■ Original article

Epileptic Disord 2021; 23 (4): 590-610

Epileptic Disorders

Clinical semiology of temporal lobe seizures in preschool children: contribution of invasive recording to anatomical classification

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Received February 26, 2020;
Accepted January 25, 2021

ABSTRACT

Objective. Focal seizure semiology is often inadequately studied, specifically in preschool children. Among drug-resistant epilepsies amenable to surgery, temporal lobe seizure semiology has been widely described in this age group. Nevertheless, a systematic anatomo-electroclinical study has never been performed.

Methods. We retrospectively reviewed the charts of patients younger than six years old at the time of video-EEG recording who were operated on for temporal lobe epilepsy in our centre between 2010 and 2016. In order to describe the electroclinical semiology and establish anatomo-clinical correlations, we reviewed all the recorded seizures on scalp and invasive video-EEG and analysed pre- and postsurgical clinical data, MRI scans, and surgical and pathological data. We classified patients into the following four anatomical groups: mesio-temporal, temporal lateral, polar, and mesio-lateral, and for each group we selected video-EEG samples for educational purposes.

Results. Twenty-eight patients fulfilled the selection criteria. Twenty-three patients (82%) were explored with invasive electrodes that consisted of foramen ovale electrodes in 11 (39%) and stereoelectroencephalography in 12 (43%). The majority of the 53% of patients with mesio-temporal epilepsies had specific ictal semiology, as described in adults. The others had subtle seizures or seizures limited to apnoea. The other groups also had some features comparable to adults, although no child reported the classic auras of lateral epilepsies. In total, 11% had infantile spasms (IS); post-ictal examination provided lateralization signs in 28%. With a mean post-surgical follow-up duration of 5.5 years, 89% of the patients were classified as Engel Class I.

Significance. Preschool children were shown to have non-specific seizures, notably subtle events or IS. However, careful video-EEG analysis can provide arguments for localizing the epileptogenic zone within the temporal lobe in most cases. Seizures with apnoea are characteristic of mesial temporal onset in patients with long-term epilepsy-associated tumours.

VIDEOS ONLINE

Key words: subtle seizures; apnoea; epilepsy surgery; ganglioglioma

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doi:10.1684/epd.2021.1302

Temporal lobe epilepsy (TLE) is frequently encountered in children, and TLE patients are generally considered good candidates for epilepsy surgery. Temporal lobe semiology in children has been widely studied based on video-EEG recording material, and compared to TLE in adults in terms of semiology [1-4] and value of lateralizing signs [5]. Nevertheless, electro-clinical semiology of TLE in

the subgroup of preschool children has been, to date, poorly evaluated, and rarely on the basis of anatomical classification provided by invasive recordings. We report our findings of a series of preschool children who entered our programme of presurgical evaluation, focusing on clinical and EEG semiology according to the anatomical origin of seizures within the temporal lobe.

Patients and methods

Between 2010 and 2016, we identified 89 consecutive patients from our database who had a surgical resection preceded by a pre-surgical investigation for drug-resistant TLE. We selected a preschool patient subgroup, aged six years and younger, at the time of surgery in order to study the electro-clinical semiology of temporal lobe seizures at a young age. A comprehensive epileptic history was obtained for each patient.

The accurate topography of the seizure onset was assessed by a surface video-EEG recording completed for the majority of patients with an invasive recording. The decision to operate and the modalities of surgery were decided by a multidisciplinary team based on the criteria we use for all patients in this age range [6]. Scalp video-EEGs were performed according to the guidelines published for presurgical video-EEG monitoring [7, 8]. The 10-20 system with 19 electrodes can be used from the age of three months. For TLE, additional temporo-basal electrodes are of great value in detecting interictal spikes and assessing the seizure onset [9, 10]. When possible, a clinical examination was performed during the seizures.

When scalp EEG was not sufficient to determine the seizure onset, invasive explorations were required. A foramen ovale electrode (FOE), combined with scalp electrodes, was very helpful when there were strong arguments for a temporo-mesial epilepsy, in particular to detect subtle seizures [11, 12]. As for other localizations, stereoelectroencephalography (SEEG) was indicated when the anatomical-electroclinical correlations obtained by non-invasive and/or FOE investigations were insufficiently concordant or inconclusive regarding the localization of the supposed epileptic zone [13]. Based on a previous report from out tertiary centre, the ictal onset zone matched the lesion in only 32% of patients with symptomatic epilepsies of this age group [14].

After the age of two, SEEG is the most appropriate invasive method for exploration of temporal epilepsies. The involvement of Wernicke's area, which could be a specific indication for subdural recording, is an exceptional issue in preschool children. All the seizures recorded were reviewed by two neurophysiologists (DT, MC).

We classified the epilepsy according to the precise origin of the seizures within the temporal lobe and followed the anatomical classification proposed by Maillard *et al*. [15]:

- mesial structures (hippocampus amygdala and para hippocampal structures);
- temporal pole;
- neocortical temporal structures (lateral and basal) (T1 to T3 excluding the pole);
- and mesio-temporal and lateral regions.

We excluded multilobar epilepsies even if the temporal lobe was part of the epileptogenic zone. These encompass so-called Temporal plus epilepsies, as reported by Barba *et al*. [16], defined as epilepsies with a typical temporal semiology but a seizure onset zone which includes extratemporal zones, mainly insular and sometimes frontal or parietal areas (six of our patients). These also include multilobar epilepsies defined by onset zones in at least two lobes, either temporal or extratemporal; the seizure semiology is concordant with the corresponding epileptogenic zone (as in seven of our patients).

Pathological studies of the surgical material were analysed in one laboratory (Lariboisiere Hospital). Results were classified according to the International League Against Epilepsy (ILAE) classification for dysplasia [17] and classification of tumours [18].

The outcome of epilepsy was assessed using Engel's classification [19].

Results

Twenty-eight patients (nine female and 19 male), aged six years and under, were studied (*table 1*). The mean age at epilepsy onset was 12.8 months (range: 0-54); the first seizure consisted of infantile spasms (IS) in three, complex febrile seizures in eight, and focal seizures in 17 patients. Altogether, three patients presented with some IS during the course of epilepsy that were controlled with medication at the time of the surgery in all cases but one (Patient 22). None of the patients had electrophysiological features of hypsarrhythmia. All patients had associated focal seizures with the exception of one (Patient 22) who had late-onset IS as the only seizure type following herpetic encephalitis. On MRI, a structural lesion was found in all the patients but one (Patient 19).

All patients underwent tailored temporal surgery at a mean age of 3.6 years (range: 0.5 to 6.4). Five patients (18%) were under two years of age at the time of the surgery. Before surgery, all the children underwent a surface video-EEG recording. Twenty-three patients

▼ **Table 1.** Demographic data.

Patient number	Sex	Age at epilepsy onset (months)	Type of first seizure	Infantile spasms	Lesion on MRI	Side
1	M	2	FS	0	Mesiotemporal tumour	R
2	F	4	FS	0	Polar and mesial blurring	L
3	M	0	FS	0	Polar blurring	L
4	F	9	FS	0	Polar and mesial tumour	R
5	M	3	FS	0	Choroid plexus papilloma and HS	L
6	M	6	CFS	0	HS	L
7	M	2	FS	0	HS	R
8	F	2	FS	0	Polar and mesial tumour	R
9	M	9	CFS	0	HS	L
10	M	10	CFS then FS at 24 months	0	HS and Polar blurring	L
11	F	9	CFS then FS at 25 months	0	HS and temporo-basal blurring	R
12	F	54	CFS	0	HS	L
13	M	14	CFS	0	HS and polar blurring	L
14	M	6	FS	0	Polar, mesial and sublenticular tumour	R
15	F	12	FS	0	Amygdala tumour	R
16	M	36	FS	0	Polar blurring	L
17	M	4	FS	0	Polar blurring	R
18	M	18	FS	0	Polar and STG tumour	R
19	M	2	FS	0	No	R
20	M	17	IS	1	Temporobasal tumour	R
21	F	4	IS	1	Lateral blurring	L
22	F	36	IS	1	Mesio-temporal encephalomalacia and polar atrophy	R
23	M	36	FS	0	Mesial, STG and MTG tumour	R
24	M	6	FS	0	HS and polar blurring	L
25	M	30	FS	0	Polar and mesial tumour	L
26	F	15	CFS then FS at 36 months	0	HS and polar blurring	L
27	M	10	CFS then FS at 16 months	0	HS and polar blurring	L
28	M	3	FS	0	Polar, lateral and mesial blurring	L

HS: hippocampal sclerosis; R: right; L: left; MTG: mesial temporal gyrus; STG: lateral temporal gyrus; CFS: complex febrile seizures; FS: focal seizures.

(82%) were explored with invasive electrodes that consisted of FOE (DIXI or ALCIS electrodes) in 11 (39%) and SEEG (DIXI or ALCIS electrodes) in 12 (43%). Two patients underwent both FOE exploration and SEEG because the former led to the hypothesis of a temporo-mesial epilepsy being discarded (*table 2*). In the nine patients for whom FOE was contributory, both a temporo-mesial onset for some of the seizures as well as the occurrence of subtle seizures were confirmed.
The patients were classified into four anatomo-electroclinical groups, and for each group, the clinical and EEG data are provided (*table 3*), as well as one or more video-EEG samples of a "typical" seizure.

Mesio-temporal seizures (15 patients)

Of these patients, 27% were explored with SEEG. We distinguished three subgroups according to ictal semiology that we defined as follows:

- **(a) Typical mesial seizures** are very similar to those in adults and are characterized by the following signs:

▼ **Table 2.** Seizure recordings.

Patient number	FO	SEEG	Seizure semiology	Motor post-ictal deficit	Speech deficit	Seizures with apnoea
1			See video 3		NR	yes
2		1	Behavioural arrest, staring, apnoea with desaturation and cyanosis, R side paresis	1	NR	yes
3			See video 1		NR	yes
4			Subtle Sz with motor arrest		NR	no
5	1		See video 4		NR	no
6		1	Motor arrest, alteration of awareness, tachycardia, wide eyes, post-ictal R side paresis	1	NR	no
7		1	Behavioural change (becomes excited) then staring, wide eyes, deviation of eyes to the R, stiffness of the R arm +/- secondary generalization.		NR	no
8	1		Goes to her parents and grasps them with a fearful expression, tachycardia, vomiting, face pallor, staring, may report frightening visual hallucinations		no	no
9	1		Fearful expression and goes to his parents, cyanosis of the lips, tight lips		no	no
10	1	1	Says «I feel sick» then puts a hand on his stomach, short loss of contact, face pallor, cyanosis of the lips, chewing		no	no
11	1		Fearful expression, loss of contact, chewing, swallowing.		NR	no
12	1		1) Motor arrest, loss of contact, head and trunk deviation to the L, deambulation, post-ictal R arm paresis. 2) During sleep, subtle Sz with awakening and chewing.	1	no	no
13			Warns of the Sz, goes to his parents, anxious expression, staring, face pallor, chewing and nausea.		no	no
14			Motor arrest, face reddening, global hypotonia, alteration of awareness		NR	no
15	1		See video 2		no	no
16		1	See video 5		no	no
17		1	See video 6	1	no	no
18		1	Raising of arms, deviation of eyes and head to the R, face reddening, staring, post-ictal confusion		no	no
19		1	See video 7	1	NR	no
20		1	Deviation of eyes and head to the R, few clonic movements of the upper body, chewing during sleep, subtle Sz: awakening and deviation of eyes to the R		NR	no
21		1	Modified breathing, alteration of awareness, deviation of eyes to the L, R arm hypertonia, no post-ictal deficit		no	no
22	1	1	Staring then asymmetric spasms (head deviation to the R and raising of L arm) in cluster		no	no
23	1		1) Subtle Sz with motor arrest, staring and modified breathing. 2) Sz with vomiting and R-side face contraction		NR	no

▼ **Table 2.** Seizure recordings (*continued*).

Patient number	FO	SEEG	Seizure semiology	Motor post-ictal deficit	Speech deficit	Seizures with apnoea
24			Motor arrest, pallor and secondary upper limb hypertonia and clonic movements		no	yes
25	1		1) Subtle Sz during sleep with arousal. 2) During wakefulness: motor arrest, staring, tight lips, face reddening, mydriasis, few clonic movements of eyelids, hypertonia of R limbs, post-ictal R arm paresis	1	no	no
26	1		See video 8		yes	no
27			Motor arrest, staring, R hand hypotonia, face pallor, sweating, chewing and throat noises, post-ictal R side paresis and dysarthria.	1	yes	no
28		1	During sleep: eyes open, deviation of eyes and head to the L, yawns, L hand automatisms, R hemi-body hypertonia associated with clonic movements.		yes	no

FO: foramen electrode; SEEG: stereo-electroencephalography; R: right; L: left: NR: not relevant; Sz: seizure.

– dysautonomic signs such as face rubefaction, pallor or cyanosis, mydriasis;
– oro-alimentary signs such as nausea, chewing, salivation, vomiting;
– emotional and subjective signs such as fear, epigastric sensation;
– contact changes with staring;
– motor signs such as automatisms, typically homolateral to the seizure onset, and posturing manifestation, often contralateral to the epileptic focus.

A typical temporo-mesial case is shown in Patient 3 *(video sequence 1 and figure 1)*.

A typical amygdala seizure with fear is shown in Patient 15 *(video sequence 2 and figure 2)*.

- **(b) Seizures with apnoea**. Three patients presented this type of seizure in which the dominant sign was apnoea with deep desaturation requiring oxygenotherapy at home (Patient 1) (*video sequence 3, figure 3*).
- **(c) Subtle seizures.** These are often overlooked and identified based on video-EEG. During sleep, patients may present with a somewhat "odd" awakening. During wakefulness, there are subtle changes in behaviour compared to the child's baseline activity. This category includes hypomotor seizures characterized by behavioural arrest (Patient 5) (*video sequence 4, figure 4*).

Temporal pole seizures (two patients)

These are subtle seizures, and may thus be easily overlooked. In the video provided for this type of seizure, the seizures were identified based on SEEG and the clinical signs consisted of a hypomotor seizure with speech arrest, motor arrest, and automatisms (Patient 16) (*video sequence 5, figure 5*). More symptomatic seizures are due to propagation to adjacent structures; an example of a patient presenting with a hyperkinetic seizure mimicking frontal lobe semiology is provided (Patient 17) (*video sequence 6, figure 6*).

Neocortical temporal seizures (five patients)

In these seizures, there is a lack of "typical" semiology which makes the diagnosis difficult in the absence of depth electrodes. In our patients, the diagnosis was based on SEEG. The clinical signs frequently observed consisted of asymmetric tonic posturing and/or subtle seizures. In the case of a dominant hemisphere, post-ictal aphasia can be present (Patient 19) (*video sequence 7, figure 7*).

Mesial and neocortical seizures (six patients)

These seizures have a temporo-mesial onset with latero-temporal propagation. After the typical mesial semiology, contralateral motor manifestations are systematically observed, related to suprasylvian propagation via the temporal neocortex. In the case of a dominant hemisphere, in speaking patients, post-ictal aphasia can be seen (Patient 26) (*video sequence 8, figure 8*).

▼ **Table 3.** Topography, surgery and follow-up.

Patient number	Mesial	Pole	Lateral	Mesio-lateral	Number of surgeries	Age at last surgery (years)	Type of resection	Pathology	Follow-up (years)	Engel Class
1	1				1	0.5	Anteromesial	GG	3.3	1
2	1				2	0.5	Anteromesial, basal and anterolateral	FCDIIa	3.9	1
3	1				1	0.9	Anteromesial	FCDI + ganglioneuronal tumour	5.9	1
4	1				1	1.7	Anteromesial	GG	3.9	1
5	1				1	2.7	Anteromesial and papilloma	Papilloma + HS	5.9	1
6	1				1	3.3	Anteromesial	FCDIIa + SH	2.9	1
7	1				2	3.1	Anteromesial and superior temporal gyrus	Negative	4.8	3
8	1				1	3.5	Anteromesial	FCDIa	5.4	1
9	1				1	3.3	Anteromesial	FCDIa + HS	9.4	1
10	1				1	5.8	Anteromesial	FCDI + HS	8.2	3
11	1				1	6.4	Anteromesial	FCDII + HS	7.2	1
12	1				1	6.3	Anteromesial	FCDI et SH	8.5	1
13	1				1	4.1	Anteromesial	Isolated HS	1.8	1
14	1				1	1.6	Anteromesial	GG	1.9	1
15	1				1	4.9	Anteromesial	GG + HS	9.7	1
16		1			1	5.6	Anteromesial and basal	FCD + DNT	5.8	1
17		1			1	4.9	Anteromesial	FCDIIb et HS	7.3	1
18			1		1	2.9	Pole and superior temporal gyrus	DNT	4.9	1
19			1		1	2.4	Temporal superior gyrus and amygadala	FCDIIb	7.4	1
20			1		1	3.1	Lateral	DNT	5.8	1

▼ **Table 3.** Topography, surgery and follow-up (*continued*).

Patient number	Mesial	Pole	Lateral	Mesio-lateral	Number of surgeries	Age at last surgery (years)	Type of resection	Pathology	Follow-up (years)	Engel Class
21			1		1	4.3	Pole and lateral	FCDIIa	5.3	1
22			1		1	4.1	Mesial and lateral	Herpetic encephalitis	8.1	3
23				1	1	2.7	Mesial and lateral	GG	4.7	1
24				1	1	3.9	Anteromesial	FCDIIb + HS	4.2	1
25				1	1	4.4	Pole and anterior hypocampus	GG	4.4	1
26				1	1	5.6	Anteromesial	FCD + HS	9.4	1
27				1	1	5.5	Anteromesial	GG	2.1	1
28				1	1	3.7	Complete temporal disconnection	FCDIIa	2.5	1

GG: ganglioglioma; HS: hippocampal sclerosis, FCD: focal cortical dysplasia; DNT: dysembryoplasic neuro-epithelial tumour.

Post-ictal contralateral paresis was observed in 10 patients and could not be linked to a specific topography within the temporal lobe. More often, the deficit was found during clinical examination or careful review of the video, but rarely reported by the parents. It has a high lateralizing value. Post-ictal aphasia was impossible to assess in 12 patients (43%), either because the patients were too young or had a speech delay (and not cooperative post-ictally), or because of tiredness. Three patients (11%) had clear-cut post-ictal aphasia. Post-ictal language was normal in 11 children (39%) (*table 2*).

Surgery

Thirty surgical procedures were performed on twenty-eight patients: two patients were operated on twice (Patients 2 and 7) because of an incomplete mesial resection. The description of the surgical resection for each patient is reported in *table 3*.

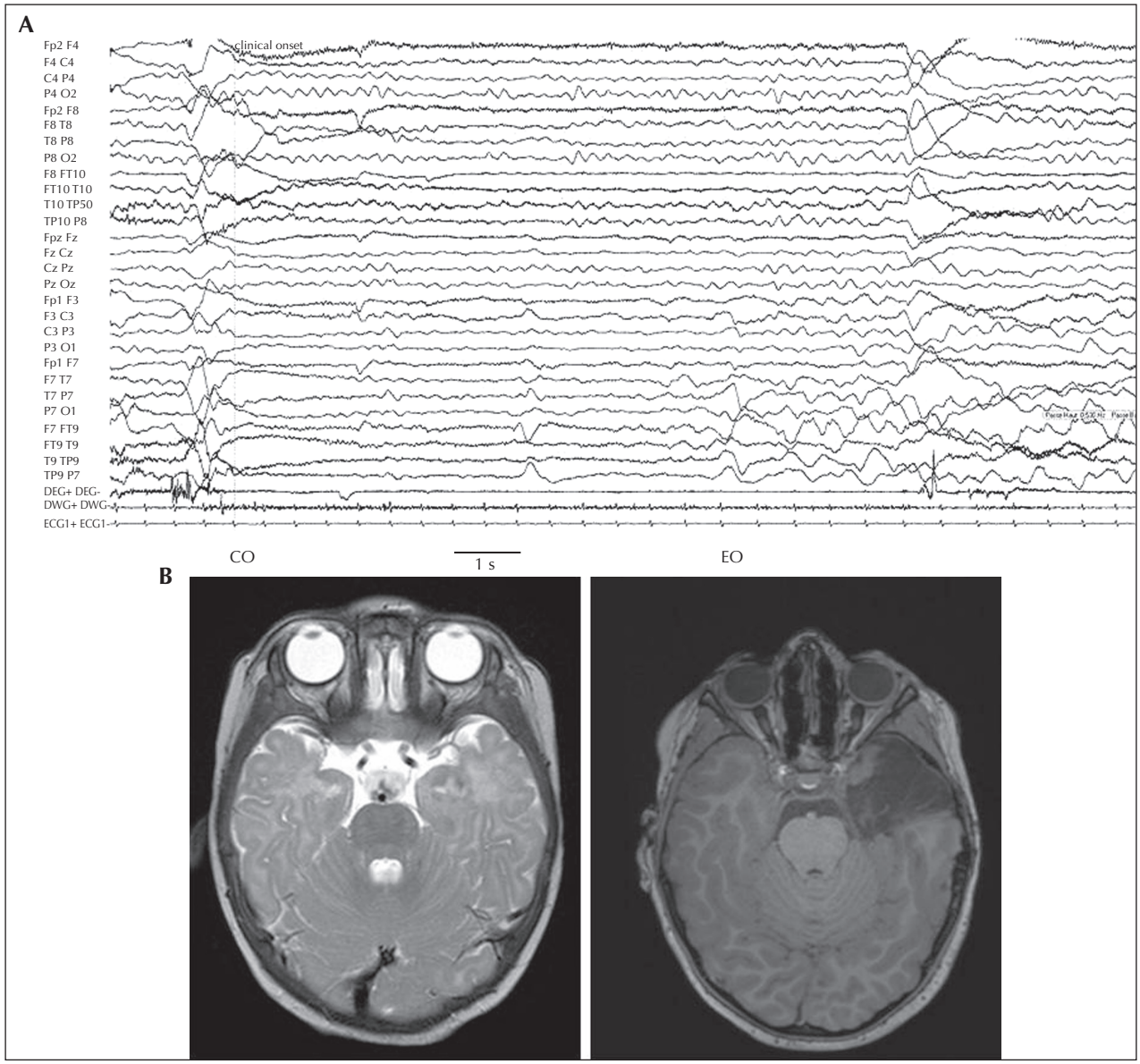
With a mean follow-up duration of 5.5 years (range: 1.8-9.7) and according to Engel's classification, 89% (25) of the patients were classified as Engel Class I, whereas three were Engel Class III (Patients 7, 10 and 22). The reasons for failure were incomplete temporal resection in Patients 10 and 22, while a mutation in the *FGF12* gene in Patient 7 could explain the unexpected post-surgical failure.

Pathological studies revealed focal cortical dysplasia (FCD) in 13, an isolated tumour in 10, and a tumour associated with a FCD in two. Isolated HS was found in one patient, encephalitis in one and histology was negative in one patient. HS was found in 11 patients (one isolated, two associated with a tumour, and eight associated with FCD).

According to the ILAE pathological classification, FCD I was found in one patient, FCD II in four, FCD IIIa in eight, and FCD IIIb in two. Isolated tumours were found in 10 patients including seven gangliogliomas, two dysembryoplastic neuroepithelial tumours and one choroid plexus papilloma associated with HS corresponding to the so-called "long-term epilepsy-associated tumour" [18].

Discussion

To our knowledge, this is the first series reporting temporal lobe electro-clinical semiology in preschool children, including a large number of toddlers (19%). Moreover, it is the first report in which clinical semiology is based on electrophysiological findings and not on the topography of the lesion based on MRI [4] or on seizure freedom after surgery [1-3, 20]. Indeed, epilepsy onset is not always determined by the lesion itself, which can be missing or does not match with

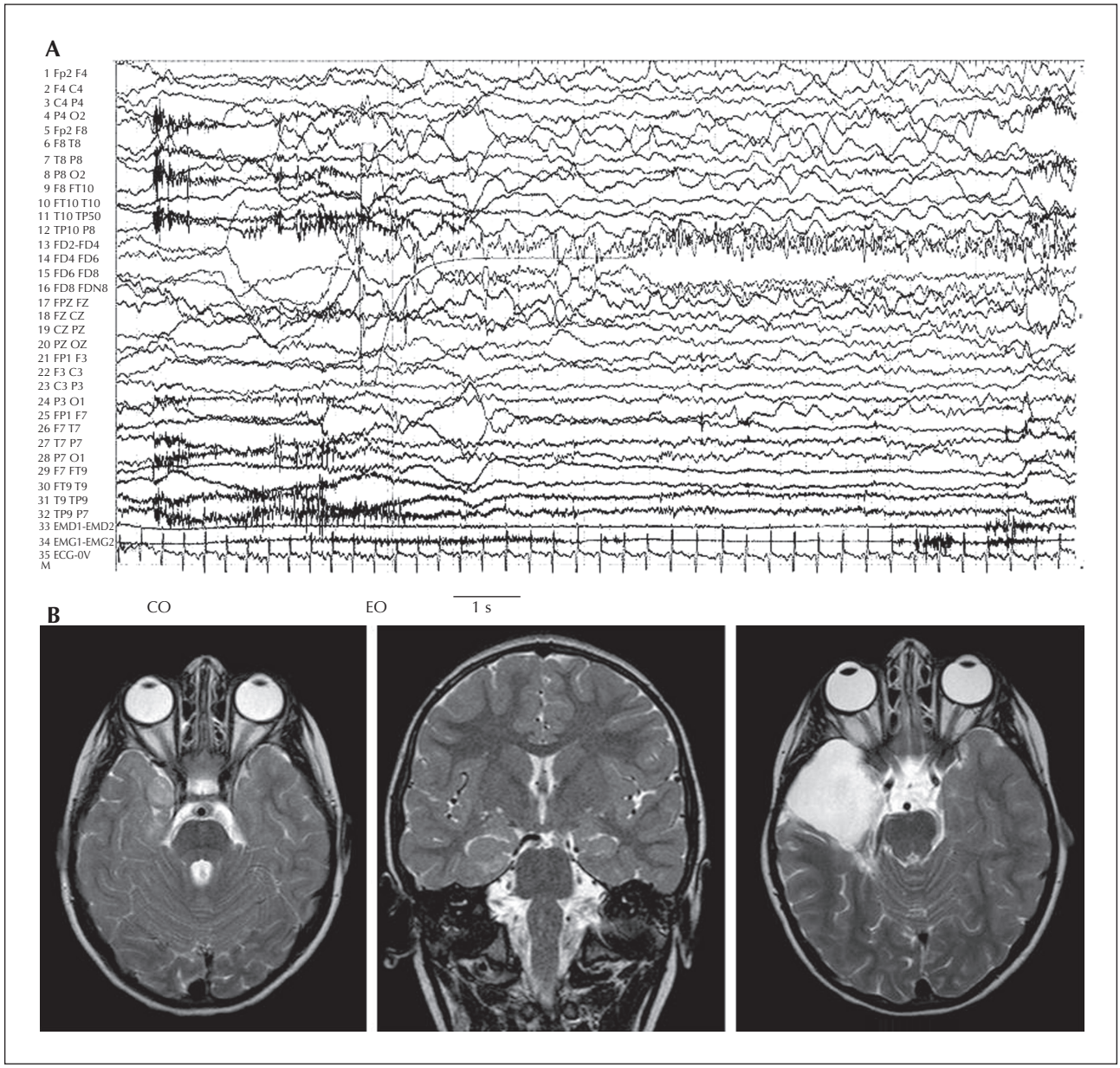


■ **Figure 1.** Patient 3. (A) Ictal EEG (*see video sequence 1*) showing electrical onset with left temporal delta activity after the clinical onset (longitudinal montage with supplementary temporo-basal electrodes). CO: clinical onset; EO: electrical onset. (B) Preoperative MRI showing left temporal pole dysplasia and postoperative follow-up showing a temporal pole resection.

the epileptic focus, and can arise from remote areas (perilesional onset in the case of a tumour or tuberous sclerosis complex, with difficulties in assessing the onset of seizures in the case of dual pathology).
Epilepsy freedom after surgery cannot always be considered an accurate "a posteriori" localizing element for two reasons:

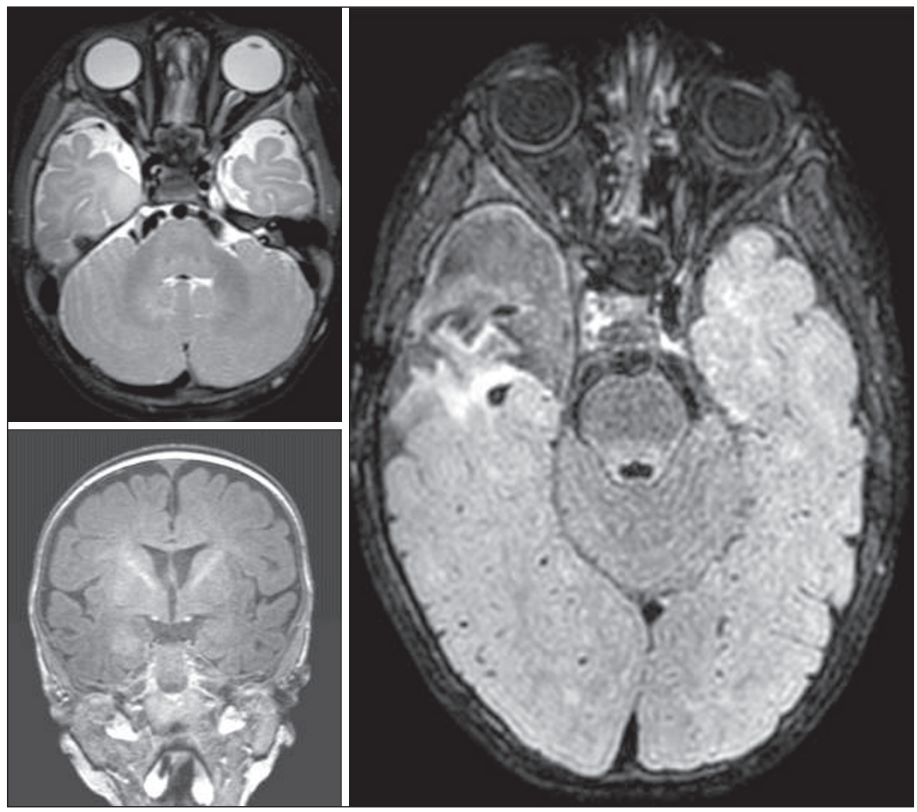
- the extent of the resection can go beyond the epileptic focus;
- in patients with neocortical epilepsy, mesial structures are frequently involved (and vice versa), but their implication may result from spreading rather than a primary seizure onset, thus the entire resection is not always required to obtain seizure freedom.

FOE was indicated to detect subtle seizures in patients with a temporal lesion, developmental or behavioural delay, and a few apparent seizures [12], but was never



■ **Figure 2.** Patient 15. (A) Scalp EEG recording combined with a foramen ovale electrode (FOE) (*see video sequence 2*). Spike-and-wave discharge is seen on the FOE, then diffuse delta activity on the right hemisphere (longitudinal montage with supplementary temporo-basal electrodes and the FOE, the electrode FO 2 being the most posterior). The seizure probably originates from the amygdala which is not explored with FOE. CO: clinical onset; EO: before electrical onset. (B) Preoperative contrast-enhanced MRI showing a right amygdala lesion and postoperative follow-up showing a temporal pole resection.

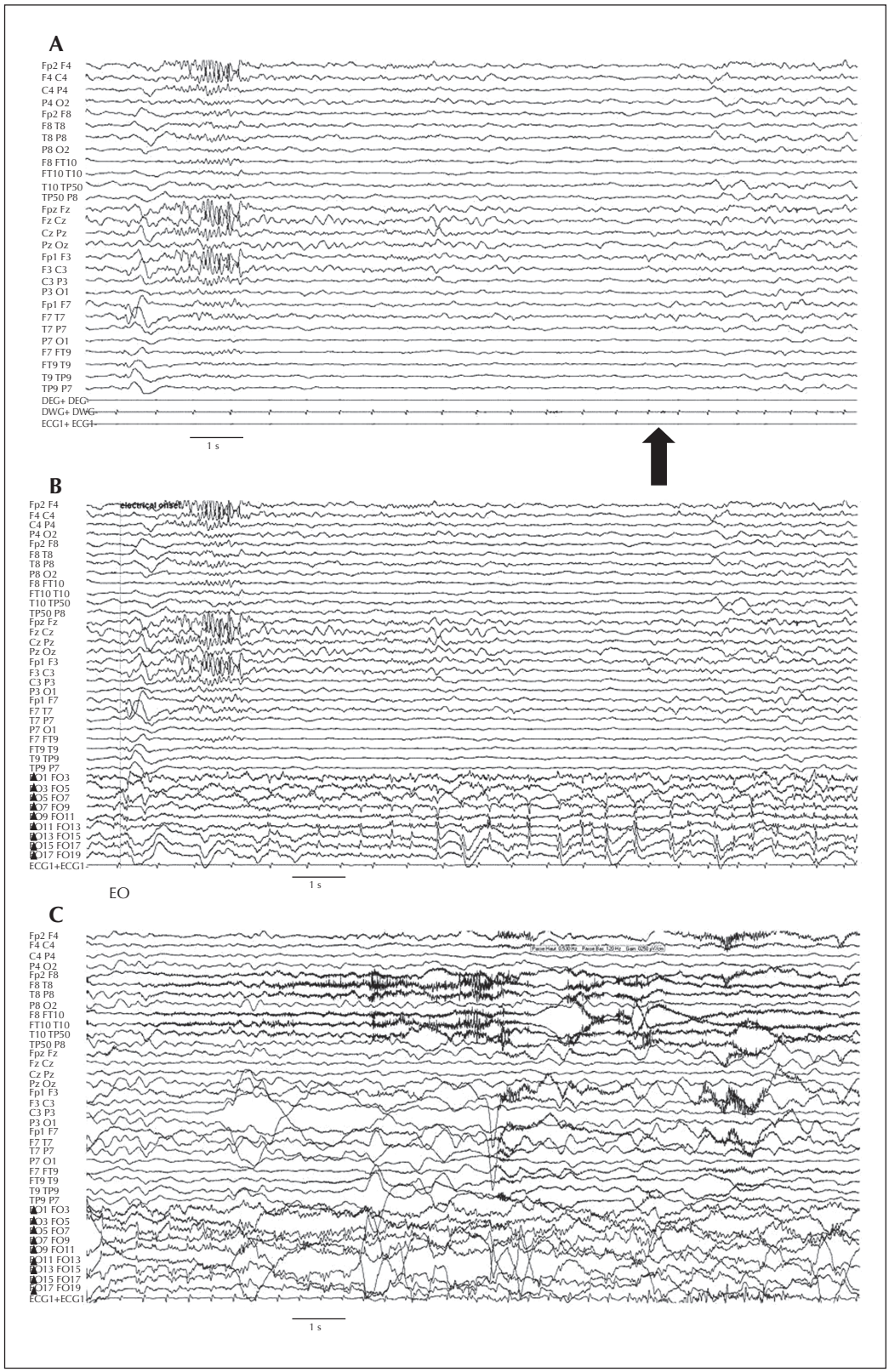
indicated to rule out a bitemporal epilepsy, which exceptionally occurs in children. SEEG was carried out in 43% of patients, allowing for greater precision of topography regarding the seizure onset, and thus based on reliable clinical semiology, as previously described [15]. In the literature, invasive exploration is undertaken in up to 50% of children with lesional epilepsies, irrespective of the type [21]. For temporal epilepsies, temporal lobe epilepsies are invasively explored in 7-27% of cases [22-24]. We have already pointed out that we tend to do more invasive explorations than most teams [25].



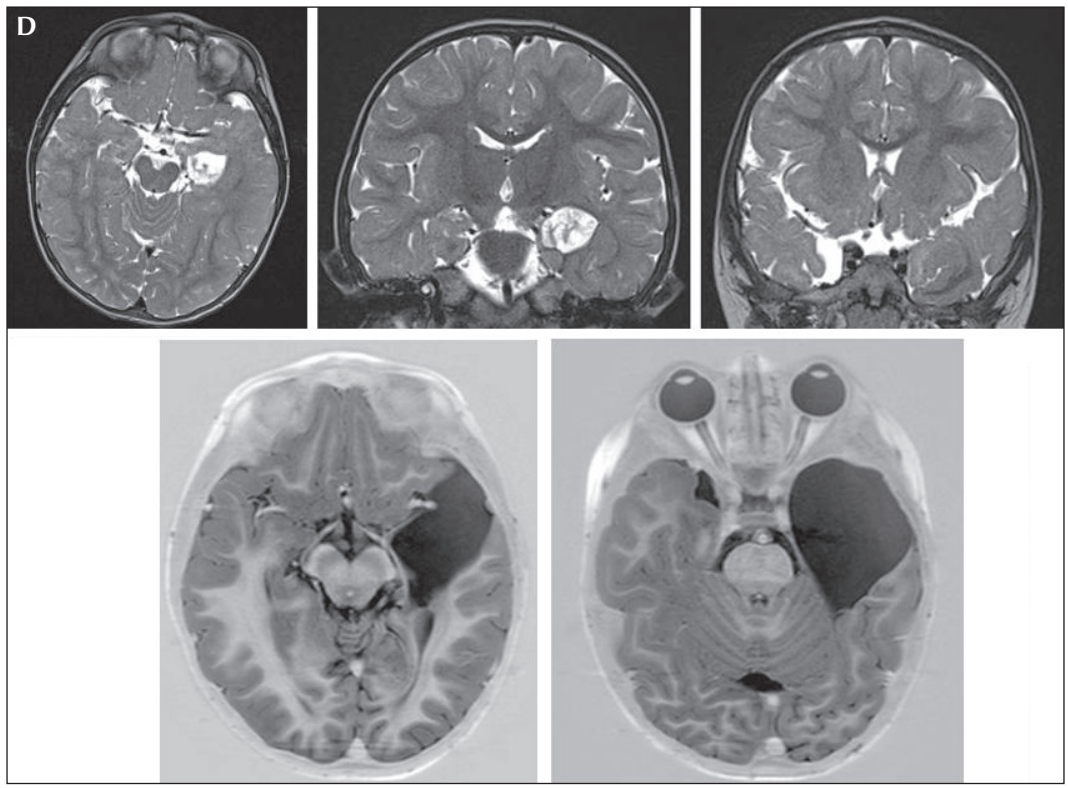
■ **Figure 3.** Patient 1. Preoperative MRI showing right mesial temporal thickening with hypersignal on T2-weighted sequence and postoperative MRI.

Specific semiology in children

The paediatric literature addresses temporal lobe semiology in general and focuses on changes in semiology with age. For most authors [2, 3], some clinical signs change with age (mainly motor manifestations), whereas others (neurovegetative features notably) are present at all ages; TLE semiology is very similar to that of adults after the age of six years [1]. In the literature, it appears that clinical signs frequently reported in preschool children are dominated by motor manifestations (IS, tonic and clonic seizures), described as either symmetrical [3] or asymmetrical [4]. Motor manifestations were not observed as frequently in our series. The first reason for this is that all authors pool together mesial seizures (unrelated to tonic or clonic seizures) with neocortical seizures (typically associated with motor manifestations that occur early). The second reason is that most authors include IS, which are frequent, as part of the motor manifestations. In our patients, three presented with IS, preceded by or associated with focal seizures. In a large series of localization-related IS, we did not find any specific correlation between IS and the temporal lobe, compared to other cortical areas [26]. Tonic-clonic generalization is infrequent in the literature [1-4, 20], as in our patients, with the exception of one child in whom a *FGF12* mutation, a possible aetiological factor, was found. Most authors underline the frequent occurrence of hypomotor seizures at this age [2, 3, 20, 27], as in our patients. Our series emphasizes the difficulty in making a diagnosis due to the frequency of so-called "subtle" seizures that are frequently overlooked and often discovered by looking carefully at the video recording and sometimes only by using depth electrode recordings. As for so-called "behavioural seizures", subtle and hypomotor seizures can be misdiagnosed as non-epileptic behavioural disorders especially in children with psychiatric comorbidity and/or intellectual disability, both frequently associated with epilepsy in general and TLE in particular [28]. This type of subtle seizure has previously been reported in preschool children irrespective of topography [29]. We have also previously reported that these subtle seizures may arise from the prefrontal lobe, making their localization value fairly poor in the absence of electrophysiological findings [30]. Automatisms are less complex in preschool children,



■ **Figure 4.** Patient 5. FOE recording combined with scalp electrodes (longitudinal montage with supplementary temporo-basal electrodes) helps distinguish awakening from seizure. To illustrate the utility of the FOE, the same seizure is presented with and without the FOE. For clinical signs see *video sequence 4*. (A) Recording without the FOE showing electrical onset before the clinical onset characterized by left anterior temporal and frontal rhythmic delta activity (arrow). (B) Same view with the FOE of electrical onset (EO) showing spike-and-wave discharge on FO starting on the anterior contacts (the electrode FO 1 being the most posterior). (C) Seizure continuation showing discharges of polyspikes on the FOE and delta activity on the left scalp electrodes.



■ **Figure 4.** Patient 5. (D) Preoperative MRI showing left choroid plexus papilloma and hippocampal sclerosis and image postoperative examination (*continued*).

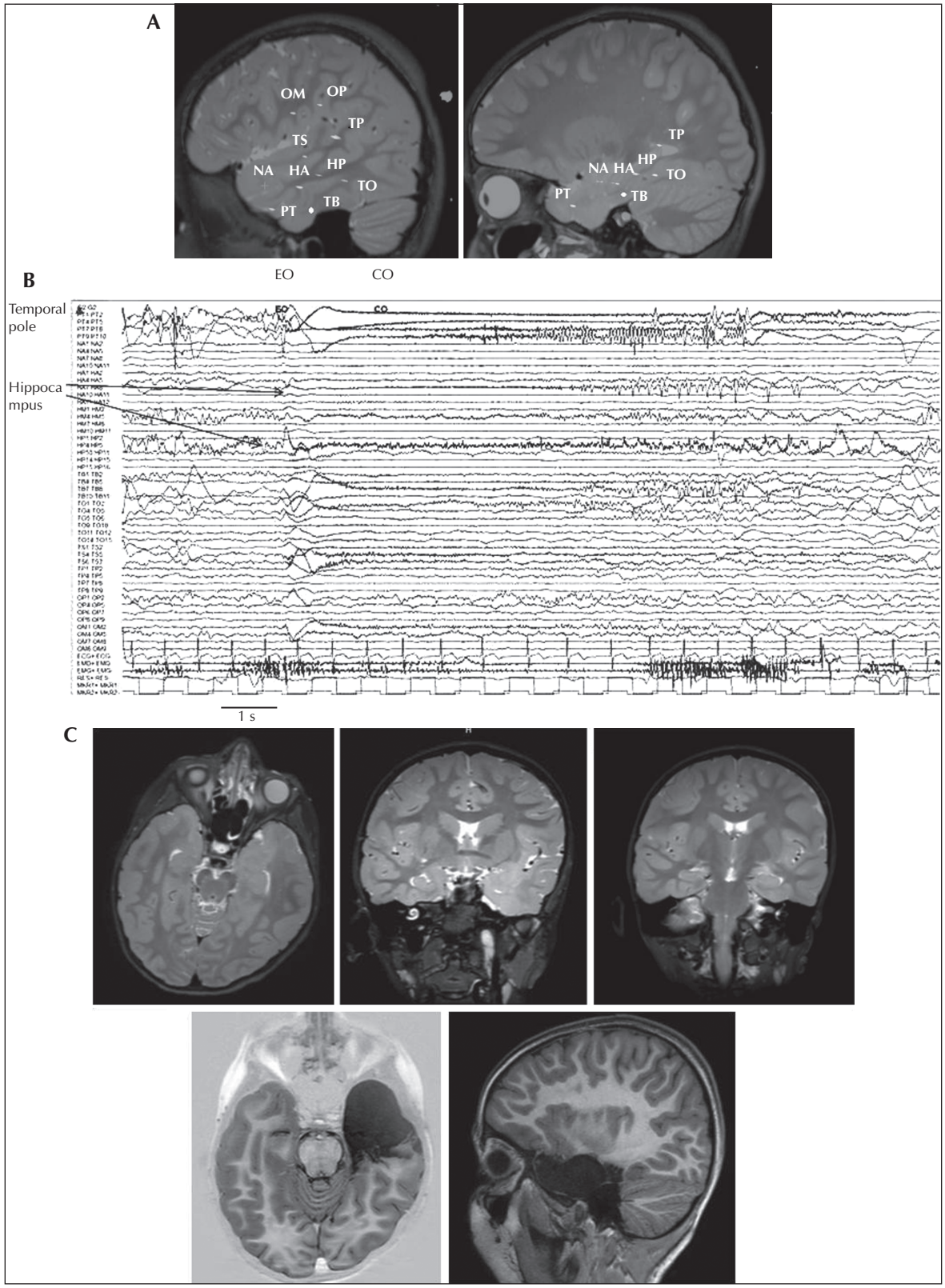
compared to older children. They are mainly oral, or subtle bimanual [2-4]. It is also notable that lateralizing ictal and post-ictal features are more frequent in older children [2]. In our experience, post-ictal paresis is rarely reported by the parents, but is found in 24% of patients during the video-EEG recording. Post-ictal aphasia is much more difficult to assess because of the basic speech status of the patient and post-ictal lack of cooperation or because of sleepiness after the seizure. In 22 children, however, a speech assessment was possible, and in only six, aphasia was found. The clinical signs that are reported to be unchanged relative to age are neurovegetative signs, emotional manifestations and auras [2, 3]. However, Fontana *et al*. underlined the rarity of auras in children younger than six years of age [27]. Regarding emotional signs frequently reported in TLE, we only observed negative emotions such as fear. None of our patients presented with positive emotions, unlike the 100 children reported by Fogarasi *et al*. [31]. For these authors, emotions have a localizing value for the temporal lobe in 26% and positive emotions have a lateralising value in the right hemisphere. The difference relative to our results is probably due to the younger age of our children. This is in accordance with our observation that auras are rare in infants and young children during focal seizures, irrespective of the localization [25]. Bitemporal epilepsies, which are significant in adult patients, are rarely encountered in children and were notably not encountered in any our patients [32].

Anatomical correlations

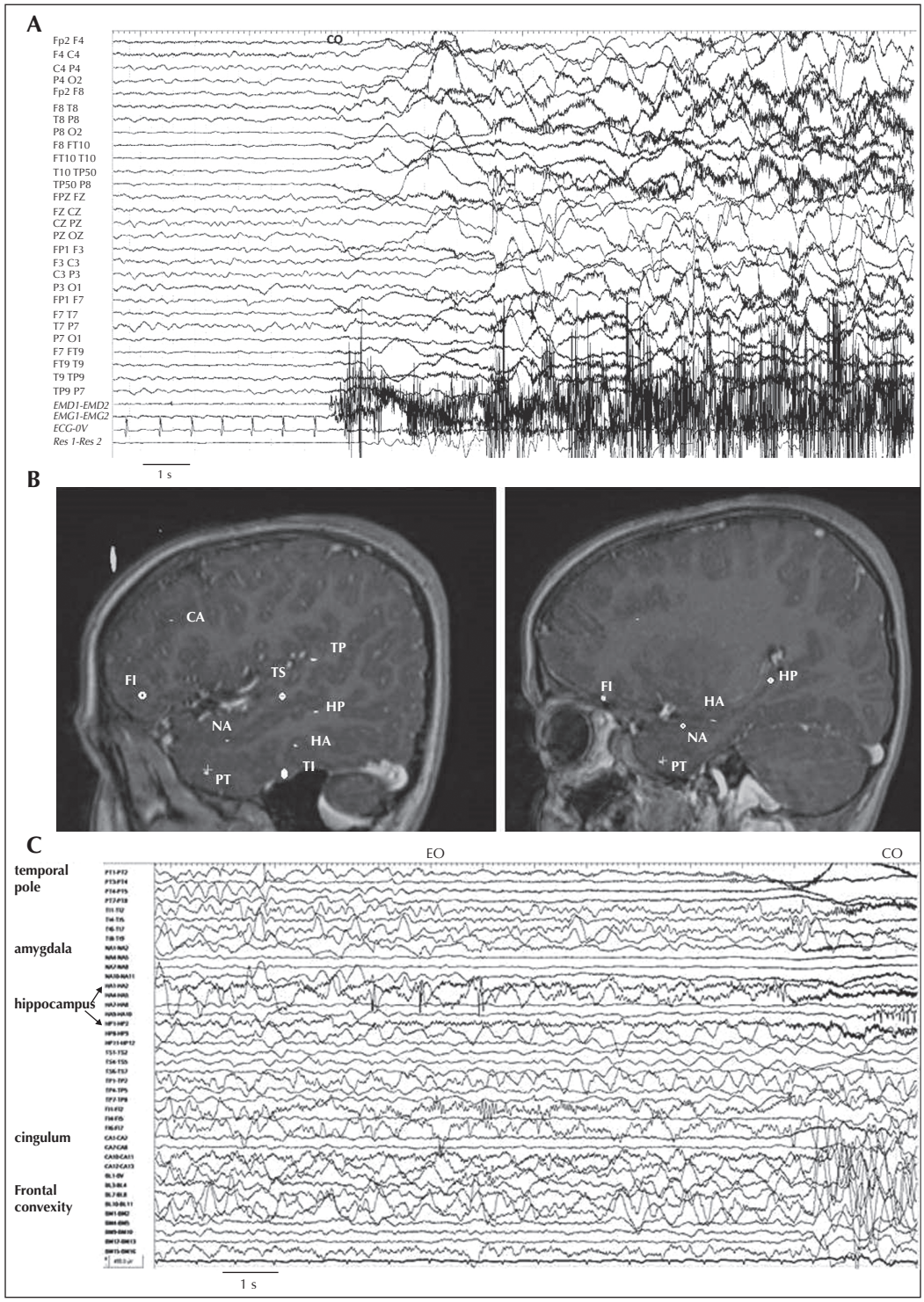
In adult patients, a topographic classification of TL seizures has been proposed [15]. Our series shows that, even in preschool patients, there are clinical seizure characteristics that support the hypothesis of precise topography, and that this classification can be used.

• *Mesial structures*

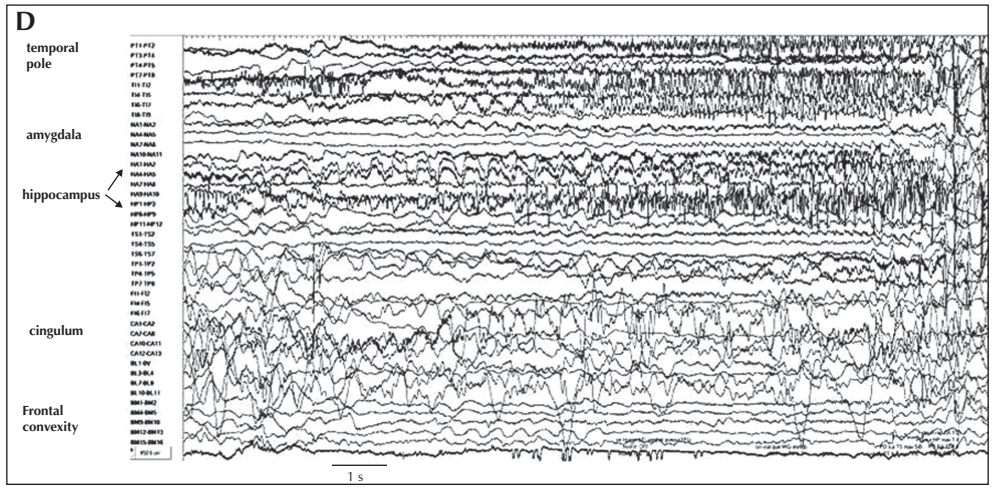
Topographic classification of TL seizures is particularly relevant to mesio-temporal epilepsies. We have shown that, even in toddlers, the semiology of mesio-temporal epilepsy can be very similar to that in adults [33-35], except for subjective signs that are missing in infants and pre-language children. In contrast, children can express some subjective signs, such as fear or pain, at



■ **Figure 5.** Patient 16. (A) SEEG with electrode implantation scheme (left: lateral view; right: hippocampal view). (B) Ictal SEEG (*see video sequence 5*) showing the ictal discharge almost restricted to the temporal pole. EO: electrical onset; CO: clinical onset. (C) Preoperative MRI showing left temporal pole blurring without hippocampal sclerosis and postoperative follow-up.



■ **Figure 6.** Patient 17. (A) Ictal EEG (longitudinal montage with supplementary temporo-basal electrodes). CO: Clinical onset. The ictal discharge is not visible because of the artefacts. (B) SEEG with electrode implantation scheme (left: lateral view; right: hippocampal view). (C, D) Ictal SEEG (*see video sequence 6*). (C) Electrical onset (EO) on the temporal pole with propagation to the hippocampus and the cingulum (CA 1-2), followed by slow waves on the frontal convexity, visible before clinical onset (CO).



■ **Figure 6.** Patient 17. (D) Seizure continuation showing a widespread temporal discharge and frontal slow waves on the convexity and the cingulum (*continued*).

as young as two or three years old, as shown in *video sequence 2*.

The occurrence of seizures with apnoea and desaturation as a dominant and/or isolated manifestation in infants is not very widely reported in adults but has been observed in children [29, 36]. Apnoea has been reported to be frequently associated with hypomotor seizures [29] and is significantly more frequent in young children with TLE [36]. More precisely, apnoea can appear as a predominant sign in children with mesio-temporal epilepsies. Three patients from our series had this type of seizure and the severity of the desaturation required oxygenotherapy at home. Regarding aetiology, there is a strong link with the existence of tumours, mainly ganglioglioma (two patients in our series and three previously reported patients) [37, 38]. Regarding the anatomical location for apnoea, the amygdala and the head of the hippocampus are clearly involved. Stimulation of these structures in adult patients, investigated using SEEG for epilepsy, can induce apnoea [39] and apnoea can be responsible for sudden unexpected death in epilepsy (SUDEP) or near missed SUDEP [40] in adult patients with complex partial seizures. These seizures, even when rare, must be identified and treated early due to the risk of infant apnoea syndrome.

● ***Pole structures***

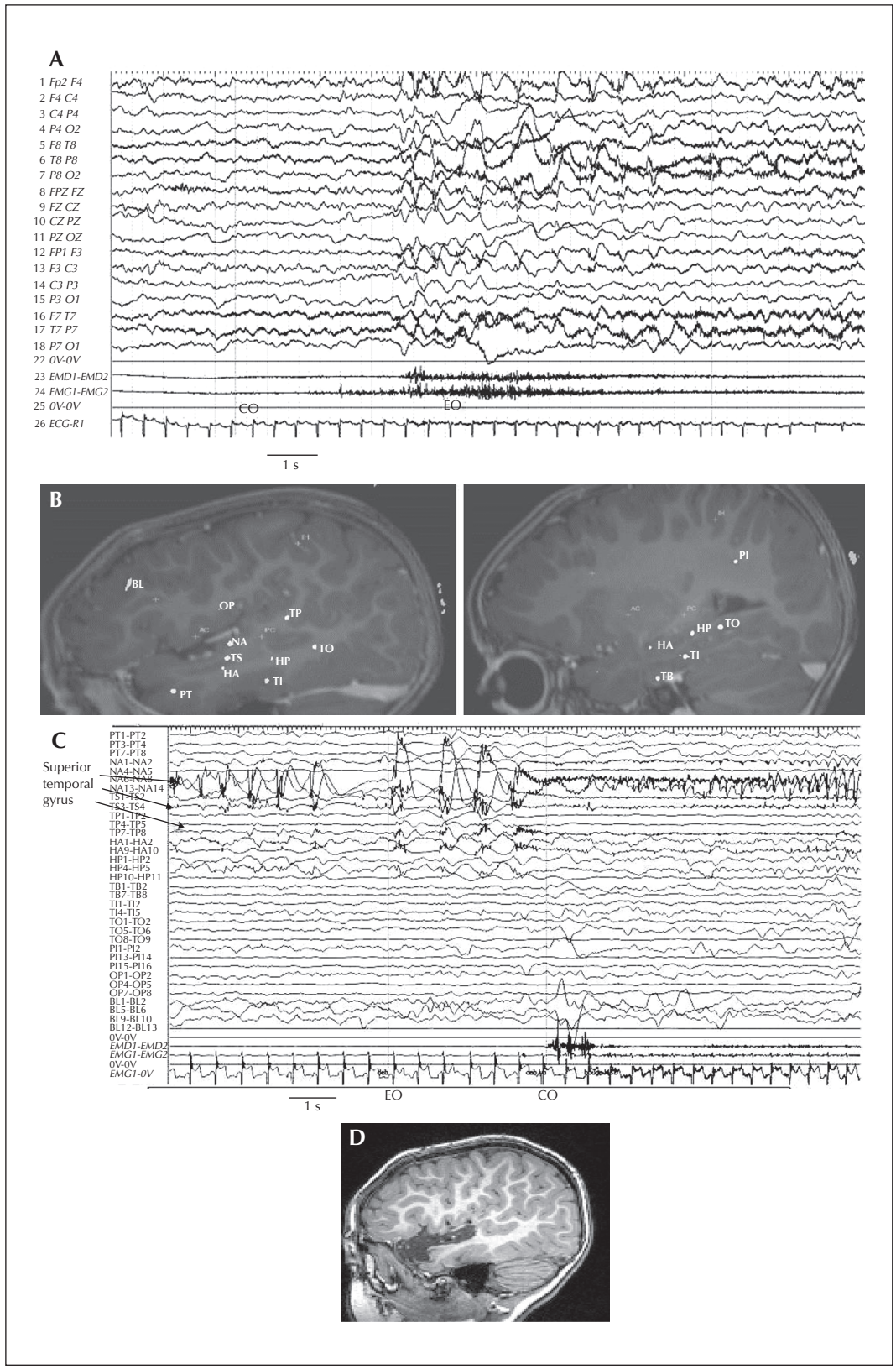
Temporo-polar epilepsies are not considered to have very specific semiology [41]. The most striking reported feature, compared to mesial epilepsies, is the early loss of consciousness. In our patients, seizures limited to the temporal pole were characterized by a behavioural modification, while other ictal features were related to propagation. In these cases, the apparent semiology was challenging to interpret as it results from a spread to other structures. One example is provided by our patient who presented with a hyperkinetic seizure; on SEEG, the seizure began within the temporal pole and then spread to the cingulum via the anterior hippocampus. Hyperkinetic seizures are classically related to the frontal lobe with different onset zones, such as the orbito-frontal or frontal mesial structures [42]. Their occurrence in TLE has previously been reported in adult patients [43, 44], but to our knowledge never in young children, except for the report by Brokhaus and Elger [1].

● ***Neocortical structures***

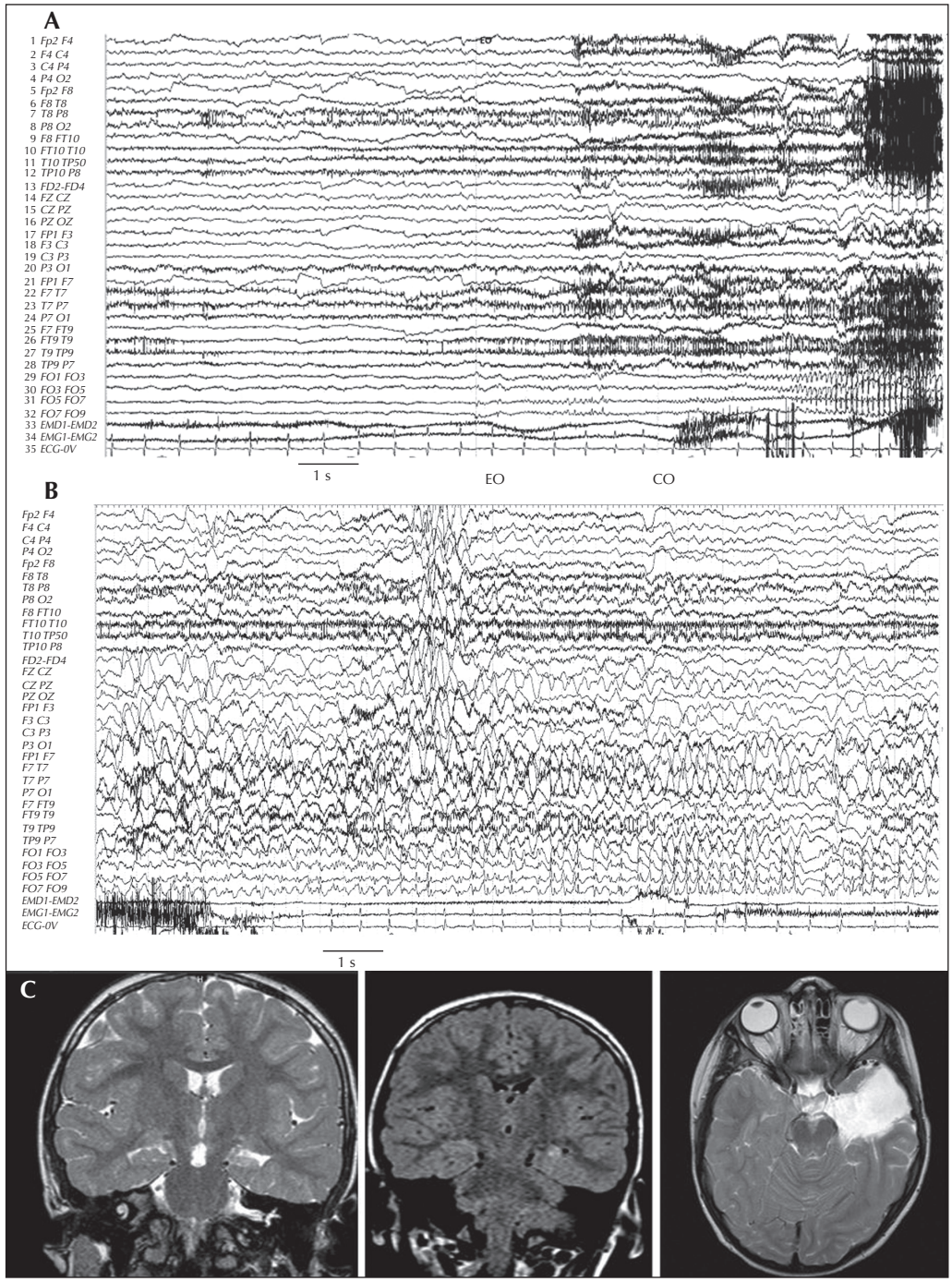
Children with temporal lateral seizures have non-specific semiology. This is also the case in adults. In the larger series in which semiology was objectively analysed, the initial loss of contact was significantly more frequent in temporal lateral than temporal mesial seizures [15]. The other statistically significant features were early unilateral clonic movements, a shorter duration, and the occurrence of tonic-clonic generalization [15, 45].The most characteristic feature of temporal lateral epilepsies is the auditory aura which significantly differentiates them from mesial epilepsies [15]. Auditory illusions or hallucinations are clearly only reported exceptionally by preschool children.

Conclusion

This series illustrates that in young children with TLE, fairly good anatomical-electroclinical correlations are possible, which contrasts with established opinions



■ **Figure 7.** Patient 19. (A) Surface recorded seizure (longitudinal montage) showing diffuse spike-and-wave discharge, more pronounced on the right temporal electrodes, visible after the clinical onset. EO: electrical onset; CO: clinical onset. (B) SEEG with electrode implantation scheme (left panel: lateral view; right panel: hippocampal view). C) Ictal SEEG (*see video sequence 7*) showing electrical onset (EO): polyspikes on the superior temporal gyrus (STG), maximum on the anterior part (NA 9-13), followed by a fast discharge in the same region. (D) Postoperative MRI showing the temporal superior gyrus and amygdala resection.



■ **Figure 8.** Patient 26. (A, B) Ictal EEG with FOE (longitudinal montage with supplementary temporo-basal electrodes, the electrode FO 1 being the most posterior) (*see video sequence 8*). (A) Electrical onset (EO) at the anterior contacts of FOE; the child says that she is having a seizure. CO: clinical onset. (B) Seizure continuation showing spike-and-wave discharge on both temporal electrodes and FOE; the discharge is faster on FOE. (C) Preoperative MRI showing polar and temporo-basal white/grey matter blurring with hippocampal sclerosis and postoperative follow-up.

[29, 46]. The predominance of motor manifestations reported in young children was not found in our series, probably because previous authors included IS and mesial- and lateral-onset epilepsies were pooled together. Temporal lobe semiology, mainly mesio-temporal, has some similarities with the semiology in adults and can be identified using electro-clinical data; the particularities in young children are the occurrence of overlooked subtle seizures and the occurrence of seizures with apnoea which have a clear mesio-temporal origin and strong link with so-called "long-term epilepsy-associated tumour". ■

Supplementary material.
Summary slides accompanying the manuscript are available at www.epilepticdisorders.com.

Acknowledgements and disclosures.
We thank the physicians who referred the patients for epilepsy surgery, Pr Veronique Sébille (Nantes University) for statistical analysis, our EEG technologists Mrs M. Benghezal and Mrs I. Cheramy, and Mrs. Aisha Yu, a native English speaker, for careful revision of the manuscript.
None of the authors have any conflicts of interest to disclose. None of the authors received any financial support.

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Legends for video sequences

Signed consent forms authorising video publication have been obtained for all identifiable patients.

Video sequence 1

(Patient 3) shows a nine-month-old boy with left temporo polar dysplasia. At birth, he had episodes with throat noises. No diagnosis was made. At the age of five months, he started having daily seizures with motor arrest, orientation to the left, nausea, chewing and reddening of the face. Clinical examination showed symmetrical motricity and a mild developmental delay. Seizures on video consisted of staring, throat noises, oro-alimentary automatisms, nausea, reddening of the face, paresis of the right arm, orientation to the left, and a suspicion of visual field loss on the right.

Video sequence 2

(Patient 15) shows a four-year-old girl with right amygdala ganglioglioma. Seizures started at the age of 12 months, and were described as staring and pallor. At the time of the recording, the girl was right-handed, with normal neurological examination, good social interactions, and normal schooling. She suffered from attention deficit/hyperactivity disorder. During the seizures, recorded on video, she was able to warn her mother, saying that she was having a seizure and that she was afraid, after which she grasped her mother and subsequently experienced a loss of contact, modified breathing, and cyanosis.

Video sequence 3

(Patient 1) shows a six-month-old boy with right temporo mesial ganglioglioma. Episodes started at the age of two months associated with loss of contact, pallor, hypotonia, facial cyanosis, and desaturation, necessitating oxygen treatment at home. No precise diagnosis was made. The neurological examination and the interictal EEG were normal. Seizures recorded on video consisted of staring preceding the desaturation. Right temporal rhythmic delta activity is visible. Note the respiratory arrest and desaturation on the lower two lines of the recording.

Video sequence 4

(Patient 5) shows a 2.5-year-old boy with left choroid plexus papilloma and hippocampal sclerosis. The seizures started at the age of 3.5 months. The parents described a reddening of the face, staring, and chewing. At the time of the recording, he was right-handed, with normal neurological examination. He had disabling hyperactivity. The parents reported rare seizures, about one a month, but eight were recorded during sleep over five days. The parents were not aware at all that the child had been having seizures during sleep. On video, recorded seizures were discreet with awakening, tachycardia and chewing.

Video sequence 5

(Patient 16) shows a six-year-old boy with left temporopolar dysplasia. Seizures began at age three without any obvious trigger; the first seizures were described as staring, face reddening and chewing and occurred weekly. Neurological examination showed right-handedness with normal cognitive development; he had mild syntaxic and fine motor difficulties with normal behaviour and sociability. SEEG revealed subtle seizures from the temporal pole with minimal propagation that are shown on the video; the patient stopped talking, experienced motor arrest, and had some manual automatisms.

Video sequence 6

(Patient 17) shows a 4.8-year-old boy with right temporopolar dysplasia. The first seizure started at the age of four months and the description by the parents was: face cyanosis, anarchical gesticulation of the four limbs and clonic movements of the eyelids. From the onset to the time of recording, the seizure rate was high with up to 20 seizures per day. He has developed a mental deficit with a language delay and presented with some autistic spectrum features and aggressiveness. On video recordings, all the seizures were stereotypical: he rushes to his parents, then becomes anarchically agitated, gesticulating the trunk and four limbs, initially symmetrically and secondarily predominant in the right hemibody with hypertonia of the left arm; the eyes are positioned to the right with facial pallor and at the end of the seizure, facial cyanosis and chewing is evident. Post-ictally, he has paresis of the left arm.

Video sequence 7

(Patient 19) shows a 25-month-old boy. At one month of life, he had a first epileptic seizure characterized by staring, head deviation to the left and changes in respiratory function. Neurological examination showed right-handedness with normal motor development. He had a mild developmental delay including speech delay. MRI was normal. SEEG was performed at the age of 25 months to explore the hypothesis of an epileptogenic zone either in the right temporal region (which could be internal or within the depth of a sulcus) or insular. The video shows changes in respiratory function, shaking of the head, an often deviated gaze to the right, hypertonia of the left arm and leg, and post-ictal paresis of the left part of the body.

Video sequence 8

(Patient 26) shows a 5.5-year-old girl with left temporopolar dysplasia and hippocampal sclerosis. During the first months of life, she presented with some faintness described as facial pallor and an upward gaze while drinking from her bottle. At the age of 15 months, she had a complex febrile seizure, 10 days after a vaccination, and two years later started to have focal seizures. From the age of 3.5 years, she had seizures limited to a headache and/or an epigastric sensation. Clinical examination showed right-handedness with normal examination including language, and normal cognitive development. The video shows longer seizures that start in the same way, consisting of motor activity arrest, pallor, chewing, followed by clear-cut aphasia which remained for a couple of minutes post-ictally. This semiology was explained by mesial onset and lateral propagation.

Key words for video research on www.epilepticdisorders.com

Phenomenology: staring and oroalimentary automatisms (*video 1*), fear (*video 2*), apnoea (*video 3*), behavioural arrest (*video 4*), subtle seizure (*video 5*), hyperkinetic seizure (*video 6*), hypertonia of the right hemibody (*video 7*), pallor, chewing, aphasia (*video 8*)
Localization: mesio-temporal (*videos 1, 3, 4*), amygdala (*video 2*), temporal pole (*videos 5, 6*), neocortical temporal (*video 7*), left mesio-temporal and neocortical (*video 8*)
Syndrome: temporal lobe epilepsy
Aetiology: focal cortical dysplasia type IIIb (*videos 1, 5*), ganglioglioma and hippocampal sclerosis (*video 2*), ganglioglioma (*video 3*), plexus choroid papilloma and hippocampal sclerosis (*video 4*), focal cortical dysplasia type IIIa (*videos 6, 8*), focal cortical dysplasia type IIb (*video 7*)

TEST YOURSELF

(1) What are the particularities of temporal lobe seizures in young children?

(2) What are the characteristics of mesio-temporal seizures in preschool children?

(3) Which aetiology suggests the occurrence of seizures with apnoea?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".