Review Article
Temporal Lobe Epilepsy Semiology

Robert D. G. Blair

Division of Neurology, Department of Medicine, Credit Valley Hospital, University of Toronto, Mississauga, ON, Canada L5M 2N1

Correspondence should be addressed to Robert D. G. Blair, rdgblair@mac.com

Received 22 October 2011; Accepted 26 December 2011

1. Introduction

Epilepsy has been recognized since antiquity. It affects millions of people worldwide and remains one of the most common and frightening neurological conditions. The word is derived from the Greek word which means to “seize” or “take hold of.” Epilepsy encompasses a heterogeneous group of disorders with various manifestations including seizures in addition to other signs, symptoms, and features that define a phenotype.

The taxonomy and terminology of epilepsy has undergone a number of changes over the years. An early classification system generated confusion and heated discussion over equating the term “complex partial seizures” (CPSs) and “temporal lobe epilepsy” (TLE) [1]. The 1981 classification of epileptic seizures represented a consensus at that time [2]. A further revision was the Classification of Epilepsies and Epileptic Syndromes accepted in 1989 [3]. Yet another modification and change in philosophy was initiated by the Executive Committee of the International League Against Epilepsy (ILAE) which took office in 1997. The ILAE task force published the Revised Terminology and Concepts for Organization of the Epilepsies in 2010 [4].

Temporal lobe seizures are the most frequent site of origin of partial seizures. They represent approximately two thirds of the intractable seizure population coming to surgical management. Jackson in the 19th Century [5] was the first to link seizures characterized by a “dreamy state” to lesions near the uncus in the temporal lobe (hence the term “uncinate fits”). Gibbs and Lennox suggested the term psychomotor epilepsy to describe a characteristic EEG pattern together with emotional, mental, and autonomic phenomena for seizures originating in the temporal lobe [6]. Researchers at the Montreal Neurological Institute (MNI) described the psychic phenomena as experiential hallucinations based on clinical observations and intraoperative stimulation studies [7, 8]. Gastaut proposed the term CPSs for partial onset seizures associated with loss of consciousness [3]. Videotape and computer technology has permitted careful review of captured seizures and their associated EEG telemetry thus providing detailed descriptions of the features of temporal lobe seizures [9–12]. Frontal lobe seizures (FLS) are the second most frequent site of origin of partial seizures and are often difficult to differentiate from temporal lobe seizures but some features may help (see Table 1).
2. Cardinal Semiology of Temporal Lobe Seizures

2.1. Prodrome. Some patients experience preictal events, which may be helpful in predicting a coming seizure. Prodromes may last several minutes, hours, or, occasionally, even days. Examples of prodromes include headache, personality change, irritability, anxiety, or nervousness. These phenomena should not be confused with seizure onset. Often, prodromes are recognized by family and friends but, not by the patient (especially changes such as irritability or exhilaration).

2.2. Aura. Auras (from the Latin for breeze, Greek for air) are in fact simple partial seizures and can occur in isolation but occur in the majority of patients at the onset of a CPS. They can last from seconds to a long as 1-2 minutes before consciousness is lost.

The types of auras patients report may correlate with the site of seizure onset. Some authors have questioned the localizing value of the aura as a marker of ictal origin in CPSs [13–15]. Many authors, however, have noted a close association of some sensory auras with temporal lobe seizures. Examples include viscerosensory symptoms such as a rising epigastric sensation and experiential phenomena such as fear, déjà and jamais vu, visceral and auditory illusions, and complex auditory or visual hallucinations [16–20]. Gustatory and olfactory hallucinations are also relatively specific for TLE, as are elementary auditory hallucinations [21]. Although auras often have localizing value, they do not often have lateralizing significance.

Semiological seizure classifications associate the anatomical focus of seizure origin with the clinical features of seizures [22–24]. If, however, the seizure begins in an area inaccessible to scalp EEG recordings, then localization will be inaccurate. Similarly, if seizures begin in “noneloquent” cortex, the subsequent spread to “eloquent” cortex may lead to false localization of the seizure focus. Seizure semiology in the latter case signifies seizure propagation rather than seizure origination.

2.3. Altered Consciousness. CPSs are associated with altered consciousness and amnesia for the event; typically, behavioural arrest and staring with a duration of 30 seconds to 1 to 2 minutes. Consciousness has several facets, including cognition, perception, affect, memory, and voluntary motility [25]. Impaired awareness should be distinguished from a temporary block of verbal or motor output or of verbal comprehension with maintained consciousness.

Loss of consciousness in CPSs (as well as in “Absence” episodes) has been shown to be associated with decreased activity in the “default mode network.” This network includes the precuneus/posterior cingulate, medial frontal, and lateral parietal cortices as detected by functional MRI (fMRI) [26]. For more detailed discussions on default mode network in TLE, the reader may refer to a dedicated article in this special issue.

2.4. Amnesia. Individuals with CPSs may be unaware that they had a seizure minutes earlier. They may also be unable to recall events which occurred before seizure onset. The degree of retrograde and anterograde amnesia is variable. For example, patients may have experienced an aura which prompted them to signal the onset of a seizure when they were in the epilepsy monitoring unit (EMU) but, subsequently, not recall having done so. Postictal amnesia likely results from bilateral impairment of hippocampal function. Stimulation of medial temporal lobe structures producing an after-discharge affects the formation and retrieval of long-term memories [27, 28].

2.5. Automatisms. Automatisms represent coordinated involuntary motor activity that is stereotyped and virtually
always accompanied by altered consciousness and subsequent amnesia. No uniform classification of this phenomenon has been developed. One system divides automatisms into de novo and preservative automatisms [29]. De novo automatisms are said to occur spontaneously at or after seizure onset. They might be classified as “release” phenomena, which include actions normally socially inhibited or “reactive” phenomena when they appear to be reactions to external stimuli. For example, the patient may drink from a cup placed in his hand or chew gum placed in his mouth. Preservative automatisms might represent continuation of complex motor acts initiated prior to seizure onset, for example, opening and closing a door repeatedly. Automatisms occur in almost two thirds of CPSs of mesial temporal lobe onset [16, 20, 30]. They often involve the hands (fumbling, picking, fidgeting) or mouth (chewing, lip smacking, swallowing).

Less common automatisms associated with temporal lobe seizures include vocalizations, ictal speech, and affective behaviours (out of context fear). Additionally, even less common behavioural, such as, crying (dacrystic), laughing (gelastic), and so-called “leaving behaviours,” for example, running out of the house or down the street during a seizure (cursive) have been reported [31–34]. A rare automatism, whistling, has also been recently reported to occur during temporal lobe seizures [35].

Temporal lobe seizures can be simple partial, complex partial, or secondarily generalized. A number of features of TLE semiology have lateralizing or localizing value (see Table 2).

3. Mesial Temporal Lobe Seizures and Neocortical Temporal Lobe Seizures

TLE is the most common symptomatic partial epilepsy in adolescents and adults but, extratemporal or neocortical epilepsy is more common in young children [36, 37]. Mesial temporal sclerosis (MTS), hippocampal sclerosis (HS), is the most common cause of TLE, representing greater than 80% [38]. Other causes include perinatal injury, malformations of cortical development (MCD), arteriovenous malformations (cavernous hemangiomas, meningioangiomatosis), infections of the central nervous system (CNS), glial tumors (e.g., ganglioglioma, dysembryoblastic neuroepithelial tumors, astrocytomas, oligodendrogliomas, meningiomas, or CNS metastasis), hamartomas, head trauma, and limbic encephalitis [38, 39]. MTS involves neuronal loss in the hilar region of the hippocampus (CA1, CA3, CA4, and the dentate gyrus, with relatively sparing of the CA2 region). It is typically bilateral but greater on one side [16]. Temporal lobe epilepsy with mesial temporal sclerosis usually presents between 6–10 years of age but can present from infancy to the 30s [16]. MTS is usually a progressive disorder and seizures initially controlled with antiepileptic drugs can later become intractable in 60–90% [16, 17].

The association between MTS and depression, anxiety, and other psychiatric comorbidities is controversial [16]. Impairment of cognition and memory may be the result of frequent seizures and/or medication side effects. Psychiatric symptoms can occur ictally and be mistaken for primary psychiatric illness. For more details on this topic, the reader may refer to the dedicated articles in this special issue.

The semiological features of mTLE were said to include typical auras such as rising epigastric sensations, déjà vu, affective phenomena (fear or sadness), or experiential phenomena followed by unilateral motor signs (frequently ipsilateral contraction of face or mouth, head deviation) and bilateral motor phenomena in the face or axial muscles. Behavioural arrest and oral automatisms are common and bitemporal spread heralds alteration in consciousness, amnesia, autonomic phenomena (change in heart rate and respirations), and prominent motor automatisms (tonic and dystonic posturing) [40].

Familial mesial temporal lobe seizures are a heterogeneous syndrome characterized by predominantly psychic, autonomic auras, and dysmnestic symptoms including déjà vu usually evolving to CPSs and/or secondary generalization [41–44].

The semiological features of neocortical TLE are said to include auditory, vestibular and complex visual hallucinations, and aphasia and focal sensory-motor phenomena [40]. Some authors have reported that early onset unilateral motor automatisms, without dystonic posturing, can localize seizure origin to the contralateral temporal lobe neocortex [45]. nTLE-originating temporal lobe seizures are seemingly less common than mesial temporal lobe seizures and as a result are less well characterized. nTLE is associated with structural abnormalities including MCD, vascular malformations, neoplasms, and traumatic brain injuries [46–48]. For more details on this topic, the reader may refer to the dedicated paper in this special issue.

A rare hereditary syndrome, autosomal dominant partial epilepsy with auditory features (ADPEAF), has been described [49–52]. It usually begins in adolescence or early adulthood. The cause, in greater than 50% of families described, is a mutation of leucine-rich glioma inactivated gene (LGI1). The most common auditory symptoms are simple unformed sounds (humming, buzzing, ringing), less frequently, complex sounds (voices, songs) or distortions (volume changes may occur). Some patients experience seizures precipitated by specific sounds (pieces of music, telephone ringing) [50]. Some also reported experiencing olfactory, visceral, vertiginous, experiential, or autonomic auras [50]. The diagnosis requires exclusion of structural lesions, in addition to a characteristic family history.

Some authors found no differences in the auras of patients with mTLE and nTLE onset [53]. Most authors, however, note that CPSs of mesial temporal origin typically begin with oroalimentary or hand automatisms in contrast to those of neocortical onset, which often begin with staring, without automatisms or epigastric phenomena.

There are reciprocal connections between mesial and neocortical temporal cortex. There is evidence that these connections are activated to produce an aura [54, 55]. This suggests that the type of aura in TLE is more indicative of the pattern of seizure spread than the site of seizure onset. Gloor’s hypothesis posits that experiential phenomena
Table 2: Semiological Features (TLE) - Lateralizing or Localizing Value.

| Feature                          | Location                        |
|----------------------------------|---------------------------------|
| **Automatism**                   |                                 |
| Unilateral limb automatism       | Ipsilateral focus               |
| Oral automatism                  | (m)/Temporal lobe               |
| Unilateral eye blinks            | Ipsilateral to focus            |
| Postictal cough                  | Temporal lobe                   |
| Postictal nose wiping            | Ipsilateral temporal lobe       |
| Ictal spitting or drinking       | Temporal lobe focus (R)         |
| Gelastic seizures (m)            | (m)/Temporal, hypothalamic, frontal (cingulate) |
| Dacrystic seizures (m)           | (m)/Temporal, hypothalamic      |
| Unilateral limb automatisms      | Ipsilateral focus               |
| Whistling                        | Temporal lobe                   |
| **Autonomic**                    |                                 |
| Ictal emeticus                   | Temporal lobe focus (R)         |
| Ictal urinary urge               | Temporal lobe focus (R)         |
| Piloerection                     | Temporal lobe focus (L)         |
| **Motor**                        |                                 |
| Early nonforced head turn        | Ipsilateral focus               |
| Late version                     | Contralateral focus             |
| Eye deviation                    | Contralateral focus             |
| Focal clonic jerking             | Contralateral perirolandic focus|
| Asymmetrical clonic ending       | Ipsilateral focus               |
| Fencing (M2E)                    | Contralateral (supplementary motor) |
| Figure 4                         | Contralateral to the extended limb (temporal) |
| Tonic limb posturing             | Contralateral focus             |
| Dystonic limb posturing          | Contralateral focus             |
| Unilateral ictal paresis         | Contralateral focus             |
| Postictal Todd’s paresis         | Contralateral focus             |
| **Speech**                       |                                 |
| Ictal speech arrest              | Temporal lobe (usually dominant hemisphere) |
| Ictal speech preservation        | Temporal lobe (usually nondominant) |
| Postictal aphasia                | Temporal lobe (dominant hemisphere) |

are positive expressions elicited by activation of neurons interconnected in a matrix that includes components of the hippocampal formation as well as elements of the temporal isocortex [54]. He proposed that these phenomena could be initiated by activation of different parts of the matrix and therefore did not distinguish between mesial and neocortical onset of seizures. He based his hypothesis on concepts of parallel distributed processing as applied to parallel distributed cortical networks for higher cognitive functions [56, 57].

4. **Lateralizing Features in Temporal Lobe Epilepsy**

Unilateral upper limb automatisms are associated with an ipsilateral seizure onset [58, 59]. Some authors, however, dispute this and found no lateralizing value of upper limb automatisms in isolation [11, 60, 61]. Other authors, however, reported that early onset unilateral motor automatisms without dystonic posturing can localize seizure origin to the contralateral temporal lobe neocortex [45]. Unilateral ictal blinking (winking) is a rare automatism and usually indicates an ipsilateral focus [62]. Postictal nose rubbing or wiping is also usually associated with an ipsilateral focus [63]. Postictal head turning or head tilt occurring early especially with preserved consciousness is usually ipsilateral to seizure onset [59]. It is most often contralateral to the seizure focus when it occurs later. It is also more forceful and appears involuntary, earning the characterization of versive [59, 64]. Eye deviation is usually associated with forced head turning and occurs in the same direction. Version occurs in both temporal and extratemporal onset seizures and is frequently followed by dystonic or tonic posturing occurring just before or concurrently with secondary generalization [60].

Unilateral tonic limb posturing is associated with a contralateral seizure focus. This was reported in an early study 3 decades ago [65]. There has been an agreement with this observation in several studies since then [59, 66]. Dystonic posturing of the arms and leg reliably predicts seizure
onset in the contralateral hemisphere [59–61]. It has been attributed to spread from the amygdale and hippocampus to the ventral striatum and pallidum through the fornix and stria terminalis [61]. The typical hand posture includes wrist flexion, finger extension at the interphalangeal joints, and flexion at the metacarpal-phalangeal joints, thumb adduction. Asymmetric tonic limb posturing (figure of 4 sign) is usually observed during the early tonic phase of a seizure, just before secondary generalization. One arm is flexed at the elbow and the other arm is extended at the elbow, hence, the appearance of a figure of 4. Seizure onset is contralateral to the extended arm [61].

Unilateral ictal paresis or “immobile limb,” [67] is an infrequent sign contralateral to the seizure focus. It involves sudden loss of tone in one upper limb while the other upper limb maintains tone and movement (automatism) [68].

Lower facial weakness (mild to severe) has been noted contralateral to a unilateral temporal lobe focus in 3/4 of a sample of 50 patients. The weakness was reportedly more prominent with mimetic movements. Facial asymmetry may, therefore, be a useful sign in temporal lobe epilepsy in combination with other semiological features [69].

Rare autonomic phenomena include ictus emeticus, ictal urinary urge, and ictal spitting or drinking localizing to a right temporal origin and piloerection to a left temporal origin [59, 70]. Ictal whistling and postictal coughing localize to a temporal lobe origin but are not lateralizing [36, 70, 71]. Postictal vomiting has no lateralizing or localizing value [70].

Language disturbances in association with temporal lobe seizures can include expressive, receptive, or global aphasia as well as dyslexia. Speech arrest at seizure onset (before altered consciousness) or ictal or postictal aphasia reliably implies dominant hemisphere seizure origin [72–74]. Speech arrest, as the origin of seizure, can occur if the patient is talking at seizure onset. It can also be confirmed if the patient is unable to speak despite clear attempts to do so and is able to recall this following termination of the clinical event. Its mechanism may be the involvement of Wernicke’s area, Broca’s area, or the dominant baso-temporal area since electrical stimulation of these areas produces speech arrest without loss of consciousness or motor impairment [73]. Postictal aphasia is a very reliable lateralizing sign (80–90%) [59, 75], but specific postictal language testing must be utilized by the EMU in order to detect this phenomenon. Anomia and paraphasic errors are easy to demonstrate during seizures [59, 72]. Paraphasic errors and alexia are clearly associated with CPSs originating in the dominant hemisphere [74]. Ictal speech preservation reliably predicts seizures of nondominant hemisphere origin [58, 72]. Seizures of nondominant hemispheric origin, however, may interfere with speech function on the basis of postictal confusion [70, 76].

5. Age and the Semiology of Temporal Lobe Epilepsy

Age at first seizure can influence the semiology of temporal lobe seizures. Several studies have noted a predominance of mesial temporal foci in younger onset patients and neocortical foci in older onset patients [77, 78]. This may suggest that mesial temporal structures are more susceptible to early development of epileptogenesis. For more details on this topic, the reader may refer to the dedicated article in this special issue. Several authors have noted an association between the occurrence of auras and a mesial temporal origin [16, 79]. This correlates with a predominance of HS in patients with a younger onset of TLE. Not all studies are in agreement on this association [80]. There is also an association of epigastric auras with HS, which also favours a younger onset population with TLE [52, 77, 78].

Several studies have noted that elderly patients (>60 years) tend to have seizures that become less elaborate and shorter in duration and have a lesser tendency to go on to secondary generalization [81]. Thus, aging may have an independent effect on seizure semiology. For more details on this topic, the reader may refer to the dedicated article in this special issue.

Blinking is a rare automatism that has been noted by some to occur in patients with older onset of TLE [77]. It has been noted to have a lateralizing value when it is unilateral. It is usually ipsilateral to the side of seizure onset [82].

6. Semiology of Childhood Onset Temporal Lobe Epilepsy

The semiology of TLE in childhood seems to be influenced by age-related mechanisms. Ictal features in young children do not seem to provide many localizing or lateralizing clues to the ictal origin [83–85].

Young preschool children often manifest an arousal type of reaction as the initial event with eye opening, sitting up, or axial jerking [83]. Some exhibit epileptic spasms resembling “infantile spasms” [83, 86]. Most studies in young children describe initial motor features such as tonic, dystonic, and clonic movements bilaterally. They tend to be symmetrical and more typical of secondarily generalized seizures [87, 88].

Older children may exhibit semiological features similar to adults. The occurrence of initial motor features decreases parallel with age and mostly disappears in school age children. Older children exhibit auras, psychomotor arrest, and automatisms. They are mainly oral and manual and tend to be less complex than in adults [89]. Automatisms tend to become more complex in parallel with increasing age [84, 88, 90]. There also seems to be an age-dependent increase in the occurrence of lateralizing signs such as unilateral dystonic, tonic, and clonic components. Also, asymmetric epileptic spasms, ictal speech and unilateral blinking, and ictal spitting and post ictal nose wiping are more common in older patients with TLE [84].

Interestingly, auras with autonomic and emotional features seem to be unaffected by the maturational process [90].

Secondary generalization of temporal lobe seizures is uncommon in childhood [87, 91, 92]. This may be related to age-dependent cortical maturation, immature dendritic development, and myelination together with imperfect synchronization of both hemispheres [93].
There are age-dependent changes in epileptic manifestations despite a seemingly identical underlying pathophysiology. An example is the progression from West syndrome to Lennox-Gastaut Syndrome (LGS) and the development of CPSs and secondarily generalized tonic-clonic seizures (GTCS) [94].

The locus of seizure origin within the temporal lobe and the underlying etiologies vary with age. For example, epilepsy, secondary to MCD, presents at a mean age of 7 years (range 1–26 years) [10, 89]. Neoplasms and cerebrovascular disease are commonest in later life [94].

7. Localization Reliability of Semiological Features in Temporal Lobe Epilepsy

Semiaology of temporal lobe seizures that occur during sleep or wake seem to reliably show the same lateralizing features in individual patients [92]. Secondary generalization occurs more frequently in temporal lobe seizures occurring during sleep. This is also a feature of rapid drug withdrawal in patients in an EMU before possible surgical treatment of their epilepsy. Other semiological features, however, are not substantially altered and seizures recorded under these circumstances, therefore, appear to be the same as the patient’s habitual seizures [92, 95].

Localizing the onset of seizures based on ictal semiology has been reportedly accurate in the majority of patients with partial epilepsy [96]. False localizations raise the possibility of multifocal epilepsy [91]. Seizure semiology has reportedly been accurate in localizing ictal onset in only 59–67% of patients with multifocal epilepsy [97, 98].

Kotagal and associates analyzed 31 Engel class 1 patients, who had complex partial seizures of temporal lobe onset to detect symptom clusters and seizure progression. The 18 most common symptoms were found to form a tight cluster with several subclusters: (i) epigastric aura, ictal emesis, alimentary, and hand automatisms; (ii) behavioral arrest, complete loss of consciousness, staring and bilateral facial contraction; (iii) unilateral dystonic posturing of an arm, mimetic automatisms, complex gestures, ictal speech, partial loss of consciousness; (iv) looking around, agitation, vocalizations, and whole-body movements. A strong association of epigastric sensation and ictal vomiting was noted with right temporal origin foci. The commonest order or sequence of symptoms consisted of behavioral arrest followed by alimentary and hand automatisms, then random gazing and whole-body movements [99].

Frontal lobe onset is the second most common site of origin of complex partial seizures. An aura of an indescribable sensation or generalized body sensation is said to occur exclusively in frontal lobe complex partial seizures, while an oroalimentary aura was more frequent and occurred earlier in mesial temporal seizures. Perseverative automatisms and emesis are said to occur only in temporal lobe seizures. Olfactory and experiential auras followed by behavioral arrest, alimentary and distal upper limb automatisms, loss of consciousness, and purposeless looking around coupled with whole-body movements were typical of mesial temporal lobe seizures. Bicycling movements and hypermotor activity were noted to be typical of frontal lobe seizures [100].

8. Orgasmic Aura’s Lateralizing and Localizing Semiological Features in TLE

Orgasmic auras most frequently represent a right hemispheric or nondominant hemispheric ictal onset. Some are associated with genitalia sensations but many are purely experiential. They have good lateralizing as well as localizing value when they represent the initial manifestation of the seizure [101–103].

Auras often provide valuable localization information but not useful lateralization features [104]. Orgasmic auras represent a useful exception. Most reported cases of ictal orgasm are revealed after a long epileptic history because patients are reluctant to disclose such intimate details. It is likely that many more patients have experienced orgasmic auras but never reported this feature. There are often other clinical features in reported cases that are typical of TLE such as fear, déjà vu, epigastric sensations, olfactory, and gustatory hallucinations [101]. Epileptic discharges from limbic elements of the temporal lobe commonly produce emotional phenomena such as fear, hallucinations, or illusions, and erotic feelings represent an extension of these experiential phenomena.

The occurrence of erotic ictal phenomena is more frequent in females as opposed to the more common nonerotic ictal genital sensations reported by males. This suggests that the neural organization of psychosexual behavior differs in male and female brains. This could point to anatomic as well as functional dimorphism of the limbic components of the temporal lobe in humans [101].

Physiological orgasm can occur without any physical stimulation, which points to a central neural mechanism and may be further evidence in support of a nondominant right hemispheric origin of the ictal onset.

9. Semiology of Benign Mesial Temporal Lobe Epilepsy

Benign mTLE (bMTLE) is defined as mTLE with at least 24 months of seizure freedom with or without AEDs. It was recognized many years ago [105]. More recent studies have attempted to establish the clinical features and prognosis of this group [106]. The epidemiological study of this entity is difficult because it can only be documented after a long period of observation. bMTLE is easily treated with one AED, such as, carbamazepine or oxcarbazepine. Most studies of TLE have focused on medically intractable patients.

In bMTLE, seizures begin in late adolescence to mid-adult life. Past histories are devoid of many risk factors, such as, head injury, stroke, or substance abuse. Some 1/3 have a family history of febrile seizures. The neurological exam is usually normal. Viscerosensory or experiential auras are the commonest ictal symptoms. Déjà vu often represents the only type of seizure for many years. Infrequent partial seizures are noted in about 2/3 of patients before treatment,
and secondarily generalized seizures are rare and tend to occur during sleep [107]. Two thirds have normal interictal EEG’s, but the majority of bMTLE with MRI signs of HS have interictal EEG abnormalities [107]. Because of this mild clinical picture, many patients are misdiagnosed as panic disorders or GI disturbances. This is especially true since many experience seizures that consist of a déjà vu phenomenon only. Since a number of nonepileptic individuals experience déjà vu phenomena, many of these patients do not come to neurological attention unless they develop other seizure manifestations.

Evidence of HS may be seen in 30–40% of bMTLE patients [106, 107]. Since febrile seizures, HS, early age onset of seizures and interictal EEG abnormalities are negative prognostic factors, other factors, both genetic and environmental must play a pivotal role in causation and severity of seizures [108].

Clinical neuroimaging, EEG features, and seizure outcome of bMTLE patients are similar to familial mTLE, [109, 110] suggesting that genetic predisposition is an important causal factor in bMTLE.

References

[1] H. Gastaut, “Clinical and electroencephalographical classification of epileptic seizures,” Epilepsia, vol. 11, no. 1, pp. 102–113, 1970.

[2] F. E. Dreifuss, J. Bancaud, and O. Henriksen, “Proposal for revised clinical and electroencephalographic classification of epileptic seizures,” Epilepsia, vol. 22, no. 4, pp. 489–501, 1981.

[3] J. Roger, F. E. Dreifuss, M. Martinez-Lage et al., “Proposal for revised classification of epilepsies and epileptic syndromes,” Epilepsia, vol. 30, no. 4, pp. 389–399, 1989.

[4] A. T. Berg, S. F. Berkovic, M. J. Brodie et al., “Revised terminology and concepts for organization of seizures and epilepsies: report of the ILAE Commission on Classification and Terminology, 2005–2009,” Epilepsia, vol. 51, no. 4, pp. 676–685, 2010.

[5] J. H. Jackson and P. Stewart, “Epileptic attacks with a warning of a crude sensation of smell and with the intellectual aura (dreamy state) in a patient who had symptoms pointing to a gross organic disease of the right temporal sphenoidal lobe,” Brain, vol. 22, pp. 534–549, 1899.

[6] F. A. Gibbs, E. L. Gibbs, and B. Fuster, “Psychomotor epilepsy,” Archives of Neurology and Psychiatry, vol. 60, pp. 331–339, 1948.

[7] H. H. Jasper and W. A. Hawke, "Electroencephalography, IV: localization of seizure waves in epilepsy," Archives of Neurology and Psychiatry, vol. 39, pp. 885–901, 1938.

[8] W. G. Penfield and H. H. Jasper, Epilepsy and the Functional Anatomy of the Human Brain, Little Brown & Co., Boston, Mass, USA, 1954.

[9] A. V. Delgado-Escueta, F. E. Bascal, and D. M. Treman, “Complex partial seizures on closed circuit television and EEG: a study of 692 attacks in 79 patients,” Annals of Neurology, vol. 11, pp. 292–300, 1982.

[10] W. H. Theodore, R. J. Porter, and J. K. Penry, “Complex partial seizures: clinical characteristics and differential diagnosis,” Neurology, vol. 33, no. 9, pp. 1115–1121, 1983.

[11] S. F. Berkovic and P. F. Bladin, “An electro clinical study of complex partial seizures,” Epilepsia, vol. 25, pp. 666–669, 1984.

[12] F. J. Rugg-Gunn, N. A. Harrison, and J. S. Duncan, “Evaluation of the accuracy of seizure descriptions by the relatives of patients with epilepsy,” Epilepsy Research, vol. 43, no. 3, pp. 193–199, 2001.

[13] A. Janati, W. J. Nowack, S. Dorsey, and M. Z. Chesser, “Correlative study of interictal electroencephalogram and aura in complex partial seizures,” Epilepsia, vol. 31, no. 1, pp. 41–46, 1990.

[14] P. Williamson, H. Wieser, and A. Delgado-Escueta, “Clinical characteristics of partial seizures,” in Surgical Treatment of the Epilepsies, J. Engel Jr., Ed., pp. 101–120, Raven Press, New York, NY, USA, 1987.

[15] C. Ajmone-Marsan, "Commentary: clinical characteristics of partial seizure," in Surgical Treatment of the Epilepsies, J. Engel Jr., Ed., pp. 121–127, Raven Press, New York, NY, USA, 1987.

[16] R. M. Sadler, “The syndrome of mesial temporal lobe epilepsy with hippocampal sclerosis: clinical features and differential diagnosis,” Advances in Neurology, vol. 97, pp. 27–37, 2006.

[17] J. A. French, P. D. Williamson, V. M. Thadani et al., “Characteristics of temporal lobe epilepsy: I. Results of history and physical examination,” Annals of Neurology, vol. 34, no. 6, pp. 774–780, 1993.

[18] P. Gloo, A. Olivier, L. F. Quesney, E. Andermann, and S. Horowitz, “The role of the limbic system in experimental phenomena of temporal lobe epilepsy,” Annals of Neurology, vol. 12, no. 2, pp. 129–144, 1982.

[19] M. Pfänder, S. Arnold, A. Henkel et al., “Clinical features and EEG findings differentiating mesial from neocortical temporal lobe epilepsy,” Epileptic Disorders, vol. 4, no. 3, pp. 189–195, 2002.

[20] A. Fogarasi, I. Tuxhorn, J. Janszky et al., “Age-dependent seizure semiology in temporal lobe epilepsy,” Epilepsia, vol. 48, no. 9, pp. 1697–1702, 2007.

[21] W. Penfield and H. H. Jasper, Epilepsy and the Functional Anatomy of the Human Brain, Little Brown & Co., Boston, Mass, USA, 1954.

[22] S. R. Benbadis, P. Thomas, and G. Pontone, “A prospective comparison between two seizure classifications,” Seizure, vol. 10, no. 4, pp. 247–249, 2001.

[23] H. Lüders, J. Acharya, C. Baumgartner et al., “Semiological seizure classification,” Epilepsia, vol. 39, no. 9, pp. 1006–1013, 1998.

[24] W. T. Blume, H. O. Lüders, E. Mizrahi, C. Tassinari, V. M. Thadani et al., “The role of the limbic system in experimental phenomena of temporal lobe epilepsy,” Annals of Neurology, vol. 20, no. 5, pp. 924–937, 1986.

[25] E. Halgren, C. L. Wilson, and J. M. Stapleton, “Human medial temporal-lobe stimulation disrupts both formation and retrieval of recent memories,” Brain and Cognition, vol. 4, no. 3, pp. 287–295, 1985.
Epilepsy Research and Treatment

S. M. Mirsattari, D. H. Lee, and W. T. Blume, "Contralateral J. H. Cross, P. Jayakar, D. Nordli et al., "Proposed criteria for P. D. Williamson, J. A. French, V. M. Thadani et al., "Char- F. Andermann, E. Kobayashi, and E. Andermann, "Genetic F. Cendes, I. Lopes-Cendes, E. Andermann, and F. Ander- 121–124, 2004.

1182–1188, 1998.

1126–1132, 2010.

1560–1565, 2009.

Epilepsia, vol. 39, no. 11, pp.

Epilepsia, vol. 39, no. 11, pp.

Epilepsy, vol. 50, no. 6, pp. 1560–1565, 2009.

Epilepsia, vol. 50, no. 6, pp. 1542–1546, 2009.

Epilepsia, vol. 50, no. 6, pp. 925–959, 2006.

Epilepsia, vol. 47, no. 6, pp.

Epilepsia, vol. 50, no. 6, pp. 781–787, 1993.

Pediatric Neurology, vol. 12, no. 3, pp. 201–206, 1995.

Epilepsia, vol. 30, no. 4, pp. 389–399, 1989.

Epilepsia, vol. 40, no. 2, pp. 227–235, 1996.

Epilepsia, vol. 50, no. 2, pp. 554–557, 1998.

Epilepsia, vol. 46, no. 10, supplement, pp. 61–67, 2005.

Epilepsia, vol. 49, no. 6, pp. 1046–1054, 2008.

Canadian Journal of Neurological Sciences, vol. 31, no. 1, pp.
[65] D. W. King and C. Ajmone-Marsan, "Clinical feature and ictal patterns in epileptic patients with EEG temporal lobe foci," *Annals of Neurology*, vol. 2, pp. 138–147, 1977.

[66] J. Wada, "Cerebral lateralization and epileptic manifestations," in *Advances in Epileptology: XIII the Epilepsy International Symposium*, H. Akimoto, Ed., pp. 366–372, Raven Press, New York, NY, USA, 1982.

[67] E. Walker and F. Sharbrough, "The significance of lateralized ictal paroxysm occurring during complex partial seizures," *Epilepsia*, vol. 29, p. 665, 1988.

[68] L. J. Oestreich, M. J. Berg, D. L. Bachmann, J. Burchfiel, and G. Erba, "Ictal contralateral paresis in complex partial seizures," *Epilepsia*, vol. 36, no. 7, pp. 671–673, 1995.

[69] R. W ennberg, "Postictal coughing and noserubbing coexist in temporal lobe epilepsy," *Epilepsia*, vol. 33, no. 2, pp. 83–90, 2002.

[70] M. M. S. Jan and J. P. Girvin, "Seizure semiology: value in identifying seizure origin," *Canadian Journal of Neuroradiology*, vol. 35, no. 1, pp. 22–30, 2008.

[71] R. Fakhoury, B. Abou-Khalil, and E. Peguero, "Diagnosing epilepsy from the right hemisphere," *Epilepsia*, vol. 34, no. 5, pp. 859–868, 1993.

[72] A. Brockhaus and C. E. Elger, "Complex partial seizures of different age groups," *Epilepsia*, vol. 36, no. 12, pp. 1173–1181, 1995.

[73] P. Jayakar and M. S. Duchowny, "Complex partial seizures of temporal lobe origin in early childhood," *Journal of Epilepsy*, vol. 3, supplement, pp. 41–45, 1990.
[103] J. Bencaud, P. Favel, A. Bonis et al., “Manifestations sexuelles paraoxiques et épilepsie temporaire,” Revista de Neurología, vol. 123, pp. 217–230, 1970.

[104] A. Palmini and P. Gloor, “The localizing value of auras in partial seizures: a prospective and retrospective study,” Neurology, vol. 42, no. 4, pp. 801–808, 1992.

[105] S. Currie, K. W. G. Heathfield, R. A. Henson, and D. F. Scott, “Clinical course and prognosis of temporal lobe epilepsy: a survey of 666 patients,” Brain, vol. 94, no. 1, pp. 173–190, 1971.

[106] U. Aguglia, A. Gambardella, E. Le Piane et al., “Mild non-lesional temporal lobe epilepsy: a common, unrecognized disorder with onset in adulthood,” Canadian Journal of Neurological Sciences, vol. 25, no. 4, pp. 282–286, 1998.

[107] A. Labate, P. Ventura, A. Gambardella et al., “MRI evidence of mesial temporal sclerosis in sporadic “benign” temporal lobe epilepsy,” Neurology, vol. 66, no. 4, pp. 562–565, 2006.

[108] F. Pittau, F. Bisulli, R. Mai et al., “Prognostic factors in patients with mesial temporal lobe epilepsy,” Epilepsia, vol. 50, no. 1, supplement, pp. 41–44, 2009.

[109] A. Labate, A. Gambardella, E. Andermann et al., “Benign mesial temporal lobe epilepsy,” Nature Reviews Neurology, vol. 7, no. 4, pp. 237–240, 2011.

[110] S. F. Berkovic, A. McIntosh, R. Anne Howell, A. Mitchell, L. J. Sheffield, and J. L. Hopper, “Familial temporal lobe epilepsy: a common disorder identified in twins,” Annals of Neurology, vol. 40, no. 2, pp. 227–235, 1996.