PAPER

Temporal lobe epilepsy surgery: different surgical strategies after a non-invasive diagnostic protocol

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J Neurol Neurosurg Psychiatry 2005;**76**:815–824. doi: 10.1136/jnnp.2004.044016

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Received 21 April 2004
Revised version received 27 August 2004
Accepted 22 September 2004

Aim: To test a non-invasive presurgical protocol for temporal lobe epilepsy (TLE) based on ''anatomo–electro–clinical correlations''.
Methods: All consecutive patients with suspected TLE and seizure history <2 years were entered into the protocol, which included video-electroencephalographic (EEG) monitoring and magnetic resonance imaging (MRI). Three different TLE subsyndromes (mesial, lateral, mesiolateral) were identified by combined anatomical, electrical, and clinical criteria. ''Tailored'' surgery for each subsyndrome was offered. Patients with seizure history <2 years, MRI evidence of temporal mass lesion, and concordant interictal EEG and clinical data bypassed video-EEG monitoring and were directly scheduled for surgery.
Results: Lesionectomy was performed without video-EEG recording in 11 patients with tumorous TLE. Of 146 patients studied with video-EEG, 133 received a TLE diagnosis. Four were excluded for neuropsychological risks, eight refused surgery, and 121 underwent surgery. Of 132 consecutive patients who underwent surgery, 101 had at least one year of follow up. They were divided into a ''hippocampal sclerosis/cryptogenic'' group (n = 57) and a ''tumours/cortical organisation disorders'' group (n = 44). In the first group, extensive temporal lobectomy (ETL) was performed in 40 patients, anteromesial temporal lobectomy (AMTL) in 17 patients. At follow up, 47 patients were seizure free. In the second group, lesionectomy plus ETL was performed in 23 patients, lesionectomy plus AMTL in six patients, and lesionectomy alone in 15 patients. Thirty nine patients were seizure free.
Conclusions: These findings suggest that different TLE subsyndromes can be identified accurately using non-invasive anatomo–electro–clinical data and can be treated effectively and safely with tailored surgery.

Drug resistant focal epilepsy is responsible for high social and economic costs in industrialised countries.[1]

Approximately 60% of all patients with epilepsy suffer from focal epilepsy, and in one third of these patients, most of them affected by temporal lobe epilepsy (TLE), seizures are not adequately controlled with antiepileptic drugs (AED).[2] Surgery is currently accepted as an effective and safe therapeutic approach in drug resistant epilepsy, particularly in TLE, where patients become ''seizure free'' in 70–90% of cases.[3] However, surgery for epilepsy remains an underused, ''last resort'' treatment because only a small proportion of patients affected by surgically remediable epilepsies undergo this intervention. In a recent editorial,[4] Engel reported fear of morbidity and confidence in new AEDs or in vagal nerve stimulation as factors that discourage patients and their physicians from surgery, which lacks sufficient data from randomised controlled trials. However, a recent randomised controlled trial of TLE surgery found it to be superior to prolonged medical treatment in terms of efficacy and safety.[5] Moreover, although today there is agreement that TLE can be diagnosed in most patients without invasive tests,[6–8] there is no consensus between different epilepsy surgery groups regarding the optimal use of non-invasive procedures.

The aim of an ideal non-invasive presurgical protocol in TLE surgery should be to: (1) identify the epileptogenic zone and consequently to identify potential candidates for intracranial investigations—that is, some patients affected by lateral TLE; (2) minimise the cost of human and technological resources; (3) reduce the time of diagnostic evaluation, so that surgery can be offered to more patients. In our centre, we implemented a non-invasive presurgical protocol for TLE diagnosis (TLE-Prot), based on ''anatomo–electro–clinical correlations'', inspired by methodological principles first proposed by Bancaud and Talairach,[9] and later developed by their successors.[10–12] This protocol allows different types of tailored resection to be offered for TLE subtypes.[13]

We report the results of the presurgical evaluation and the outcome of epilepsy surgery in 101 consecutive patients with TLE, diagnosed by our protocol, who have been followed for at least one year after surgery.

PATIENTS AND METHODS

Patient population

Between September 1999 and January 2003, 101 consecutive patients with drug resistant TLE who underwent surgery and who had a follow up of at least one year were entered into our study. They were from a series of patients (331 patients: 184 males, 147 females; mean age, 30.3 years; SD, 10.3; range, 6–62; mean age of epilepsy onset, 13.1 years; mean epilepsy duration, 16.2; SD, 10.2; range, 1–45) with medically refractory partial epilepsy for more than one year, referred to the epilepsy surgery unit of IRCCS NEUROMED, Pozzilli, Italy. All operated patients had undergone adequate trials of at least two first line and two add on drugs from among the new AEDs.

Abbreviations: AED, antiepileptic drugs; AMTL, anteromesial temporal lobectomy; CI, confidence interval; DNET, dysembrioneuroepithelioma; EEG, electroencephalographic; ETL, extensive temporal lobectomy; ETLE, invasive presurgical evaluation; MRI, magnetic resonance imaging; MTS, mesial temporal sclerosis; Prot, protocol; TLE, temporal lobe epilepsy

Presurgical diagnostic protocol

In our unit, the presurgical evaluation consists of a multilevel and individualised investigation characterised by non-invasive and invasive procedures. The invasive procedures (ETLE-Prot) are not the focus of our present study and will not be discussed.

First diagnostic level

All patients referred for surgery had a one day admission to the hospital to collect medical history data and perform preliminary neuroradiological and electroencephalographic (EEG) studies.

Medical history

A detailed medical history was obtained from all patients as a first step. Particular attention was paid to recognising the symptoms/signs at seizure onset and auras, because it is largely accepted in the literature that the initial seizure semiology usually provides valuable information about the seizure onset zone.[14–16] A full clinical general and neurological examination was carried out in all patients.

EEG monitoring

The patients were monitored for 24 hours. Three 30 minute interictal EEG ''standard'' samples in the awake state, including hyperventilation and photic stimulation, and all sleep recordings were evaluated to assess the presence of background abnormalities, interictal slow activity, and epileptiform activity (focal (over one to three channels), regional (over three or more channels), hemispheric (over all channels of one side), and diffuse (over all channels of both sides)).[17]

Neuroradiological evaluation

The patients underwent brain 1.5 Tesla magnetic resonance imaging (MRI) examination and, in selected cases, brain computed tomography scans. The MRI scans were reviewed by a neuroradiologist, experienced in the field of epilepsy, masked to the clinical and outcome data. At review, MRI data were classified as follows: mesial temporal sclerosis (MTS) or lesion (low grade tumours, dysembrioneuroepithelioma (DNET), other tumour, dysplasia, or other). The presence of MTS was evaluated qualitatively by visual inspection of MRI: atrophy (on T1 weighted sequences) and increased mesial temporal signal intensity (on T2 weighted and fluid attenuated inversion recovery sequences) were considered markers of MTS. Volumetry was not performed.[18 19]

Second diagnostic level

The patients admitted to the second diagnostic level were evaluated by longterm video-EEG monitoring. They also underwent a comprehensive neuropsychological and psychiatric assessment, the results of which are beyond the scope of this paper and will be reported elsewhere.

The video-EEG recording technique[20] was performed with collodion fixed scalp electrodes (16 EEG channels, positioned according to international 10–20 system, and one electrocardiographic channel to monitor ictal heart rate findings).[21] All patients admitted to video-EEG monitoring had at least one seizure recorded.

Ictal EEG findings

We categorised ictal events as seizures with subjective phenomena only (auras) and seizures with symptoms and/or signs, with or without loss of contact (simple partial or complex partial seizures).

Each ictal event was correlated with the corresponding EEG changes, when present.

According to the site, we distinguished between focal, regional, hemispheric, or diffuse discharges.

According to the EEG seizure pattern appearance and course, ictal EEG changes were classified into ictal onset pattern (first sudden change of frequency with attenuation or appearance of a new rhythm), ictal core pattern, late patterns, and post ictal pattern.

In patients with TLE, we used a classification of EEG ictal core patterns according to well known studies[22–24] concerning ictal scalp–intracranial recordings, which showed a strict correlation between ictal scalp morphology of discharge and different temporal lobe structures involved at seizure onset, as follows:

- Type 1: antero–temporal 5–9 Hz discharge, associated with a highly probable onset in the mesial temporal structures.
- Type 2: temporal 2–4 Hz discharge, associated with a highly probable onset in the lateral temporal structures.

EEG ictal onset in extratemporal regions excluded the patients from the TLE-Prot.

Ictal clinical findings

In selecting the patients with TLE, ictal onset semiology characterised by focal motor, sensory, visual phenomena, or complex motor manifestations (forced head turning or hypermotor behaviour) suggesting a non-temporal lobe seizure onset, led to exclusion from the TLE-Prot.

In implementing the TLE diagnostic grid, despite the fact that no ictal behaviour is specific for temporal lobe seizures, we considered some initial symptoms/signs as highly suggestive of temporal seizures and classified them into a mesial or lateral cluster (table 1).

TLE protocol (fig 1)

The first diagnostic step in selecting patients to admit to the TLE-Prot was the evaluation of the MRI examination. If MRI showed an extratemporal lesion the patient was scheduled for the ETLE-Prot, whereas in all other cases the patient was admitted to the TLE-Prot (if other investigations performed in the first diagnostic level consistently pointed to TLE).

(A) If MRI showed a temporal tumour and seizure history was one to two years, the proposal of a lesionectomy

Table 1 Grid of anatomical, EEG, and clinical criteria used for the identification of TLE subsyndromes

	Mesial cluster	Lateral cluster
Anatomical criteria	Mesial temporal sclerosis or hippocampal atrophy and/or medial structural lesion	Focal atrophy of temporal lateral neocortex and/or lateral structural lesion
Ictal EEG criteria	5–9 Hz discharge, well localised to temporal regions	2–5 Hz ictal discharge well localised to temporal regions
Ictal clinical criteria	Rising epigastric aura, isolated or associated with other vegetative symptoms	Psychic/experiential aura
	Early oroalimentary automatisms (''machonnement'')	Auditive or vestibular symptoms
	Dystonia of contralateral superior limb	Staring without oroalimentary automatisms

EEG, electroencephalographic; TLE, temporal lobe epilepsy.

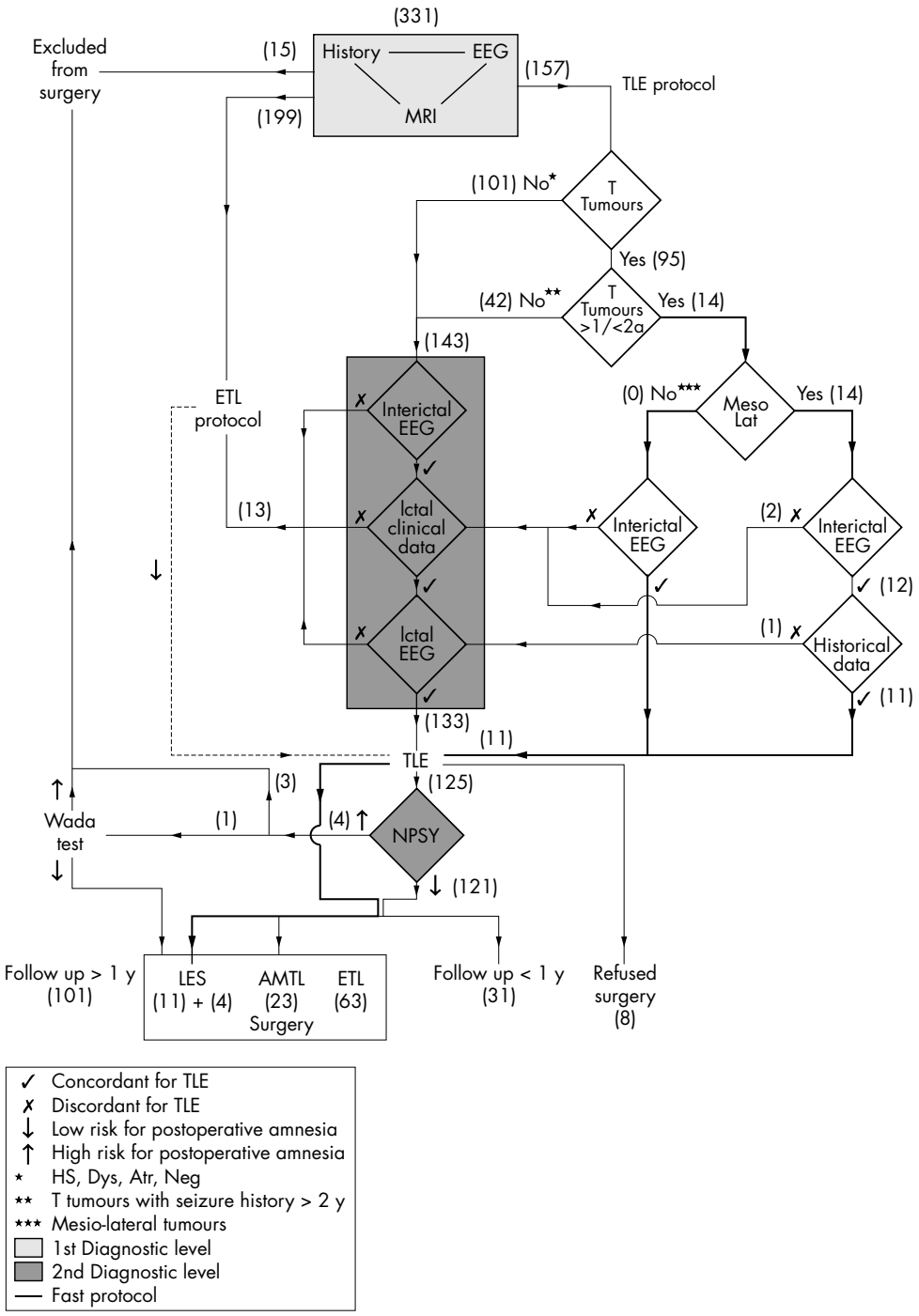

Figure 1 Decisional algorithm for the diagnosis of TLE and surgical strategy. AMTL, anteromesial temporal lobectomy; Atr, atrophy; Dys, dysplasia; ET, extratemporal; ETL, extensive temporal lobectomy; HS, hippocampal sclerosis; I/Ictal, interictal; INV, invasive investigations; L, lateral; LES, lesionectomy; M, mesial; NPSY, neuropsycological assessment; TLE, temporal lobe epilepsy.

was evaluated without admission to level 2 diagnostic procedures: when the tumour involved mesiolateral structures and interictal EEG was concordant, and when the tumour involved mesial or lateral structures only and both interictal EEG and the patient and eyewitness history clinical data were concordant, a diagnosis of TLE was made and lesionectomy was offered (fast TLE-Prot); if interictal EEG and, in mesial or lateral tumours, clinical data were discordant, the patients were scheduled for the second diagnostic level.

(B) If MRI showed a temporal tumour and the seizure history was longer than two years, and in all other cases but extratemporal lesions, the patient was admitted to level 2 diagnostic procedures and underwent video-EEG. If both interictal and ictal electro–clinical data revealed a diagnosis of TLE, surgery was offered and the operation was individualised according to the scheme for the identification of individualised TLE surgery. If interictal EEG and/or ictal electroclinical data were discordant, the patient was scheduled for the ETLE-Prot.

Table 2 Results of investigations and postoperative outcome in 57 patients with hippocampal sclerosis

Patient	Sex	Age	Risk factors	Epilepsy onset	Epilepsy duration	MRI	Ictal clinical symptoms/ signs	Slow I/ictal EEG	Epileptiform I/ictal EEG	Ictal EEG onset	Ictal EEG pattern	Epileptogenic zone	Surgery	Histopathology	Seizure outcome	Follow up (months)
1	M	44	PA+FC	16	28	L HS	Res+Veg+O-Aut+Esp	T Focal	T Focal	Focal	2	L Mes Lat	L ETL	HS	2	59
2	F	40	FC	25	15	R HS	Res+Dys+O-Aut	T Focal	T Focal	Focal	1	R Mes	R AMTL	HS	1a	39
3	M	28	FC	4	24	R HS	Staring+O-Aut	–	BT Focal	Focal	1	R Mes Lat	R ETL	HS	2	25
4	M	40	PA	15	25	L Lat atrophy	Res+Aud+O-Aut	T Reg	T Reg	Focal	2	L Mes Lat	L ETL	HS	1b	49
5	M	35	FC	6	29	L HS	Veg+O-Aut	T Reg	T Focal	Focal	2	L Mes Lat	L ETL	HS	2	19
6	F	32	FC	20	12	L HS	Res+Veg+Esp+Staring	T Focal	BT Focal	Focal	2	L Mes Lat	L ETL	HS	1b	14
7	M	24	FC	18	6	L HS	Veg+Esp	–	T Focal	Focal	2	L Mes Lat	L ETL	HS	1a	19
8	M	47	FC	6	41	L HS	Res+Veg+Staring+O-Aut	T Reg	T Focal	Reg	2	L Mes Lat	L ETL	HS	1a	31
9	F	12	FC	6	6	L HS	Res+O-Aut	T Focal	T Focal	Focal	2	L Mes Lat	L ETL	HS	1a	33
10	M	38	FC	12	26	R HS	Res+O-Aut	T Reg	BT Focal	Focal	1	R Mes	R AMTL	HS	1a	36
11	M	21	PA+FC	6	15	L HS	Res+Dys+O-Aut	T Focal	T Focal	Focal	1	L Mes	L AMTL	HS	1a	24
12	F	32	FC	6	26	L HS	Res+Veg+Esp+O-Aut	T Reg	BT Focal	Reg	2	L Mes Lat	L ETL	HS	2	12
13	M	31	FC	14	17	R HS	Res+Veg+O-Aut	T Reg	T Focal	Focal	1	R Mes	R AMTL	HS	1a	34
14	M	53	FC	43	10	L HS	O-Aut	T Focal	T Focal	Focal	1	L Mes	L AMTL	HS	1a	13
15	M	29	FC	13	16	R HS	Res+Veg+Esp+Aud+ Staring+O-Aut	T Reg	–	Focal	2	R Mes Lat	R ETL	HS	1b	32
16	F	35	FC	4	31	L HS	Res+Veg+O-Aut	T Reg	T Focal	Focal	2	L Mes Lat	L ETL	HS	1a	39
17	M	24	PA	17	7	R HS	Res+Veg+O-Aut	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1a	29
18	M	41	FC	6	35	L HS	Esp+Res+O-Aut	T Focal	T Focal	Focal	1	L Mes Lat	L ETL	HS	1a	13
19	F	19	FC	7	12	L HS	Res+Aud+Esp	T Focal	T Focal	Focal	2	L Mes Lat	L ETL	HS	1a	35
20	M	26	–	8	18	L HS	Res+O-Aut+Dys	T Focal	T Focal	Focal	1	R Mes	R AMTL	HS	1a	14
21	F	21	FC	8	13	R HS	Res+O-Aut	BT Focal	BT Focal	Focal	1	R Mes	L AMTL	HS	1a	48
22	F	26	FC	14	12	L HS	Res+Veg+Dys+O-Aut	BT Focal	T Focal	Focal	1	L Mes	R AMTL	HS	1a	28
23	M	53	FC	16	37	R HS	Res+Veg+O-Aut	T Focal	T Focal	Focal	1	R Mes	R AMTL	HS	1a	40
24	M	46	PA	5	41	R Mes Lat atrophy	Esp+Staring+O-Aut	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1a	12
25	M	28	–	8	20	L HS	Res+Veg+Esp+Staring	T Focal	T Focal	Focal	1	L Mes Lat	L ETL	HS	1a	19
26	M	22	PA	4	18	L HS	Esp	T Focal	BT Focal	Focal	2	L Mes Lat	L ETL	HS	1a	24
27	F	31	FC	16	15	R HS	Res+Veg+Esp+O-Aut	T Focal	T Focal	Focal	1	R Mes Lat	R ETL	HS	1a	33
28	F	17	FC	6	11	R HS	Res	T Focal	BT Focal	Focal	1	R Mes	R AMTL	HS	1a	35
29	F	37	PA	4	33	L Lat atrophy	Staring+O-Aut	BT Focal	BT Focal	Focal	2	L Mes Lat	L ETL	HS	3	13
30	M	42	FC	10	32	R Lat atrophy	Esp+O-Aut	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	2	16
31	F	33	FC	8	25	R HS	Res+O-Aut	BT Focal	BT Focal	Focal	1	R Mes	R AMTL	HS	1a	30
32	F	33	PA+FC	4	29	L HS+Lat atrophy	Res+Dys	T Reg	T Reg	Reg	2	L Mes Lat	L ETL	HS	1a	13
33	M	51	–	11	40	R HS	Res+Veg+O-Aut	T Focal	T Focal	Focal	1	R Mes	R AMTL	HS	1a	23
34	M	34	FC	7	27	L HS	Res+O-Aut+Staring	BT Focal	BT Focal	Focal	1	L Mes Lat	L ETL	HS	1a	49
35	F	29	–	27	2	L HS	Res+Veg+Esp+Staring+ O-Aut	BT Focal	BT Focal	Focal	2	L Mes Lat	L ETL	HS	4	14
36	F	43	FC	14	29	R HS	Res+Veg+O-Aut+Dys	T Focal	BT Focal	Focal	2	R Mes Lat	R ETL	HS	1a	28
37	M	21	FC	4	17	R HS	Res+Veg+O-Aut	T Reg	T Focal	Focal	1	R Mes	R AMTL	HS	2	60
38	F	31	FC	6	25	R HS	Res+O-Aut+Dys	T Focal	T Focal	Focal	1	R Mes	R AMTL	HS	1a	24
39	F	41	–	23	18	R HS	Res+Staring+O-Aut	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1a	14
40	F	36	FC	4	32	R HS+Lat atrophy	Esp+Staring+O-Aut+Dys	BT Reg	BT Reg	Focal	1	R Mes Lat	R ETL	HS	1a	20
41	M	39	FC	14	25	L HS+Lat atrophy	Res+Staring+O-Aut	T Focal	T Reg	Reg	2	L Mes Lat	L ETL	HS	1a	20

Table 2 Continued

Patient	Sex	Age	Risk factors	Epilepsy onset	Epilepsy duration	MRI	Ictal clinical symptoms/ signs	Slow I/ictal EEG	Epileptiform I/ictal EEG	Ictal EEG onset	Ictal EEG pattern	Epileptogenic zone	Surgery	Histopathology	Seizure outcome	Follow up (months)
42	F	41	FC	23	18	R HS	Staring	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	2	17
43	M	36	PA	4	32	L Lat atrophy	Veg+Esp	T Focal	BT Focal	Focal	1	L Mes Lat	L ETL	HS	1a	33
44	M	21	–	8	13	L HS	Res+Veg+O-Aut+Dys	T Focal	T Focal	Focal	1	L Mes	L AMTL	HS	1a	36
45	M	38	FC	8	30	L HS	Res+Veg+Dys	T Focal	T Focal	Focal	1	L Mes	L AMTL	HS	1a	33
46	M	29	–	7	22	R HS	Res+Esp+Dys	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1a	40
47	F	24	–	6	18	L HS	Res+Esp+Staring+O-Aut+Dys	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1a	18
48	M	49	–	27	22	L HS	Res+O-Aut	T Focal	T Focal	Focal	1	L Mes	L AMTL	HS	1a	22
49	M	21	FC	4	17	L Lat atrophy	Staring+O-Aut	T Reg	T Focal	Focal	2	L Mes Lat	L ETL	neg	1b	36
50	F	40	–	25	15	R HS	Staring+O-Aut+Dys	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1a	12
51	F	37	FC	20	17	L HS	Esp+Staring+O-Aut	T Focal	T Focal	Focal	1	L Mes Lat	L ETL	HS	1a	34
52	F	36	–	7	29	R HS	Res+Esp+O-Aut	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1b	41
53	M	28	FC	4	24	L HS	Res+Veg+Staring+O-Aut	T Reg	T Reg	Reg	2	L Mes Lat	L ETL	HS	1a	36
54	F	44	FC	28	16	L Lat atrophy	Staring+O-Aut	T Focal	T Focal	Focal	2	L Mes Lat	L ETL	neg	1a	42
55	M	62	FC	32	30	R HS	Res+Esp+Staring+O-Aut	BT Focal	BT Focal	Focal	1	R Mes Lat	R ETL	HS	1a	33
56	F	25	–	21	4	R HS	Res+Staring+O-Aut	T Focal	T Focal	Focal	2	R Mes Lat	R ETL	HS	1a	36
57	M	41	–	20	21	L HS	Veg+O-Aut	BT Focal	T Focal	Focal	2	L Mes Lat	L ETL	HS	3	30

AMTL, anteromesial temporal lobectomy; Aud, auditory symptoms; BT, bitemporal; Dys, contralateral upper limb dystonia; EEG, elecroencephalography; Esp, psycho-esperienitial symptoms; ETL, extensive temporal lobectomy; FC, febrile convulsions; G, ganglioglioma; HS, hippocampal sclerosis; I/ictal, interictal; L, left; Lat, lateral; Mes, mesial; MRI, magnetic resonance imaging; O-Aut, oroalimentary automatisms; PA, perinatal asphyxia; R, right; Res, rising epigastric sensation; Reg, regional; T, temporal; Veg, vegetative symptoms.

When neuropsychological data disclosed memory deficits contralateral to the side of the epileptogenic zone, suggesting a high risk for postoperative amnesia after temporal lobe surgery, the patient was scheduled for the Wada test or excluded from surgery.

Of 331 patients who underwent the preliminary assessment, 157 were scheduled for the TLE-Prot. MRI evaluation of these patients showed 84 hippocampal scleroses, 56 tumours (48 low grade neoplasms, eight cavernomas), 11 findings suggesting dysplasia (in four cases a hippocampal sclerosis was also evident), and four focal neocortical atrophies. In two patients, MRI showed no abnormalities. Among the 56 patients with MRI evidence of a tumour, 14 patients had a history of epilepsy for one to two years. In this group, in eight patients the lesion was located in the mesial structures and in six patients in the lateral aspects. No patient in this group had a lesion involving both the mesial and lateral structures. Three of 14 patients were excluded from a fast surgical protocol and were admitted to the second level because of non-concordance of EEG interictal or clinical data with MRI. In the 143 patients admitted to video-EEG monitoring, 929 seizures (mean number of seizures for each patient, 6.5; mean duration of monitoring days, 5.6) were recorded.

One hundred and fifty nine patients were admitted to the ETLE-Prot and 15 patients were excluded from surgery.

Scheme for the identification of individualised TLE surgery

To propose individualised and limited surgery strictly according to the epileptogenic zone, we developed a diagnostic grid (table 1) based on anatomical and ictal electroclinical criteria considered to be suggestive of epileptogenesis in the mesial or lateral temporal lobe. Patients were subdivided into the following three groups:

(1) Mesial TLE: when the anatomical criterion for mesial cluster, the EEG criterion for mesial cluster, and at least two of the clinical criteria for mesial cluster were met.

(2) Lateral TLE: when the anatomical criterion for lateral cluster, the EEG criterion for lateral cluster, and at least two of the clinical criteria for lateral cluster were met.

(3) Mesiolateral TLE: when at least one criterion for mesial cluster and at least one criterion for lateral cluster were met, or when the anatomical and the EEG criteria were concordant with one clinical criterion alone.

If brain imaging was negative, only the presence of EEG and clinical criteria were taken into account for the classification.

This scheme was used to define different surgical strategies: lesionectomy (fast TLE-Prot, tumorous lateral TLE), anteromesial temporal lobectomy (AMTL) in mesial-TLE, and extensive temporal lobectomy (ETL) in hippocampal sclerosis/cryptogenic or lesional mesiolateral TLE.

Surgery

All operations were performed by the same epilepsy surgeon (VE).

Both ETL and AMTL included microsurgical resection of the amygdala and en bloc excision of the hippocampal formation and parahippocampal gyrus. These interventions differed in the extent of the neocortical resection. Non-dominant ETL included excision of 4–4.5 cm of the superior temporal gyrus and the middle temporal gyrus, and 5–6 cm of the inferior temporal gyrus, whereas dominant ETL included excision of 4–5 cm of the middle and inferior temporal gyrus, although the superior gyrus was left intact. In AMTL, the extent of the neocortical excision was 3 cm for

all the first three temporal gyri (sparing the superior gyrus in the dominant hemisphere). Lesionectomy consisted of complete removal of the foreign epileptogenic tissue alone.

Seizure outcome assessment

Seizure outcome was determined by the patient's report to the neurologist during the scheduled follow up visits, including 60 minutes awake EEG standard recordings,[25] and it was classified according to Engel.[26] During the first year of follow up, AEDs were kept constant in all patients.

Statistical analysis

Descriptive statistics were used to summarise the data for all variables. The confidence intervals (CI) for the proportion of seizure free patients in each group were calculated. The χ^2 test was used to test for differences in outcome between patients with foreign tissue lesions and patients with hippocampal sclerosis.

RESULTS

A lesionectomy was offered to 11 patients with TLE after the fast TLE-Prot. After video-EEG monitoring, another 133 patients were diagnosed as having TLE, and 13 were admitted to the ETLE-Prot. Four of the 133 patients with TLE were excluded from surgery for psychiatric or neuropsychological disturbances. Therefore, surgery was offered to a total of 140 patients with TLE. Eight of these patients refused surgery.

Of the 132 consecutive patients with TLE who underwent surgery, 101 had a follow up of at least one year (range, 12–60 months; mean duration, 30). No patients were lost to follow up, although 31 patients had a follow up period of less then one year and therefore were excluded from our study. All patients with TLE were divided in a ''hippocampal sclerosis/cryptogenic'' group (57 patients) (table 2) and a ''tumours/cortical organisation disorders'' group (44 patients) (table 3).

Hippocampal sclerosis/cryptogenic group

General characteristics

Fifty seven patients (25 females and 32 males; mean age, 33.9 years; SD, 10.2; range, 12–62; mean age of epilepsy onset, 12.4 years; SD, 8.7; range, 4–43; mean epilepsy duration, 21.5 years; SD, 9.5; range, 2–41) were classified into this group. Risk factors for epilepsy were found in 44 patients (febrile convulsions in 35 and perinatal asphyxia in nine patients).

Table 4 shows the results of presurgical investigations, type of operation and histopathology.

Seizure outcome

Forty seven (95% CI, 70.1 to 91.2) patients were classified as Engel class I (42 as class Ia, five as Engel class Ib), seven as Engel class II, two as Engel class III, and one as Engel class IV.

Tumours/cortical organisation disorders group

General characteristics

Forty four patients (21 females and 23 males; mean age, 32.3 years; SD, 12.7; range, 76–63; mean age of epilepsy onset, 19.7 years; SD, 14.7; range, 1–57 years; mean epilepsy duration, 12.5 years; SD, 11.9; range, 1–45) were classified into this group. Risk factors for epilepsy were found in eight patients (febrile convulsions in five and perinatal asphyxia in three patients). Pathology showed 21 low grade tumours, eight DNETs, five cavernous angiomas, nine cortical dysplasias, and one focal Rasmussen encephalitis.

Table 4 shows the results of the presurgical investigations, the type of surgery, and the histopathology.

Seizure outcome

Thirty nine (95% CI, 75.4 to 96.2) patients were classified as Engel class I (36 as Engel class Ia, three as Engel class Ib) and five as Engel class II.

Morbidity

Permanent postoperative complications occurred in two patients (2%): one patient developed a hemiplegia after ETL, whereas the other developed a hemianopia after lateral lesionectomy.

With regard to the neuropsychological outcome, no patients suffered from serious or clinically evident neuropsychological morbidity at the one year follow up assessment.

The seizure outcome of patients with foreign tissue lesions (39 in Engel class 1) was not significantly different from that of patients with hippocampal sclerosis (47 in Engel class 1) ($\chi^2 = 0.75$; $p = 0.39$).

DISCUSSION

TLE is the ''model'' surgically remediable epileptic syndrome, first because it is the most frequent among focal epilepsies, and second because it is often resistant to medical treatment. Moreover, because TLE often reflects epileptogenesis localised in ''discrete'' and anatomically well circumscribed structures (the amygdalo–hippocampal complex), whose radiological and electroclinical seizure correlates are well known, it may be considered particularly eligible for surgical treatment.[5 6 27] Therefore, almost all centres offer surgery for TLE after a non-invasive diagnostic investigation, reporting good seizure outcome and surgery safety. However, common guidelines regarding the relative weight of the different presurgical investigations are still not well established. As a consequence, several protocols emphasise neuroimaging (MRI),[28] whereas other protocols emphasise video-EEG recording.[29] Few studies have focused on the problem of implementing a ''reproducible'' methodology to allow TLE surgery with an optimal cost–benefit ratio, both in terms of seizure outcome/quality of life and costs.[29] However, even in the best published TLE surgical series, only 80% of patients have ''good outcome'', often including patients in Engel class II who are not seizure free.[4] Moreover, several non-invasive protocols include very expensive procedures, such as MRI volumetric analysis, functional MRI, single photon emission computed tomography, positron emission tomography, and the Wada test. This reduces the cost effectiveness of surgery for epilepsy and the number of patients to whom it can be offered.[28–31]

Another open issue concerns the identification, by means of non-invasive techniques, of different TLE subsyndromes (mesial, lateral, and mesiolateral TLE)[32] so that different surgical strategies can be offered as appropriate. In fact, the ''classic'' surgical approach in TLE tends to consider standard surgical procedures (temporal lobectomy or selective amygdalohippocampectomy),[29 33 34] with the risk of removing cortical regions not involved in epileptogenesis or not resecting the whole epileptogenic zone.

When MRI shows a tumour with a brief (less than two years) seizure history, we consider ictal video-EEG recording unnecessary when the patient and eyewitness history, clinical semiology, and/or interictal EEG are concordant with the tumour site.[35] Recurrent seizures are recognised as a potential cause of neuronal damage, and hence have been hypothesised to modify the extent of the epileptogenic zone in time.[36] Therefore, in tumorous epilepsy with a brief history, it is more likely that the epileptogenic zone is restricted to the lesion, and surgery may be limited to lesionectomy alone.

The results of lesionectomy alone for the treatment of TLE as a result of a temporal lobe mass lesion have been controversial. Jooma and colleagues[37] and Cascino and colleagues[38] reported that lesionectomy led to a ''seizure

Table 3 Results of investigations and postoperative outcome in 44 patients with a foreign tissue lesion

Patients	Sex	Age	Risk factors	Epilepsy onset	Epilepsy duration	MRI	Ictal clinical symptoms/ signs	Slow I/ictal EEG	Epileptiform I/ictal EEG	Ictal EEG onset	Ictal EEG pattern	Epileptogenic zone	Surgery	Histopathology	Seizure outcome	Follow up (months)
1	F	17	FC	7	10	R Mes	Res+Veg+O-Aut+Dys	T Reg	T Focal	Focal	2	R Mes Lat	Les+R ETL	HS+Dyspl	1a	48
2	M	29	–	28	1	R Mes	–	T Focal	T Focal	–	–	R Mes	Les	DNET	1a	18
3	F	29	–	27	2	L Mes	Res+Dys+O-Aut	T Reg	T Reg	Focal	2	L Mes Lat	Les+L ETL	G	1b	40
4	M	51	–	11	40	R Mes Lat	Veg+O-Aut	BT Focal	BT Focal	Focal	2	R Mes Lat	Les+R ETL	Dyspl	1a	36
5	M	30	–	28	2	R Lat	Staring+O-Aut+Dys	T Reg	T Reg	Focal	1	L Mes Lat	Les+L ETL	Cav	1a	29
6	M	28	–	5	23	R Lat	Staring	T Reg	T Reg	Focal	2	R Lat	Les	Dyspl	1a	30
7	M	22	–	5	17	L Mes	Res+Veg+Aud+Esp+O-Aut	T Reg	T Focal	Focal	2	L Mes Lat	Les+L ETL	G	1b	53
8	M	21	FC	19	2	R Mes	–	T Focal	BT Focal	–	–	R Mes	Les	G	1a	13
9	F	26	–	25	1	L Lat	–	T Focal	T Focal	–	–	L Lat	Les	DNET	1a	32
10	M	40	–	30	10	R Mes	–	T Focal	T Focal	–	–	R Mes	Les	DNET	1a	34
11	F	34	–	16	18	L Lat	Veg+Esp+O-Aut	T Reg	BT Focal	Focal	1	L Mes Lat	Les+L ETL	Dyspl+HS	2	46
12	F	29	–	23	6	R Mes	Veg+O-Aut	T Focal	T Focal	Focal	1	R Mes	Les+R AMTL	G	1a	31
13	F	58	–	57	1	L Lat	–	T Focal	T Focal	–	–	L Lat	Les	G	1a	35
14	M	45	PA	3	42	R Mes Lat	Res+Veg+Aud+O-Aut	T Reg	T Focal	Focal	2	R Mes Lat	Les+R ETL	HS+Dyspl	2	46
15	M	46	–	37	9	L Mes Lat	Esp+Staring	T Focal	T Focal	Focal	2	L Mes Lat	Les+L ETL	DNET	1a	22
16	F	63	–	61	2	L Mes	–	T Focal	T Focal	–	–	L Mes	Les	DNET	1a	14
17	M	39	PA	18	21	R Lat	Staring+Aud	T Reg	T Focal	Focal	2	R Lat	Les	Dyspl	1a	35
18	M	8	FC	6	2	R Mes	–	T Focal	T Focal	–	–	R Mes	Les	DNET	1a	12
19	M	23	–	5	18	L Mes Lat	Res+Veg+O-Aut	T Reg	T Reg	Focal	2	L Mes Lat	Les+L ETL	DNET	1a	34
20	F	24	–	22	2	L Lat	–	T Focal	T Focal	–	–	L Lat	Les	Cav	1a	46
21	M	41	–	30	11	L Mes	Res+Esp+O-Aut	T Focal	T Focal	Focal	1	L Mes Lat	Les+L ETL	G	1a	16
22	F	46	–	36	10	R Mes	Res+Veg	T Focal	T Focal	Focal	1	R Mes	Les+R AMTL	G	1a	57
23	M	42	–	13	29	L Mes Lat	Staring+Veg	T Reg	T Reg	Focal	2	L Mes Lat	Les+L ETL	HS+Dyspl	2	18
24	M	36	–	34	2	R Lat	Aud+Esp	T Reg	T Reg	Focal	2	R Lat	Les	DNET	1a	21
25	F	24	–	17	7	R Mes	Staring	BT Focal	BT Focal	Focal	2	R Mes Lat	Les+R ETL	Dyspl	1a	26
26	F	55	–	53	2	L Mes	–	T Focal	T Focal	–	–	L Mes	Les	G	1a	34
27	F	27	–	14	13	L Lat	Esp+O-Aut	T Focal	T Focal	Focal	1	L Mes Lat	Les+L ETL	Cav	1a	56
28	F	11	FC	7	4	R Lat	Veg+Staring	T Focal	T Reg	Focal	1	R Mes Lat	Les+R ETL	G	1a	29
29	M	21	–	20	1	R Mes	Res	T Focal	T Focal	–	–	R Mes	Les	G	1a	37
30	M	7	–	1	6	R Lat	Staring	BT Reg	BT Reg	Reg	2	R Mes Lat	Les+R ETL	Dyspl	2	14
31	M	37	–	25	12	L Mes	Res+Staring+O-Aut+Esp	T Focal	T Focal	Focal	1	L Mes Lat	Les+L ETL	G	1a	54
32	M	29	–	1	28	R Mes	Esp+Veg+Staring+O-Aut	T Focal	T Focal	Focal	2	R Mes Lat	Les+R ETL	G	1a	17
33	M	17	–	15	2	R Lat	–	T Reg	T Reg	–	–	R Lat	Les	G	1a	40
34	F	21	–	8	13	R Mes	Res+Esp+O-Aut	T Reg	T Reg	Focal	2	R Mes Lat	Les+R ETL	G	1a	53
35	F	40	–	34	6	L Mes	Res	T Focal	T Focal	Focal	1	L Mes	Les+L AMTL	G	1a	54
36	M	31	–	26	5	L Lat	Esp+Staring	T Focal	T Focal	Focal	2	L Lat	Les	G	1b	35
37	F	32	–	1	31	L Mes	Veg	T Focal	T Focal	Focal	1	L Mes	Les+L AMTL	G	1a	16
38	F	34	–	13	21	L Mes	Res+Esp+Staring+O-Aut	T Focal	T Focal	Focal	2	L Mes Lat	Les+L ETL	G	1a	34
39	M	35	–	28	7	L Lat	Veg	T Reg	T Reg	Reg	2	L Mes Lat	Les+L ETL	G	2	12
40	F	52	–	7	45	L Lat	Res+O-Aut+Staring	BT Focal	BT Focal	Reg	2	L Mes Lat	Les+L ETL	G	1a	23
41	M	22	PA+FC	7	15	R Mes	Res+Staring+O-Aut+Dys	T Reg	T Focal	Reg	2	R Mes Lat	Les+R ETL	G	1a	40
42	F	31	FC	27	4	L Lat	Esp+O-Aut	T Focal	T Focal	Focal	1	L Mes	Les+L AMTL	Rasm	1a	13
43	F	28	–	1	27	R Mes	Res+Veg+O-Aut	T Focal	T Focal	Focal	1	R Mes	Les+R AMTL	Cav	1a	15
44	F	39	–	18	21	R Lat	Veg+Esp+O-Aut	T Reg	T Reg	Focal	1	R Mes Lat	Les+R ETL	Cav	1a	35

AMTL, anteromesial temporal lobectomy; Aud, auditory symptoms; BT, bitemporal; Cav, cavernoma; DNET, dysembrioblastic neuroepithelial tumour; Dys, contralateral upper limb dystonia; Dyspl, dysplasia; EEG, elecroencephalography; Esp, psycho-esperiential symptoms; ETL, extensive temporal lobectomy; FC, febrile convulsions; G, ganglioglioma; HS, hippocampal sclerosis; I/Ictal, interictal; L, left; Lat, lateral; Les, lesionectomy; Mes, mesial; MRI, magnetic resonance imaging; O-Aut, oroalimentary automatisms; PA, perinatal asphyxia; R, right; Rasm, Rasmussen encephalitis; Reg, regional; Res, rising epigastric sensation; T, temporal; Veg, vegetative symptoms.

Table 4 Global results of presurgical evaluation, type of surgery, and histopathology in the 101 operated patients with follow up after surgery >1 year

	Hippocampal sclerosis/ cryptogenic group		Tumorous/ COD group	
	N	(%)	N	(%)
Interictal EEG abnormalities	55	95.6	44	100
Focal	34	61.8	24	54.5
Regional	12	21.8	16	36.4
Bitemporal	9	16.4	4	9.1
Epileptiform abnormalities	56	98.2	44	100
Focal	37	66.1	27	61.4
Regional	4	7.1	11	25
Bitemporal	15	26.8	6	13.6
Ictal clinical findings				
Rising epigastric sensation	41	71.9	16	36.4
Other vegetative symptoms	23	40.3	16	36.4
Psyco-experential phenomena	19	33.3	13	29.5
Auditory symptoms	3	5.3	4	9.1
Oroalimentary automatisms	21	36.8	14	31.8
Staring	46	80.7	20	45.4
Controlateral dystonia	13	22.8	4	9.1
Ictal EEG findings				
Focal	52	91.2	29	65.9
Regional	5	8.8	4	9.1
Pattern type 1	26	45.6	13	29.5
Pattern type 2	31	54.4	20	45.4
Epileptogenic zone				
Mesial	17	29.9	13	29.5
Lateral	–	–	8	18.1
Mesiolateral	40	70.1	23	52.2
Surgery				
ETL	40	70.2	–	–
AMTL	17	29.8	–	–
ETL+lesionectomy	–	–	23	52.3
AMTL+lesionectomy	–	–	6	13.6
Lesionectomy	–	–	15	34.1
Histopathology				
MTS	55	96.5	–	–
Low grade tumours (ganglioglioma, diffuse astrocytoma)	–	–	21	47.7
DNET	–	–	8	18.2
Cavernoma	–	–	5	11.4
Dysplasia	–	–	9 (+4 MTS)	20.5
Focal Rasmussen encephalitis	–	–	1	2.2
Negative	2	3.5	–	–

AMTL, anteromesial temporal lobectomy; COD, cortical organisation disorders; DNET, dysembrioneuroepithelioma; EEG, electroencephalographic; ETL, extensive temporal lobectomy; MTS, mesial temporal sclerosis.

free'' condition in only 20% of patients with temporal lobe mass lesions, whereas Fried and colleagues[39] reported a seizure free outcome in 80% of patients.[35] Kraemer *et al* reported that 73% of the patients affected by TLE as a result of vascular malformations were seizure free (no seizures in the last year of follow up, according to Dukes's classification[40] after lesionectomy).[35] In general, it is agreed that if the seizure disorder exists for more than a year before surgical resection, lesionectomy alone may not be efficacious.[37 41] Other studies[42 43] corroborate the assumption that a shorter seizure disorder duration correlates with a better prognosis when lesionectomy is the surgical approach.

In our series, 14 patients with TLE had a short seizure history and MRI evidence of a temporal mass lesion: three of these patients were excluded from ''fast'' lesionectomy and were admitted to diagnostic level 2 for video-EEG recording because of lesional pathology not colocalised with EEG interictal findings, whereas 11 patients had lesionectomy with excellent outcome (all in Engel class 1a). Two of the three abovementioned patients who were entered into diagnostic level 2 had extensive temporal lobectomy (one in Engel class 1a and one in Engel class 1b), whereas the other patient had lesionectomy (Engel class 1a).

Although some studies[44] report that ictal video-EEG recording may be not mandatory in patients with TLE, we decided to admit most lesional or non-lesional patients with TLE to video-EEG recording. This strategy is obviously ''obligatory'' in patients with normal MRI or absent or bilateral interictal EEG epileptiform abnormalities. Moreover, in our opinion, a tailored operation cannot be offered without having ictal EEG and clinical data, which allow the three different TLE subsyndromes to be identified. Therefore, we developed a diagnostic grid for TLE surgery based on correlations among anatomical, electrical, and clinical data, in accordance with principles first planned by Bancaud and Talairach,[9] and later developed by their successors,[10–12] obtained by non-invasive investigations. According to many studies that evaluated the localising value of a single diagnostic criterion,[22 23 45–52] the data obtained from presurgical evaluation were grouped in clusters strongly suggesting the location of epileptogenesis in the mesial or lateral aspect of the temporal lobe. Then, based on our experience, specific combinations of different criteria were used for localisation. Although most authors agree about the localising value of clinical signs and of neuroradiological findings in suggesting the epileptogenic zone, to our knowledge, only a few clinical studies[53] have used EEG ictal patterns for localising purposes. In our classification scheme, we particularly stressed the localising value of the ictal scalp EEG pattern in TLE, according to the theories of Pacia and Ebersole,[23] based on ''the most comprehensive systematic study of scalp–intracranial ictal EEG findings in patients with temporal lobe epilepsy''.[54] Their findings confirmed and extended previous studies on this issue,[55 56] revealing that > 5 Hz ictal EEG pattern over the temporal regions is the characteristic pattern associated with mesial temporal seizures. In contrast, there is comparatively little information about neocortical temporal lobe seizures, and in most cases the available descriptions are shown in negative terms, emphasising the absence of features typical of mesial temporal lobe seizures. In light of this, the studies of Ebersole's and Foldvary's groups should still be thought of as important contributions; moreover, they depicted neocortical temporal lobe seizures in positive terms. Both authors agree that low frequency scalp EEG ictal patterns in temporal lobe seizures are highly related to the lateral neocortical seizure onset zone. Compared with other non-invasive diagnostic protocols[31 33 57] for TLE surgery, aimed at selecting those patients who should benefit from standard temporal lobectomy and identifying those patients who should be investigated invasively or excluded, our diagnostic grid enables TLE classification to be refined into the mesial, temporal, and mesiotemporal subtypes. Hence, surgery can be specifically planned, as far as possible, according to the extension of the epileptogenic zone in the three different TLE subtypes (that is, AMTL in mesial-TLE, ETL in mesiolateral-TLE, and lesionectomy in lesional lateral-TLE), avoiding unnecessarily extensive surgery. As suggested by studies based on stereotactic intracerebral EEG recordings,[58] during which the simultaneous involvement of both the amygdala, the hippocampus, and the temporal pole at the onset of temporal lobe seizures was often observed, the so called ''mesial'' temporal lobe seizures probably do not always arise solely from the amygdalo–hippocampo–parahippocampal complex. Therefore, in the absence of intracerebral recordings, in mesial-TLE, we decided to use the AMTL resection, which includes the temporal pole in addition to the mesial temporal structures, rather than the selective amygdalo–hippocampectomy.

The usefulness of our T-Prot was confirmed by the excellent outcome (overall, 85.1% in Engel class 1), although

we cannot exclude the possibility that a similar outcome would have been achieved without using a tailored resection. Given that the diagnostic approach we describe is based on a global assessment that correlates and combines a set of criteria, we could not carry out an analysis of the performance of each diagnostic criterion (anatomical, ictal EEG, and ictal clinical) in terms of sensitivity and specificity. In our classification method, it is not possible to assign a patient to a group using single criteria, and this made it impossible for us to test the performance of a single criterion. In any case, the performance of the full set of criteria was very satisfactory.

Although our findings need to be confirmed by further studies with a longer follow up duration and by randomised controlled trials comparing our protocol with other established protocols, they suggest that in most patients with TLE different subsyndromes can be accurately identified using non-invasive anatomo–electro–clinical data, and can be treated effectively and safely with a tailored operation.

ACKNOWLEDGEMENTS

We thank the nursing staff and the team of neurophysiology technicians (L Cacciola, F Orabona, and R Ponticelli) who performed meticulous video-EEG recordings in all patients.

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Competing interests: none declared

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