Symptoms in focal sensory seizures
Clinical and electroencephalographic features

Carlo Di Bonaventura[a], Anna T. Giallonardo[a,*],
Jinane Fattouch[a], Mario Manfredi[a,b]

[a] *Department of Neurological Sciences, Epilepsy Unit, University of Rome ''La Sapienza'',
Viale dell'Università 30, 00185 Rome, Italy*
[b] *Neuromed Institute of Pozzilli (IS), University of Rome ''La Sapienza'', 00185 Rome, Italy*

Summary   *Purpose*: Aura is a brief subjective symptom that may represent the initial manifestation of a partial epileptic seizure with objective signs or constitute the entire epileptic attack (focal sensory seizure (FSS)). We studied the electro-clinical features of FSSs recorded in 28 patients. *Methods*: Using long-term surface video-EEG recordings, we examined 28 patients (from a consecutive series of 64) with stereotyped FSSs and complex partial seizures (CPS) preceded in at least one instance by identical subjective manifestations (overall 255 FSSs and 39 CPS were recorded). FSSs were subdivided according to the type of sensation into somatosensory, visual or oculosensory, viscerosensory, experiential, cephalic and diffuse warm sensations. The EEG discharges accompanying FSSs were examined by two of the authors either blinded as to the type and timing of the seizure, or unblinded, i.e. after receiving complete clinical information including timing of the patient's warning. *Results*: The ictal pattern accompanying FSSs was identified blind in 13 patients and unblind in 8 patients. In seven patients, the ictal discharge remained undetected. In the cases with recognizable ictal abnormalities, two main patterns could be distinguished, static and dynamic. FSSs whose ictal discharge could be recognized by blind EEG examination more frequently consisted of somatosensory and visual or oculosensory manifestations, and the discharge generally involved the centro-parieto-occipital regions. The ictal discharge of viscerosensory and experiential FSSs more easily remained undetected; when identified, it generally involved the fronto-temporal regions. *Conclusions*: FSSs are often accompanied by ictal abnormalities recognizable on surface EEG. A thorough knowledge of their EEG accompaniments may be a useful diagnostic aid in patients with partial epilepsy.
© 2004 Published by Elsevier Ltd on behalf of BEA Trading Ltd.

Introduction

According to the 1981 definition of the ad hoc Commission on Classification and Terminology of the International League against Epilepsy,[1] aura is that part of a seizure which takes place before consciousness is impaired and can be subsequently

*Corresponding author. Tel.: +339-6-49914727;
fax: +339-6-49914302.
E-mail address: annateresagiallonardo@uniroma1.it
(A.T. Giallonardo).

recollected. In simple partial seizures, the aura may constitute the entire epileptic attack; in complex partial seizures (CPS) it represents the initial manifestation before consciousness begins to fade. While the ad hoc Commission definition fails to distinguish subjective from objective ictal manifestations, Luders et al.[2] have proposed a more restrictive definition, limiting the term aura to the subjective warning symptoms at the beginning of a seizure.

Auras are common phenomena, particularly in partial epilepsies of temporal origin. The reported incidence varies from 20 to 80%.[3—7] Aura can be recognized as an epileptic manifestation only when the subjective symptoms are followed by objective, unmistakably epileptic signs such as motor or autonomic events or impaired consciousness, or when simultaneous electroencephalographic (EEG) recordings document irrefutable ictal discharges. In agreement with the classification of Luders et al.[2] in this paper we use the term aura to describe a seizure consisting only of symptoms perceived by the patient alone. According to the new ILAE classification proposal,[8] we refer here to these events as focal sensory seizures (FSSs).

Compared with the clinical manifestations of auras,[9—13] the EEG accompaniments have received less attention, possibly because they are not always easy to detect by surface electrodes.[14,15]

Our primary aim in this study was to clarify the clinical and surface EEG features of FSSs. We therefore sought to determine the extent of the ictal EEG accompaniments and provide criteria identifying them. We selected for study patients with video-EEG recorded FSSs undergoing presurgical assessment. To avoid including events of non-epileptic origin (e.g., psychogenic seizures), we selected patients whose recordings showed stereotyped FSSs, followed—on at least one occasion—by indisputable motor or autonomic signs or by unresponsiveness.

Material and methods

From a total of 1150 patients admitted to our Epilepsy Clinic over the 5-year period, 1995—2000, 64 consecutive patients with drug-resistant partial epilepsy were referred to the video-EEG laboratory. From this group we selected 28 patients (14 men, and 14 women) aged 21—53 years (mean age 33 years) who had, during the recording session, purely subjective seizures similar to the episodes reported in clinical history. Their seizures began at the age of 4—31 years (mean age 13 years), and the duration of the epilepsy ranged from 1 to 39

years (mean 20 years). Of the 28 patients, 17 had simple and 11 had complex seizures; and 11 of the 28 patients had secondary generalization. The frequency of seizures ranged from 3 to 4 events per month to 2—3 per day. In 14 patients epilepsy was symptomatic and in 14 cryptogenic. In symptomatic cases, the lesion was constituted by mesial sclerosis (4 cases), focal dysplasia (3 cases), neoplasm (3 cases), anoxic-ischemic focal encephalopathy (2 cases), cavernoma (1 case), and an undetermined focal lesion (1 case). The lesion was located in nine cases in the right and in three cases in the left hemisphere. In two patients out of the four with mesial sclerosis the lesion was bilateral. In cryptogenic cases the interictal EEG abnormalities were located in five cases on the right, in six on the left hemisphere, and in three cases were bilateral; one patient had a normal interictal EEG (Table 1).

After detailed clinical and neuroradiological examination, including magnetic resonance imaging scan (MRI), patients underwent long-term video-EEG monitoring (Biologic System, Telefactor or Grass, 19 channels, International 10—20 System) under constant surveillance of experienced technical and medical personal ready to interact with the patient on occasion of the seizures. The recordings were obtained during the daytime and lasted for 8 h a day. Patients were asked to signal when their subjective sensations began and ended, and the episodes were considered FSSs only when the patient could respond to external cues during the ictal sensation and was able to describe the events in detail after the attack. Criteria for inclusion were a clinical history of recurring auras, at a frequency of at least two a month; stereotyped ictal clinical features; video-EEG recording of at least one episode of purely subjective symptoms immediately signaled by the patient, analogous to the episodes reported in the clinical history; besides, a video-EEG recording of at least one episode of subjective symptoms accompanied or followed by unmistakably epileptic objective signs such as motor or autonomic manifestations or impaired consciousness (hereafter referred to as a ''major episode''). Patients were excluded when the diagnosis of epileptic aura remained in doubt, the subjective symptoms changed in subsequent seizures or were in disagreement with clinical history, or the ictal EEG tracings were obscured by artifacts related to the warning signal or to involuntary ictal movements. In line with Palmini and Gloor,[7] FSSs were subdivided according to the type of sensation, in somatosensory, simple visual or oculosensory, viscerosensory, cephalic, diffuse warm, and experiential sensations. We consider this subdivision more detailed and clinically oriented than the

Table 1 Clinical and electroencephalographic characteristics of FSSs.

Patient	Sex	Age	Clinical features in FSSs	Number of recorded FSSs	Mean FSS duration (s)	EEG	EEG features pattern	Localization
1	M	48	Viscerosensory	40	5	+	Focal voltage reduction → rhythmic theta	Fp2-F8-T4 F4-C4
2	M	29	Viscerosensory	1	16	+	Low voltage fast activity → rhythmic theta → rhythmic delta	T4-T6-O2
3	F	26	Visual or oculosensory	33	8	+	Focal spike-and-waves complexes	C3-P3
4	M	27	Visual or oculosensory	8	8	+	Focal voltage reduction → rhythmic theta → rhythmic delta	F8-T4-T6-O2
5	F	53	Viscerosensory	14	8	±-	Focal voltage reduction → rhythmic theta → rhythmic delta	F8-T4-T6 F4-C4
6	F	41	Experiential	18	7	−	—	——
7	F	23	Visual or oculosensory	20	42	+	Focal spike-and-waves complexes	T6-P4-O2
8	M	44	Viscerosensory	1	6	±	Focal voltage reduction → rhythmic theta	F8-T4-T6
9	M	26	Viscerosensory	4	16	+	Focal voltage reduction → rhythmic theta → rhythmic delta	F8-T4-T6-O2
10	F	29	Viscerosensory	1	10	−	—	—
11	M	30	Experiential	1	71	+	Low voltage fast activity → rhythmic theta → rhythmic sharp waves	F8-T4-T6
12	M	50	Somatosensory	1	110	+	Rhythmic theta → sharp waves → spike-and-waves	F3-C3
13	F	27	Viscerosensory	2	4	−	—	—
14	M	51	Somatosensory	25	4	−	—	—
15	F	21	Experiential	2	5	±	Focal voltage reduction → rhythmic theta	Fp1-F7-T3-T5
16	M	30	Experiential	2	12	+	Low voltage fast activity → rhythmic theta	F8-T4-T6-O2
17	F	26	Warmth	1	12	±	Focal voltage reduction → rhythmic theta	F8-T4-T6 Fp2-F4
18	M	23	Experiential	2	2	−	—	—
19	F	38	Experiential	2	15	+	Focal voltage reduction → rhythmic theta	F8-T4-T6
20	M	38	Cephalic	2	4	±	Rhythmic theta	F8-T4 F4-C4
21	F	31	Viscerosensory	6	5	−	—	—
22	F	25	Visual or oculosensory	1	12	+	Focal voltage reduction → rhythmic theta → rhythmic delta	F8-T4-T6
23	M	39	Experiential	20	6	±	Focal voltage reduction → rhythmic theta → rhythmic delta	F8-T4-T6
24	M	41	Cephalic	10	15	+	Low voltage fast activity → rhythmic theta	F8-T4-T6
25	M	24	Viscerosensory	6	7	±	Rhythmic theta	Fp1-F7-T3
26	F	26	Somatosensory	1	7	+	Focal voltage reduction	C4-P4 T4-T6
27	F	26	Viscerosensory	10	8	±	Focal voltage reduction → rhythmic theta	F8-T4-T6
28	F	29	Viscerosensory	21	4	−	—	—

classification proposed by the ad hoc Commission[1] and Luders et al.[2]

Two authors (C.D.B. and A.T.G.), separately, first inspected the ictal EEG tracing of each patient blindly, i.e. in random order, without the aid of the video tape, knowing only that the tracing belonged to a patient under presurgical examination, possibly experiencing a subjective seizure. The same EEG were subsequently reviewed unblinded, i.e. after the examiner had received detailed clinical information including age, duration and etiology of the epilepsy, type of seizure, interictal and ictal EEG recordings of a major episode, and the presence of a recorded episode including the exact time of the patient's warning. The examiners were asked to identify under blinded and unblinded conditions the EEG correlates of FSSs. If a definite EEG correlate could be recognized, the following features were recorded: (a) location and number of electrodes involved at onset; (b) pattern and frequency of discharges; and (c) duration of electroencephalographic changes. FSSs were then subdivided according to the examiners' ratings into three categories: EEG [+], i.e. episodes identified during blind examination (subsequently confirmed unblinded); EEG [±], episodes identified only after unblinded examination; and EEG [−], episodes with no recognizable EEG correlate under blinded or unblinded conditions.

Results

We recorded 255 FSSs in 28 patients (range 1—40 per patient, mean 9 per patient). All patients' FSSs by definition exhibited stereotyped clinical features. FSSs with definite electrographic correlates also exhibited stereotyped EEG findings (pattern and localization) (Table 1).

Clinical manifestations

According to the classification of Palmini and Gloor (1992),[7] 3 patients had somatosensory FSSs (27 episodes), 4 patients simple visual or oculosensory FSSs (62 episodes), 11 patients viscerosensory FSSs (106 episodes), 2 patients cephalic FSSs (12 episodes); 1 patient a diffuse warm FSS (1 episode); and 7 patients experiential FSSs (47 episodes). In 8 patients, two different subjective sensations were reported in close succession, the most common combination being viscerosensory and experiential sensations (5 patients). Other combinations were viscerosensory and visual (or vice versa) (2 patients), and experiential and visual sensations (1 patient). In these cases, FSSs were categorized according to the first reported sensation, which remained unchanged in the subsequent seizures.

Single manifestations differed widely within the same category. Somatosensory sensations were described as ''a feeling of tremor in one arm'' (1 patient), paresthesias in the scrotal region (1 patient) or in the hands (1 patient). Visual sensations were described as negative (''I cannot see . . . on one side . . .'' in one patient and ''a narrowing of the visual field'' in another patient) or positive phenomena (''a light like a white flash . . . a dizziness . . . on one side . . .'' in one patient); and an oculosensory sensation was reported as ''the feeling that my eyes are moving'' (one patient). The most frequent visceral sensations were epigastric, variously described as ''a hole in the stomach . . . ,'' ''a pain . . . ,'' ''something in the stomach . . . ,'' and ''the stomach coming up . . . .'' (seven patients), nasal or pharyngeal (''a smell . . . a taste . . . ,'' in one patient); cardiac, described as a feeling of accelerated heart beats (two patients) or buccal, described as ''a swollen mouth . . . the sensation of mounting saliva'' in the remaining two patients. Two patients had cephalic sensations: one said they were ''indescribable'' and the other referred to a ''feeling of lightness.'' Only one patient reported a diffuse sensation of warmth within the chest. Seven patients reported experiential phenomena: the classic ''deja vu''—''deja vecu'' (two patients), anxiety and fear (two patients) undefined dizziness (one patient), undefined olfactory sensation (one patient) or a ''feeling of being lost'' (one patient).

In every patient at least one recorded major episode began with the same sensation(s) as FSSs. In the 28 patients 39 of these major episodes (1—6 per patient) were observed. In addition, 9 patients had 15 major episodes not preceded by subjective sensations but showing objective manifestations comparable to those in seizures starting with a subjective warning. None of the patients had convulsive seizures.

Clinical-EEG correlates

Because the stereotyped ictal manifestations and their EEG correlates remained appreciably unchanged in subsequent episodes in the same patient, we analyzed the findings for each patient irrespective of how many FSSs were recorded. We distinguished clinical-EEG correlates of three types.

EEG [+] seizures
In 13 patients (124 episodes) FSSs were accompanied by definite changes in the EEG tracing, recognizable by blinded and unblinded inspection. Two patterns could be distinguished, static and dynamic

Table 2 Ictal EEG patterns.

	EEG [+]	EEG [±]
Static pattern		
Focal voltage reduction	1 (1)	—
Rhythmic theta waves	—	2 (8)
Focal spike-and-waves complexes	2 (53)	—
Dynamic pattern		
Focal voltage reduction → rhythmic theta waves	2 (42)	4 (14)
Focal voltage reduction → rhythmic theta waves → rhythmic delta waves	3 (13)	2 (34)
Low voltage fast activity → rhythmic theta waves	2 (12)	—
Low voltage fast activity → rhythmic theta waves → rhythmic delta waves	1 (1)	—
Low voltage fast activity → rhythmic theta waves → rhythmic sharp waves	1 (1)	—
Rhythmic theta waves → sharp waves → spike-and-waves	1 (1)	—

No. of patients (No. of FSSs).

(Table 2). The static pattern (three patients) consisted of a focal isolated voltage reduction or of focal rhythmic, spike and wave complexes (Fig. 1). These abnormalities suddenly interrupted the background activity and lasted throughout the seizure: when the episode ended the EEG abruptly returned to the preceding pattern.

The dynamic pattern (10 patients) was characterized by a EEG activity that changed rapidly as the seizure evolved. Close inspection showed three possible changes: a focal voltage reduction followed by recruiting theta or theta—delta waves (Fig. 2); focal low-voltage fast activity followed by recruiting theta or theta—delta waves; or focal recruiting theta waves followed by sharp waves or spike-and-wave complexes. The abnormalities began and subsided suddenly, emerging clearly from background activity.

EEG [±] seizures

In 8 patients (56 episodes) FSSs were accompanied by subtle EEG changes identifiable only by unblinded inspection. Once the ictal EEG pattern had been identified in one episode, the same pattern could be easily distinguished during the ensuing seizures. Static (two patients) and dynamic (six
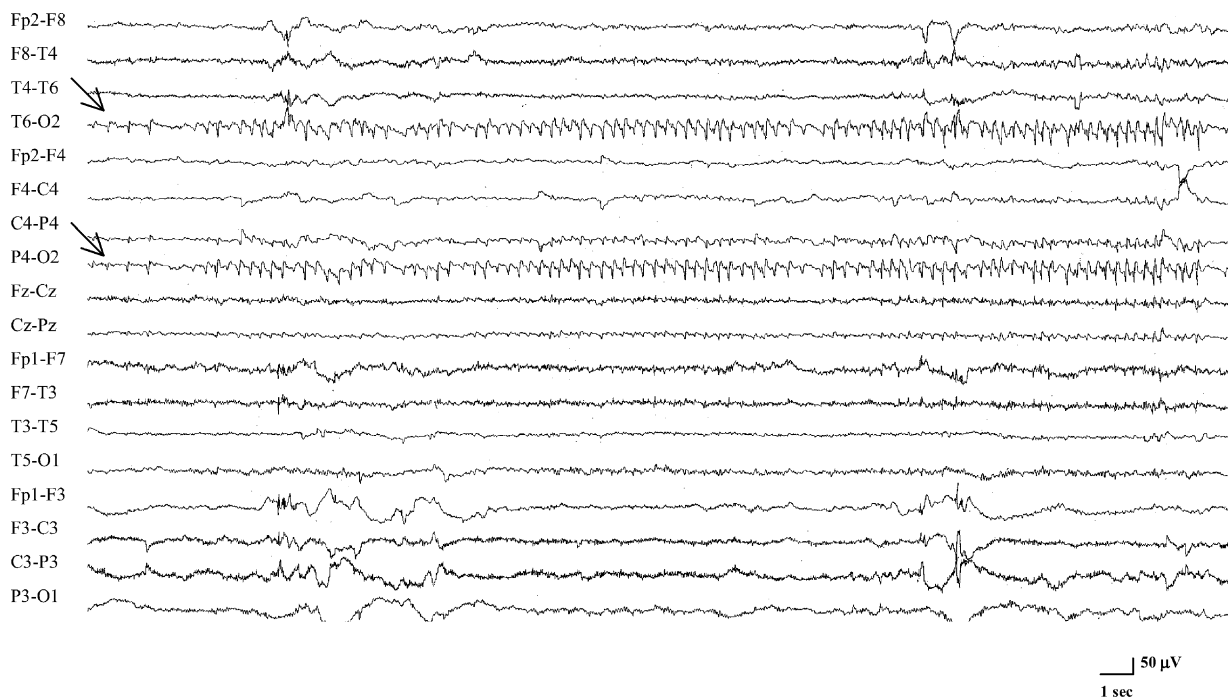

Figure 1 Patient 7 ''I see white lights ... on the left side ...'' accompanied by a static pattern (monomorphous, repetitive spike-and-waves in right occipital sites). EEG seizure onset (↘).
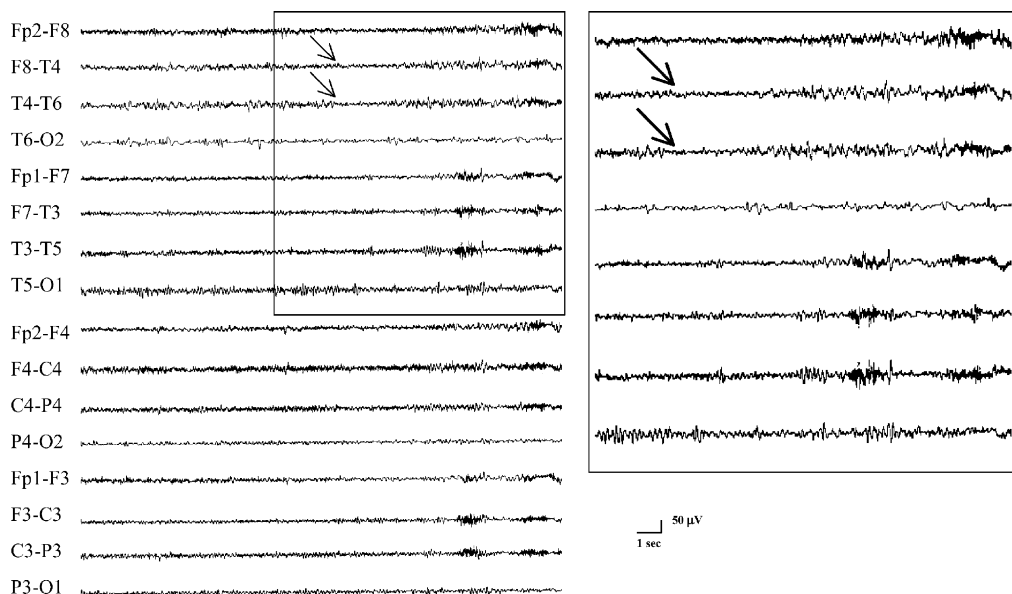
Figure 2 Patient 1 ''a sensation in the throat ...'' accompanied by a dynamic pattern (focal voltage reduction followed by rhythmic theta waves localized in right temporal sites). EEG seizure onset (↘).

patients) patterns, similar to the ones observed in EEG [+] seizures, could be recognized either as focal isolated rhythmic theta waves bursts or as a focal voltage reduction followed by recruiting theta or theta—delta waves (Table 2). These ictal abnormalities were more difficult to recognize because they lacked clear-cut ictal features, and therefore resembled the focal physiological desynchronization following sensory stimulation or interictal paroxysms. Conversely, the most useful clues for identifying an ictal pattern under unblinded conditions were, besides the time of the warning signal, the ictal pattern at the beginning of a major episode, its stereotypy in subsequent episodes in the same patient, and the type of subjective ictal sensation reflecting the presumed site of the ictal discharge.

EEG [−] seizures
In 7 patients (75 episodes) FSSs had no identifiable EEG correlate. The observers failed to recognize specific ictal EEG activity even under unblinded conditions. In these patients, close inspection of the EEG preceding the major seizure failed to disclose a recognizable local discharge preceding the ictal pattern accompanying the objective seizure manifestation.

Viscerosensory and experiential FSSs could have any type of ictal EEG correlate (EEG [+], [±] or [−]) whereas visual and oculosensory FSSs were invariably accompanied by EEG [+] discharges. The other types of clinical seizures were observed in few patients and had variable EEG correlates (Table 3).

There was no relation of the length of the seizures to the EEG pattern or observers' ability to detect the EEG onset.

Discharge site

Ictal EEG activity was localized in the fronto-temporal region in seven patients, in the temporal region in eight, in the temporo-occipital region in two, in the temporo-opercular region in one, in the fronto-central region in one and in the centro-parietal region in two. The clinical manifestations agreed well with the site of the discharge. Somatosensory FSSs originated in fronto-central or centro-parietal sites, visual and ocular sensory FSSs in a temporal, temporo-occipital or centro-parietal sites, viscero-sensory FSSs in temporal, temporo-occipital or temporo-opercular, experiential FSSs in temporal or fronto-temporal,

Table 3 Seizure semeiology and ictal EEG correlates.

FSSs	EEG [+]	EEG [±]	EEG [−]
Somatosensory	2 (2)	—	1 (25)
Visual or oculosensory	4 (62)	—	—
Viscerosensory	3 (45)	4 (31)	4 (30)
Experiential phenomena	3 (5)	2 (22)	2 (20)
Cephalic	1 (10)	1 (2)	—
Diffuse warmth	—	1 (1)	—

No. of patients (No. of FSSs).

Table 4 Seizure semeiology and ictal EEG localization in EEG [+] and EEG [−] FSSs.

FSSs	Temporal	Fronto-temporal	Temporo-occipital	Temporo-opercular	Fronto-central	Centro-parietal
Somatosensory	—	—	—	—	1 (1)	1 (1)
Visual or oculosensory	1 (1)	—	2 (28)	—	—	1 (33)
Viscerosensory	5 (56)	1 (6)	—	1 (14)	—	—
Experiential phenomena	2 (3)	3 (24)	—	—	—	—
Cephalic	—	2 (12)	—	—	—	—
Diffuse warmth	—	1 (1)	—	—	—	—

No. of patients (No. of FSSs).

and cephalic, diffuse warm FSSs in an indefinite anterior (fronto-temporal) area (Table 4).

Interobserver variability

There was agreement between the two examiners in recognizing the ictal pattern accompanying the clinical manifestations of EEG [+] and EEG [−] FSSs whereas in four of the eight EEG [±] FSSs they disagreed. In these cases, after reviewing and discussing the EEG tracings, disagreements were resolved by consensus.

FSSs and major seizures

The sensory experience accompanying FSSs was replicated at the beginning of each major episode in all patients. Correspondingly, in patients with EEG [+] and EEG [±] ictal discharges, FSSs and major seizures began with similar ictal patterns. In patients with EEG [−] ictal discharges, the ictal patterns in FSSs and major seizures could not be compared because no recognizable EEG correlate accompanied the subjective phase of the seizure. The pattern and localization of EEG discharges accompanying major seizures is beyond the scope of the present paper and is not described.

Of the 28 patients, 9 also had major seizures not preceded by subjective sensations recollected by the patient and described at the end of the episode. These unheralded episodes were recorded from patients with various types of subjective sensations and did not belong in a specific category of FSSs. In seven patients with EEG [+] and EEG [±] FSSs unheralded seizures began with focal EEG discharges resembling those of major seizures signaled by the patient. It is therefore possible that recollection of the initial subjective sensation was canceled by the subsequent development of the major episode.

Discussion

In the majority of our patients FSSs have recognizable EEG features that could be helpful in the diagnosis of epilepsy. Using surface electrodes, we were able to determine specific ictal EEG accompaniments in as many as 21 of the 28 patients (180 out of 255 episodes) studied and to provide several criteria for identifying them. In nearly half of the patients (13 out of 28; 124 episodes out of 255), FSSs could be recognized by examining the EEG tracing blind, the only information being that the tracing belonged to a patient possibly experiencing subjective ictal episodes. In the remaining eight subjects (56 FSSs), specific cues—including clinical details of the seizure and the exact time when the subject experienced the warning—were needed to identify the ictal pattern correctly. This finding underlines how important it is that EEG recordings should be inspected with a specific aim in mind, by a reader who has a thorough knowledge of the subject's clinical history and of the recording conditions. Once the first seizure has been identified, the stereotyped EEG pattern made the ensuing seizures easily recognizable. This learning effect depends mainly on the selection criteria, our population being constituted only by patients with stereotyped FSSs and probably accounts for our surprisingly homogeneous results: in all patients having more than one FSS, the EEG pattern invariably remained unchanged.

Despite the use of surface recordings, we found a high percentage of patients who had FSSs with recognizable EEG correlates (21 out of 28, 65%). Published studies report widely different rates largely depending on the recording methods used. Surface recordings give values varying from 11 to 37% of episodes[14–18] but the number of cases is limited and inclusion criteria are not always stringent, because some investigators also classify as aura simple partial seizures with motor signs. The low percentage of EEG correlates in studies using surface electrodes depends on the fact that the EEG discharges

generally consist of brief, spatially limited bursts, intermingled with interictal transients and physiologic rhythms[19,20] often originating from deep brain regions and therefore difficult to collect through surface leads.[14,15] Deep-electrode recordings yield definitely higher percentages, ranging from 80 to 100% of cases.[16,19,21] Only Sperling and O'Connor[15] report a percentage as low as 52% of EEG tracings with evident correlates. In the only study using subdural electrodes, Devinsky et al.[17] reports noticeable ictal discharges in 90% of the cases.

In our study, the EEG accompaniments of FSSs occurred in two main patterns, static and dynamic. Whereas the dynamic pattern changed during the course of its development closely abiding to what is expected in a recruiting discharge, the static pattern remained unchanged throughout the attack and was easily confused with interictal abnormalities or physiological transients.

We found no relation between the clinical features and the type of EEG patterns, but in agreement with the literature our study confirms the good correspondence between the type of sensory experience and the site of the ictal discharge. In our patients, somatosensory sensations always involved the central regions[10,22] and viscerosensory and experiential phenomena always corresponded to discharges originating in the temporal region.[5,7,10,19,23–26] Visual and oculosensory sensations were accompanied by discharges originating from a wide temporo-parieto-occipital area.[10,22] Only three patients reported sensations in the head and sensations of diffuse warmth and no definitive localizing conclusions can be drawn. The fronto-temporal site of the discharges accompanying these types of clinical manifestations is nevertheless in line with a previous observation.[27]

A larger percentage of manifest ictal EEG patterns was found in somatosensory and visual FSSs than in viscerosensory and experiential FSSs. A possible explanation is that somatosensory and visual phenomena reflect the involvement of neocortical (parietal and occipital) areas near to the recording electrodes, whereas viscerosensory and experiential events classically reflect the involvement of mesial temporo-frontal structures, deeply embedded in the brain.[14,15,19,28] Many investigators have examined the relationship between ictal sensory experiences and the lateralization of the EEG discharge.[5,7,25,29] We could not investigate this association since in our population the presurgical selection purposely excluded from video-EEG recording patients whose seizures started with, or displayed during their development, prominent dysphasic disturbances, indicating involvement of the dominant hemisphere.

The stringent criteria used in selecting patients for video-EEG recording are also responsible for another limitation of our study. Because the ictal events observed in our patients probably do not encompass the entire range of subjective epileptic manifestations, but are forcibly biased toward events originating or affecting areas far from the perirolandic and perisylvian regions, elementary somatosensory sensations may be underrepresented.

The EEG discharges accompanying isolated sensory manifestations corresponded closely with those constituting the initial phase of a major seizure. This finding confirms the high localizing value of FSSs.[15,17,20] In one third of our patients (9 of 28) we also recorded major seizures without sensory warning symptoms. These events seem casually distributed in the various types of seizures and are not a hallmark of a particular kind of FSS. The same holds true regarding the etiology of seizures. We observed FSSs in patients with lesional as well as cryptogenic partial seizures. We found no relationship between the type of seizure, its EEG accompaniments and the presence of a documented organic lesion.

In a condition such as epilepsy whose diagnosis largely relies on clinical history, partial seizures limited solely to subjective manifestations pose serious diagnostic problems. Among the more common, yet not the only, pitfalls are fear and anxiety accompanying panic attacks, epigastric sensation secondary to cardiac and gastrointestinal disorders, visual hallucinations preceding migraine, and paresthesias symptomatic of transient cerebrovascular insufficiency. Identifying the clinical and EEG features of FSSs provides useful clues in classifying these seizures and may be helpful in selecting candidates for surgical treatment.

In conclusion, FSSs, namely, symptoms perceived by the patient alone, are often the minimal and occasionally the only manifestation of partial epilepsy in patients attending a video-EEG laboratory. In two thirds of the cases, they are accompanied by recognizable EEG ictal discharges whose pattern and localization correspond closely to the initial EEG events associated with major seizures in the same patient. In these cases, FSSs have high localizing value, and will provide conclusive evidence for the diagnosis of many epileptic conditions.

Acknowledgements

References

1. Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia* 1981;**22**:489—501.
2. Luders H, Acharya J, Baumgartner C, et al. Semeiological seizure classification. *Epilepsia* 1998;**39(9)**:1006—13.
3. Delgado-Escueta A, Bacsal F, Treiman D. Complex partial seizures on closed-circuit television and EEG: a study of 691 attacks in 79 patients. *Ann Neurol* 1982;**11**:292—300.
4. Theodore WH, Porter RJ, Penry JK. Complex partial seizures: clinical characteristics and differential diagnosis. *Neurology* 1983;**33**:1115—21.
5. Gupta AK, Jeavons PM, Hughes RC. Covanis A aura in temporal lobe epilepsy: clinical and electroencephalographic correlation. *J Neurol Neurosurg Psychiatry* 1983;**46**:1079—83.
6. Janati A, Nowack WJ, Dorsey S, Chesser M. Correlative study of interictal electroencephalogram and aura in Complex Partial Seizures. *Epilepsia* 1990;**31(1)**:41—6.
7. Palmini A, Gloor P. The localizing value of auras in partial seizures: a prospective and retrospective study. *Neurology* 1992;**42**:801—8.
8. Engel Jr J. A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: report of the ILAE task force on classification and terminology. *Epilepsia* 2001;**42(6)**:796—803.
9. Lennox WG, Cobb S. Epilepsy XIII. Aura in epilepsy: a statistical review of 1359 cases. *Arch Neurol* 1933;**30**:374—87.
10. Penfield W, Jasper H. *Epilepsy and functional anatomy of the human brain*. Boston: Little, Brown; 1954.
11. Gastaut H, Broughton R. *Epileptic seizures: clinical and electroencephalographic features, diagnosis and treatment*. Springfield, IL: Charles C. Thomas; 1972. p. 124—34.
12. Ajmone-Marsan C. Commentary. Clinical characteristics of partial seizures. In: Engel Jr J, editor. *Surgical treatment of the epilepsies*. New York: Raven Press; 1987. p. 121—7.
13. Williamson P, Wieser H, Delgado-Escueta A. Clinical characteristics of partial seizures. In: Engel Jr J, editor. *Surgical treatment of the epilepsies*. New York: Raven Press; 1987. p. 101—20.
14. Devinsky O, Kelley K, Porter RJ, Theodore WH. Clinical and electroencephalographic features of simple partial seizures. *Neurology* 1988;**38**:1347—52.
15. Sperling MR, O'Connor MJ. Auras and subclinical seizures: characteristics and prognostic significance. *Ann Neurol* 1990;**28**:320—8.
16. Lieb JP, Walsh GO, Babb TL, et al. A comparison of EEG seizure patterns recorded with surface and depth electrodes in patients with temporal lobe epilepsy. *Epilepsia* 1976;**17**:137—60.
17. Devinsky O, Kufta CV, Ito B, et al. Electroencephalographic studies of simple partial seizures with subdural electrode recordings. *Neurology* 1989;**39**:527—33.
18. Manford M, Hart YM, Sander JW, Shorvon SD. National General Practice study of Epilepsy (NGPSE): partial seizures patterns in a general population. *Neurology* 1992;**42**:1911—7.
19. Gloor P, Olivier A, Quesney LF, et al. The role of the limbic system in experiential phenomena of temporal lobe epilepsy. *Ann Neurol* 1982;**12**:129—44.
20. Sperling MR, Lieb JP, Engel J, Crandall PH. Prognostic significance of independent auras in temporal lobe seizures. *Epilepsia* 1989;**30(3)**:322—31.
21. Bancaud J, Bonis A, Munari C, et al. Localizing value of the clinical manifestations of the partial seizures. *Acta Neurochir Suppl* 1984;**33**:7—15.
22. Engel Jr J. Approaches to the localization of the epileptogenic lesion. In: Engel Jr J, editor. *Surgical treatment of the epilepsies*. New York: Raven Press; 1987. p. 75—96.
23. Van Buren J. The abdominal aura: a study of abdominal sensations occurring in epilepsy and produced by depth stimulation. *Electroencephalogr Clin Neurophysiol* 1963;**15**:1—19.
24. Currie SW, Heathfield R, Scott D. Clinical course and prognosis of temporal lobe epilepsy: a survey of 666 patients. *Brain* 1971;**94**:173—90.
25. Mullan S, Penfield W. Illusions of comparative interpretation and emotion. *Arch Neurol Psychiatry* 1959;**81**:269—84.
26. Penfield W, Perot P. The brain's record of auditory and visual experience. A final summary and discussion. *Brain* 1963;**86**:595—696.
27. Rasmussen T. Surgical therapy of frontal lobe therapy. *Epilepsia* 1963;**4**:181—98.
28. Halgren E, Walter RD, Cherelow DG, Crandall PH. Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. *Brain* 1978;**101**:83—117.
29. Taylor D, Lochery M. Temporal lobe epilepsy: origin and significance of simple and complex auras. *J Neurol Neurosurg Psychiatry* 1987;**50**:673—81.