Surgical treatment of temporal lobe epilepsy: clinical, radiological, and histopathological findings in 178 patients

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Abstract

The surgical treatment of pharmacoresistant temporal lobe epilepsy is increasing rapidly. The correlation of preoperative MRI, histopathological findings, and postoperative seizure control is reported for 178 patients with chronic medically intractable temporal lobe epilepsy who were operated on between November 1987 and January 1993. Histopathologically there were distinct structural abnormalities in 97.2% of the surgical specimens. Signal abnormalities on MRI were present in 98.7% of patients with neoplastic lesions (n = 79), 76.6% of patients with non-neoplastic focal lesions (n = 55), and 69.2% of patients with Ammon's horn sclerosis (n = 39). Overall, structural abnormalities were detected by MRI in 82.7% of all patients. The mean postoperative follow up period was three years. Some 92% of the patients benefited from surgery: 103 patients (61.7%) were seizure free, 26 (15.5%) had no more than two seizures a year, and 24 (14.4%) showed a reduction of seizure frequency of at least 75%. Fourteen patients (8.4%) had a <75% reduction of seizure frequency. The percentage of patients who were completely free of seizures after operation was 68.5% for patients with neoplastic lesions, 66.7% for Ammon's horn sclerosis, and 54.0% for patients with non-neoplastic focal lesions. By contrast, none of the patients in whom histopathological findings were normal became seizure free postoperatively. The data show that the presence of focal lesions or Ammon's horn sclerosis as determined by histopathological examination is associated with improved postoperative seizure control compared with patients without specific pathological findings. Brain MRI was very sensitive in detecting neoplasms; however, its sensitivity and specificity were limited with respect to non-neoplastic focal lesions and Ammon's horn sclerosis. Improvement of imaging techniques may provide a more precise definition of structural lesions in these cases and facilitate limited surgical resections of the epileptogenic area rather than standardised anatomical resections.

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Temporal lobe epilepsy is the most common form of epilepsy, with a prevalence of around 0.1% in the general population.¹ Despite optimal pharmacotherapy, about 30% of the patients do not become seizure free.² Recently, there has been increased interest in the relation between structural lesions and epileptic seizures. Abnormalities that are often found in association with intractable seizures include low grade tumours,³⁻¹³ non-tumorous disorders,¹⁴⁻¹⁹ and Ammon's horn sclerosis.²⁰⁻²⁴ Moreover, the presence of a "dual pathology"—that is, a focal lesion in association with Ammon's horn sclerosis—has been found in many cases.²⁵⁻²⁷ Magnetic resonance imaging has been found to be valuable for both identification and localisation of various lesions.²⁸⁻³⁸ Most recent reports on temporal lobe epilepsy have been limited to small numbers of patients and predominantly consider specific aspects. Here we provide a comprehensive report on 178 patients with a mean follow up period of three years. The relation between clinical, radiological, and histopathological findings and postoperative seizure control is presented in detail.

Patients and methods

Between November 1977 and January 1993 227 patients underwent surgical treatment for temporal lobe epilepsy at the Department of Neurosurgery, University of Bonn. The patients had a well documented chronic and medically intractable epilepsy lasting for a minimum of two years. For all patients adequate periods of treatment with at least two first line anticonvulsant agents such as carbamazepine, phenytoin, phenobarbitone, or valproic acid were required before referral for presurgical evaluation. Histopathological evaluation of surgical specimens showed uncertain findings in 49 cases. These specimens did not show focal lesions; however, they were not appropriate to allow definite conclusions as to the presence or absence of Ammon's horn sclerosis. Thus these 49 patients were omitted, leaving 178 patients in this series.

All patients underwent continuous video EEG monitoring by cable telemetry with scalp and sphenoidal electrodes. If the site of the seizure origin remained undetermined, bilateral subdural strip electrodes and—more recently—additional stereotactic depth electrodes were used to identify the epileptogenic area. Extraoperative electrocorticographic studies were done in 74 of the 178 patients (41.8%). Of these, 61 patients had subdural

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electrodes only, and 13 had both subdural and depth electrodes. The site and extent of the resection were based mainly on the ictal and interictal EEG pattern and—if present—on the site of a localised structural lesion as determined by MRI. Other information was used to confirm the location of the epileptogenic area and to assess the risks and prognosis of resecting this. The information included a detailed evaluation of seizure history and semiology, neuropsychological studies, psychiatric evaluation, and intracarotid amylorbarbitone testing in most cases. Studies with single proton emission computed tomography (SPECT) have been performed in a subgroup of patients. A detailed analysis will be published elsewhere.³⁹

In all, 168 MRI examinations were available for review. Of these, about 50% were performed at the University of Bonn Medical Centre. The rest were done at various other institutions. At our institution, MRI was carried out with a 1.5 Tesla unit (Philips Gyroscan S15) with at least sagittal T1 weighted images (TR/TE/slice thickness = 500–600 ms/15–25 ms/8–10 mm) and axial proton density weighted and T2 weighted images (TR/TE/slice-thickness = 2000–2500 ms/20–30 and 80–120 ms/6–8 mm). Usually, spin echo scans were performed. T2 weighted gradient echo scans were done only rarely. If a tumour was suspected, additional coronal and axial T1 weighted images with and without gadolinium-DTPA were acquired with similar parameters. If there was prior evidence for a temporal focal lesion, axial images were often acquired with a modified angulation parallel to the long axis of the temporal lobe—that is, about 25° off the orbitomeatal line.

Examinations with MRI from other institutions included a wide range of technical variations. Employing systems of various manufacturers and field strengths, slices were generally 1–2 mm thicker than those obtained at our institution, whereas acquisition of one T1 weighted and one T2 weighted scan in two different planes was standard. Some-

times, no proton density weighted images had been performed. The modified “temporal” orientation for axial images was not used in these examinations. The differences in imaging techniques reflect the fact that there is no standardised protocol for the examination of patients with temporal lobe epilepsy.

All preoperative MRI studies were retrospectively evaluated by a neuroradiologist with no other information available. Findings on MRI were classified as follows: tumour, non-tumorous lesion, signal abnormalities of “uncertain” significance, Ammon’s horn sclerosis, and no detectable abnormality. The term “uncertain” was used with respect to the interpretation of imaging studies if the findings were not conclusive due to insufficient image quality, questionable partial volume effects, or the lack of appropriate sequences or sectional planes. The diagnosis of Ammon’s horn sclerosis was based on the following criteria: increased signal on T2 weighted images or temporomesial atrophy with an enlarged temporal horn of the lateral ventricle. Volumetric measurements of hippocampal size were not performed.

The following surgical procedures were performed: anterior temporal lobectomy (standard or “keyhole”) with hippocampectomy ($n = 144$), anterior temporal lobectomy without hippocampectomy ($n = 8$), extended lesionectomy with hippocampectomy ($n = 9$), extended lesionectomy without hippocampectomy ($n = 15$), and selective amygdalohippocampectomy ($n = 2$) (fig 1). Thirteen patients in whom anterior temporal lobectomy with hippocampectomy was performed had been operated on previously at other institutions for tumours; however, their seizure outcome had not been improved.

All operations were performed under general anaesthesia without intraoperative electrocorticography. In every case, complete removal of a radiologically identified lesion was intended. A standard anterior temporal lobectomy consisted of a 4.5 cm (non-dominant hemisphere) and 4 cm (dominant hemisphere) resection of the superior and middle temporal gyri and a 5.5 cm (non-dominant hemisphere) and 5 cm (dominant hemisphere) resection of the inferior temporal gyrus. This was followed by removal of the amygdala, the parahippocampal uncus, and total resection of the hippocampus and parahippocampal gyrus. If there was adequate memory performance of the non-operated temporal lobe as evaluated by neuropsychometric and intracarotid amylorbarbitone testing, the anterior 3 to 3.5 cm of the parahippocampal gyrus and hippocampus were resected.

In cases with insufficient contralateral memory performance, resection of the hippocampus was limited to 1–2 cm, or the hippocampus was preserved completely. Extended lesionectomy was performed by an anterolateral or posterolateral approach depending on the location of the lesion. In some patients, these procedures were combined with amygdalohippocampectomy. Selective amygdalohippocampectomy was

Figure 1 Schematic presentation of different resective procedures used for this series: standard anterior temporal lobectomy with and without hippocampectomy (A); “key-hole” resection with hippocampectomy (B); extended lesionectomy with and without hippocampectomy (C); and selective amygdalohippocampectomy (D).

performed by the trans-sylvian route as described by Wieser and Yasargil.⁴⁰

All glass slides of operative specimens were reviewed by two neuropathologists. Sections stained with haematoxylin and eosin were available from all cases. In most specimens there were also slides with Nissl stains and combined haematoxylin-eosin-luxol-fast blue stains available. In selected cases additional special stains (elastic van Gieson, reticulin, Bodian) were used. In all tumour cases some or all of the following immunohistochemical reactions were carried out: glial fibrillary acid protein, synaptophysin, neurofilament protein, neuron specific enolase, and S 100 protein. Tumours were classified according to the revised World Health Organisation classification for tumours of the nervous system.⁴⁰ Histological findings were divided into four groups: neoplasm, non-neoplastic focal lesion, Ammon's horn sclerosis, and absence of specific histopathological alterations.

Follow up information, available from 167 patients, was based on regular outpatient visits to hospital at three to 12 month intervals. Postoperative observation time ranged from 12 to 72 months (mean three years). With respect to the postoperative seizure state, patients were assigned to four different outcome classes as described by Engel *et al*³⁰: (a) seizure free or only auras since surgery, (b) rare seizures (≤ 2 per year), (c) reduction of seizure frequency $\geq 75\%$, and (d) unchanged ($< 75\%$ reduction of seizure frequency). Student's *t* test was used for statistical comparison of continuous variables if the distribution was normal, and Wilcoxon's test if it was not. Discrete variables were compared by cross tabulation.

Results

CLINICAL FINDINGS

There were 82 male and 96 female patients with ages ranging from 3 to 64 years and a mean age of 27.7 years. All patients had complex partial seizures, 78 of them with secondary seizure generalisation.

Patients' ages at onset of seizures ranged between 1 and 45 (mean 15.9) years.

Table 1 Main histopathological diagnoses in 178 specimens from patients with temporal lobe epilepsy

Histopathological diagnosis	Patients No (%)
Neoplasm	79 (44.4)
Non-neoplastic lesion	
Glioneuronal hamartia	19 (10.7)
Glioneuronal hamartoma	5 (2.8)
Vascular malformation	14 (7.9)
Old necrosis	3 (1.7)
Abundant neurons in white matter	3 (1.7)
Chronic encephalitis	2 (1.1)
Other	9 (5.0)
Ammon's horn sclerosis	39 (21.9)
Normal	5 (2.8)
Total	178 (100.0)

In some specimens more than one pathological abnormality was present. Only 72 specimens were sufficiently preserved to permit an evaluation as to the presence of Ammon's horn sclerosis. The incidence of Ammon's horn sclerosis in this subgroup was 72.2%.

Duration of epilepsy varied between two and 52 years with a mean of 12.2 years. Patients' mean age at surgery was 26.2 years with neoplastic lesions, 26.5 years with non-neoplastic focal lesions, and 29.2 years with Ammon's horn sclerosis. Mean age at onset of seizures was 14.2 years in patients with neoplastic lesions, 14.7 years in patients with non-neoplastic focal lesions, and 17.9 years in patients with Ammon's horn sclerosis. As determined by Student's *t* test the patients' ages at the time of surgery and the ages at the first onset of seizures were significantly lower in patients with focal lesions than in those without focal lesions ($P < 0.01$). There was considerable overlap between the two groups. No statistically significant differences were present for duration of seizures between any of these groups.

HISTOPATHOLOGICAL FINDINGS

Table 1 gives the results of histopathological examination of the specimens obtained at operation. In 106 patients (56.7%), a definite histopathological statement as to the presence or absence of Ammon's horn sclerosis was impossible. In some cases, segmental neuronal loss and gliosis were obscured by infiltrating tumour or inflammation. More often, however, the portion of the hippocampal formation that was actually submitted for pathological evaluation consisted only of small tissue fragments that did not permit a proper orientation during embedding and a reliable identification of the different segments of Ammon's horn. In many cases, the lack of an appropriate specimen was due to the extensive use of ultrasonic aspiration. Most specimens contained minor and non-specific changes such as focal gliosis of the end folium or other parts of the hippocampal formation, subpial gliosis, or diffuse white matter gliosis irrespective of the presence or absence of a specific focal lesion or classic Ammon's horn sclerosis. As representative specimens of the hippocampus were not available in many cases, it was impossible to give a specific figure for the number of entirely normal samples for the whole series.

The most frequent tumour was a ganglioglioma, and all but two tumours were of low histopathological grade (table 2). Dysembryoplastic neuroepithelial tumours were present in eight patients. This entity has recently been included in the 1993 World Health Organisation (WHO) classification of CNS

Table 2 Histopathological diagnoses in 79 tumours

Diagnosis	Patients No (%)
Ganglioglioma (WHO grade I)	29 (36.7)
Ganglioglioma (WHO grade II)	3 (3.8)
Anaplastic ganglioglioma (WHO grade III)	1 (1.3)
Pilocytic astrocytoma (WHO grade I)	18 (22.7)
Astrocytoma (WHO grade II)	6 (7.6)
Anaplastic astrocytoma (WHO Grade III)	1 (1.3)
Oligodendroglioma (WHO grade II)	10 (12.6)
Oligoastrocytoma (WHO grade II)	1 (1.3)
Pleomorphic xanthoastrocytoma	1 (1.3)
Ependymoid	1 (1.3)
Dysembryoplastic neuroepithelial tumour	8 (10.1)
Total	79 (100.0)

Table 3 MRI diagnoses in 77 histopathologically verified neoplastic lesions

Histopathological diagnosis	MRI diagnosis				
	Tumour No	Non-tumorous lesion No	"Uncertain" No	Normal No	Total No
Ganglioglioma (WHO grade I)	21	1	5	1	28
Ganglioglioma (WHO grade II)	3	—	—	—	3
Ganglioglioma (WHO grade III)	1	—	—	—	1
Astrocytoma (WHO grade I)	16	1	—	—	17
Astrocytoma (WHO grade II)	5	—	1	—	6
Astrocytoma (WHO grade III)	1	—	—	—	1
Oligodendroglioma (WHO grade II)	10	—	—	—	10
Oligoastrocytoma (WHO grade II)	1	—	—	—	1
Pleomorphic xanthoastrocytoma	1	—	—	—	1
Dysembryoplastic neuroepithelial tumour	7	—	1	—	8
Epidermoid	1	—	—	—	1
Total	67	2	7	1	77

tumours⁴¹ and its detailed histopathology remains to be determined. In the present series, two tumours that had been previously designated as gangliogliomas were reclassified as dysembryoplastic neuroepithelial tumours. Among non-neoplastic focal lesions, glioneuronal hamartias and cavernomas were most often found (table 1). Ammon's horn sclerosis was the only abnormality in 39 cases (17.2%). Correlation of focal lesions and Ammon's horn sclerosis showed that Ammon's horn sclerosis was significantly more frequent in patients without focal lesions. There was a small group, however (25.0%), of patients with Ammon's horn sclerosis and focal lesions ("dual pathology"). Only five patients had neither Ammon's horn sclerosis nor focal lesions. More detailed descriptions on the histopathological findings have recently been published elsewhere.^{19 42}

MRI FINDINGS

Brain MRI showed structural abnormalities in 139 of 168 cases (82.7%). There were signal abnormalities on MRI in 76 of the 77 histopathologically verified tumours (98.7%), and in 67 cases (87.0%) the MRI diagnosis

was that of a tumour. In seven tumour cases MRI showed signal abnormalities of uncertain relevance, and in one case no signal abnormality was found. In six of these eight cases, histopathological examination showed a circumscribed ganglioglioma (table 3).

Of 47 histopathologically verified non-neoplastic focal lesions, MRI was abnormal in 36 cases (76.6%). Cavernomas and heterotopias were correctly assessed in every case, whereas hamartias and hamartomas were often missed or interpreted as neoplasms. In 11 cases (23.4%), MRI was normal. In 10 of these patients, histopathological examinations showed microscopic glioneuronal hamartias (table 4).

Of the 39 patients with histopathologically verified Ammon's horn sclerosis and no focal lesion, MRI was abnormal in 27 cases (69.2%). In 21 of them (53.8%), Ammon's horn sclerosis was correctly diagnosed, whereas in two cases a tumour was suspected due to high signal intensity. All five patients with inconspicuous histopathological findings had normal MRI (table 5). Figures 2–5 are representative MRI findings from our patients.

FOLLOW UP

There was no operative or postoperative mortality. The postoperative course was complicated in 18 of 178 patients (10.1%). In particular, six patients had deep wound infection and two had meningitis. Neurological impairment was encountered in seven patients: aphasia (one), third nerve paresis (two), hemianopsia (one), and hemiparesis (three). Deep vein thrombosis was found in two cases and peripheral nerve deficits due to inadequate positioning on the operating table in one case. Almost all of these complications resolved completely without further sequelae. Permanent morbidity occurred in three patients (1.7%) and included hemiparesis in two cases, and hemianopsia in one case.

Table 6 shows the outcome of patients with respect to seizure control. In total, 91.6% of the patients were seizure free or had a worthwhile improvement after surgery, whereas 8.4% had no appreciable reduction in seizure frequency or were unchanged. We did not encounter any significant differences in seizure outcome with respect to the side of operation (right *v* left). The most favourable

Table 4 MRI diagnoses in 47 histopathologically verified non-neoplastic focal lesions

Histopathological diagnosis	MRI diagnosis				
	Non-tumorous lesion No	Tumour No	"Uncertain" No	Normal No	Total No
Hamartia	—	5	4	10	19
Hamartoma	—	3	2	—	5
Cavernoma	11	—	—	—	11
AV malformation	2	—	1	—	3
Necrosis	—	1	1	1	3
Heterotopia	3	—	—	—	3
Abscess wall	—	1	—	—	1
Encephalitis	—	1	—	—	1
Gliosis	—	1	—	—	1
Total	16	12	8	11	47

Table 5 MRI diagnoses in 44 patients with and without definite Ammon's horn sclerosis (AHS)

Histopathological diagnosis	MRI diagnosis				
	AHS No	Tumour No	"Uncertain" No	Normal No	Total No
AHS	21	2	4	12	39
No AHS	—	—	—	5	5
Total	21	2	4	17	44

AHS = Ammon's horn sclerosis.

Figure 2 Ammon's horn sclerosis: MRI with T2 weighted temporally oriented image (left) and T1 weighted coronal image (inversion recovery) (right) shows increased signal (T2 weighted) and right hippocampal atrophy (T1 weighted) (arrows).

results were in patients with neoplastic lesions and Ammon's horn sclerosis. With these histopathological diagnoses, 68.5% and 66.7% of patients were seizure free. The ratio of seizure free patients with non-neoplastic focal lesions was somewhat lower (54.0%). These differences were statistically significant ($P < 0.05$). None of the five patients in whom there were no specific histopathological findings became seizure free postoperatively (table 7). The differences in seizure outcome between patients without specific histopathological alterations and those with the diagnosis of a focal lesion or Ammon's horn sclerosis were highly significant ($P < 0.001$).

Discussion

The surgical management of epilepsy involves the identification of the epileptogenic cortex

and its excision as completely as possible.^{36 43} Despite complex diagnostic tools the definition of the epileptogenic area remains hypothetical. It is generally accepted, however, that structural abnormalities play a significant part in epileptogenesis and that altered brain has to be removed completely to achieve seizure control. Brain MRI has proved to be highly sensitive both for detection and localisation of structural lesions.^{3 15 28 32 38 44-47} This study of 178 patients treated surgically for temporal lobe epilepsy was designed to evaluate the sensitivity and specificity of MRI for identifying structural abnormalities and to define the relevance of histopathological findings with respect to seizure control.

In our series, histopathological examination showed definite structural abnormalities in 173 of 178 specimens (97.2%). Forty nine patients who did not show a focal lesion and whom histopathological findings did not allow definite conclusions as to the presence or absence of Ammon's horn sclerosis were excluded from the total series of patients operated on during the respective period. Neoplastic lesions occurred most often (44.4%), and all but two tumours were histopathologically benign. Among non-neoplastic focal lesions (30.9%), hamartias and cavernomas were predominant. The relatively low rate of definite Ammon's horn sclerosis (21.9%) reflects the fact that many specimens submitted for pathological evaluation consisted only of small tissue fragments that did not allow a reliable identification of the different segments of Ammon's horn. In many cases the lack of an appropriate specimen was due to the extensive use of ultrasonic aspiration. Regarding only specimens in which the hippocampal formation was well preserved ($n = 72$), 72.2% showed Ammon's horn sclerosis. This number includes coincidence of Ammon's horn sclerosis with focal lesions as a dual pathology (25.0% of focal lesions). Thus Ammon's horn sclerosis represents the most

Figure 3 Cavernoma. There is a right temporomesial lesion with central hyperintensity and peripheral signal void typical for cavernoma on axial T1 weighted MRI. Note enlarged tip of inferior horn.

Figure 4 Glioneuronal hamartia. MRI with four coronal T2 weighted slices shows a partly cystic lesion with pronounced cortical and subcortical hyperintensity in the right mesial temporal lobe.

important structural abnormality in temporal lobe epilepsy, whereas low grade gliomas and non-neoplastic focal lesions are found with a lower frequency.^{12 23 24 26 47 48} The fact that almost all lesions found in our patients were histopathologically benign corresponds well

with the usually long history of seizures, which was similar in patients with and without lesions, although the age at time of surgery and the age at the first onset of seizures was significantly lower in patients with focal lesions than in those without focal lesions.

Figure 5 Ganglioglioma (WHO grade I). Proton density (left) and T2 weighted (right) axial MRI illustrate a minimally space occupying lesion of the left uncus with pronounced hyperintensity in both images.

Table 6 Seizure outcome in 167 patients divided into four classes related to the operation side

Outcome class	Patients		
	Right No (%)	Left No (%)	Total No (%)
I	59 (59.6)	44 (64.7)	103 (61.7)
II	16 (16.2)	10 (14.7)	26 (15.5)
III	15 (15.1)	9 (13.2)	24 (14.4)
IV	9 (9.1)	5 (7.4)	14 (8.4)
Total	99 (100.0)	68 (100.0)	167 (100.0)

The high anatomical resolution and the lack of artifacts from overlying bone have been stressed as advantages of MRI over CT; MRI is more sensitive in detecting small structural lesions than CT with the exception of small areas of calcification for which CT is more sensitive.^{13,46,49-52} In view of these findings, MRI is used in most institutions including our own as the initial imaging procedure. Abnormalities are found on MRI in 40% to 70% of patients with temporal lobe epilepsy,^{28,48} and 20% to 25% of these abnormalities are missed by CT.^{46,50-52} In our series, MRI showed structural abnormalities in 82.7%. Sensitivity and specificity of MRI were highest in tumours (98.7% and 87.0%). In non-tumorous lesions, MRI was abnormal in 36 of 47 cases (76.6%); however, only 16 of these lesions (34.0%) were recognised to be non-tumorous. It is remarkable that none of the hamartias and hamartomas were recognised as lesions by MRI. This may be in part due to the small size and variable distribution pattern of these lesions. In some patients, however, the glioneuronal malformations were of considerable size and were therefore classified as probable neoplasms based on MRI criteria. Special attention has been paid to the sensitivity of MRI for detection of Ammon's horn sclerosis. By contrast with Sperling *et al.*,³⁸ who did not find MRI abnormalities in patients with Ammon's horn sclerosis, others have shown abnormal signals in 50% to 80%.^{2,16,24} Even severity of pathological changes seems to correlate with the relative intensity of the abnormal signals.^{16,20,21} In our series, MRI was abnormal in 69.2% and Ammon's horn sclerosis was correctly diagnosed in 53.8%. These abnormalities were best seen on T2 weighted and proton density weighted images in the coronal plane. Abnormal high intensity signal in two of our cases gave rise to the diagnosis of a tumour. In 12 cases, MRI was inconspic-

uous; however, in all of these cases the quality of the images was unsatisfactory. Volumetric or signalometric measurements of the hippocampus may represent a valuable diagnostic tool for demonstration and quantification of hippocampal sclerosis.^{34,53,54} These measurements have not been performed at our institution so far.

With respect to seizure outcome, about 92% of our patients benefited from surgery. These results correspond favourably with those reported from other centres.^{30,55-57} We did not encounter significant differences in seizure outcome comparing the side of surgery (right *v* left). Patients with Ammon's horn sclerosis or focal lesions had a significantly better outcome than patients without specific histopathological findings. It seems to be remarkable that none of the patients in whom specific structural changes were absent became seizure free postoperatively. This indicates that histopathological evaluation provides prognostically relevant information.

Controversy exists with respect to the surgical approach in temporal lobe epilepsy and intraoperative techniques. At some centres the indication for surgery is based on the location of interictal events as determined by scalp EEG, and the resection is carried out under local anaesthesia and tailored to the results of intraoperative electrocorticography (ECoG) and functional mapping.⁵⁸⁻⁶⁰ Other centres base the selection of surgical candidates on extraoperative recordings of seizure onset through depth electrodes. This is followed by an anatomically standardised resection that may be either a standard anterior temporal lobectomy with resection of both mesial and lateral structures⁶¹ or a more limited resection such as a selective amygdalohippocampectomy.⁴⁰ In the present series, we have combined features of these two opposite approaches. The definition of the epileptogenic area was based on extraoperative evaluation of the seizure focus. In earlier non-lesional cases, resection of lateral structures was a standardised removal of the anterior 4 to 5 cm of the temporal lobe. More recently, we have limited and individualised the lateral resections according to the patient's EEG and functional localisation results. In our experience, combined subdural and hippocampal depth electrodes have proved to be valuable to distinguish the foci of mesial and lateral seizures. Obviously, no single approach to resective surgery is suitable for all patients. Although in most cases the location of a structural lesion roughly indicates the epileptogenic area the relation is not absolute and the extent of resection should be tailored to extraoperative electrocorticographic findings and maximal interictal epileptiform activity.

In conclusion, detailed histopathological evaluation of temporal lobe epilepsies emphasises the role of structural abnormalities for epileptogenesis and allows important prognostic conclusions. Magnetic resonance imaging is reliable in identifying tumorous lesions. Imaging techniques need further improvement,

Table 7 Seizure outcome related to histopathological diagnoses

Outcome class	Histopathological diagnosis				Total No (%)
	Neoplasm No (%)	Non-neoplastic lesion No (%)	Ammon's horn sclerosis No (%)	Normal No (%)	
I	50 (68.5)	27 (54.0)	26 (66.7)	— (—)	103 (61.7)
II	13 (17.8)	7 (14.0)	6 (15.4)	— (—)	26 (15.5)
III	9 (12.3)	8 (16.0)	5 (12.8)	2 (40.0)	24 (14.4)
IV	1 (1.4)	8 (16.0)	2 (5.1)	3 (60.0)	14 (8.4)
Total	73 (100.0)	50 (100.0)	39 (100.0)	5 (100.0)	167 (100.0)

however, for detection of non-tumorous focal lesions and Ammon's horn sclerosis. Further improvement of morphological and functional imaging techniques and refined electrophysiological examinations can be expected to allow a more precise preoperative definition of the epileptogenic area. This will further facilitate the use of tailored resections instead of classic standardised operations.

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