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Localizing and lateralizing features of auras and seizures

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ABSTRACT

The symptomatology of auras and seizures is a reflection of activation of specific parts of the brain by the ictal discharge, the location and extent of which represent the symptomatogenic zone. The symptomatogenic zone is presumably, though not necessarily, in close proximity to the epileptogenic zone, the area responsible for seizure generation, the complete removal or disconnection of which is necessary for seizure freedom. Knowledge about the symptomatogenic zone in focal epilepsy is acquired through careful video/EEG monitoring and behavioral correlation of seizures and electrical stimulation studies. Ictal symptomatogy provides important lateralizing and/or localizing information in the presurgical assessment of epilepsy surgery candidates. As the initial symptoms of epileptic seizures, many types of auras have highly significant localizing or lateralizing value. Similarly, motor signs during focal and secondary generalized seizures, language manifestations, and autonomic features offer reliable clues to the delineation of the epileptogenic zone. Some focal epilepsies (e.g., neocortical temporal lobe epilepsy, insular lobe epilepsy, temporal-plus epilepsies, and parieto-occipital lobe epilepsy) generate seizure manifestations that mimic temporal lobe epilepsy, potentially contributing to surgical failure. To optimize surgical outcome, careful interpretation of ictal symptomatology in conjunction with other components of the presurgical evaluation is required.

1. Introduction

The goal of the presurgical evaluation is to identify the epileptogenic zone (EZ), the cortical area generating seizures, the complete removal or disconnection of which is necessary for seizure freedom. However, no diagnostic method directly assesses the location and extent of the EZ. There are five cortical zones (symptomatogenic zone [SZ], irritative zone, ictal onset zone, epileptogenic lesion, and functional deficit zone) obtained from different testing modalities used to estimate the EZ [1]. Favorable surgical outcomes are obtained when these zones overlap significantly.

2. General principles of ictal symptomatology

The SZ, the cortical area(s) producing signs and symptoms when activated by the ictal discharge, is generally in close proximity to the EZ [2]. Electrical stimulation (ES) studies have shown that most of the cortex is silent. This implies that clinical signs and symptoms emerge only when epileptic activity spreads to activate SZ areas. When ictal signs and symptoms are considered sequentially, they provide useful clues to propagation patterns and reflect the anatomical sites involved during seizure propagation. In the course of a clinical seizure, multiple SZs may be activated at once and one sign or symptom may obscure others, potentially producing variable or misleading findings. Some initial signs and symptoms are generated by activation of a well-defined cortical area with a high degree of certainty (e.g., localized paresthesias in a restricted dermatome due to excitation of the primary sensory area in the contralateral hemisphere). In such cases, the EZ and SZ are likely to be closely related. Conversely, seizures may arise from different areas and evolve to the same SZ, producing similar clinical symptoms (e.g., orbitofrontal or mesial parieto-occipital seizures can indistinctly produce supplementary sensorimotor area [SSMA] semiology). However, seizures tend to follow preferential pathways, so even if the symptom itself has no useful localizing or lateralizing value, it may shed light on the network activated. For example, a dialeptic component characterized by motionless staring and unresponsiveness, evolving to an automotor phase (oral or gestural automatisms), is a common manifestation of temporal lobe epilepsy (TLE), whereas an isolated dialeptic phase is of no localizing value. Identification of the SZ requires careful integration of information gathered from the clinical history, analysis of video/EEG (VEEG) recordings, and ES as each component in isolation has limitations [3]. The age, intellect, mood, and mental status of the patient and observers and their ability and willingness to describe ictal symptoms affect the value contributed by the clinical history. The VEEG evaluation is dependent on the recording of reproducible ictal signs and symptoms of sufficient quality with appropriate technical interventions. Finally, although ES is the gold standard for measuring the effect of activation of a cortical area, findings often raise concern that distant areas are activated.

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3. Localizing/lateralizing value of auras and clinical seizures

3.1. Auras

As the first ictal symptoms, auras can provide important localizing and lateralizing value useful in determining the relationship between the SZ and the EZ (Table 1). Auras are reported by most patients with temporal and parieto-occipital epilepsies and have been associated with a favorable surgical outcome after temporal lobectomy [4]. The localizing value of auras in identifying the most likely lobe of origin was found to be as good as that of EEG and imaging [5].

Somatosensory auras include tingling, numbness, electrical shock-like feelings, thermal sensations, and pain. When originating from the primary somatosensory area (SI), they involve discrete parts of the body contralateral to the ictal discharge. The second sensory area (SII) gives rise to similar symptoms that are bilateral or ipsilateral to the seizure focus. The SSMA produces sensory symptoms that are poorly localized and involve primarily contralateral proximal body parts. Various types of somatosensory auras are elicited by stimulation of the insular cortex. Well-localized somatosensory auras with evolution to motor features are usually associated with an EZ in the perirolandic area close to the SZ [6]. Somatosensory illusions, including sensations of swelling, shrinking, and movement of body parts, are often elicited from nondominant inferior parietal lobe or temporo-parieto-occipital (TPO) junction activation.

Visual auras include both simple and complex manifestations. Simple visual auras such as static, flashing, or moving lights in different shapes and colors are characteristic of activation of the primary visual cortex and contiguous visual association areas. Complex visual auras of people, scenes, objects, and illusions suggest activation of the temporo-occipital junction or basal temporal cortex. Stimulation of the precuneus, posterior cingulum, or mesial parieto-occipital region produces blurry vision or visual motions [7]. Visual phenomena lateralized to one hemifield as an early ictal manifestation are highly suggestive of an ictal onset in the contralateral occipital lobe. Visual symptoms restricted to the lower or upper quadrant predictably localize to the contralateral supra- or infracalcarine fissure, respectively. Visual distortions (e.g., micropsia, macropsia, metamorphopsia, and palinopsia) suggest activation close to the geniculostriate radiation.

Auditory auras, like visual auras, range from simple to complex in character. Simple auditory auras include ringing and buzzing sounds. Complex auditory phenomena include voices and music. The SZ of simple auditory auras is in the primary auditory cortex. Complex auditory hallucinations and illusions are produced by activation of auditory association areas in the temporo-occipital cortex. Although each hemisphere has bilateral innervation for auditory information, the contralateral ear is better represented in the auditory cortex.

Vertiginous auras include sensations of rotation or movement in all planes that are usually associated with visual or auditory symptoms. The SZ is close to visual and auditory association areas in the temporo-parietal junction. ES studies produced easily elicited vestibular symptoms and rotatory sensations in a lateral cortical temporoparietal area extending above and below the Sylvian fissure, including the parietal operculum and the middle and posterior part of the superior and middle temporal gyri [8].

Olfactory auras are typically unpleasant sensations, often associated with gustatory phenomena. The areas shown to consistently produce olfactory sensations when stimulated include the amygdala, olfactory bulb, and insular cortex. Stimulation in the posterior part of the orbitofrontal region generates olfactory illusions [9]. For many patients, *gustatory auras* are hard to differentiate from olfactory disturbances. Stimulation of the parietal operculum and mesiobasal temporal regions generates gustatory hallucinations [10].

Autonomic auras include cardiorespiratory (e.g., palpitations and shortness of breath), gastrointestinal, genitourinary (genital sensations,

Table 1
Localization and lateralization of epileptic signs and symptoms.
Source. Modified from Rona [61].

Seizure type	Subtype	Symptomatogenic zone[a]	Lateralization	Epilepsy syndrome[b]
Auras	Somatosensory	Primary somatosensory cortex (areas 1,2, and 3b)	CL[c]	PLE
		Secondary somatosensory areas (parietal operculum/SSII)	IPSI (if unilateral)	PLE, TLE
		SSMA	CL (mostly)	PLE, FLE
	Simple visual	Primary visual cortex (areas 17, 18, and 19)	CL	OLE
	Complex visual	Temporo-occipital junction and basal temporal cortex	CL (if unilateral)	TLE, OLE
	Simple auditory	Primary auditory cortex (area 41)	CL (if unilateral)	TLE
	Complex auditory	Auditory association cortex (areas 42 and 22)	CL (if unilateral)	TLE
	Vertiginous	Temporo-occipital junction	NonLAT (often right)	TLE
	Olfactory	Orbitofrontal region, amygdala, and insula	NonLAT	MTLE, FLE
	Gustatory	Parietal operculum and basal temporal cortex	NonLAT	TLE
	Autonomic	Insula, amygdala, anterior cingulum, and SSMA	NonLAT	TLE, FLE
	Abdominal	Anterior insula, frontal operculum, mesial temporal lobe, and SSMA	NonLAT	MTLE
	Fear	Amygdala, hippocampus, and mesial frontal lobe	NonLAT	TLE, FLE
	Déjà vu/jamais vu	Uncus, entorhinal cortex, and temporal neocortex	NonLAT (often ND)	TLE
	Multisensorial	Mesiobasal limbic cortex, temporal neocortex, TPO junction	NonLAT	TLE, PLE
	Cephalic/whole body	Amygdala, entorhinal cortex, and temporal neocortex/SSII and SSMA	NonLAT	NTLE, FLE
Simple motor	Myoclonic/negative myoclonus	Primary motor cortex (area 4) and premotor cortex (area 6)/primary somatosensory area	CL (if unilateral)	FLE
	Clonic	Primary motor cortex, premotor cortex, and SSMA	CL	FLE
	Tonic	Primary motor cortex and SSMA	CL (if unilateral)	FLE
Complex motor	Hypermotor	Anterior cingulum, orbitofrontal region, frontopolar region, opercular–insular cortex, and medial intermediate frontal area	NonLAT	FLE
	Automotor	Mesial temporal and anterior cingulum	NonLAT	TLE, FLE
	Gelastic	Hypothalamus, anteromesial frontal region, and basal temporal area	NonLAT	FLE, TLE
Dialeptic		Limbic temporal structures, cingulum, intermediate frontal (area 8) and orbitofrontal areas	NonLAT	
Autonomic	Tachycardia/ hyperventilation	Amygdala, insula, anterior cingulum, and medial prefrontal cortex	NonLAT (often right)	TLE
	Pilorection		IPSI	TLE
	Mydriasis		IPSI (if unilateral)	TOLE

[a] Typical symptomatogenic zones are provided.
[b] Common focal epilepsy syndromes.
[c] CL, contralateral; D, dominant; FLE, frontal lobe epilepsy; IPSI, ipsilateral; ND, nondominant; NonLAT, nonlateralizing; OLE, occipital lobe epilepsy; PLE, parietal lobe epilepsy; TLE, temporal lobe epilepsy; SSMA, supplementary sensorimotor area epilepsy; TOLE, temporo-occipital lobe epilepsy; TPO junction, temporo-parieto-occipital junction.

urinary urge), and cutaneous (feeling of warmth or cold) sensations. Autonomic symptomatology is produced by activation of the insular cortex, anterior cingulum, SSMA, or amygdala. These areas have been found to share the same subcortical relay station as the hypothalamus [11]. Abdominal auras constitute the most common type of autonomic aura. These include sensations of nausea, pain, or indescribable discomfort in the abdominal or periumbilical area that can be static, rise to the chest and throat, or descend into the lower abdominal region. The SZ for abdominal auras is the anterior insular cortex, frontal operculum, mesial temporal structures, and SSMA. In isolation, abdominal auras are highly associated with TLE (probability of 74%); when evolving into an automotor component, the probability of TLE increases to 98% [12]. When combined with vomiting, abdominal auras are suggestive of an EZ in the nondominant temporal lobe [13]. Orgasmic auras are defined as erotic thoughts and feelings, sexual arousal, and orgasm that are occasionally accompanied by viscerosensory phenomenon (i.e., vulvovaginal secretion). This has been observed more in women with right TLE [14]. Genital auras are often painful, unpleasant sensations associated with fear. The SZ is localized to the postcentral parasagittal region but, when bilateral, can arise from activation of the SII area [15].

Psychic auras include emotional symptoms (e.g., fear, anxiety, impending doom, and elation) and distortions of familiarity (e.g. déjà vu, jamais vu, and multisensorial hallucinations including revocation of complex memories). The SZ resides in the temporal neocortex or mesial temporal structures with the exception of forced thoughts, generally observed in frontal lobe epilepsy (FLE). Fear is produced by activation of the amygdala, hippocampus, mesial frontal region, or temporal neocortex [11]. Pleasant emotional auras (e.g., euphoria and satisfaction) are associated with activation of the mesiobasal temporal area [16]. Multisensorial hallucinations require activation of mesiobasal temporal, lateral temporal, or TPO junction. Out-of-body experiences are produced by stimulation in the region of the temporoparietal junction.

Nonspecific auras are those that do not fall into a more specific category. Cephalic auras are nonvertiginous head sensations such as dizziness, lightheadedness, electrical shock-like feelings, head numbness, and pressure. These sensations can arise from the amygdala, entorhinal cortex, and lateral temporal neocortex, but often are of little localizing value. Whole-body auras are generalized body sensations that may be elicited by stimulation of SII and the SSMA.

3.2. Simple motor seizures

Simple motor seizures are characterized by unnatural, relatively simple movements that are reproduced by ES of the primary and somatosensory motor areas. Seizures in this category are of several types based on the duration of contraction, rhythmicity of movement, and muscle groups involved.

Myoclonic seizures are sudden, irregular muscle jerks of short duration (<400 ms). They are usually generalized or bilateral, prominently affecting the shoulders and proximal arms, but can be focal. Unilateral myoclonic seizures are generated from the contralateral primary motor area or premotor cortex. Negative myoclonus consists of brief periods (20–400 ms) of muscle atonia that occur when a muscle is contracted (i.e., sudden drop of an extended arm). Negative epileptic myoclonus is rarely observed in patients with perirolandic epilepsy [17].

Clonic seizures are characterized by repetitive, short contractions of agonist and antagonist muscle groups, recurring at regular intervals of 0.2–5 per second. The distal extremity or face is usually affected because of its relatively large cortical representation. Spread of clonic seizures from distal to proximal (Jacksonian march) reflects propagation of epileptic activity over the motor cortex. In focal epilepsy, clonic seizures are usually preceded by an aura or an automotor or tonic phase and are part of a generalized tonic–clonic seizure (GTCS) [18]. In FLE, clonic seizures tend to occur early in the ictal sequence while consciousness is preserved, in contrast to seizures of temporal or occipital origin in which awareness is lost during the clonic component [6]. In TLE, clonic activity generally involves the face or upper extremity, usually preceded by an automotor phase. Clonic activity is typically preceded by somatosensory disturbances in seizures of parietal lobe origin and by visual auras or versive head/eye movements in seizures of occipital lobe origin. SSMA seizures usually involve bilateral clonic activity associated with tonic posturing.

Tonic seizures are characterized by sustained contraction of one or more muscle groups lasting at least 3 seconds and leading to posturing of the limbs and/or trunk. Proximal muscles are affected in a bilateral fashion, although asymmetric postures involving primarily contralateral muscles are observed in some cases. Preserved consciousness is common at least at the onset. If clearly unilateral, tonic seizures strongly support seizure origin in the contralateral hemisphere.

Versive seizures are characterized by sustained, forced, unnatural turning of the head or eyes to one side, having a tonic or clonic quality. Typically, the angle of the mouth is deviated to the same side and the head is hyperextended. When occurring immediately before GTCSs, the direction of version is contralateral to the hemisphere of seizure onset. In contrast to FLE, version in temporal lobe seizures occurs while consciousness is impaired later during the ictal sequence [19].

Tonic-clonic seizures are characterized by a sequence of a generalized tonic contraction followed by clonic activity lasting 1 to 2 minutes. Consciousness is usually disrupted at the beginning of the tonic phase. In focal epilepsy, tonic–clonic seizures are usually preceded by other semiological features suggesting a localized ictal onset.

3.3. Complex motor seizures

Complex motor behaviors are characterized by movements similar to those executed during common daily activities (e.g., clapping, swallowing, lip smacking, and coughing) or behaviors observed in movement disorders, such as dystonia. The term *complex* refers to the complexity of the movement, not the state of consciousness. Lüders and colleagues proposed this terminology as part of their semiological seizure classification in which the most prominent clinical manifestation(s) is emphasized [20]. Complex motor seizures include hypermotor seizures, automotor seizures, and gelastic seizures.

Hypermotor seizures are characterized by repetitive complex movements involving the proximal limbs and trunk that are rapid and violent in nature. Motor activity simulates normal movements inappropriate for the situation (e.g., thrashing, rocking, jumping, waving, bicycling, and kicking). Vocalization, laughter, and crying are commonly observed. Hypermotor seizures originate primarily in the frontal lobe and less commonly in the temporal lobe, posterior cortex, and insula [21]. Seizures arising in the ventromedial frontal region exhibit more hypermotor features than dorsolateral frontal seizures, which are more commonly characterized by head and eye version and complex gestural automatisms [22]. Hypermotor movements having a sexual quality (e.g., writhing, thrusting, and rhythmic movements of the pelvis or extremities) typically arise from the frontal lobes. Rarely, rotation around the body axis of at least 180° occurs in association with other hypermotor features, again suggesting frontal lobe origin. When preceded by version, the rotation direction is usually contralateral to the EZ, whereas in the absence of version, the direction of rotation is usually ipsilateral to the EZ [23].

Automotor seizures are characterized by repetitive, stereotyped, semipurposeful motor behaviors, involving primarily the distal limbs, mouth, and tongue. Automotor movements involving the mouth and tongue (oral automatisms) include mastication, swallowing, lip smacking, blowing, whistling, and kissing. Those involving the distal extremities (gestural automatisms) include fumbling, picking, and gesticulating movements. Awareness is generally impaired except in seizures restricted to the nondominant temporal lobe. Homogeneous perseverative automatisms, complex gestures, and upper limb automatisms prolonged

in duration are characteristics of TLE, whereas frontal lobe automatisms are more hyperkinetic and irregular in quality and involve proximal segments of the limbs [24]. Genital manipulations (e.g., grabbing, picking, and fondling) are rarely observed in seizures of frontal and temporal lobe origin.

Gelastic seizures are the rarest type of complex motor seizure. Characterized by brief periods of laughter or grimacing with or without the subjective feeling of mirth, this semiology strongly suggests the presence of a hypothalamic hamartoma. However, intracranial EEG and ES studies have identified the SZ for gelastic seizures in the anteromesial frontal and basal temporal regions in isolated cases [25].

The semiological features of complex motor seizures in young children differ from those of older patients because of the presence of more diverse pathological substrates and lack of brain maturation. Children under 3 to 4 years of age with TLE tend to have prominent tonic, clonic, or myoclonic movements that may be bilateral and symmetric, resembling generalized epilepsy. Automatisms, when present, are less elaborate and restricted to the orobuccal region. After age 6, the manifestations of TLE are similar to those of adults [26]. In very young patients, posterior cortex epilepsy typically presents with decreased motor activity (*hypomotor*), given the paucity of other features and inability to assess level of consciousness.

3.4. Dialeptic seizures

The term *dialeptic*, from the Greek "to interrupt, stand still, or pass out," describes seizures characterized by an alteration of consciousness and staring with minimal motor activity. The more commonly recognized term *absence* is a type of dialeptic seizure associated with generalized 3-Hz spike–wave complexes on the EEG. In contrast to typical absence seizures, dialeptic seizures are observed in patients with generalized and focal epilepsies. Dialeptic seizures alone provide no useful localizing or lateralizing information and can be seen in focal epilepsies arising from virtually any area. However, an aura preceding the dialeptic phase and the subsequent ictal sequence can provide clues to the structures activated by the ictal discharge.

3.5. Autonomic seizures

Symptoms of central autonomic nervous system activation are commonly observed in focal seizures. Autonomic seizures are seizures in which the predominant feature is autonomic in nature. In contrast to autonomic auras, measurable or visible autonomic signs are necessary for autonomic seizures. Autonomic features of focal seizures usually reflect sympathetic activation (e.g., tachycardia and hyperventilation), although parasympathetic activation can also occur. The cortical areas responsible for producing autonomic manifestations include the medial prefrontal cortex, amygdala, and insular cortex.

Cardiac manifestations are the most well recognized autonomic manifestation of focal seizures. Ictal tachycardia, defined as a heart rate >100 bpm, is reported in more than 50% of seizures. Early and significant tachycardia is more common in temporal than extratemporal lobe epilepsy (EXTLE) and is associated primarily with right mesial TLE (MTLE) [27]. Ictal bradycardia, defined as a heart rate <60 bpm, is much less common and has not been shown to have localizing or lateralizing value. Ictal asystole and arrhythmia are rare; both have been implicated in the pathogenesis of sudden unexpected death in epilepsy.

Autonomic features involving respiratory function include hyperventilation, apnea, and dyspnea. Ictal hyperventilation, defined as a 10% or greater increase in respiratory rate from baseline, was observed in seizures of more than 50% of children in one series and was more common in temporal than frontal lobe epilepsy [26]. In adult patients with TLE, ictal hyperventilation is more common in seizures arising from the mesial temporal than neocortical structures. Ictal apnea is most common in infants and neonates. Ictal dyspnea and stridor are rare and occur primarily during the tonic phase of GTCSs. Postictal nose wiping and cough are due to increased parasympathetic activity resulting in increased nasal and pharyngeal secretions. These behaviors are believed to be reflexive in nature, occurring postictally as they are inhibited during the ictal period (see below).

Gastrointestinal manifestations of focal seizures include epigastric phenomena, ictal vomiting, and defecation. Abdominal epilepsy, characterized by episodic abdominal pain, nausea, vomiting, and confusion, is a common autonomic manifestation of TLE in children [26]. Ictal vomiting is often a sign of nondominant temporal seizure origin (see below). Ictal defecation is rarely observed.

Cutaneous manifestations of focal seizures include ictal piloerection, pallor, and flushing. Ictal piloerection presents as goose bumps involving a limb ipsilateral to the seizure onset, having a marching quality. Piloerection is most common in TLE [28]. Ictal pallor, typically coexisting with other cutaneous signs, was associated with left temporal onset in one pediatric series [29]. Ictal flushing, involving mainly the face, has no localizing value.

Pupillary changes are not uncommon in focal epilepsy and are typically observed during GTCSs. Pupillary dilation is typically bilateral in GTCSs, but may be unilateral in focal epilepsy. Unilateral mydriasis was found to be ipsilateral to the EZ in seizures arising from the temporo-occipital region, but contralateral to the frontal focus in benign childhood epilepsy [30]. Bilateral and unilateral miosis are rarely described in seizures arising from the temporal and occipital regions.

Urogenital manifestations of focal seizures include incontinence, ictal urinary urge, orgasmic sensations, and genital sensations. These are rare phenomena that have not been extensively studied. Ictal urinary urge and orgasmic phenomena may suggest seizure origin in the nondominant temporal lobe.

4. Lateralizing motor signs in complex motor seizures

A variety of motor signs observed in focal epilepsy provide important clues related to seizure localization and lateralization (Table 2). One of the most reliable is *dystonic limb posturing*, characterized by forced, unnatural limb posturing, either in flexion or in extension, proximal or distal, having a rotatory component, often with superimposed athetosis or tremor. Dystonic limb posturing is more common in TLE than EXTLE and typically occurs following the appearance of bilateral manual automatisms or simultaneously with automatisms involving the other upper extremity. Limb dystonia is contralateral to the EZ in more than 90% of cases [31]. When associated with manual automatisms and head turning ipsilateral to the EZ, it is highly suggestive of MTLE [13]. *Unilateral tonic posturing* consists of flexion or extension only, without rotation or unnatural postures. It was observed in 17% of TLE cases and 15% of EXTLE cases, contralateral to the EZ in 40–86 and 67–89% of patients, respectively [32]. *Unilateral ictal/postictal immobile limb* refers to a sudden loss of tone in an upper limb while the opposite side expresses automatisms. It was observed in 5–28% of focal seizures, usually of temporal lobe origin, contralateral to the EZ in almost all cases [33]. Postictal limb paresis (Todd's paresis/paralysis) was found in 0.5–13% of focal seizures, always contralateral to the hemisphere of seizure origin. *Nonversive head turning* is a natural and unforced lateral movement of the head. It is reported to occur in temporal and frontal epilepsies, and usually occurs ipsilateral to the epileptogenic hemisphere. However, nonversive head turning does not have the same lateralizing value as version (see below). In seizures without secondary generalization, one or two head turns in the same direction occurred ipsilateral to the EZ in 94% of seizures, whereas preceding the secondarily GTCS (SGTCS), the first of two turns was ipsilateral and the second contralateral to the EZ [34]. *Emotional facial expression/facial asymmetry* is a less common manifestation of focal seizures. Ictal smile is suggestive of nondominant hemisphere onset in children with parieto-occipital lobe epilepsy and FLE [35]. Lower facial asymmetry was found in 70% of patients with TLE, usually contralateral to the EZ [36]. Ictal crying is a rare feature of temporal or mesial frontal epilepsy. Facial

Table 2
Lateralizing signs of focal seizures.
Source. Modified from Bianchin and Sakamoto [62].

Ictal sign	Subtype	Symptomatogenic zone or mechanism[a]	Lateralization	Epilepsy syndrome[b]
Motor signs in complex motor seizures	Dystonic limb posturing	Activation of basal ganglia	CL[c]	TLE, FLE
	Tonic posturing	Activation of SSMA, basal ganglia, cingulum, and primary motor cortex	CL	FLE, TLE
	Immobile limb	Activation of negative motor areas or exhaustion of primary motor or premotor cortex	CL	TLE
	Head turning	Exhaustion of epileptogenic hemisphere, seizures propagate to basal ganglia, or neglect of CL space	IPSI	TLE
	Facial alterations	Activation of emotional network (amygdala, prefrontal cortex, hypothalamus, orbitofrontal region, insula) or emotional facial movements in cingulum	CL (if facial weakness)	TLE
	Eye version	Frontal eye fields (area 8) and extrastriate cortex (area 19)	CL	
	Unilateral eye blinking	Mesial temporal structures	IPSI	
	Nose wiping	Ictal olfactory hallucinations, increased nasal secretions, or CL postictal immobile limb	IPSI	MTLE
Nondominant temporal signs	Automatisms with preserved responsiveness	Non-speech-dominant temporal lobe and anterior cingulum	ND	TLE, FLE
	Ictal vomiting	Mesial temporal structures, insula, and mesial frontal regions	ND	TLE
	Ictal splitting	Complex automatisms, excessive salivation, or bad mouth sensations	ND	TLE
	Ictal urinary urge	Activation of central bladder control	ND	TLE
	Peri-ictal water drinking	Hypothalamic involvement	ND	TLE
	Ictal/postictal cough	Increased secretions or direct activation of central autonomic system	ND	TLE
	Unilateral ear plugging	Superior temporal gyrus	CL	TLE
Signs during secondary generalized tonic–clonic seizures	Head version	Premotor area (areas 6 and 8)	CL	FLE, TLE
	Asymmetric tonic limb posturing	SSMA and precentral area	CL	TLE, FLE
	Asymmetric ending of clonic jerks	Exhaustion of hemisphere of seizure onset	IPSI	
Language manifestation	Ictal/postictal aphasia	Anterior and posterior language areas	D	TLE
	Ictal speech	Inhibition of D hemisphere or overexcitement of ND hemisphere	ND	TLE

[a] Typical symptomatogenic zones are provided.
[b] Common focal epilepsy syndrome.
[c] CL, contralateral; D, dominant; FLE, frontal lobe epilepsy; IPSI, ipsilateral; ND, nondominant; NonLAT, nonlateralizing; TLE, temporal lobe epilepsy; MTLE, mesial temporal lobe epilepsy.

expressions of fear and anger suggest frontal lobe origin. *Eye movements* are important, often unrecognized, features of focal seizures. Eye version is a forced, sustained, conjugate deviation of the eyes typically accompanying head version. It reliably lateralizes to the contralateral hemisphere when occurring before secondary generalization. In the absence of generalization, eye version may be ipsilateral or contralateral to the epileptogenic lesion, depending on whether occipital or frontal regions are activated. Unilateral eye blinking is observed ipsilateral to the EZ in about 80% of cases and often suggests activation of the amygdala or mesial temporal structures [37]. Nystagmus is a rare phenomenon, observed predominantly in parieto-occipital epilepsies. The direction of the fast phase is usually contralateral to the EZ. *Nose wiping* is defined as wiping or rubbing of the nose during or within 60 seconds of seizure termination. It is most common in seizures of mesial temporal origin (50–85% of cases) and is performed with the hand ipsilateral to the epileptogenic temporal lobe in 75–90% of cases [38].

Focal seizures in young children are often devoid of these aforementioned motor signs. Reliable lateralizing signs were seen in 58% of seizures in infants 1–32 months of age in one series and were primarily simple motor movements [39]. In another series of children up to 3 years of age, focal ictal signs were seen in only 23% of cases [40]. Unilateral automatisms, dystonic limb posturing, version, postictal dysphasia, and nose wiping, present in up to 75% of children under 13 years of age, did not have the same localizing and lateralizing value as observed in adults.

5. Nondominant temporal signs

The following semiologies have been shown to reliably localize seizure onset to the nondominant temporal lobe (Table 2). One of the most common is *automatisms with preserved awareness* in which oral or manual automatisms occur while the patient is able to respond. Awareness is preserved because of a lack of seizure propagation to the language-dominant temporal lobe or extratemporal structures [41]. *Ictal vomiting* usually occurs in the absence of other gastrointestinal symptoms during the phase of unresponsiveness, followed by amnesia for the behavior postictally [42]. It is also an early manifestation of benign childhood epilepsies. *Ictal spitting* is most often observed in nondominant TLE and less commonly in seizures arising from the dominant temporal, insular, or frontal regions [43]. Similarly, *ictal urinary urge* and *peri-ictal water drinking*, drinking occurring during or within 2 minutes of termination of an automotor seizure, are usually seen in seizures arising from the nondominant hemisphere, often the temporal lobe [44,45]. *Ictal/postictal cough* can be observed during the ictal and postictal periods of seizures arising from the temporal and extratemporal regions, though more often in the former [46].

6. Speech and language manifestations of complex motor seizures

Speech disturbances are observed in the majority of temporal lobe complex motor seizures (Table 2). Types of language disturbances include vocalizations, abnormal speech, ictal speech, and postictal aphasia [47]. In a series of patients with TLE, vocalizations occurred in 49% and abnormal speech (e.g., speech arrest, dysphasia, dysarthria, and nonidentifiable speech) was observed in 51% of cases. *Ictal speech,* defined as clearly intelligible speech during the period of altered consciousness, was reported in 34% of patients and localized to the nondominant temporal lobe in 83% of cases. *Postictal aphasia* was observed in 12% of patients and virtually always localized to the dominant temporal lobe. Vocalizations, dysarthria, dysphasia, speech arrest, and nonidentifiable speech had no lateralizing value.

7. Lateralizing signs of secondarily generalized tonic–clonic seizures

Compared with primary generalized tonic–clonic seizures, most SGTCSs have considerable motor asynchrony and asymmetry. However,

two semiological features at the initial phase of generalization and one at its termination provide useful clues to seizure lateralization (Table 2). *Head version* is characterized by forced, prolonged head turning assuming an unnatural position with the chin elevated and head hyperextended [48]. The movement may be tonic or clonic and is associated with eye version to same side. Tonic contraction of the face ipsilateral to the head movement is often observed. The direction of head version is contralateral to the EZ in more than 90% of cases, especially when occurring in the 10 seconds preceding SGTCS. Ipsilateral head version at the end of a SGTCS occurs in as many as 15–20% of patients with focal seizures and is more common in FLE than TLE. In seizures of temporal lobe origin, initial ipsilateral (nonversive) head turning of brief duration often precedes contralateral version immediately prior to the SGTCS; this finding is highly accurate for seizure lateralization [49]. *Asymmetric tonic limb posturing (ATLP),* or the *figure-4 sign,* is tonic limb posturing in which the elbow contralateral to the epileptogenic hemisphere assumes an extended position and the opposite (ipsilateral to seizure onset) flexes over the chest during the tonic phase of a SGTCS. The posture resembles a figure-4 [50]. This sign provides correct lateralization in 90% of cases and is most common in seizures arising from the temporal lobe. ATLP can change sides during a seizure, so only its initial appearance should be considered in seizure lateralization. *Asymmetric ending of clonic jerks* refers to the asymmetric termination of clonic activity at the end of SGTCSs. This is proposed to be due to ongoing ictal activity in the hemisphere contralateral to seizure origin after the seizure terminates in the epileptogenic hemisphere. Asymmetric clonic activity at seizure termination usually involves the arm, although the face and leg may be affected. Persistent clonic activity provides lateralizing value, suggesting ipsilateral seizure onset [51].

8. Using symptomatology to optimize surgical outcome

Over several decades, observations made of patients treated with temporal lobe resection led to the delineation of other focal epilepsies more challenging to identify than MTLE. The causes of temporal lobe surgical failure include incomplete removal of the EZ, presence of a contralateral temporal focus, and presence of an extratemporal lesion. Regions producing electroclinical features mimicking TLE include the orbitofrontal, cingulate, insular, opercular, neocortical temporal, and TPO regions [52].

8.1. Neocortical temporal lobe epilepsy

Ictal symptomology can be used to help distinguish neocortical TLE (NTLE) from seizures arising from the mesial temporal structures. Although abdominal sensations, fear, and dreamy states are more commonly observed in MTLE, patients with NTLE more often report psychic, visual, auditory, and vertiginous auras [53]. Dystonic limb posturing and oral automatisms occurring within 20 seconds of seizure onset have been reported to be more common in MTLE (52% vs 26% and 69% vs 11%, respectively). However, the early appearance of facial grimacing/twitching and contralateral motor activity is suggestive of NTLE [53,54]. NTLE seizures are generally longer than those of mesial temporal origin, are associated with loss of contact earlier in the ictal sequence, and more often evolve into SGTCSs [55]. Because of their more diverse pathological substrates, neocortical temporal seizures tend to have more heterogeneous symptomatology than MTLE seizures. NTLE should be considered in patients with temporal EEG findings who lack typical semiological features of MTLE.

8.2. Parieto-occipital lobe epilepsy

The tendency for rapid propagation of parieto-occipital seizures to the frontal and temporal lobes produces potentially misleading electroclinical features [56,57]. The most reliable semiology of posterior cortex seizures includes contralateral somatosensory auras and simple (elementary) visual auras, highly suggestive of parietal and occipital seizure origin, respectively. Patients with parietal epileptogenic lesions also experience vertigo, disturbances of body image, and visual illusions or hallucinations, suggesting activation of visual association areas. Tonic posturing is commonly observed in parietal epilepsy when the EZ involves the superior parietal lobule, whereas the EZ includes the inferior parietal lobule in the majority of patients who demonstrate automatisms [57]. Other manifestations of occipital seizures include contralateral eye deviation, blinking, sensation of eye movements, nystagmus, and contralateral head version. Contralateral version and clonic activity, hypermotor or asymmetric tonic seizures, and oral or gestural automatisms are observed when posterior cortex seizures spread to the dorsolateral frontal, SSMA, and limbic structures, respectively [56].

8.3. Insular lobe epilepsy

As a result of rapid propagation through extensive connections between insular and adjacent regions, insular seizures produce a variety of visceral, motor, and somatosensory symptoms that mimic seizures arising from many other areas. Seizures arising from temporal structures virtually always invade the insula [58]. Pure insular epilepsy has a typical ictal sequence beginning with laryngeal discomfort with thoraco-abdominal constriction or dyspnea, followed by unpleasant paresthesias or warmth in the perioral region or involving large somatic areas, dysarthria or dysphonia, and ending with focal motor manifestations. Seizures restricted to the insular lobe do not result in impairment of consciousness. Unilateral sensory symptoms and focal motor activity are usually contralateral to the EZ. This behavioral sequence was not observed in mesial temporal or frontal seizures that spread to the insular lobe in which consciousness is altered early in the ictal sequence [59]. Insular seizures should be suspected in patients with electroclinical manifestations suggestive of MTLE when mesial temporal origin is otherwise deemed to be unlikely and in those with ictal symptoms suggesting spread to the opercular region (e.g., vomiting, hypersalivation, laryngeal constriction, lip/face paresthesias or tonic–clonic activity, dysarthria, and auditory hallucinations).

8.4. Temporal-plus epilepsies

Temporal-plus epilepsies include those in which the EZ involves the temporal lobe and its complex network (e.g., orbitofrontal region, insula, frontal/parietal operculum, and TPO junction). Although the presence of hippocampal sclerosis on MRI cannot be replied on to differentiate TLE from temporal-plus epilepsy, ictal symptomatology can provide useful information. Patients with pure TLE are more likely to have abdominal auras evolving into automatisms and postictal amnesia. In contrast, temporal-plus epilepsies are often associated with gustatory, auditory, or vertiginous auras, version, piloerection, ipsilateral tonic signs, and postictal dysphoria [60].

9. Conclusions

The objective of the presurgical evaluation for epilepsy surgical candidates is to delineate the EZ through a detailed assessment of the concordance of its components. As the EZ and the SZ are related, the location and extent of the EZ can be indirectly estimated through seizure symptomatology. Detailed history and video analysis provide important clues for seizure localization and lateralization. Many seizure signs and symptoms have highly significant localizing or lateralizing value. Some focal epilepsy subtypes, including neocortical temporal lobe epilepsy, insular lobe epilepsy, temporal-plus epilepsies, and parieto-occipital epilepsy, are characterized by electroclinical features mimicking TLE, potentially contributing to surgical failure. To optimize surgical outcome, careful interpretation of ictal symptomatology in conjunction with other components of the presurgical evaluation is required.

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