term scalp-recorded VEEG monitoring [8]. The rationale underlying ictal recordings includes confirmation of seizure type, evaluation of ictal semiology, and surgical localization. Trained personnel perform peri-ictal speech and memory testing and ensure the patient's safety. Ictal scalp recordings alone may suffice if results are concordant with those of other neurodiagnostic studies [8]. This method has some limitations for identification of ictal EEG changes in patients with simple or frontal lobe partial seizures. Myogenic and electrode artifacts during seizures may obscure an ictal electrographic seizure pattern, and patients may not have a typical, habitual seizure during prolonged EEG recordings despite the withdrawal of AEDs [8, 32].

Invasive procedures become necessary when noninvasive means of preoperative investigation cannot determine in a reliable manner the epileptogenic zone for surgery or due to its close proximity to the eloquent cortex. They should be indicated only if the noninvasive evaluation results in at least a reasonable hypothesis regarding the possible localization of the epileptogenic zone.

39.3 Temporal Lobe Epilepsy

Temporal lobe epilepsy is the most frequent form of adult refractory epilepsy and also presents the best prognosis after surgical treatment. These refractory forms can be classified as neocortical or mesial temporal epilepsies. While the neocortical forms are similar to extratemporal neocortical epilepsies in terms of evaluation and surgical treatment, most patients with mesial temporal epilepsy present clinical, electrophysiological, and imaging signs and symptoms suggestive of mesial temporal sclerosis (MTS) with hippocampal sclerosis.

Fig. 39.1 Video electroencephalogram (EEG): ictal EEG showing rhythmical theta activity in electrodes in the right mesial temporal lobe

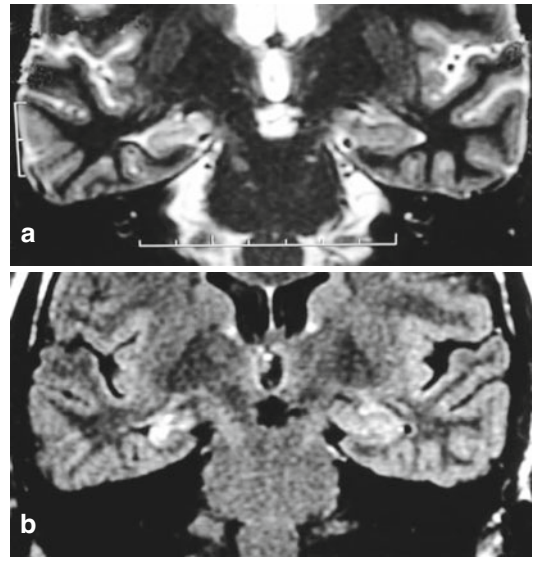

Fig. 39.2 Right mesial temporal sclerosis. (**a**) Hippocampal atrophy on T1-weighted imaging. (**b**) Signal increase in fluid-attenuated inversion recovery sequence

MTS typically causes complex partial seizures in young adult patients with a history of prolonged febrile seizures during childhood [2, 19]. Despite intensive investigation, it has not been determined whether MTS is the cause (disorders of neuronal migration in the hippocampus) [15] or the consequence (hippocampus vulnerable to the tissue damage induced by prolonged seizures) of the febrile episodes.

The difficulty in controlling seizures increases progressively over the years, with the disorder becoming refractory to medicamentous treatment within a period of 1–15 years [31]. The seizures usually arise with epigastric or psychic auras followed by behavioral arrest and oroalimentary or hand automatisms. The latter are usually homolateral to the temporal lobe involved, while the contralateral limb presents dystonic postures [21]. The difficulty in speaking during the seizure and during the postictal period may suggest lateralization to the dominant temporal lobe [16].

Electrophysiological investigations demonstrate the presence of rhythmic activities in the mesial electrodes of the interictal EEG. Ictal investigation by VEEG, in addition to correlating the clinical seizure with the electrographic seizure (lateralized rhythmic theta activity is usually observed; Fig. 39.1), permits radioisotope study (SPECT), which identifies local metabolic changes, facilitating the location of the epileptogenic area.

Over recent years, improvements in imaging techniques such as magnetic resonance imaging (MRI) have represented a major advance, permitting a precise evaluation of the changes mainly located in the hippocampus. MTS is characterized by atrophy of this structure in the T1-weighted sequence or in the volumetric reconstructions and by an increase of the signal in T2-weighted and the fluid-attenuated inversion recovery sequence (Fig. 39.2) [9].

In patients with MTS, the congruence of these data (clinical, interictal EEG, VEEG, SPECT, and MRI) suggests a better prognosis for surgical treatment. When doubts exist, especially regarding the lateralization of the seizures, invasive evaluation can be used by bilateral implantation of deep electrodes into the hippocampus.

Surgery is the recommended treatment. Randomized studies have demonstrated that after 1 year, seizure control is at least twice as efficient in patients who submit to surgical treatment as in patients receiving medicamentous treatment [59].

Surgical treatment of MTS is based on two interventions: anterior temporal lobectomy and selective amygdalohippocampectomy sparing resection of the temporal neocortex. No statistical difference has been demonstrated between these two methods regarding the efficiency of seizure control or the prevention of cognitive disorders [31].

Anterior temporal lobectomy starts with resection of the temporal neocortex including the middle and inferior temporal gyri, permitting a better view of the mesial structures. The posterior extension differs according to the side: about 5–6 cm in the nondominant hemisphere and 3–4 cm in the dominant hemisphere [51]. Using a subpial technique, the entire lateral temporal parenchyma and uncus are removed up to the limit consisting of the medial margin of the tent. This permits the exposure laterally through the pia mater, of cranial nerve III and the posterior cerebral artery, and of the internal carotid, posterior communicating, and choroidal arteries medially. This resection in a posterior direction creates a space that permits the lateral retraction of the hippocampus, facilitating the coagulation of its hilus. The next step is the opening of the ventricular cavity in order to expose the amygdala and the hippocampus. To facilitate the location of these structures, a corticectomy is performed from the superior temporal sulcus, 3 cm posterior to the tip of the temporal lobe, in the direction of the free margin of the previously exposed tent. The amygdala is the first structure to be removed using, as a reference, the upper limit of the line formed between the emergence of the choroidal artery into the internal carotid artery and its entry into the choroidal fissure of the temporal horn (choroidal point). This limit is important in order to prevent damage to the optic tract [58]. Resection of the hippocampus starts from the anterior portion (head) and extends posteriorly through its medial surface, with coagulation of the hilus and of the branches originating from the posterior cerebral artery. The hippocampus is removed in a block of 2.5–3 cm up to the posterior portion of the cauda, permitting the complementary removal of the parahippocampal gyrus in its more medial portion (Figs. 39.3 and 39.4).

Regarding the results of anterior temporal lobectomy, 65–80 % of patients have been reported to remain seizure-free. This difference is due to different criteria for the evaluation of the results and to refractoriness for surgical indication. In our series of 60 patients with temporal lobe epilepsy followed up for at least 2 years, 69 % showed results compatible with Engel Ia or Ib [12]. However, if we consider only patients with MTS, this rate reaches 80 % (Fig. 39.5).

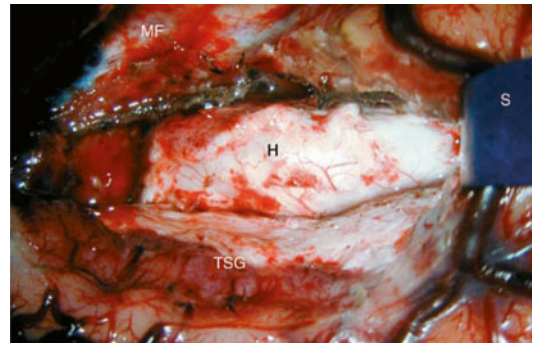

Fig. 39.3 Surgical exposure of right hippocampus (*H*). *TSG* temporal superior gyrus, *MF* medial fossa, *S* spatula

Several complications have been reported regarding this procedure: superior quadrantopsy, hemianopsia, hemiplegia due to lesion of the choroidal artery, and memory and speech disorders. In our series we observed a patient with late osteomyelitis, a patient with temporary dysfunction of cranial nerve III, and two asymptomatic patients with a chronic subdural hematoma revealed by postoperative MRI.

39.4 Extratemporal Epilepsies

While about 80 % of patients with temporal epilepsy present with an anatomical substrate (hippocampal sclerosis), the extratemporal epilepsies are heterogeneous in terms of their etiology. Recent advances in imaging examinations have led to a rapid increase in the number of identifiable lesions, especially cortical dysplasias, which play an important role in the genesis of extratemporal epilepsies. However, approximately 50 % of refractory extratemporal epilepsies are not definitely identified by neuroimaging studies [52].

Historically, the first surgeries for extratemporal epilepsies date back to the work of Rasmussen and Penfield at the Neurological Institute of Montreal [44, 45, 47]. These investigators classified the pre- and postcentral gyri as a specific region, denoted the central area. On this basis, surgical epilepsies were identified as being 56 %

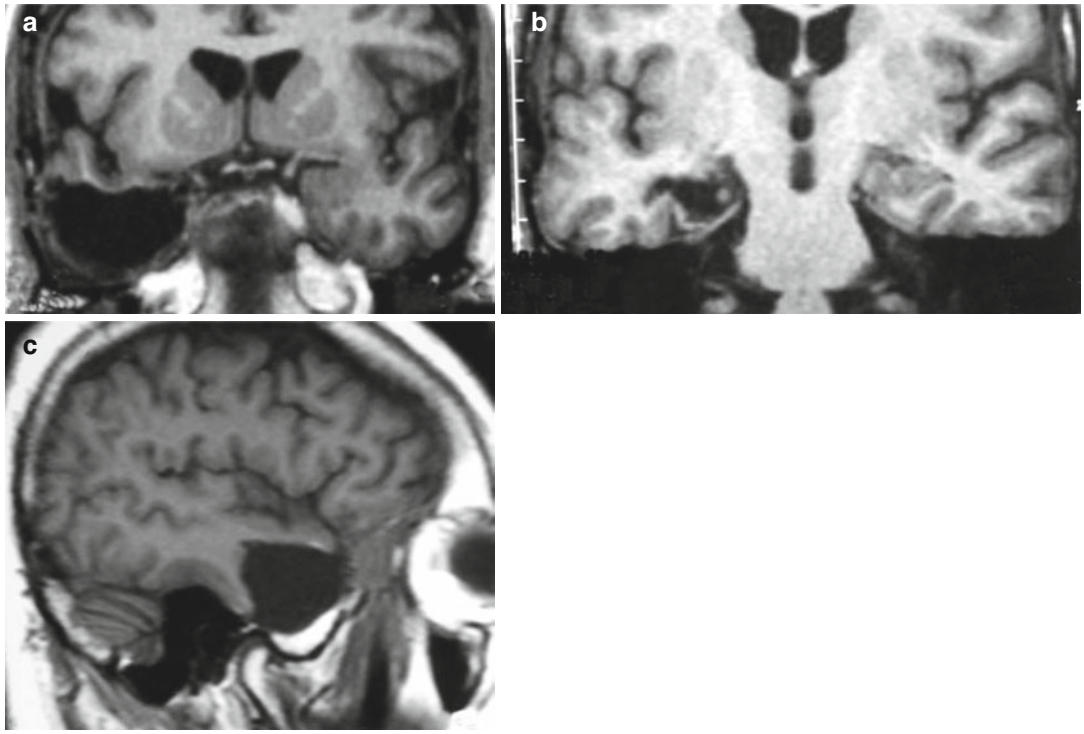

Fig. 39.4 Postoperative magnetic resonance imaging (MRI) showing resection of the right neocortex (**c**), amygdala (**a**), and hippocampus (**b**)

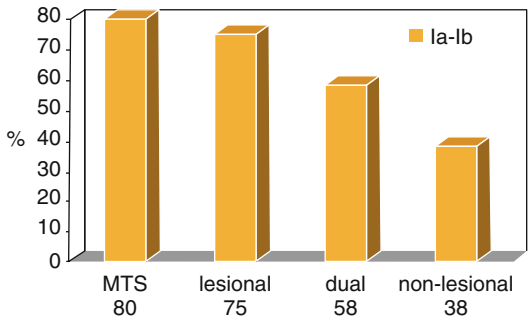

Fig. 39.5 Results in temporal lobe surgery. Percentage of patients with Engel Ia–Ib results. *MTS* mesial temporal sclerosis, *Dual* MST, and lesional

temporal, 18 % frontal, 7 % in the central region, 6 % in the parietal lobe, and 1 % in the frontal lobe [45]. Multilobar resections or hemispherectomy was used to treat 11 % of the patients. Other series focusing on surgical treatment of extratemporal epilepsies identified 45–64 % of the surgeries as being performed after seizures starting in the frontal lobe, 7–13 % starting in the parietal lobes, 2–23 % as being occipital, and 23–44 % as multilobar foci [25]. In the surgical series published by Eriksson et al. [13], while 75 % of adult surgeries involved the temporal lobe, only 25 % of children's surgeries involved this lobe.

The semiology of extratemporal syndrome is varied and is not well characterized, even when limited to a single lobe. Extratemporal epilepsies also tend to spread rapidly, impairing their location on the basis of clinical characteristics. In some cases, especially those involving the frontal lobe, the seizures rapidly cross to the contralateral side, also impairing their lateralization [38].

The presence of a focal finding in MRI is probably the most important factor in the approach to extratemporal epilepsies, since a focal lesion classifies the epilepsy as lesional, with a better surgical prognosis. Other imaging studies such as ictal or interictal SPECT, PET, and spectroscopy can be of help for the focal diagnosis when MRI is normal [36].

Most patients with extratemporal epilepsies refractory to medicamentous treatment present extensive irritative multilobar surface areas in their EEG monitoring [36, 38]. In a series of 30 patients with localized forms of cortical dysplasia, more than half of the subjects presented a greater distribution of interictal findings than the structural lesion, with two-thirds of them being multilobar [35]. The identification of lobar distribution, in turn, is insufficient for a precise topographic definition. In addition, extensive neocortical areas located in the interhemispheric and basal regions, and therefore "far" from the surface electrodes, may be initial firing foci. These factors, together with the property of extratemporal foci, especially of the frontal lobe, to rapidly propagate also to the contralateral hemisphere, lead to an inconclusive, or even "false-positive," location of the epileptogenic foci [24]. Thus, it is necessary, especially when the structural lesion cannot be located, to use invasive monitoring (i.e., the placement of deep electrodes in the cerebral parenchyma or subdural electrodes on the cortical surface) [35].

Deep electrodes are fine cables with cylindrical contacts along their terminal extremity, which are placed inside the encephalic parenchyma. They are used when there is a suspicion of epileptogenic areas deeply located or, more commonly, in patients with temporal epilepsy who cannot be correctly lateralized due to the rapid propagation of the impulse to the contralateral temporal lobe. They are commonly implanted stereotaxically by trephining along the direction of the hippocampus (entering through the occipital lobe) or orthogonal to the axis of the hippocampus (entering through the temporal lobe; Fig. 39.6).

The need for implantation involves a 1–4 % risk of infection and a risk of cerebral hemorrhage of 3 % in parasagittal placement and of 1 % in lateral placement [42]. The placement of bilateral intrahippocampal electrodes may be associated with a postoperative decline of verbal memory [41].

Subdural electrodes are used to map the surface of the encephalon and to delimit the region of seizure onset in the neocortex. The electrodes are fine platinum or stainless steel disks attached to a fine plastic surface arranged in the configuration of striae or plates. They are placed in intimate contact with the cerebral parenchyma by craniotomy, and they can be placed in mesial structures. Subdural electrodes are used not only for ictal recording and for the determination of the zone of seizure onset but also for cortical stimulation and mapping of cortical function on this area [52]. Six cases of infection (two cases of meningitis, one abscess, and three infections of the surgical wound) were reported in a series of 350 patients. In another series of 131 patients, 2.5 % of them presented small hematomas that did not require surgical drainage [41]. Analyses of a limited amount of cortical surface and enhancement or reduction of the epileptiform activity over the underlying cortex are disadvantages of this method (Figs. 39.7, 39.8, 39.9, 39.10, and 39.11) [7].

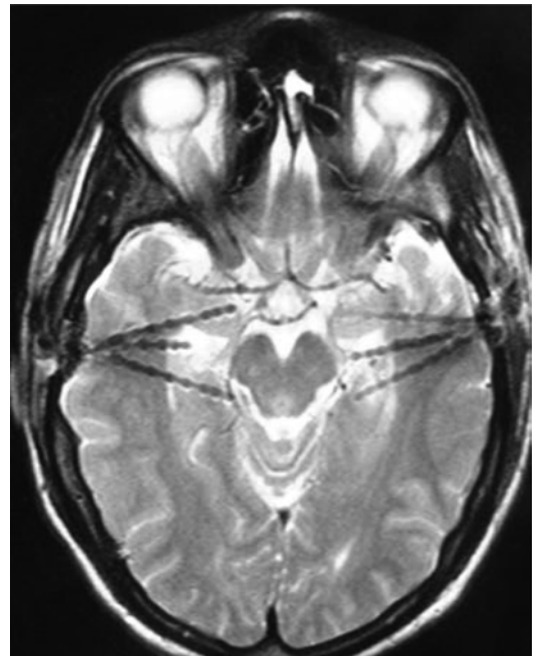

Fig. 39.6 Deep bilateral temporal electrodes

Eloquent areas such as the motor cortex, sensitive cortex, language areas (Broca's area, Wernicke's area, supplementary motor area, basal temporal area), and visual areas can be identified by cortical stimulation either in the operating theater (motor and language) or during the period of

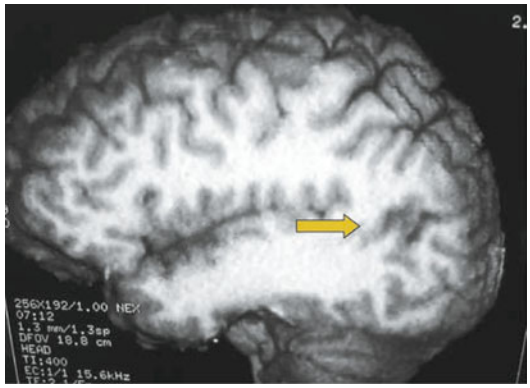

Fig. 39.7 Gliotic area near the left angular gyrus (*arrow*) in a 40-year-old man with partial seizures

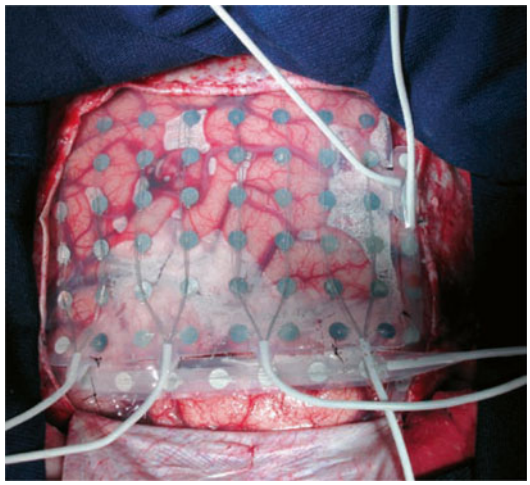

Fig. 39.8 Subdural electrodes in the same patient as shown in Fig. 39.7

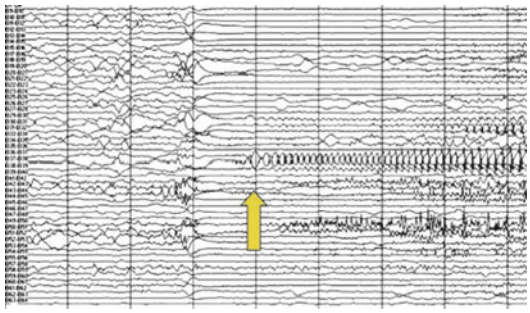

Fig. 39.9 Ictal EEG recording during a partial seizure from the patient of Fig. 39.7. Repetitive spikes in contacts B38, 42, 51 corresponding to the area surrounding the language area. The *arrow* marks the electrographic seizure onset

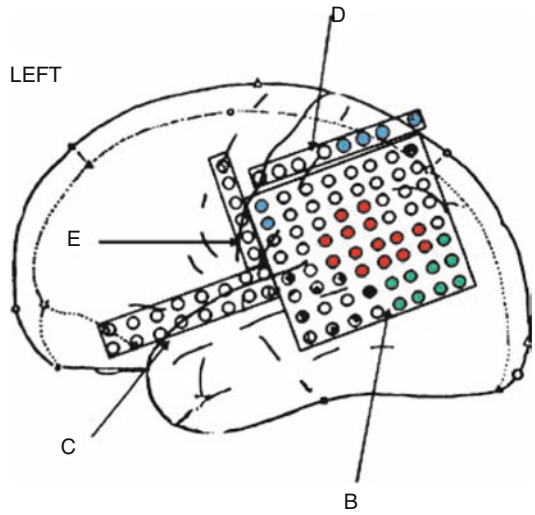

Fig. 39.10 Results of electrical stimulation of the cortex from the patient in Fig. 39.7 with subdural electrodes in the left hemisphere. Key: *red* ictal zone, *green* visual area, *blue* sensitive area, and *black and white* language area

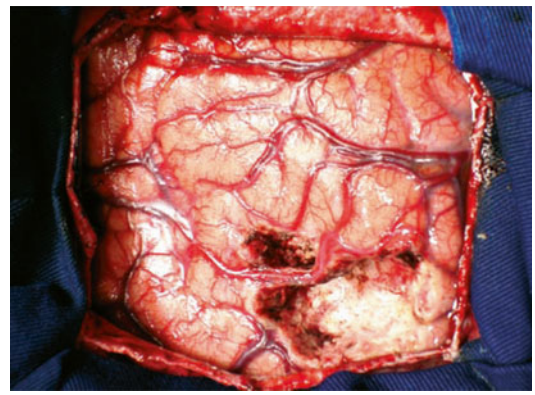

Fig. 39.11 Postresection with preservation of the arterial supply for the adjacent cortex from the patient in Fig. 39.7

invasive VEEG evaluation when subdural electrodes are implanted.

The interictal epileptiform activity can be recorded directly from the cerebral cortex during surgery (electrocorticography) and is considered to be an indispensable technique for defining the irritative zone in the intraoperative evaluation. During the subdural grid implantation, it is useful for better defining the areas of the cerebral cortex to be evaluated. After electrode removal and cortex resection, it is a tool for determining any residual epileptiform activity.

Patients with extratemporal epilepsy who submit to surgical treatment are divided into two major groups: patients with lesional epilepsies and patients with cryptogenic epilepsies whose imaging exams do not identify lesions. Patients with lesional epilepsies have a better prognosis regarding seizure control. In a representative study of 60 patients with extratemporal epilepsy, 61 % of the patients with identified structural lesions were seizure-free over a period of 5 years, as opposed to 20 % of nonlesional patients [60]. In a review of frontal epilepsies from 1987 to 1994, 72 % of the lesional patients had adequate seizure control (Engel I or II), after 5 years, as compared to 40 % of nonlesional patients [29].

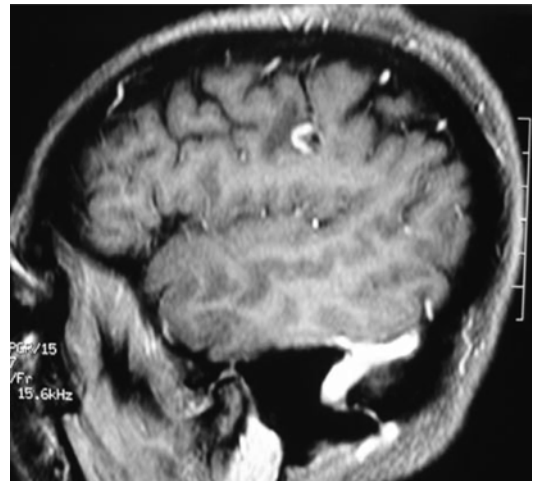

Fig. 39.12 A 35-year-old woman with left frontal granuloma

39.4.1 Lesional Epilepsies

Of patients with refractory extratemporal epilepsies, 50 % present identifiable structural lesions, representing up to 80 % of patients in the surgical series [36]. Focal abnormalities such as tumors, vascular lesions, and cortical abnormalities show different degrees of differentiation from the parenchyma, a fact that influences the surgical programming [25].

Neoplasias such as low-grade astrocytomas, oligodendrogliomas, dysembryoplastic neuroepithelial tumors, and others may often be the origin of epileptic foci (Figs. 39.12, 39.13, and 39.14). Epileptogenic tumors usually grow slowly, occur in young individuals, and involve the gray matter. Epileptogenic control is better when resection of the entire lesion and the marginal area (extended lesionectomy) is possible, with a rate of seizure control of about 80 % [25].

Cavernomas and arteriovenous malformations are vascular lesions commonly associated with epileptic seizures [25]. Lesionectomy with the removal of marginal areas by impregnation with hemosiderin leads to seizure control in 73 % of patients [22].

As diagnostic imaging methods evolve with increasing sensitivity, anomalies of cortical development are becoming increasingly more frequent as the cause of focal epilepsy. The most common developmental anomaly is cortical dysplasia,

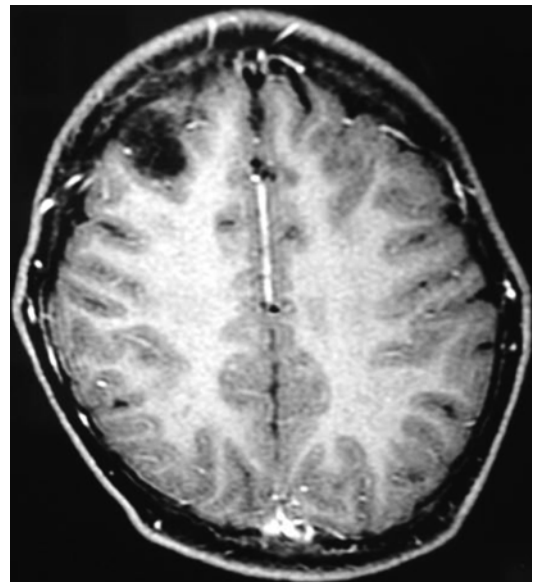

Fig. 39.13 A 13-year-old boy with a right frontal dysembryoplastic neuroepithelial tumor

followed by malformations of the cortical gyri such as polymicrogyria. Developmental lesions are observed more frequently in children, often involving large extensions of the cortex [13]. Cortical dysplasias are associated with a particular pattern of epileptogenicity, with some authors believing that intraoperative electrocorticography is a necessary tool [37]. In large series, 49 % of

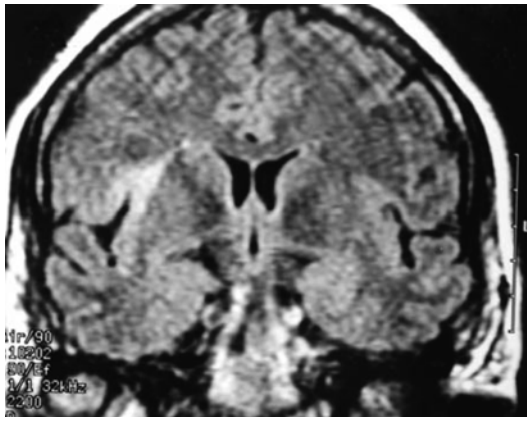

Fig. 39.14 A 51-year-old woman with a right insular dysplasia

the patients with dysplasia became seizure-free, with 58 % having submitted to complete resection and 27 % to incomplete resection. Cortical dysplasia with Taylor's balloon cells was completely removed with a 100 % success rate in a series of 16 patients [41].

39.4.2 Nonlesional Epilepsies

Patients with nonlesional epilepsy represent the greatest challenge for the surgical treatment of epilepsies, with a lower success rate ranging from 20 to 55 % of the patients. The use of invasive preoperative electrocorticography appears to play a fundamental role in this type of patient. A study conducted on 24 patients with epilepsy and normal MRI did not reveal a significant difference in seizure-free evolution in patients submitted to invasive monitoring. However, the use of invasive monitoring was of fundamental importance for the surgical strategy by establishing a clear definition between the epileptogenic zone and the motor and language areas [10].

In a study conducted by Chapman et al. [10], 78 % of patients with frontal epilepsy and a normal MRI received a diagnosis of dysplasia after a postoperative anatomopathological exam.

Among the surgical possibilities for nonlesional patients are neocortical resection and disconnection procedures such as MST and callosotomy. Resection of the cerebral cortex is denoted topectomy, corticectomy, or neocortical resection. The margins of this resection are determined by invasive subdural recordings and by the mapping of eloquent areas. Mapping can be performed by a preoperative methodology with functional MRI and magnetoencephalography in addition to cortical stimulation and somatosensory evoked potential recording using subdural electrodes.

39.5 Disconnective Surgery

The objective of disconnective surgery for the treatment of intractable epilepsies is to isolate or disconnect the epileptogenic area of the ipsilateral hemisphere or to prevent the propagation of the seizure to the contralateral hemisphere. The main types of disconnective surgery used are MST, corpus callosotomy, and hemispherectomy.

39.5.1 Multiple Subpial Transection

MST was developed by Morrel [28, 49] for the treatment of epilepsies originating from a cortex that cannot be resected, such as the motor cortex. The technique is based on the principle of the columnar organization of the cerebral cortex, whereby the fibers belonging to the long descending tracts would not be affected by the section of horizontal segments. The theory that the generation of electroencephalographic and clinical seizures depends on the horizontal propagation of the epileptiform discharge in the cortex, while functional activity such as motor and speech control depends on an intact vertical column of the cortex, is the physiological basis of this technique. MST consists of lesion of the horizontal connections that run between the cortex, which are crucial for the synchronization of neural activity (a key point of the seizures), without involving the fibers of ascending and descending projections responsible for cortical functioning. A small hook-shaped blade is used to cut through the gray matter while leaving the pia and vessels intact. The transections should be performed at right angles along the axis of the gyri at 5-mm

intervals. Invasive recording and electrocorticography can be used to determine the area to be submitted to the procedure, with an intraoperative seizure-free recording being the determinant of the functional final point of the procedure. Most patients present temporary deficits during the postoperative period, with improvement within 2–4 weeks and a return to the previous functional status. The incidence of permanent deficits is about 5 % [27].

MSTs can be used for the treatment of continuous partial epilepsy; focal seizures of the sensory, somatosensory, or visual cortex; resection with evidence of epileptiform activity in the adjacent eloquent areas; Landau-Kleffner syndrome; and Rasmussen encephalitis. The procedure is also used when the extension of resection increases the surgical risk [6].

A meta-analysis of international experience with 53 patients submitted to MST and 158 submitted to cortical resection plus MST reported a successful reduction of seizures in 95 % of the patients who submitted to resection and in 68 % of the patients submitted to MST alone [25]. Another study, however, demonstrated a late increase in the incidence of seizures in up to 19 % of the patients [34].

The efficacy of treatment of patients with Landau-Kleffner syndrome has been promising, with favorable results in 62 % of patients with isolated MST [48].

39.5.2 Corpus Callosotomy

Median section of the corpus callosum is a palliative form of treatment for patients with generalized tonic-clonic, tonic or myoclonic seizures, or seizures with drop attacks refractory to medicamentous treatment. Some authors perform corpus callosotomy in patients with primarily generalized epileptiform discharges or in patients who show secondary bilateral generalization [17]. First introduced by Van Wagenen and Herren in 1939 [53], section of the corpus callosum is performed in order to prevent the bilateral synchrony of a cortical epileptiform activity that may result in seizures with bilateral motor manifestations.

The corpus callosum performs interhemispheric connection, unifying motor function and sensory, visual, and somatosensory perception. The axons that connect the frontal lobes occupy the more rostral portions, whereas the connections of the parietal, temporal, and occipital cortex occupy the more caudal portion. In a feline model of generalized epilepsy, Musgrave and Gloor [30] demonstrated that total section of the corpus callosum and of the anterior commissure leads to the disappearance of the bilateral synchrony of epileptiform discharges of the slow-wave-spike type.

Direct right frontal craniotomy is performed, with the anterior limit located 2 cm from the coronary suture and the medial limit located in the superior sagittal suture, exposing the superior sagittal sinus. An opening of the dura mater is performed toward the superior sagittal sinus until the interhemispheric portion is exposed. The interhemispheric fissure is dissected following the ascending branch of the pericallosal artery, with identification of the cingulate gyrus and of the corpus callosum. The anterior two-thirds or three-quarters of the corpus callosum is then opened longitudinally, while the splenium is left intact because it contains fibers connecting the supplementary motor areas. Complete section of the corpus callosum is reserved for patients whose response to anterior section is unsatisfactory [50]. Phillips and Sakas [40] reported that surgical section of the corpus callosum reduced the frequency of seizures by 70 % in 16 of 20 patients (80 %). Gates and dePaola [18] reported a reduction of generalized tonic-clonic seizures in 50–80 % of their patients.

Feichtinger et al. [14] evaluated the efficacy of anterior callosotomy by radiosurgery in a group of eight patients with intractable drop attacks. One of these patients required radiosurgical callosotomy of the middle third after 17 months and two patients were submitted to partial hemispherotomy. This was an effective and safe noninvasive method for the treatment of these patients.

In addition to the complications inherent to the surgical act, the typical complications of corpus callosotomy are mainly neuropsychological [3]. Varied degrees of disconnection syndrome

characterized by lethargy, apathy, mutism, incontinence, apraxia, and a bilateral Babinski sign may be present during the first postoperative week. There is a direct relationship between symptoms and the extension of section. The more posterior the disconnection, the greater the possibility of neuropsychological alterations.

Total callosotomy should be performed as the initial procedure only in patients whose initial cognitive deficit is so severe that no functional impairment will derive from the institution of disconnection syndrome [25].

39.5.3 Hemispherectomy

The first description of hemispherectomy was reported in 1928 by Walter Dandy [1] for the treatment of a patient with glioblastoma multiforme. Ten years later, Mackenzie described the use of this technique for a patient with epilepsy. In 1950, Krynauw reported the use of hemispherectomy in a patient with hemiplegia since childhood, intractable epilepsy, and behavioral alteration [55].

In 1966, Oppenheimer and Griffith [33] reported a late complication of hemispherectomy consisting of small and repeated bleedings in the subdural space causing obstructive hydrocephalus and superficial hemosiderosis of the central nervous system. In the 1970s, numerous modifications were introduced in the anatomical technique of hemispherectomy in order to prevent late complications. In 1982 Rasmussen [43] introduced the surgical technique of functional hemispherectomy, anatomically incomplete but functionally complete, leaving more brain tissue and considerably reducing late complications. More recently, the hemispherotomy technique of Delalande and the peri-insular hemispherotomy technique of Villemure [57] represent an attempt to obtain complete functional disconnection with a smaller cerebral resection.

This technique is indicated for patients who present severe motor seizures associated with other types of seizures that originate from the same brain hemisphere and who present contralateral hemiparesis, with a nonfunctioning hand and homonymous hemianopsia. Patients with Rasmussen and Sturge-Weber syndrome and hemimegalencephaly are frequent candidates for this type of treatment. Other malformation disorders and cerebral ischemias leading to unilateral cortical atrophy or porencephaly also benefit from this surgery.

Anatomical hemispherectomy was the original technique [54]. Lobar or en bloc resection of the brain hemisphere is performed, with preservation of the base nuclei. Peacock et al. [39] recommend anatomical hemispherectomy for patients with a small lateral ventricle and with dysplastic syndromes, because of the risk of hemorrhage and because of the consistency of brain tissue.

The technique of functional hemispherectomy proposed by Rasmussen [43] consists of temporal lobectomy with amygdalohippocampectomy followed by suprasylvian central corticectomy extending to the fornix. The initial disconnection is performed and then extended by callosotomy and by disconnection of the temporal and parieto-occipital lobes. The frontal lobe is disconnected through a plane corresponding to the wing of the sphenoid bone.

Hemispherotomy, proposed by Delalande in 1992, consists of a disconnection technique above the resection. The lateral ventricle is accessed and callosotomy is performed, followed by transection of the ganglia from the base to the inferomesial frontal region. In 1995, Villemure [57] described peri-insular hemispherotomy. The frontal, parietal, and temporal opercles are resected by means of suprainsular and infrainsular corticectomies, and callosotomy is performed through the lateral ventricle. Disconnection of the frontal lobe on the coronal plane follows the level of the greater wing of the sphenoid bone. After callosotomy, the procedure is continued with subpial aspiration through the fimbria-fornix. Amygdalohippocampectomy is then performed (Figs. 39.15 and 39.16).

The complication of hemispherectomy [56], especially in patients with incomplete motor deficit, is flaccid hemiplegia, which usually improves over a period of months. Preservation of arteries and veins in functional hemispherectomy is important for the prevention of cerebral

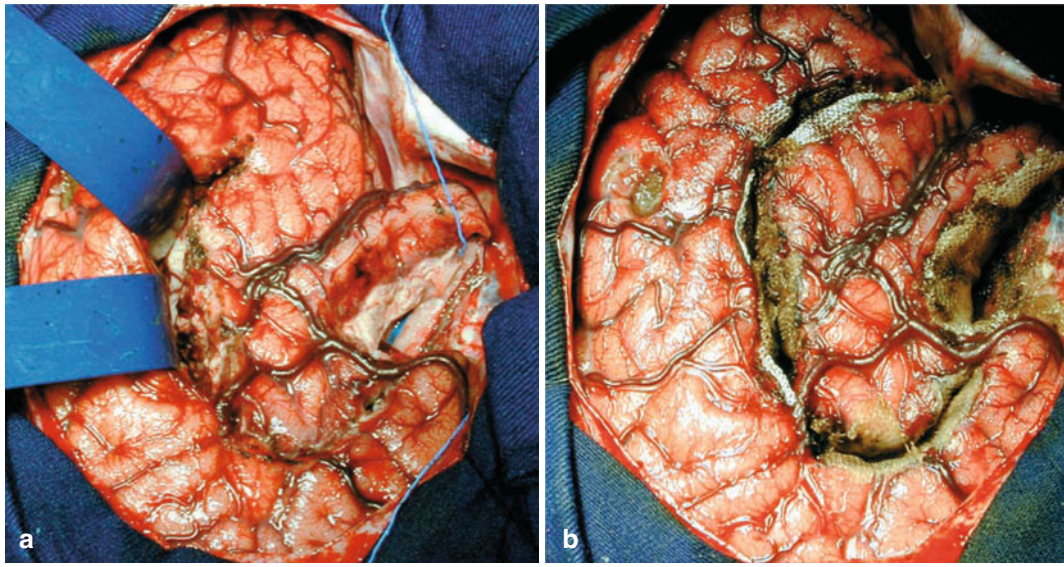

Fig. 39.15 Operative view of a right hemispherotomy. (**a**) Temporal lobe resection and intraventricular exposure. (**b**) Final view, with a small amount of tissue removed

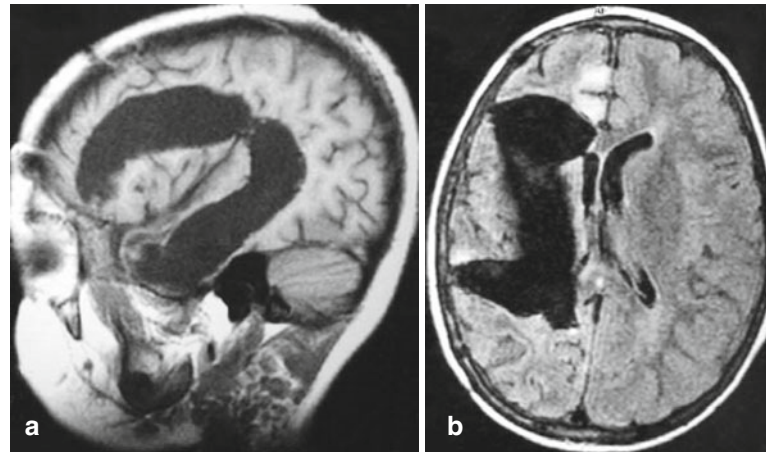

Fig. 39.16 Postoperative, posthemispherectomy MRI. (**a**) Saggital view (**b**) Axial view

infarctions and edema during the immediate postoperative period, as well as late progressive brain atrophy. Hydrocephalus can be an acute complication requiring a temporary or definitive external shunt. Many patients develop aseptic meningitis, which is characterized by low fever, headache, irritability, and lethargy. Infections and hemorrhages are late complications. Hemispherectomy, when properly indicated, provides an excellent result in terms of the control of epileptic seizures, leading to intellectual and behavioral improvement of the patient.

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