

Panic attack semiology in right temporal lobe epilepsy

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Received December 27, 2002; Accepted April 17, 2003

ABSTRACT – *Background:* Panic attack semiology as a manifestation of epileptic seizures may lead to difficulties and delay in diagnosis. We present a case series to demonstrate the association of ictal panic and anxiety symptoms with partial seizures lateralized to the right temporal lobe.

Methods: From 112 consecutive patients with intractable temporal lobe epilepsy (59 right, 53 left) referred for video-EEG monitoring, five patients were identified whose seizures had been diagnosed as panic attacks in the past. Their ictal symptomatology included feelings of panic and impending doom, hyperventilation, palpitation, diaphoresis, shortness of breath and generalized paresthesiae. Ictal panic was not identified in 72 patients with extratemporal epilepsy investigated during the same period.

Results: EEG documented a right anterior to mid-temporal focus in all five patients. Brain MRI or pathology showed right mesial temporal sclerosis in four and a right temporal ganglioglioma in one. Ictal tachycardia was documented with EEG-EKG recording in the latter patient, prior to right anterior temporal lobectomy and amygdalohippocampectomy. Reinvestigation of this patient five years later for recurrent seizures, no longer associated with panic symptomatology, showed right temporal ictal onsets with seizure spread to the left temporal lobe, now associated with ictal bradycardia.

Conclusions: Our case series provides further evidence to support a relationship between panic attack symptomatology and ictal involvement of the right mesial temporal region [Published with video sequences].

KEY WORDS: anxiety, bradycardia, hyperventilation, ictal tachycardia, panic, seizures

Introduction

Some symptoms of panic attacks such as anxiety, fear, derealization or depersonalization, altered autonomic functions and cardiovascular fluctuations may be found both ictally and interictally in epileptic patients. Several animal and human studies have provided evidence for right hemispheric dominance in the cerebral control of heart rate and blood pressure modulation, typically ascribed to sympathetic lateralization in the right

hemisphere [1-5], although parasympathetic effects have been implicated by others [6]. Although it is recognized that some symptoms of complex partial seizures overlap with panic and autonomic symptoms, there are relatively few documented cases of panic disorder that are later found to be due to focal epileptic disorders. We present a case series to demonstrate the association of ictal panic and anxiety symptoms with partial onset seizures lateralized to the right temporal lobe. We review the relevant literature.



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Presented in part at the American Epilepsy Society meeting, December 2001, Philadelphia, PA, USA

Methods

From 112 consecutive patients with intractable temporal lobe epilepsy (59 right, 53 left) referred for video-EEG monitoring at the Toronto Western Hospital between February 1997 and April 2001, five patients were identified whose seizures had been diagnosed as panic attacks at some point in the past. A history of ictal panic was not identified in 72 patients with extratemporal epilepsy investigated during the same period.

Results

All five patients were ultimately diagnosed with right temporal lobe epilepsy. The individual case histories are given below. The apparent trend to a right temporal localization and lateralization for ictal panic in this study group did not reach statistical significance ($0.05 < P < 0.1$, Chi-Square test, two-tailed, with correction for continuity).

Patient 1

Patient 1 was a 48-year-old man whose first seizures had occurred at age 16. Investigations led to the discovery of a right temporal lobe tumor, which was resected. Pathology was consistent with ganglioglioma. Following the resection of his tumor, over the next 20 years, he experienced paroxysmal, brief (15 – 30 s) episodes of sudden onset palpitations, diaphoresis, shortness of breath, generalized paresthesias, anxiety and feelings of warmth. These episodes occurred two to three times a week. He was treated with phenytoin for many years, and subsequently switched to carbamazepine.

Ten years prior to investigations at this hospital, he began to experience lapses of awareness during some of his spells and he started to have more frequent attacks, which could not be completely controlled with valproate, lamotrigine, clobazam, gabapentin, vigabatrin, or various combinations thereof. He was also followed for chronic depression and anxiety. Medically-trained witnesses of some of his attacks had documented their uncertainty as to whether they had witnessed simple partial seizures or panic attacks.

At the time of admission to the Epilepsy Monitoring Unit (EMU) for video-EEG investigation, he was experiencing 3 – 6 events per day. Sixteen partial onset seizures were recorded during his EMU admission. During these episodes he activated the event marker as he sensed his aura of fear, and subsequently had an anxious expression on his face. He then experienced deep regular breathing and was able to verbalize that he felt anxious, that he was hyperventilating, and that his heart was pounding. With larger events, he stated that he was unable to think clearly and would stop what he was doing until the end of the spell.

Ictal, or, more frequently, postictal noserubbing [7] with the right hand accompanied some of the events (see videosequences).

In all seizures, the first definite ictal electrographic activity was preceded by the onset of clinical symptomatology. Seizure onsets were localized to the right midtemporal region as low amplitude, 10 – 14 Hz rhythmic ictal activity with phase reversal at T4 (*figure 1*). Brain MRI showed the previous right anterolateral temporal resection with sparing of the mesial temporal structures (*figure 2*). The patient underwent repeat right temporal lobe surgery, this time with amygdalohippocampectomy and further resection of the structures posterior to the previous excision (*figure 3*). Electrocorticography prior to this resection revealed active spiking from the second and third temporal gyri at the posterior margins of the previous resection and overlying the preserved amygdala and anterior hippocampal structures. Surgical pathology demonstrated traces of residual ganglioglioma in a parahippocampal specimen from the posterior portion of the resection.

Following this second surgery, the patient remained seizure-free on valproate monotherapy for nearly two years. Seizures subsequently recurred, however, initially as infrequent nocturnal convulsions over the next two years. During the fifth postoperative year diurnal seizures returned, no longer associated with panic symptomatology or aura of any kind. Reinvestigation with continuous ambulatory EEG at this time captured two such seizures, both of right temporal origin with subsequent spread to the left temporal lobe, now associated with ictal bradycardia (*figure 4*).

Patient 2

Patient 2 was a 25-year-old woman, first diagnosed with panic attacks at the age of 16. Her attacks consisted of brief episodes of hyperventilation, palpitations, feelings of fear and impending doom, lasting between 30 s to 2 min. Three years later, a diagnosis of epilepsy was reached when oral automatisms and staring were noted and treatment was started with antiepileptic medications. She was allergic to phenytoin and carbamazepine, and did not tolerate valproate, clobazam or topiramate. When admitted to the EMU for localization of her seizures, she was taking lamotrigine monotherapy with spells occurring approximately five times per week.

There was also a recent history of episodes beginning with her typical subjective symptomatology, but progressing to involve unusual movements of all extremities, not associated with alteration of consciousness according to the patient's own description, raising the possibility of additional nonepileptic events. The onset of these episodes had been noted to coincide with the patient's leaving home to live with her boyfriend and the concurrent commencement of a new job, both of which had been significant emotional stressors.

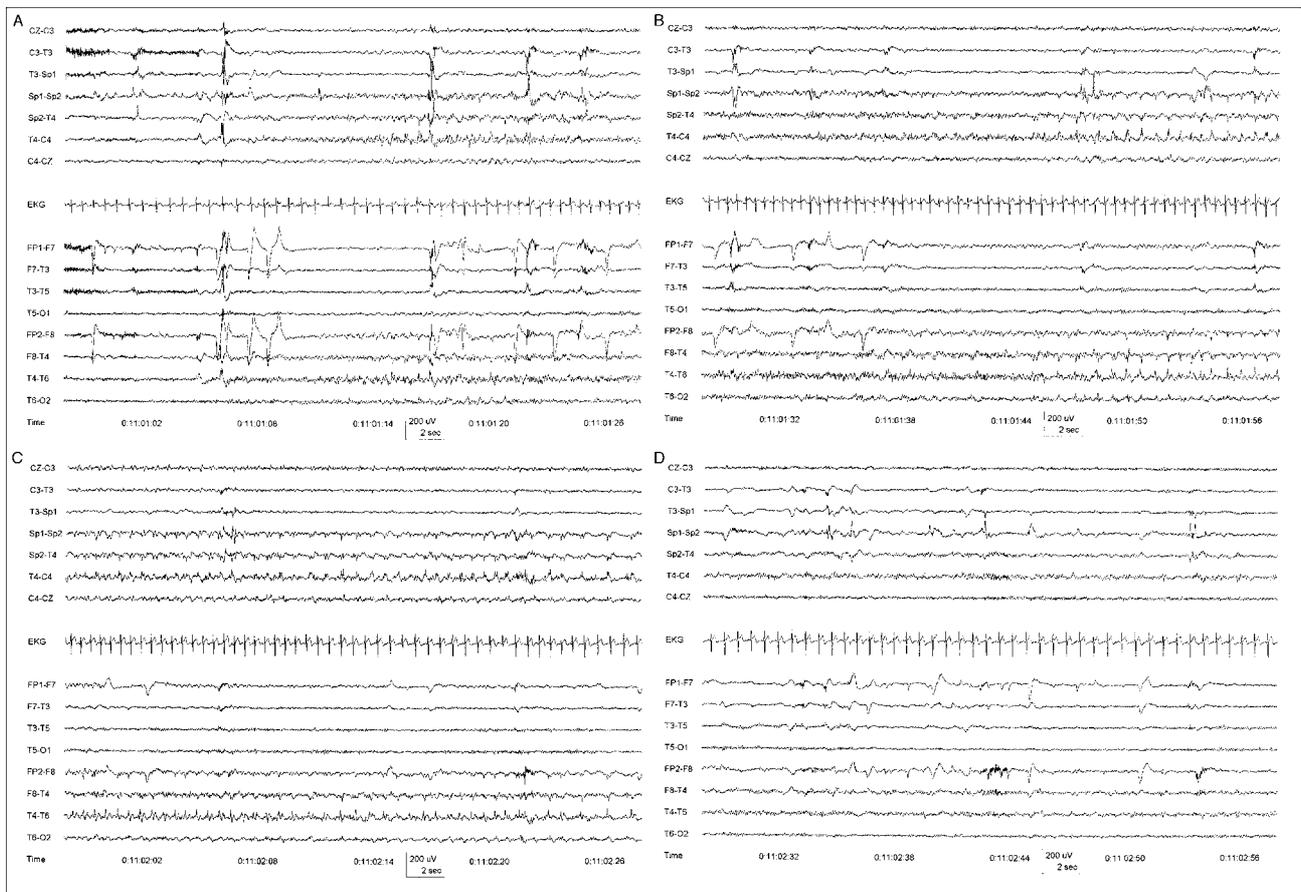


Figure 1. A-D. Continuous EEG recording shows right temporal seizure in patient 1 prior to epilepsy surgery. Electrographic seizure onset near 0:11:01:08 (A). Ictal activity maximum at T4 > F8, Sp2. EKG shows ictal tachycardia with increase in heart rate from 80 to 140 bpm during seizure (B, C) and return to baseline heart rate postictally (D).



Figure 2. Patient 1 MRI before repeat epilepsy surgery shows a remote right anterolateral temporal tumor resection cavity with preservation of mesial temporal structures.

During 6 days of video-EEG monitoring, three types of events were recorded. The first episode was marked only by hyperventilation, lasting approximately 2 min, with no EEG changes. Subsequently, four episodes were recorded commencing with hyperventilation followed by long periods, lasting more than 5 min, of repetitive bilateral finger flexion and extension progressing to large amplitude atetoid movements of both arms and legs, all in the setting of completely preserved awareness and responsivity. There

were no associated ictal EEG changes with these events. Ultimately, two complex partial seizures were recorded, associated with early hyperventilation (approximate doubling of the baseline respiration rate from 18 to 32 per minute during the first 60 s of the clinical seizure, preceding the first ictal electrographic changes by 30 s), tachycardia, oral automatisms and eye deviation to the left. Two secondarily generalized seizures were then recorded, both of which arose from sleep. Ictal hyperventilation during sleep could not be appreciated on video prior to the arousal associated with generalization in these two seizures.

The ictal electrographic onsets were all localized to the right anterior temporal region. The spells unassociated with EEG changes were interpreted to represent nonepileptic events, although the possibility of psychogenic elaboration of simple partial seizures not apparent on scalp EEG, could not be excluded [8, 9]. MRI showed right mesial temporal sclerosis. The patient underwent a right selective amygdalohippocampectomy and has been free of all episodes for more than 18 months.

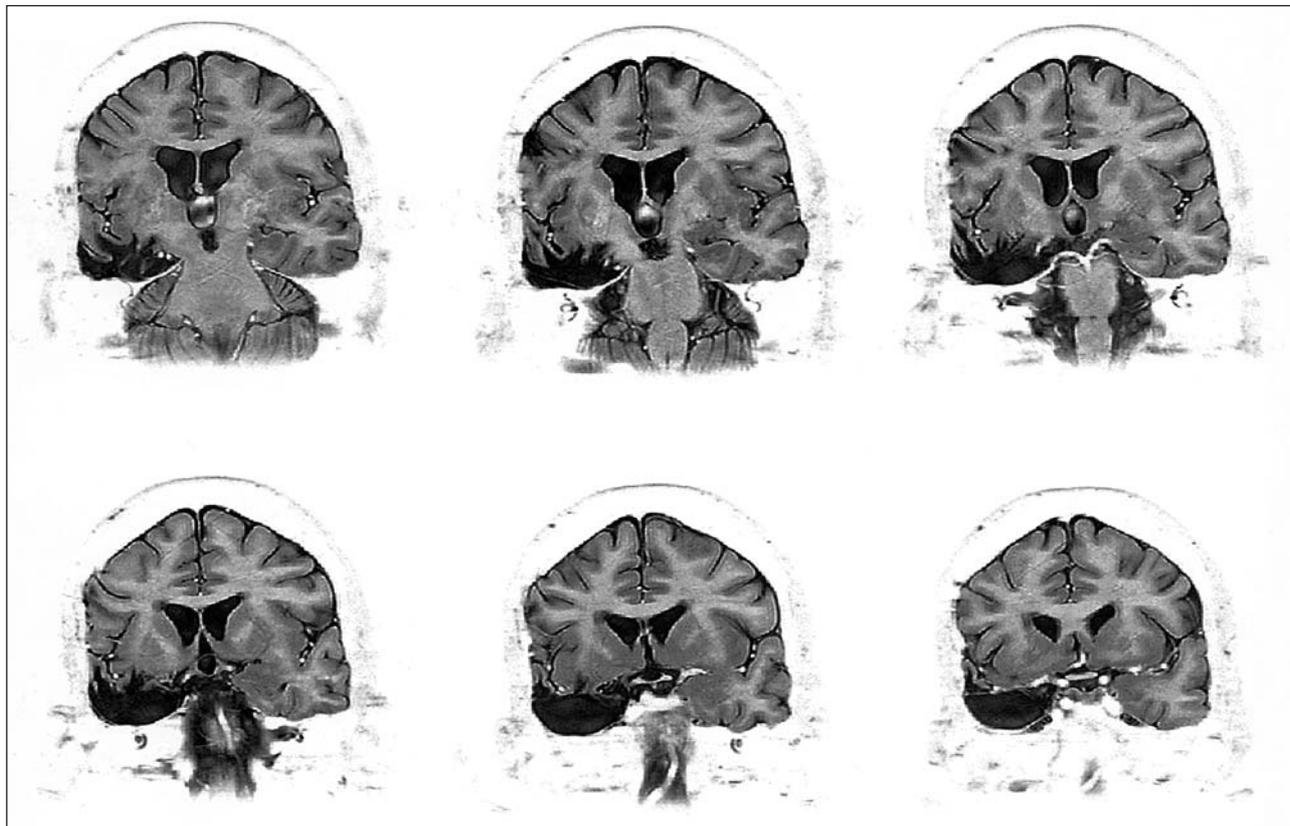


Figure 3. Patient 1 MRI after epilepsy surgery shows a right anterior temporal resection cavity with excision of amygdala/uncus, hippocampus and parahippocampal gyrus.

Patient 3

Patient 3 was a 34-year-old woman with a normal birth history and uneventful childhood. At age 20, she developed persistent headaches and was diagnosed with hydrocephalus attributed to congenital aqueductal stenosis. A right-sided ventriculo-peritoneal shunt was inserted and revised four years later. That same year, when pregnant, she experienced her first seizure, a generalized convulsion. She was started on phenobarbital, switched to phenytoin, and then to carbamazepine and clobazam. During the year before her admission to the EMU, she was experiencing episodic olfactory hallucinations of "medicinal" smells described to last 1 to 2 min, followed by a feeling of fear lasting 30 min with her whole body feeling numb, and her head heavy. These events occurred 2 to 3 times a day. Over the same year, she had also experienced depression and decreased emotional control. She was seeing a psychiatrist for these problems and had been given a diagnosis of major depression, anxiety disorder and panic attacks. The patient was reluctant to take the prescribed psychotropic medications.

The patient was referred to the EMU for diagnostic purposes. After discontinuing her antiepileptic drug therapy, a

number of simple partial seizures were recorded associated with her typical aura of a medicinal smell and a feeling of fear, all localized to the right anterior temporal region. No complex partial seizures were recorded. MRI showed right mesial temporal sclerosis, as well as encephalomalacia along the right occipital ventricular shunt insertion. She is currently taking carbamazepine monotherapy, with infrequent seizures, and does not want to consider surgical management at this time.

Patient 4

Patient 4 was a 39-year-old woman, first diagnosed with panic attacks at age 9. Her episodes were characterized by feelings of intense fear, panic and palpitations, lasting about 2 min. Years after the onset of her spells, automatisms and occasional falls were noted and she was diagnosed with epilepsy. Her events were significantly more frequent near the beginning of her menses. She had a long history of diagnosed anxiety treated with anxiolytics, with what amounted to agoraphobic behavior perimenstrually, her panic and anxiety at these times raising the suspicion, in retrospect, of an epileptic "aura continua" state. Treatment with various antiepileptic drugs invariably de-

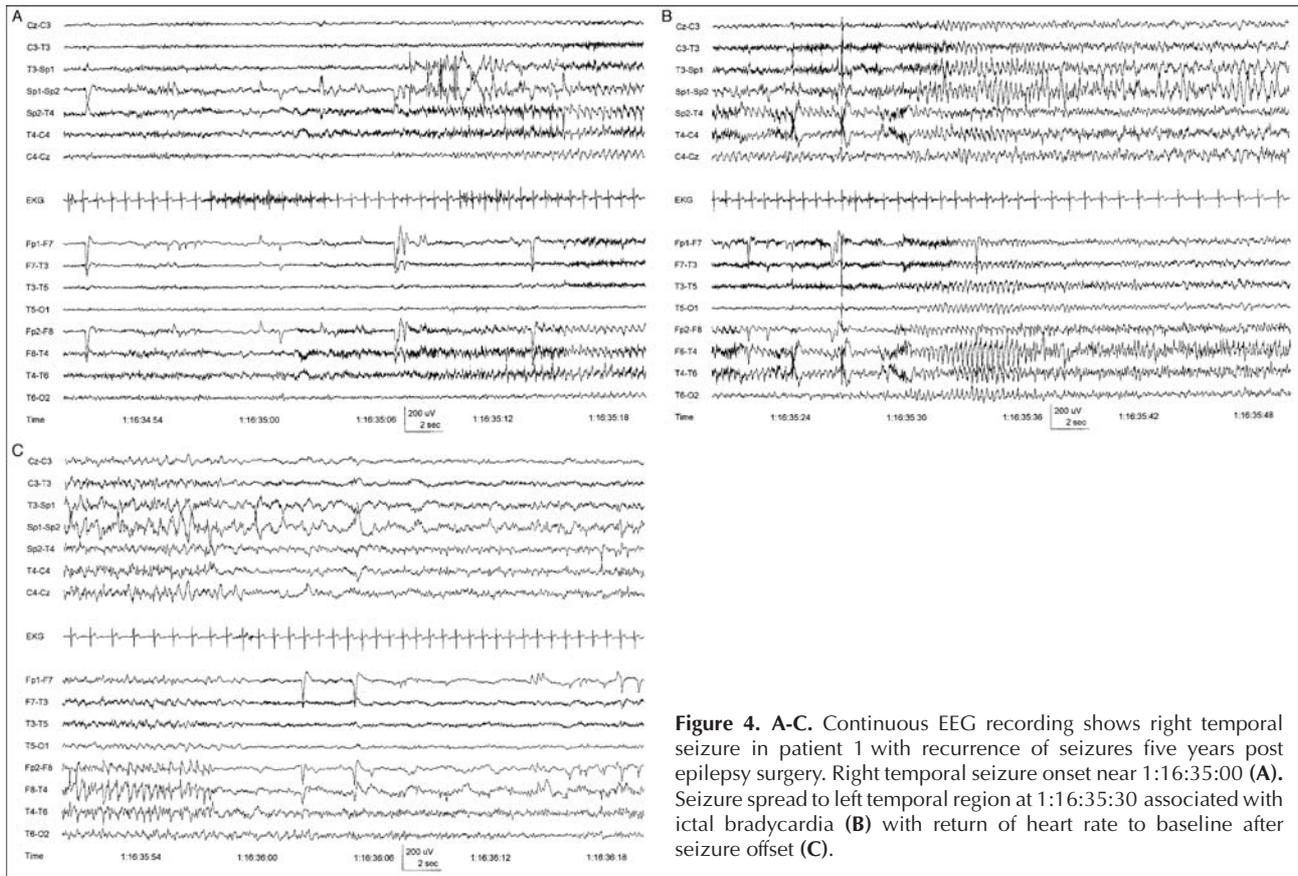


Figure 4. A-C. Continuous EEG recording shows right temporal seizure in patient 1 with recurrence of seizures five years post epilepsy surgery. Right temporal seizure onset near 1:16:35:00 (A). Seizure spread to left temporal region at 1:16:35:30 associated with ictal bradycardia (B) with return of heart rate to baseline after seizure offset (C).

creased the frequency of events but never completely eliminated them, and she tolerated the medications poorly.

Video-EEG monitoring documented right temporal lobe ictal onsets for her typical events, with an exceedingly active unilateral right anterior temporal interictal epileptiform abnormality, at times recorded continuously for minutes on end, giving credence to the possibility of an “aura continua” etiology to her periods of agoraphobic behavior (figure 5). MRI showed right mesial temporal sclerosis. Initially reluctant to consider surgical management, she is now awaiting right anterior temporal resection.

Patient 5

Patient 5 was a 24-year-old woman with a history of two febrile seizures during infancy. She was treated with phenobarbital until age 8, though there was no seizure recurrence until age 20. Typical events consisted of an aura of a “head rush” quickly followed by progression to complex partial seizures associated with hypersalivation and terminating with a cough. One prolonged event, which had commenced with the same aura, did not progress to the typical complex partial seizure but instead was followed

by pronounced hyperventilation and fear as well as an altered sense of visual perception suggestive of depersonalization or derealization. This event led to her being taken to a hospital emergency department for evaluation, where a diagnosis of panic attack was offered.

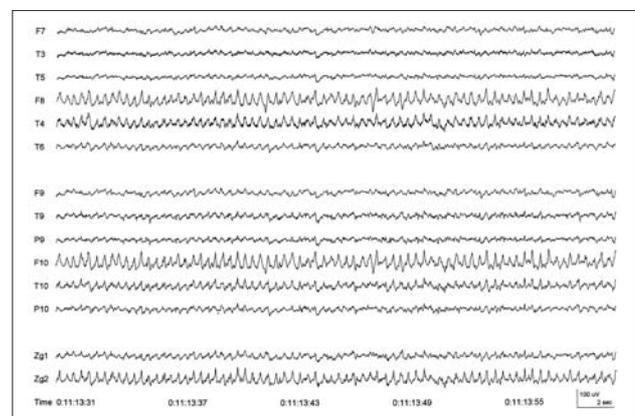


Figure 5. Example of interictal EEG recording in patient 5 (asymptomatic at the time) shows continuous right anterior temporal sharp wave activity (F8, F10, Zg2 > T4, T10; Cz monopolar referential montage).

The patient was referred for video-EEG monitoring after trials of multiple, different antiepileptic drugs (valproate, gabapentin, topiramate, carbamazepine, and combinations thereof) had not been able to completely control her seizures. Three of her typical complex partial seizures were recorded with a brief aura prior to loss of awareness with blank stare, postictal noserubbing and postictal coughing [10]. Ictal onsets were lateralized to the right frontotemporal area, maximal over the anterior temporal region. MRI showed right mesial temporal sclerosis. The patient subsequently underwent a right selective amygdalohippocampectomy and has been seizure free more than 18 months after her operation.

Discussion

Partial onset seizures presenting with panic attack symptomatology may pose a diagnostic challenge. Fear, an affective symptom associated with idiopathic panic attacks, is also seen as an ictal symptom in 10–15% of patients with temporal lobe epilepsy [11]. Fear was described to be the most common experiential phenomenon produced by direct electrical brain stimulation in temporal lobe epilepsy, localized to the anteromedial temporal region including the amygdala [12]. Other than fear, focal seizures and panic attacks may both present with autonomic symptoms such as tachycardia, blood pressure fluctuations, hyperventilation and dyspnea. Our case series demonstrates this overlap of symptomatology and the need to differentiate between these two distinct diagnostic entities, which have very different treatment implications. Hemispheric control of autonomic function has been the subject of debate for many years. Despite some contradictory results in the literature [6], several investigators have shown right hemispheric dominance for sympathetic heart rate and blood pressure modulation. Hilz *et al.* analyzed autonomic heart rate and blood pressure modulation in 15 patients with refractory epilepsy during hemispheric inactivation with the intracarotid amobarbital procedure (IAP) [1]. They confirmed previous IAP studies indicating sympathetic lateralization in the right hemisphere and parasympathetic predominance in the left hemisphere [4, 5]. In patients with right hemispheric partial onset seizures, it is then conceivable that ictal electrical stimulation of ipsilateral hemispheric autonomic centers could evoke tachycardia, hyperventilation and other symptoms resembling panic semiology. The principal site of autonomic representation in the brain is thought to involve the insular cortex [1-3]. Spread of right mesial temporal ictal activity to insular cortex may be responsible for the autonomic panic attack symptomatology, although this remains speculative and other possible networks of activation (for example, direct temporolimbic-hypothalamic pathways) may be responsible.

Our demonstration in patient 1 that panic symptomatology and ictal tachycardia both disappeared after right

mesial temporal resection of uncus/amygdala, anterior hippocampus and adjacent parahippocampal gyrus strongly supports the preoperative localization of initiation of these symptoms and signs within the structures subsequently resected. Eventual recurrence of seizures originating from the residual posterior temporal lobe structures on the right side, now associated with ictal bradycardia during seizure spread to the left temporal region, suggests that only the left hemispheric (parasympathetic) autonomic centers could now be activated by the ictal activity: ie. that the right anteromesial temporal region was necessary for ictal activation of the right hemispheric autonomic centers.

Changes in respiratory rate were not found with inactivation of either hemisphere in one study using the IAP [1]. This raises the possibility that ictal hyperventilation may not necessarily represent direct ictal effects upon autonomic centers, but may instead arise in response to the affective feelings of fear and panic. The observation that hyperventilation was not seen in patient 2 with seizures starting in sleep, but was clearly evident with diurnal events, lends some support to the possibility of an affective component to ictal hyperventilation.

Diagnostic difficulties are not limited to the clinical differentiation of attacks. Simple partial seizures unassociated with EEG changes may be misinterpreted as nonepileptic events if their symptomatology is entirely compatible with panic attacks [13]. Likewise the scenario of psychogenic elaboration of simple partial seizures unassociated with EEG changes [8, 9]. Patient 2, in all likelihood, demonstrated both of these phenomena prior to the video-EEG confirmation of an epileptic etiology obtained with the recording of larger events. Although it is presumably only a small proportion of patients diagnosed with anxiety and panic attacks who are actually suffering from a focal epileptic disorder, the possibility of an epileptic “aura continua” etiology for even prolonged periods of pathological anxiety is suggested by the “interictal” EEG findings in patient 4.

There are previous reports in the literature describing patients with lateralized epilepsy initially presenting as panic disorder [13-26]. It has been suggested that partial seizures initially misdiagnosed as panic attacks may be a clue to the diagnosis of symptomatic right temporal lobe epilepsy [19]. Although all five of the patients reported in this study did suffer from right temporal lobe epilepsy, this lateralizing trend did not reach significance. However, a review of the previously published cases of definite epilepsy (those with well-described scalp or intracranial EEG plus or minus concordant neuroimaging findings [13-21]) does show that in a vast majority of patients, a right hemispheric, and especially temporal lobe, focus for the epileptic disorder was identified, with 83% of the reported patients found to have a structural lesion (*table 1*). Combining the results from these studies does show a signifi-

Table 1. Reported cases of probable and definite epilepsy presenting with panic semiology.

Reports	Patients	R Temporal	Other Sites	Structural lesions
Ghadirian <i>et al.</i> (1986)	1	1	0	Meningioma
Devinsky <i>et al.</i> (1989)	1	1	0	None
Laidlaw <i>et al.</i> (1993)	1	1	0	None
Alemayehu <i>et al.</i> (1995)	2	0	2 R parietal	Astrocytoma
Meyer <i>et al.</i> (2000)	1	1	0	MTS
Daly <i>et al.</i> (2000)	1	0	1 R frontal	Cortical dysplasia
Tassinari <i>et al.</i> (2000)	7	7	0	2 MTS, 2 cavernomas, 2 tumors, 1 cerebral lupus
Thompson <i>et al.</i> (2000)	3	1	2 L temporal	1L MTS, 2 None
Huppertz <i>et al.</i> (2002)	1	0	1 L temporal	Cortical dysplasia
Sazgar <i>et al.</i> (2003)	5	5	0	4 MTS, 1 ganglioglioma
Total	23	17	6	19

MTS = Mesial temporal sclerosis; R = Right; L = Left.

cant bias for a right hemispheric lateralization (20:3, right:left; $P < 0.001$, Chi-Square test, two tailed, with correction for continuity). Notwithstanding, review of the additional published cases of possible or questionable epilepsy presenting as panic attacks [22-26] does not show evidence of lateralization (table 2), and the existence of proven cases with extratemporal or left temporal localization [16, 18, 20, 21] shows that the association of panic attack semiology with right temporal lobe epilepsy is not invariant. Seizure spread, to preferentially activate right hemispheric autonomic centers, might underlie panic symptomatology in patients with extratemporal or left temporal onsets. However, it is equally possible that individual differences in lateralization or localization of autonomic centers could be responsible.

In keeping with an asymmetry of hemispheric representation for panic symptomatology Reiman *et al.*, using positron emission tomography to measure baseline cerebral blood flow in patients with idiopathic panic disorder, demonstrated increased blood flow in the region of the right parahippocampal gyrus [27]. Panic symptomatology in both epileptic and nonepileptic patients may thus implicate structural or functional alterations similarly localized

to the right mesial temporal region. Activation of the same temporolimbic structures could contribute, via different mechanisms, to the pathophysiology of both idiopathic panic disorder and epileptic panic symptomatology.

In our case series, the clinical history provided important clues to the diagnosis of epilepsy such that by the time of our assessment diagnostic uncertainty was not an issue. In the early stages of presentation, however, diagnostic uncertainty in similar patients is to be expected. The past medical or surgical history contributed to the diagnosis in three of our five patients and included febrile convulsions, previously resected brain tumor, and shunted hydrocephalus (though the latter condition is presumably unrelated to the subsequently discovered mesial temporal lobe epilepsy and sclerosis). Other differentiating factors in favor of a diagnosis of epilepsy as opposed to idiopathic panic attacks, include short duration of attacks, stereotyped aura (apart from fear), ictal unresponsiveness, oral or motor automatisms, postictal confusion, nocturnal occurrence, lack of response to anxiolytic medications and, most importantly, EEG confirmation of the diagnosis of epilepsy. □

Table 2. Reported cases of possible/questionable epilepsy associated with panic attacks.

Reports	Patients	R Temporal	Other lateralized sites	Bilateral seizure onsets
Wall <i>et al.</i> (1985)	1	1	0	0
Weilburg <i>et al.</i> (1987)	3	1	L temporal; 1 L hemisphere	0
Edlund <i>et al.</i> (1987)	6	3	2 L temporal	1 bifrontotemporal
Dantendorfer <i>et al.</i> (1995)	3	1	1 L parieto-occipital	1 bitemporal
Pegna <i>et al.</i> (1999)	1	0	1 L temporal	0
Total	14	6	6	2

R = Right; L = Left



Video Caption

- First seizure: Clinical onset approximately 12:28:39
- Scalp EEG onset 12:28:45
- (No speech)
- Second seizure: Clinical onset approximately 15:48:05
- Scalp EEG onset 15:48:15
- 15:48:10 Hangs up phone
- 15:48:54 "Bad one..."
- 15:49:03 "More and more anxious..."
- 15:49:07 "My heart's picked up..."
- 15:49:15 "...can't think clearly..."
- 15:49:28 "...dizzy...funny with vision..."
- 15:49:43 Phone rings "Hello...Yeah...I'll call you back. Bye" Hangs up phone
- 15:49:53 "Starting to tremor now...hotter..."
- 15:50:05 "There's some relief...the panic is going..."
- 15:50:24 "I hung up on the phone...wasn't thinking clearly"
- 15:50:53 "It's gone now"
- Third seizure: Clinical onset approximately 19:29:21
- Scalp EEG onset 19:29:26
- 19:30:19 "Heart is pounding..."
- 19:30:36 "Boy oh boy..."

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