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Seizure Semiology: Its Value and Limitations in Localizing the Epileptogenic Zone

Krikor Tufenkjian, Hans O. Lüders

Epilepsy Center, Department of Neurology, University Hospitals Case Medical Center, Cleveland, OH, USA

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Correspondence
Krikor Tufenkjian, MD
Epilepsy Cener,
Department of Neurology,
University Hospitals of Cleveland,
11100 Euclid Ave. Lakeside 3200,
Cleveland, OH 44106, USA
Tel +1-216-844-3650
Fax +1-216-844-5066
E-mail Krikor.Tufenkjian@Lahey.org

Epilepsy surgery has become an important treatment option in patients with medically refractory epilepsy. The ability to precisely localize the epileptogenic zone is crucial for surgical success. The tools available for localization of the epileptogenic zone are limited. Seizure semiology is a simple and cost effective tool that allows localization of the symptomatogenic zone which either overlaps or is in close proximity of the epileptogenic zone. This becomes particularly important in cases of MRI negative focal epilepsy. The ability to video record seizures made it possible to discover new localizing signs and quantify the sensitivity and specificity of others. Ideally the signs used for localization should fulfill these criteria; 1) Easy to identify and have a high inter-rater reliability, 2) It has to be the first or one of the earlier components of the seizure in order to have localizing value. Later symptoms or signs are more likely to be due to ictal spread and may have only a lateralizing value. 3) The symptomatogenic zone corresponding to the recorded ictal symptom has to be clearly defined and well documented. Reproducibility of the initial ictal symptoms with cortical stimulation identifies the corresponding symptomatogenic zone. Unfortunately, however, not all ictal symptoms can be reproduced by focal cortical stimulation. Therefore, the problem the clinician faces is trying to deduce the epileptogenic zone from the seizure semiology. The semiological classification system is particularly useful in this regard. We present the known localizing and lateralizing signs based on this system. **J Clin Neurol 2012;8:243-250**

Key Words semiology, localization, epilepsy surgery.

Introduction

This review summarizes semiological findings that are valuable in localizing the epileptogenic zone. The information is based on literature review with results that are consistent with our experience. The discussion will focus on the signs that are more prominent and consistent. While there are other signs described anecdotally; however their infrequency makes them unreliable as an evaluation tool for epilepsy surgery.

Classification of Paroxysmal Events

Paroxysmal events can be classified into epileptic and non-epileptic events. Non-epileptic paroxysmal events are classified into organic (for example cardiogenic events such as syncope) and psychogenic events. For a diagnosis of non-epileptic psychogenic paroxysmal events the EEG should not show any epileptiform discharges during a typical paroxysmal event. Preserved awareness, eye flutter and the ability of the bystanders to modify or alleviate were found to be significant markers for these events.[1] The presence of a normal alpha activity while the patient is unresponsive and has amnesia for the event is essentially pathognomonic for psychogenic non-epileptic seizures.

Epileptic Paroxysmal Events

Auras

Somatosensory auras

These are abnormal somatosensations (usually "tingling" or "numbness") that are limited to a clearly defined somatosensory region of the body. The contralateral primary sensory cor-

tex is usually the symptomatogenic zone of somatosensory auras of unilateral, distal distribution. Bilateral and more widespread somatosensations can be produced from the supplementary sensorymotor area (SSMA) and also the second somatosensory area (S2) located in the superior bank of the Sylvian fissure and/or the posterior insula.[2] In addition, stimulation of S2 can also produce unpleasant sensations of heat or pain.[3] It is also important to remember that low intensity cortical stimulation of motor areas may elicit muscle contractions that are not sufficient to result in actual movements. The patient may interpret such a mild motor activity as a somatosensory aura. Poorly localized or lateralized "all body sensations" have no localizing or lateralizing value and should not be classified as somatosensory auras.

Visual auras

These are visual hallucinations that usually consist of flashing lights of different colors that may blink and move in the visual field. Frequently the patient reports the visual hallucinations in front of both eyes without a clear lateralization. However, occasionally the visual aura may be lateralized to one visual field (contralateral to the symptomatogenic zone) and may even be localized to the upper or lower visual field. Not infrequently the patient reports amaurosis either during or after the epileptic seizure.[4] In these cases the flashing lights may appear on top of the blind visual field. The symptomatogenic zone for the visual hallucinations is Broadmann's area 17 and 18. Complex visual hallucinations and visual illusions are more likely to involve the association cortex (parieto-temporal) or the adjacent lobes and frequently are part of psychic auras.

Auditory auras

These are simple auditory hallucinations, like hearing a "buzz" or a "noise". The symptomatogenic zone is Heschell's gyrus in the superior temporal gyrus. Usually the patient has difficulty lateralizing the sound. Besides, even when the sound is perceived as lateralized to one side it is not a reliable sign to lateralize the epilepsy.[5]

Olfactory auras

Most of the time these are hallucinations of unpleasant smells. They have no lateralizing value but are most frequently seen in patients with mesial temporal lobe epilepsy. Interestingly a relatively high percentage of these patients have neoplasms that involve the amygdala.[6]

Gustatory auras

These auras consist of unpleasant taste. Cortical stimulation studies have found the insula to be a symptomatogenic zone for this aura.[3] These auras have no lateralizing value.

Autonomic auras

These are subjective sensations suggesting possible autonomic alterations such as palpitations, sweating, "goose bumps", etc. The episodes should be diagnosed as "autonomic seizures" when there is objective documentation of autonomic alterations [for example when tachycardia is documented with an electrocardiogram (ECG) recording]. The symptomatogenic zone of most autonomic auras is most likely the insular cortex.

Abdominal auras

Abdominal auras are frequent and are usually secondary to temporal lobe epilepsies. However, occasionally abdominal auras may also be triggered by extratemporal epilepsies (mainly frontal lobe and insula). These auras are the result of either a sensation produced by increased peristalsis or 2) as a sensory phenomena resulting from direct activation of the sensory cortical areas of the abdominal viscera. Van Buren[8] assessed peristaltic motility with gastric balloons during spontaneous and induced abdominal auras. His studies showed no effect upon gastromotor function except of rare occasions of gastric inhibition.

The abdominal auras have been described by a plethora of symptoms such as nausea, tenseness, knot, external weight or squeezing, rolling, turning or whirling movement in the abdomen, tickling, tingling or electric shock sensation, pain, vibrating, fluttering or butterflies sensation, gas or pressure within the abdomen, an empty, hungry feeling, sensation of warmth, sensation of sudden descent in an elevator, burning or heartburn. The sensation begins usually in the epigastrium or stomach in the midline and can remain localized there but not infrequently rises to the chest, throat, head or even face.[7] Almost invariably when the aura rises to the neck or face the patient loses consciousness. Epigastric sensations closely resembling epigastric auras can be elicited by electrical stimulation of the insula. There are also reports of abdominal auras elicited by electrical stimulation of other structures such as the mesial temporal structures, the basal ganglia, the supplementary motor area, the pallidum and the centrum medianum of the thalamus.[8] However, in these studies the possibility that the stimulation produced after discharges spreading to other structures was not excluded.

Psychic auras

These are complex hallucinations and/or illusions that usually affect different senses. Psychic auras include phenomena such as autoscopy, fear, elation, déjà vu and jamais vu. Autoscopic phenomena have several variations. The more common ones are seeing a double of the whole or part of the body, feeling of presence without optical image, out of body experience with observing the self from an elevated position, as well as nega-

tive phenomena with failure to perceive one's own body. The temporal lobe is usually involved with these phenomena but they have no lateralizing value.[9] Some of these sensations can be elicited by electrical stimulation of the temporal lobe convexity or the junction of the posterior temporal lobe with the occipital or parietal lobe. Mesial temporal structures were concurrently involved in most cases.[10]

Autonomic seizures

Autonomic seizures are epileptic seizures in which the main symptomatology is an autonomic alteration that can be documented objectively (for example; an ictal tachycardia that is documented with an ECG recording and can be with or without an associated sensation of tachycardia). Pilomotor seizures are one type of autonomic seizures. These seizures have a somatotopic distribution and may spread in a "Jacksonian march" like pattern. Pilomotor seizures tend to be ipsilateral to the seizure onset zone, but the localization value is poor.[7]

There are rare autonomic signs that are not usually classified as autonomic seizures because they are not the dominant semiological feature of the seizure. These are: 1) Ictal vomiting and ictal retching that is observed mainly in temporal lobe seizures in adults. It has no definite lateralizing value but there is some evidence supporting the role of the insula as a visceral sensory cortex.[7] 2) Ictal spitting is a rare sign that also occurs in temporal lobe epilepsy but has no lateralizing value.[11] 3) Ictal hypersalivation is another uncommon sign found more frequently in mesial temporal lobe epilepsy particularly in the non-dominant hemisphere.[12]

Dialeptic seizures

Dialepsis is an alteration of consciousness consisting of unresponsiveness during the seizure and amnesia of the episode post-ictally. Dialeptic seizures are seizures in which the main symptomalogy is dialepsis. The epileptogenic zone of patients with dialeptic seizures tends to be at a distance from the primary or supplementary motor areas. On the other hand, in seizures originating at or near the primary and supplementary motor areas the predominant symptomatology tends to be motor phenomena (motor seizures) not infrequently with preserved consciousness.

The duration of the dialeptic seizures has a localizing value with seizures originating from the mesial temporal structures being of longer duration than the ones arising from the frontal lobe.[13]

Motor seizures

These can be divided into "simple" and "complex" motor seizures. The distinction between those two types of motor seizures does not pertain to the level of consciousness but to the complexity of the ictal movement itself. Simple motor seizures refer to unnatural but simple movements, usually involving only one articulation in one plane. These movements can be reproduced by electrical cortical stimulation of the motor areas. Complex motor seizures refer to movements that imitate natural movements. The latter are usually complex involving several articulations in different planes, and tend to be repetitive. These movements cannot be elicited by electrical cortical stimulation unless a seizure discharge is triggered.

Simple motor seizures

Myoclonic seizures

These are short muscle contractions lasting <200 ms and are most often seen in generalized epilepsies.[14]

Tonic seizures

These consist of sustained muscle contractions, usually lasting several seconds which lead to "posturing". Tonic seizures in patients with focal epilepsy preferentially affect proximal muscle groups, may be uni- or bilateral in distribution and tend to be asymmetric. Frequently in patients with focal epilepsy, consciousness is not clouded, at least at the onset of such unilateral or asymmetric seizures. Preservation of consciousness during bilateral motor activity can localize a seizure focus to the supplementary motor area.[15] Tonic seizures occur most commonly in frontal lobe epilepsy (62.2%) and very rarely in temporal lobe epilepsy (1.7%). In temporal lobe epilepsy only unilateral tonic seizures occurred, whereas 32% of the tonic seizures in frontal lobe epilepsies were bilateral.[16] If clearly unilateral, tonic seizures have a high lateralizing significance, pointing to a contralateral seizure onset.

Tonic seizures originating from the SSMA appear to be bilateral from the onset but careful analysis has revealed it may begin in one part of the body and move rapidly to the other limbs.[17] Less often posturing may be unilateral or restricted to a single limb but proximal limb and axial muscle involvement is always prominent. Most often all 4 limbs are involved with abduction of the upper limbs and asymmetric flexion of the elbows. The lower limbs are abducted at the hips with the knees extended or semi-flexed.

Tonic seizures are also a part of generalized seizures. The position assumed during tonic seizures and its sequence differs between primary and secondarily generalized seizures. The typical position seen in primary generalized tonic-clonic seizures starts with tonic flexion of the body with shoulder elevation, arm elevation and the elbow semiflexion. This is followed by a tonic extension of the entire body into an opisthotonic posture. The elbows become semiflexed in front of the chest but also at times become extended, with forearm pro-

nation and either wrist flexion and finger extension or wrist extension and fist clenching. The tonic contraction of the chest and abdominal muscles produce the "Tonic epileptic cry".

Secondarily generalized seizures on the other hand tend to have a typical "motor sequence". This starts with version and pulling of the face (tonic) to the contralateral side then progresses to the M2e position and subsequently to the asymmetric tonic limb posturing called "sign of four". The motor sequence includes 4 signs that in isolation are relatively good lateralizing signs, namely the versive seizure, the tonic or clonic seizure of the face, the M2e sign and the "sign of four". However, the epileptogenic zone can be lateralized with confidence when 2 or more of these lateralizing signs are observed. The tonic face seizure and the versive seizure lateralize the seizure to the contralateral side. The fencing position (M2e) lateralizes to the hemisphere contralateral to the raised arm[18] and the asymmetric tonic limb posturing "sign of four" lateralizes to the hemisphere contralateral to the extended arm.[19] All these 4 signs have lateralizing value but do not localize the seizure origin.[20] Tonic seizures are also the prominent feature of epileptic spasms which are discussed next.

Epileptic spasms

This term is used to identify muscle contractions of relatively symmetric, either tonic or myoclonic features which affect predominantly proximal axial muscles. The predominant contraction is usually a flexion of the trunk and an extension and abduction of the arms in a "salaam position". Infrequently opistothonic posturing is seen. These seizures usually appear in clusters as the patient goes to sleep. They have variable duration and may vary in the same patient from one seizure to another. The EEG pattern with these seizures is predominantly generalized.[14] Usually epileptic spasms are seen in patients with generalized epilepsies. However, there are well documented cases of epileptic spasms occurring in patients with focal epilepsies, particularly parieto-occipital epilepsies. Some of these patients had successful resective surgeries.[21] The mechanism of these seizures has not been clearly determined but involvement of the brainstem raphe nuclei and spinal pathways has been suggested.[22]

Clonic seizures

These are myoclonic contractions that recur regularly at a rate of 0.2-5 per second. The symptomatogenic zone is usually the primary motor strip. In temporal lobe epilepsies the face, the frontal eye field and hand areas tend to be affected earlier than legs. The epileptogenic zone is usually at or in close proximity of the primary motor strip when clonus without alteration of consciousness is the first sign of the seizure. Unilateral clonic seizures have a highly lateralizing value to the contralateral hemisphere.[23] In secondarily generalized tonic-clonic seizures the clonic activity may persist longer on the side ipsilateral to the epileptogenic focus ("end of seizure paradoxical clonus"). This is probably related to the fact that the hemisphere of seizure origin tends to get "exhausted" earlier than the contralateral hemisphere. "End of seizure paradoxical clonus" is a highly reliable lateralizing sign.[24] An asymmetric seizure termination is relatively unusual in generalized epilepsy.[25]

Tonic-clonic seizures

This expression is used exclusively for generalized tonic-clonic seizures. These seizures start with tonic posturing of all the limbs followed by a "jittery" phase that progressively slows down and eventually transforms in a clonic activity of all four extremities. Occurrence of these types of seizures at the onset and relatively symmetrical involvement of all limbs is highly suggestive of generalized epilepsy. Generalized tonic-clonic seizures also occur in focal epilepsies but almost always are preceded by other seizure types and the tonic phase is usually asymmetric.

Versive seizures

Versive seizures are defined as a forced and involuntary turning of the head and eyes in one direction with an associated neck extension resulting in a sustained unnatural position of both. The frontal eye fields are the symptomatogenic zone. Versive seizures appear earlier in seizures of frontal lobe origin as opposed to temporal lobe origin and can be the first sign of frontal lobe seizures.[13] Versive seizures have a highly lateralizing value to the contralateral hemisphere, especially when they occur within 10 seconds before secondary generalization.[26,27] However, the reliability of the versive seizures requires clear differentiation between non-versive head turnings (which resemble natural movements) and epileptic versive seizures.

Complex motor seizures

Hypermotor seizures

The main manifestations in these seizures consist of complex movements (movements that involve more than one articulation in different planes and resemble normal movements) involving the trunk and proximal segments of the limbs. The proximal predominance of these movements results in large movements justifying the denomination of "hypermotor". Examples are pedaling movements, running, etc. Consciousness may be preserved during these seizures. They occur mostly during sleep. Most originate from the orbital or mesial frontal regions. However, hypermotor seizures may also occur in temporal lobe and insular epilepsies.[28,29] Occasionally

hypermotor seizures consist of automatisms that resemble sexual activity, like violent writhing, thrusting and rhythmic movements of the pelvis, arms and legs. Sometimes this is associated with picking and rhythmic manipulation of the groin or genitalia and exhibitionism.[7]

Automotor seizures

The main manifestation of automotor seizures are automatisms involving the distal segments of the hands, feet, mouth and tongue. Automotor seizures are typical of temporal lobe epilepsy but occasionally can also be seen with frontal lobe seizures. Frontal lobe automotor seizures tend to be of shorter duration than temporal lobe automotor seizures. Automotor seizures may consist of unilateral or bilateral automatisms and unilateral automatisms are more frequently an expression of an ipsilateral epileptogenic zone. The unilateral presentation of automotor seizures is likely a manifestation of limb dystonia in the contralateral limb which interrupts distal automatisms in the limb involved in the dystonia. 95% of the automotor seizures are associated with altered consciousness. However, preservation of consciousness during automotor seizures has been observed almost exclusively in patients with non-dominant mesial temporal epilepsy.

Gelastic seizures

Seizures in which the main motor manifestation is "laughing" are termed gelastic seizures. In approximately 50% of the cases with gelastic seizures hypothalamic hamartomas can be detected by MRI. However, patients with epilepsies in extra-hypothalamic localizations have also been described. For example, there are case reports of gelastic seizures observed in patients with epilepsies in the anterior cingulate region, as well as the frontal, parietal and temporal lobes.[30]

Special seizures

Seizures that cannot be classified in one of the four above mentioned types are classified as special seizures.

Atonic seizures

These seizures result in loss of postural tone with ensuing falls or head drop. Atonic seizures are seen most frequently in patients with symptomatic generalized epilepsies (Lennox-Gastaut syndrome) and are usually preceded by a generalized, proximal myoclonic seizure resulting in an abrupt fall compounding the propensity to injury. Atonic seizures can also be seen in patients with frontal and temporal lobe focal epilepsies.[31] These seizures, however, are usually not associated with myoclonic seizures. Therefore, they tend to be associated only with slower falls and only infrequently result in significant injuries.[32]

Astatic seizures

These are seizures that consist of epileptic falls. They can be the result of atonic seizures but most commonly are due to a myoclonic seizure followed by an atonic seizure. The expression astatic seizure can be used when there is evidence of a fall but the exact mechanism is unknown (i.e., atonic seizure vs. myoclonic seizure followed by an atonic seizure vs. bilateral asymmetric tonic seizure, etc).

Hypomotor seizures

The main manifestation of these seizures is a decrease or total absence of motor activity. This expression is only used in patients in whom consciousness cannot be tested during or after the seizure (newborns, infants and children under 3 years; mentally retarded patients). In patients with focal epilepsy, hypomotor seizures are seen most frequently in temporal and parietal lobe epilepsy.[33]

Akinetic seizures

These seizures are characterized by inability to perform voluntary movements. The diagnosis of akinetic seizures can only be made in patients who are conscious and cooperative, i.e., they try to perform a movement but are unable to do so (apraxia). They are thought to arise from activation of the negative motor areas in the mesial frontal and inferior frontal gyri. Results of cortical stimulation support this localization.[34]

Negative myoclonic seizures

These are seizures in which a brief movement is produced by a loss of muscle tone of less than 400 msec duration. The postcentral cerebral cortex was indicated in a case report of unilateral epileptic negative myoclonus.[35] The movement observed in these seizures is similar to the one observed in asterixis which is an example of non-epileptic negative myoclonus.

Aphasic seizures

In these seizures the patient is aphasic despite preserved awareness and memory. These are often mixed aphasias and lateralize the epilepsy to the dominant hemisphere. They can also present as status epilepticus.[36]

Additional lateralizing signs

These are semiological signs that do not represent a dominant part of a seizure or they present in the post-ictal period only. They however are valuable in lateralizing the seizure focus.

Dystonic posturing

This is a sustained (>10 sec), forced, unnatural positioning of an upper extremity on one side of the body with a clear rotational component. In patients with temporal lobe epilepsy this

is a reliable lateralizing sign to the contralateral hemisphere.[37] Although more common in temporal lobe epilepsy this sign can occur in extra temporal lobe epilepsy as well. It is thought to be related to activation of the basal ganglia through spread of the epileptifom discharges. Basal ganglia were shown to be involved during dystonic posturing on ictal SPECT.[38]

Ictal speech

Ictal verbalization is defined as the presence of clearly intelligible speech when the patient already shows unresponsiveness and/or has clear distal automatisms. It tends to lateralize the epilepsy to the non-dominant hemisphere in patients with temporal lobe epilepsy. However, exceptions to this rule are not so infrequent making this a poorly reliable lateralizing sign. Besides, pure vocalization has no lateralizing significance.[39,40]

Post ictal aphasia

Postictal aphasia lateralizes the epilepsy to the language dominant hemisphere in patients with temporal lobe epilepsy.[39] Recovery of language function after the ictal EEG pattern has stopped was found to be significantly more delayed in patients with left temporal lobe epilepsy.[41] It has been reported that post-ictal language delay is less significantly affected in frontal lobe epilepsy unless there is extension to the ipsilateral temporal lobe.[42] To diagnose post-ictal aphasia it is essential to have a patient who is cooperative postictally (i.e., clearly tries to understand language and tries to talk) who, however, is aphasic. Besides, the patient should be tested continuously and language should recover slowly (10-20 minutes) but progressively.

Todd's paralysis

This is a non-localizing but highly lateralizing sign. It is always preceded by prominent ipsilateral motor activity of the affected limb.

Post-ictal nose wipe

This lateralizes the epilepsy to the ipsilateral hemisphere in patients with temporal lobe epilepsy.[43] It is assumed that the hemisphere contralateral to the nose-wipe (which is the hemisphere of seizure onset) suffers from post-ictal neglect.

Peri-ictal water drinking

This has been reported as a lateralizing sign to the non-dominant temporal lobe epilepsy.[44] The validity of this has been contested.[45]

Unilateral eye blinking

This is infrequent but has been reported as a good lateralizing sign to the ipsilateral hemisphere. It has no localizing value.[46] The mechanism of ipsilateral eye blinking has not been elucidated.

Ictal nystagmus

Most cases described a predominantly horizontal binocular nystagmus. In these cases the fast phase of the nystagmus was opposite the seizure focus. The seizures originate from either the occipital or the temporo-occipital junction and are often associated with ictal vertigo.[47]

Limitations of using semiology as a localizing tool

Seizure semiology is subjective

There is significant inter-rater variability particularly when observers come from different epilepsy centers which may define symptoms and signs differently. This occurs despite using systematic approach to analyze the seizure semiology with video recording and highly trained individuals. An example would be misinterpretation between versive seizures and non-versive head rotations among different observers.

Seizure semiology is the manifestation of the activation of the symptomatogenic zone

That implies that it can be the result of ictal spread from a more distant epileptogenic zone. In other words seizures arising from different epileptogenic zones could activate the same symptomatogenic zone or, vice-versa, seizure originating from the same epileptogenic zone could activate different symptomatogenic zones producing different seizure semiologies.

Seizure semiology does not always permit differentiation of focal or generalized epilepsies

Focal epilepsy may trigger seizures of "generalized" semiology, and vice versa generalized epilepsies may have seizures of focal symptomatology. So for example, focal signs were found in 46% of patients in a group with juvenile myoclonic epilepsy. Focal semiologic findings in this group included unilateral clonic, unilateral myoclonic, version and asymmetric tonic seizures.[48]

Conclusions

Seizure semiology is a very useful tool; however, it requires standardization among evaluators. It is necessary to include only signs that are unambiguous and achieve a consensus of several qualified observers. It is not infrequent to see conflicting lateralizing or localizing signs occurring in a single seizure. In this situation the importance of each sign is determined based on its sequence during the seizure as well as the specificity of the sign.

Conflicts of Interest

The authors have no financial conflicts of interest.

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