Title: Structured testing during seizures: A practical guide for assessing and interpreting ictal and postictal signs during video EEG long term monitoring

Michael Owen Kinney (1) , Stjepana Kovac (2) , Beate Diehl (1, 3)

1. Department of Clinical Neurophysiology, National Hospital for Neurology and Neurosurgery, Queen Square, London, UK
2. Department of Neurology, University of Münster, Münster, Germany
3. Department of Clinical and Experimental Epilepsy, National Hospital for Neurology and Neurosurgery, Queen Square, London, UK

Keywords: Seizure, Video EEG, Ictal testing, Postictal, Semiology

Highlights

- There is currently sub-optimal ictal-postictal testing in inpatient settings.
- The European consensus protocol offers a standardised assessment of semiology.
- Ictal-postictal semiology helps determine lateralisation and localisation.
- Possible psychogenic nonepileptic attack disorder may require tailored testing.

Abstract

Background: Ictal and postictal testing carried out in long-term epilepsy monitoring units is often sub-optimal. Recently, a European consensus protocol for testing patients during and after seizures was developed by a joint taskforce of the International League Against Epilepsy – Commission on European Affairs and the European Epilepsy Monitoring Unit Association. Aim: Using this recently developed standardised assessment battery as a framework, the goal of this narrative review is to outline the proposed testing procedure in detail and explain the rationale for each individual component, focusing on the underlying neurobiology. This is intended to serve as an educational resource for staff working in epilepsy monitoring units. Methods: A literature review of PubMed was performed; using the search terms “seizure”, “ictal”, “postictal”, “testing”, “examination”, and “interview”. Relevant literature was reviewed and relevant references were chosen. The work is presented as a narrative review. Results: The proposed standardised assessment battery provides a comprehensive and user-friendly format for ictal-postictal testing, and examines consciousness, language, motor, sensory, and visual function. Conclusion: The standardised approach proposed has the potential to make full use of data recorded during video EEG increasing the diagnostic yield with regards to lateralisation and localisation, aiding both presurgical and diagnostic studies.

Introduction

Seizures have many different manifestations, with diverse clinical signs and symptoms, together referred to as ictal semiology [1–4]. Semiology can help determine the hemisphere and lobe of seizure onset. To characterise a patient's habitual seizures, admission is often required to a long-term electroencephalography (EEG) monitoring unit (LTM). A video recording of all captured seizures is correlated with the simultaneously recorded EEG to either confirm or refute the epileptic nature of the event [5], and in presurgical cases potentially identify the hemisphere (lateralisation) and cerebral lobe (localisation) of seizure onset [6]. The postictal period from seizure termination until the return of the patient's baseline, also contains useful clues to guide localisation and lateralisation [7]. LTM is an expensive resource and carries a small risk of complications [8], and so requires maximal efforts to obtain an informative study. To achieve this ictal and postictal testing should be completed, to assess consciousness, behaviour, language, sensory and motor function. The main problems with ictal-postictal assessment include the lack of well trained and experienced staff [9], poor standardisation of testing across centres, and delayed initiation of testing during seizures [10]. In a UK study, only 27% of seizures were assessed within 30 s and 50% remained unassessed [11]. Recognising this need to standardise assessments during LTM, the International League Against Epilepsy Commission on European Affairs (ILAE-CEA) and the European Epilepsy Monitoring Unit Association (EEMA) Taskforce developed and validated a standardised structured ictal-postictal testing battery [10] (ITB) (Fig. 1). The ITB is brief and can be adapted depending on the dynamic evolution of individual seizures [9]. This review outlines the main components of the proposed ITB. The focus is on the procedure of completing the assessment and the rationale behind each aspect of the ITB. The aim is to provide a helpful guide to the semiology encountered during seizures, as well as indicate how best to test the patient during and after a seizure. It is suitable for newly appointed staff working in LTM units to help empower them in carrying out a basic bedside ictal-postictal examination and understand how to adapt testing in certain situations

Initial considerations when attending seizures: patient safety

The first concern is patient safety [12] and a rapid evaluation is required to determine if cardio-respiratory resuscitation is needed. Ensuring the patient is in a safe position is vital. Once safety has been established testing can commence. Seizures typically are brief, lasting 1–2 minutes [13], so the ITB should be administered rapidly (See Table 1: Initial approach at the patient's bedside [10,12,14]). The initial section of the ITB focuses on evaluating consciousness and language. Once these have been established, more detailed motor, sensory, and visual testing can be undertaken. If the patient does not comply with an individual step in the ITB but appears to have awareness, one can proceed to the next step, as the ITB is adaptive to the seizure and former steps can be revisited. It is vital to examine the patient during the interictal state, to ensure that presumed ictal postictal deficits are indeed only transient and not present in the interictal state. This includes an assessment to ensure hearing is satisfactory, and that the patient understands the language in which testing is being completed in.

Consciousness

Consciousness is challenging to define [15], but for everyday clinical purposes is considered the inability to appropriately engage with external stimuli due to an altered state of awareness or responsiveness [16]. Consciousness can also be defined as an impairment of self-referential internal thought processes [17].

Neurobiological basis of testing

Loss of awareness or consciousness is responsible for significant morbidity, mortality, and stigma in epilepsy [18,19]. Approximately 50% of seizures with loss of awareness are not recognised by patients [20]. The localisation and lateralisation of seizure onset, prior state (sleep or wake), cognitive baseline, and type of seizure can all influence seizure recall [21], with the extent of EEG involvement predicting aura recall [22,23]. The "consciousness system"

is the network of critical cortical (medial and lateral fronto-parietal association cortices) and subcortical structures (reticular activating system and thalamo-cortical relays) which maintain consciousness [24]. Consciousness has two key components: firstly, the ability to maintain vigilance towards the environment and secondly, the ability to obtain specific cognitive information through the senses [25,26]. Therefore clinical testing of consciousness assesses responsiveness to external stimuli, appropriate orientation to environment and memory. The mechanism by which seizures impair consciousness is not fully understood, and this is an area of active research [24]. Similarities exist between the minimally consciousness state and seizures with impaired awareness with both exhibiting reduced neocortical activity [24,27]. Focal seizures could also disrupt cortical-subcortical dynamics, altering consciousness (inhibitory hypothesis) [28]. To provide objective measures of the impairment of the level and content of consciousness, various classifications have been proposed [29]. Further to this, specific psychometric scoring tools to objectively assess consciousness during seizures have been published including the ictal consciousness inventory (ICI) [30], the consciousness seizure scale (CSS) [31], and the responsiveness in epilepsy scale—versions I and II (RES-I and RES-II [32,33]) [for review see [34]]. Studies assessing their utility identified either global impairment or spared consciousness, suggesting critical “consciousness system” structures involvement or non-involvement, respectively [34]. One study of 338 seizure videos in 100 patients found that seizures with a predominant semiological manifestation of loss of consciousness could originate in any lobe of the brain [35]. A further study [36] found consciousness was most commonly impaired in bitemporal or left temporal seizures, but was more frequently spared in non-dominant temporal lobe seizures. Dominant temporal lobe seizures also result in memory and language deficits, with frontal seizures resulting in loss of orientation and expressive speech function. Dominant hemisphere focal onset seizures with automatisms were associated with loss of awareness and non-dominant seizures were associated with retained awareness. So-called “rudimentary automatisms” such as grasping, visual tracking and blinking to threat, were seen in half of focal impaired awareness seizures. These findings highlight the importance of obtaining consistent responses when determining

awareness, and not mistaking automatic behaviours as conscious voluntary actions. Forced thinking, aggressiveness, ictal aphasia, amnesia, impaired comprehension, and apraxia can all make the ictal determination of degree of impairment of consciousness challenging [37]. Despite the above findings, in seizures associated with impaired awareness, the finding of altered awareness is not reliably lateralising or localising [1,35]. The particular aura and ictal sequence can potentially offer additional localisation information. When awareness is established, more reliable localising and lateralising information can be gained. Orientation to person, place, and time are fundamental aspects of the mental status examination. Disorientation in the different domains may have a common causative mechanism, but as yet has not been clearly determined [38].

Ictal and postictal testing of consciousness

In order to establish responsiveness, the patient can be addressed by their name to test their reaction. If they respond, it is important to establish if they are experiencing any aura symptoms (i.e. disturbed content of consciousness) (Table 2: Summary of seizure semiology: lateralizing signs during ictal and postictal testing [1,4]). By engaging in this way it indicates language and attention are intact. Testing orientation (name, place, and today's date) and asking them to follow a simple command (such as "lift your arms") further tests awareness. If the patient does not follow a verbal instruction then a simple motor task can be demonstrated for the patient to mimic. This task requires attention and motor function, but is independent of language function. If the patient does complete the mimicry task, then it is important to repeat the language assessment to determine if comprehension has recovered as this may have been impaired. Failure to follow commands could be due to hearing difficulties and should always be considered and correlated with the interictal testing. Where awareness is intact, a recall task is also useful to assess working memory. This can be completed by giving two common words for the patient to recall later in the assessment. Awareness is required to complete the rest of the ITB, despite testing other specific cognitive functions. If awareness subsequently appears diminished, return to the beginning to reassess [10].

Motor assessment

Completing a motor task requires a complex harmonious interaction of a range of anatomical sites such as bones, muscles, joints, with the main cerebral areas including the primary motor cortex (M1), the supplementary motor area (SMA) and pre-supplementary motor areas which are involved in linking cognition to action and thus voluntary motor response.

Neurobiological basis of testing

Observation of any motor phenomenon is highly informative from a localising and lateralising perspective. For example, clonic jerking of the face represents activation of the contralateral face motor area in the primary motor cortex (M1). Electrical stimulation of both M1 and the SMA produces simple motor responses, i.e. myoclonic, tonic, clonic and tonic-clonic or version. Versive seizures indicate involvement of the contralateral frontal eye field and can indicate involvement of the SMA when asymmetric tonic posturing is seen. Motor function of different anatomical sites (e.g. limbs, face, or trunk) are represented on the motor cortex, in proportion to their functional importance rather than anatomical size. The face and hand for example occupy a relatively large proportion of the motor cortex and require low stimulation thresholds to stimulate function [39,40]. The primary motor cortex is required to perform voluntary action. The SMA and pre-SMA are both located in the mesial superior frontal gyrus anterior to the primary motor area and are crucial for the initiation of movement [41]. SMA electrical stimulation produces simultaneous activation of axial, proximal and distal muscles, and potentially atonic responses [42,43]. Simultaneous activation of more than one limb is often seen, as the somatotopic representation of the limbs is contained within such a small area of cortex. Cortical stimulation studies in the human SMA have identified a somatotopic organization with eye movement responses lying anterior to upper and lower limb responses [43]. Ictal and postictal paresis are hard to distinguish, but provide the same lateralisation information [44,45]. Automatisms can be ictal or postictal, and when associated with dystonia provide strong lateralisation clues [4]. Manipulative automatisms may be more common

postictally, and EEG can help to distinguish between ictal and postictal automatisms [46,47]. In the post-ictal period motor weakness, termed Todd's paresis can occur for variable periods of time, and may be related to ongoing inhibition [48,49]. It is reported in < 1% of LTM series, yet is a strong lateralising sign, indicating onset in the contralateral hemisphere in 93% of patients who experience it [50]. A rare variant is bilateral weakness from SMA seizures [51]. Case reports of prolonged postictal hypoperfusion [52] and animal model [53] evidence of cyclooxygenase pathway involvement are putative explanations.

Ictal and postictal motor testing

Seizures can result in involuntary movements, abnormal postures due to abnormal tone, as well as muscle weakness (Table 3: Motor semiology: lateralisation and localisation [1,4]). One vital concern is that the camera should be positioned with a clear view of the moving limb or body part. It is often helpful if a commentary is provided by staff present, in case the camera resolution is sub-optimal. This is particularly important for subtle jerks of the hand, face or nystagmoid jerks of the eyes. Other potential movements that might be seen include dystonic posturing, myoclonus, and automatisms (manual or oral). In order to detect subtle weakness a useful instruction to give the patient is to "lift your arms", encourage them to maintain the posture, with palms facing upwards, and observe for pronator drift, Muscle tone is the resistance muscles provide when passively moved through a range of motion around a joint. During a seizure it is usually evident that tone is increased (tonic/dystonic), with the limb being difficult to passively move. Reduced tone (hypotonia) is rare as a seizure manifestation (inhibitory seizure) and is commonly due to postictal processes.

Language

Language is the means by which humans communicate through symbols, sounds, or gestures. The language network links incoming auditory or visual stimuli, generates semantic meaning, facilitates understanding and generates a meaningful output to the interlocuter. If

the patient remains conscious during the seizure and a language deficit can be elicited, rapid involvement of language cortex by the seizure should be suspected. It is then important to attempt to assess the different components of language early in the seizure before propagation occurs and results in widespread disruption of language function. Due to time constraints, it is rarely possible to fully elucidate all aspects of language. Postictal language deficits have great lateralising value, as detailed below.

Neurobiological basis of testing

Language function is lateralised to the left hemisphere in > 90% of right handed individuals and 70% of left handed individuals. The remaining 30% of left handed individuals are either right dominant or have bilateral language representation [54]. Atypical language localisation [55] and lateralisation can be seen as a result of a shift of language function due to neuronal dysfunction in the original language dominant hemisphere [56–60]. Calculation and praxis abilities are more strongly represented in the dominant hemisphere. The prevalent model for understanding language function is that the auditory signal is initially processed via Heschel's gyrus (in sign language via primary and secondary visual cortices), comprehended via Wernicke's area, and relayed to Broca's area via the arcuate fasciculus. Broca's area allows planning of expressive language [61]. Recent functional imaging studies have identified a more widespread language network than was traditionally understood [62], including other areas such as the basal temporal lobe [62,63]. The right hemisphere has a role in prosody (providing the lyrical quality including pitch, tempo, cadence and melody), with non-dominant ictal speech on rare occasion manifesting in a foreign accent [64]. In video-EEG cohorts 10% of unselected seizures and 50% of temporal lobe seizures exhibit language abnormalities [65], including ictal speech, aphasia (ictal or postictal), paraphasias, and anomia (inability to name objects). Ictal or postictal dys/aphasia is a valuable finding with an approximately 90% lateralisation value [4,66]. The localisation value of language however is poor [65], almost certainly because language testing is rarely completed quickly enough to be of high localising value, and the seizure has spread. This was highlighted in one series [65] where ictal aphasia

was associated with seizure onsets in the parieto-occipital region, with no lobar preference for postictal dysphasia. Ictal aphasia only occurs with dominant hemisphere involvement, even when onset is in the non-dominant hemisphere. Postictal aphasia almost always indicates dominant hemisphere involvement, seen in 12% of cases of temporal lobe epilepsy [66]. The maximal duration of postictal dysphasia is not clear from published cases [49], but language recovery is related to presence of a structural etiology and hemisphere of onset [67,68]. One LTM study with 212 seizures in 60 patients identified that interictal and postictal language testing are equally accurate in lateralising language dominant temporal lobe seizures, after applying the Boston Naming Test as a confrontational naming assessment [69]. Phonemic paraphasic errors in comparison to semantic paraphasic errors are more clinically useful in providing lateralising information in temporal lobe epilepsy [70]. When robust and rapid testing is carried out, the information can help increase our ability to localise the seizure onset. A small series of patients with temporal lobe epilepsy undergoing stereo-EEG delineated three sub-lobar localisations associated with distinct patterns of language involvement [71] (See Fig. 2 [71]). Recent case studies [72] have also highlighted the role of detailed ictal testing demonstrating ictal conduction aphasia, which is a form of disconnect between the representation of words and motor function. It is characterised by the inability to repeat, with phonemic paraphasias (fluency), with intact receptive (auditory comprehension) language function but without deficits in naming, auditory or written language. This was considered to be related to a lesion of the arcuate fasciculus. However, conduction aphasia has also been observed during electrical stimulation of the posterior superior temporal gyrus. Although cortical stimulation studies [73] have certain inherent limitations, they have supported the different classical patterns of language impairment, delineated by earlier lesional studies [73]. Larger stimulation intensities engage more language network, resulting in global aphasia, and this is typically what occurs in seizures undergoing propagation. Ictal speech refers to intelligible spontaneous speech associated with altered awareness [74]. Use of words, such as “em”, “eh”, “yeah”, “yes”, “no” or isolated swear words, are not considered robust examples of ictal speech. One-third of temporal lobe epilepsy cases demonstrate ictal speech, with >

80% being of non-dominant hemisphere lateralisation [66]. Ictal “verbal help seeking” speech has been reported [75]. Anarthria is the inability to articulate words and can occur as a result of oro-facial weakness or a negative motor phenomenon. It is not a language deficit per se and is non lateralising.

Ictal and postictal testing of language

The six language focused tasks are: auditory comprehension, spontaneous speech, repetition, naming, writing and reading. Auditory comprehension is assessed by observing the response after an attempt to engage the patient by stating their name, asking them what they feel, as well as by their ability to follow a verbal command. Any subsequent spontaneous speech can be evaluated for articulation, phonation, rate, and prosody (i.e. rhythm). Repetition is assessed by giving two common words (e.g. “horse” and “table”) to repeat and recall later. Visual naming of objects (to detect anomia), naming function of objects, writing a sentence (assesses for agraphia), and reading a short paragraph (assess for alexia) are all important language skills. If the person appears to have speech arrest, it is informative to test the motor function of the tongue. The language assessment should continue until language has normalised. If concern arises that loss of awareness has occurred and it is difficult to distinguish from lack of language comprehension, attempt a non-language based task. The naming task should be adapted to patient age and intellectual development (see supporting materials in Task Force report [10]).

Vision

The processing of visual stimuli is dependent on normal eye function and the integrity of the primary visual cortex, visual association cortices and the white matter tracts connecting them. The calcarine cortex (BA 17) in the medial occipital lobe, represents the primary visual cortex. Visual association areas (BA 18, 19) in the occipital, parietal, and temporal lobe are responsible for motion perception, recognition, orientation, shape/size detection, and colour discrimination. The central visual field receives the greatest cortical representation (almost

40% of the mesial occipital lobe represents the central 15 degrees of vision [76]) due to its functional importance. There is also a retinotopic organisation of the visual cortex, with posterior regions representing the central vision and anterior regions representing peripheral vision.

Neurobiological basis of testing

Visual auras are very common in series reporting occipital lobe seizures (approximately > 70%) [77]. Visual phenomena are also seen with temporal and parietal involvement. Visual auras can either be elementary or complex visual hallucinations [78]. The former typically are seen with seizures originating in the primary visual cortex (V1). They are simple geometrical shapes, lines or blobs, or flashes of light, which can be black, white or (multi)coloured, stationary or moving (positive visual symptoms). Negative symptoms, consist of ictal visual field deficits, blurring of vision, and cortical blindness [79,80]. Visual field loss can co-exist with positive hallucinations seen within the field defect [81]. Visual field loss or bilateral blindness (amaurosis) can also be seen as a postictal feature, lasting minutes to days [49]. Complex visual hallucinations are seen with seizures involving the visual association cortex [82], including medial (limbic), lateral temporal, posterior parietal cortex, and the temporo-parietal-occipital junction [83]. In addition, direct electrical stimulation studies elicited complex hallucinations from basal temporo-occipital gyrus, temporo-occipital [84] and prefrontal cortex (the latter has mnestic and visual processing associations) [85]. In these seizures well-formed hallucinations of scenery (potentially historical memories [86]), images of people, animals, or faces are seen. Distortions/Illusions of object size, shape, colour, and motion (kinetopsia) are reported. The latter has been mapped to the superior parietal lobule and intraparietal sulcus [87], and in seizures from the temporo-parietal occipital junction [88]. Palinopsia, visual perseveration, allesthesia [89], cinematographic hallucinations [90] and tunnel vision [82] are rarely seen. The clear identification of a visual field defect establishes involvement of symptomatogenic cortex. The visual pathways are depicted in Supplementary Fig. 1. Note that the left visual hemifield is represented in the right occipital cortex, and thereby a right

occipital seizure could result in a left homonymous hemianopia. Such a deficit has a reported 100% predictive value for a contralateral onset in reported series [77] and is a very useful sign to detect. One entity to distinguish from a visual field defect is that of neglect. Visual neglect can be an ictal-postictal process, which is characterized by the inability of a person to process and perceive stimuli on one side of the body or environment rather than having a true isolated visual cortical abnormality. Hemineglect is a result of dysfunction of multiple association cortices and thus multiple sensory functions such as auditory, visual and somatosensory can be involved. The patient may not recognise the defect. Visual neglect is detected clinically as the inability to recognize a visual stimulus once it is presented simultaneously in one hemifield with another stimulus in the contralateral hemifield (For example, moving a visually presented index finger in both the right and left hemi-field, and asking the patient which finger moved). One study evaluated 33 patients with the line-bisection task; spatial neglect was found on post-ictal exam but not interictal exam for patients with right parietal foci, and was maximal for the left-positioned lines. This occurred despite there being no other signs of neglect [91]. Neglect is usually non-dominant, but is seen with dominant parietal lobe involvement. The dominant parietal cortex serves the contralateral hemisphere, and the nondominant parietal has bilateral representation [92].

Ictal and postictal evaluation of visual function

The presence of positive or negative visual symptoms should be checked, with a follow up question to clarify the location of where the symptoms are perceived in the visual field. Each temporal visual hemifield is assessed by the confrontation method (for details see supporting materials of Task Force report [10]). A visual field deficit will result in an examiner's moving finger, not being seen. This methodology presumes a central deficit, and more detailed testing may be required in visual phenomena associated with possible psychogenic non-epileptic seizures.

Sensation

Sensory function is the ability to perceive touch, pressure, pain, temperature, and is mediated centrally in the primary somatosensory cortex in the post-central gyrus.

Neurobiological basis of testing

Just as with the motor cortex, the sensory cortex (See Supplementary Fig. 2) has a somatotopic representation (sensory homunculus), demonstrated through electrical stimulation studies [93]. Unilateral somatosensory auras were present in < 10% of LTM series, and 89% of seizures were of contralateral onset [4]. In a minority of patients somatosensory aura can be of ipsilateral onset. A “sensory march” in a well localised area will increase the reliability of the sign. Cortical stimulation studies also elicited somatosensory phenomena from stimulation of mesial temporal structures, insula and secondary somatosensory region [94] (on the superior bank of the sylvian fissure) and supplementary sensory motor area [43] (mesial superior frontal gyrus). Somatosensory phenomena can have a positive or negative nature. Unilateral somatosensory auras predominant, typically with a contralateral focus, but occasionally can be ipsilateral or bilateral, particularly with insular, SMA, or secondary sensory area involvement [4]. With secondary sensory area involvement, there is more likely to be involvement of the face and distal extremities with proximal sparing [94]. In large cohort studies, somatosensory auras were present in 12% [95]. Somatosensory auras correlate with centroparietal epilepsy, particularly if there is a clear “march” but can also be found in temporal lobe, mesial frontal and multifocal epilepsy [95]. Most patients (77%) with somatosensory auras reported tingling. Less commonly, sensations of pain or thermal changes can be reported which are more suggestive of S2 (secondary somatosensory area) involvement. Somatosensory neglect can mimic sensory loss in one extremity, and this should not be mistaken as primary sensory cortex dysfunction, and so a simultaneous double stimulus should be given to test sensation (see supporting materials in Task Force report [10]), as well as testing each side in turn individually to facilitate accurate localisation.

Ictal and postictal evaluation of sensory function

If the patient has a sensory aura and has awareness it is helpful to test sensory function by asking the patient to close his/her eyes. To do this the examiner touches each of the patient's hands in turn and asks them to indicate which hand was touched. If the sensory aura involves a distinct body part, this should be tested. If sensation is intact, sensory neglect can also be determined by touching both hands simultaneously. Neglect is present when touch is not perceived on one side during simultaneous touch, but with normal sensation on the affected side when tested in isolation (see supporting materials in Task Force report [10]).

Ictal and postictal testing when psychogenic non-epileptic seizures (PNES) are suspected

Psychogenic non-epileptic seizures (PNES) result in an interruption to normal functioning and behaviour, with experiences of altered awareness, abnormal movements, and other sensory, experiential or autonomic disturbances, due to internal or external stimuli [96–98]. 1.18.

Neurobiological basis of testing

It is a result of psychological processes, rather than abnormal synchronous cerebral electrical discharges [99]. PNES is not under consciousness voluntary control, nor do the persons experiencing these events always have a recognisable internal thought trigger. Attempts at correlating PNES semiology with psychopathy found altered awareness was associated with lower emotional resilience [100]. The underlying neurobiological mechanisms of PNES have not been fully elucidated with respect to altered interior and exterior awareness [101]. It is proposed that different patients with PNES may have different underlying neurobiological processing mechanisms resulting in their expression of PNES [101]. A recent systematic review [102] found the mean frequency of epilepsy in patients with PNES was 22%, and the mean frequency of PNES in patients with epilepsy was 12%. No individual co-morbidity or semiological clue could provide pathognomonic proof of PNES. PNES is frequently

misdiagnosed (25% in LTM admissions [103,104]) especially if solely based on semiology, and LTM with EEG is required for a definite diagnosis. It has a good inter-rater reliability when the following key points are kept in mind [105]:

1 The semiology should be compatible with the ictal EEG expression.

2 The EEG should be technically satisfactory, ideally without obscuring artefact, and with good quality video.

3 The video EEG should be reviewed from some time before the event, in case an epileptic aura has triggered a PNES event [106].

4 Review for autonomic signs such as significant tachycardia or apnoea. Tachycardia onset is faster in epileptic seizures compared to PNES events, and increases in heart rate from baseline can occur up to 5 min pre-onset of PNES, with a larger increase 1 min pre-onset. Increased respiratory rate is also seen [107].

5 Certain seizure types can have misleading EEG manifestations to those not familiar with LTM. Auras have EEG manifestations in as low as 15–35% of cases. Hyperkinetic seizures including with pelvic thrusting semiology may have obscured EEG manifestations due to myogenic artefact from the seizure onset. Stereotypy is helpful in this instance to determine likelihood of an epileptic seizure. Seizures with brief bilateral asymmetric motor posturing, retained awareness and no post-ictal confusion may indicate SMA semiology and may lack clear surface EEG manifestations [108]. Behavioural arrest spells may have subtle midline rhythmic EEG slowing as the only manifestation. Clear behavioural arrest with preserved posterior dominant alpha rhythm, and no other EEG abnormality is virtually always indicative of PNES.

6 Postictal EEG patterns are helpful if the ictal EEG is obscured by artefact [109].

7 In the postictal period, level of confusion can appear similar in PNES and epileptic seizures, however deep confusion and stertorous breathing favours epilepsy. PNES can be diagnosed based on the positive features of the semiology. Contrary to widely held beliefs, PNES can be stereotyped [98].

The LTM admission should seek to record all the semiological forms of the paroxysmal events noted by the patient/eye-witnesses. It is helpful to show the video to eye-witnesses to ensure the habitual events were captured. Fig. 3 [105] outlines the key semiological features suggestive of PNES and other semiological features reported, which lack sufficient strength of evidence to form strong conclusions [105].

Ictal determination of consciousness is important in cases when trying to distinguish between epilepsy and PNES. The Ictal consciousness inventory was used in LTM and patients with PNES reported greater levels of general awareness/responsiveness and more enhanced subjective experiences in comparison to the epilepsy group [110]. It is important to consider that with attacks that occur off camera, a majority are likely to be PNES, therefore skilful camera manoeuvring is required to ensure the attack is captured and tested for correlation with the EEG [111]. Different methods of inducing PNES can be used, increasing the likelihood of capturing habitual events, particularly when the patient has been informed these procedures may increase the likelihood of an event [112,113]. Induction procedures can result in an 84% success rate at capturing events without excessive suggestion [114]. Standard hyperventilation and photic stimulation are useful in addition to suggestion. It is important to beware of the risk of triggering a non-habitual event [96].

Ictal and postictal testing in PNES events

To our knowledge, there is no specific consensus guidance on how to assess PNES during video telemetry. However, the above paragraphs clearly indicate helpful criteria for a diagnosis, and many require some interaction. At our centres, the testing procedure remains largely similar but with some caveats which are useful in PNES. It is important to ascertain the level of awareness, and the subjective memory of the event [96]. Loss of awareness is typically not absolute, and patients may comply with requests during the paroxysmal event. Memory recall tasks are performed better in PNES than epileptic seizures (63% v 4% respectively for word recall [115]). It is important to take note of the amplitude and pattern of limb movements.

The effect of an examiner holding the patient's moving limb should be noted. Resistance to eye opening can be seen, as can “eye flutter”. PNES tends to have more variable durations, and a stop-start quality, with a greater number of pauses being more characteristic of PNES. While the ILAE taskforce document didn’t specifically cover the ictal testing protocol used in PNES suspected cases, it is hoped that this template can serve as a method to be used clinically and can ideally be validated in future studies.

Conclusion

Careful and timely ictal-interictal assessment provides vital information that when correlated with the EEG patterns expressed during the seizure, allows a confident diagnosis of epilepsy to be made, and in drug refractory cases to formulate a hypothesis of seizure onset for surgical planning. Multi-disciplinary teaching is vital to ensure the success of the standardized ictal testing battery, and we would encourage colleagues to adopt this testing procedure in their LTM units and through quality improvement methodology monitor its implementation. Future research would ideally attempt to correlate the robustness of ictal testing and confidence in surgical hypothesis with invasive monitoring outcomes or epilepsy surgery seizure freedom outcomes.

Declaration of Competing Interest

MK received salary support from UCB Pharma for 6 months for sabbatical work in 2017-2018. This was paid via his employer, the Belfast Health and Social Care Trust and was not related to this present work in any way. BD is a Member of the Center for SUDEP research and receives funding by the NIH (U01NS090407 Autonomic and Imaging Biomarkers of SUDEP) and Epilepsy Research UK. SK received honoraria for lecturing and advising from Sanofi Genzyme, Novartis and Eisai and funding from the Ursula von Euch Foundation (fellowship to Stjepana Kovac).

Acknowledgment

This work was undertaken at UCLH/UCL who receives a proportion of funding from the Department of Health's NIHR Biomedical Research Centres funding scheme.

Appendix A. Supplementary data Supplementary data associated with this article can be found, in the online version.

References

[1] Foldvary-Schaefer N, Unnwongse K. Localizing and lateralizing features of auras and seizures. Epilepsy Behav 2011;20(2):160–6.
[2] Tufenkjian K, Lüders HO. Seizure semiology : its value and limitations in localizing the Epileptogenic Zone. J Clin Neurol 2012;8(4):243–50.
[3] Rossetti AO, Kaplan PW. Seizure semiology: an overview of the "inverse problem.". Eur Neurol 2010;63(1):3–10.
[4] Loddenkemper T, Kotagal P. Lateralizing signs during seizures in focal epilepsy. Epilepsy Behav 2005;7(1):1–17.
[5] Tatum WO, Rubboli G, Kaplan PW, Mirsatari SM, Radhakrishnan K, Gloss D, et al. Clinical utility of EEG in diagnosing and monitoring epilepsy in adults. Clin Neurophysiol 2018;129(5):1056–82.
[6] Kobulashvili T, Hofler J, Dobesberger J, Ernst F, Ryvlin P, Cross JH, et al. Current practices in long-term video-EEG monitoring services: a survey among partners of the E-PILEPSY pilot network of reference for refractory epilepsy and epilepsy surgery. Seizure 2016;38:38–45.
[7] Fisher RS, Engel JJ. Definition of the postictal state: When does it start and end? Epilepsy Behav 2010;19(2):100–4.
[8] Rheims S, Ryvlin P. Patients' safety in the epilepsy monitoring unit: time for revising practices. Curr Opin Neurol 2014;27(2):213–8.
[9] Hamandi K, Beniczky S, Diehl B, Kandler RH, Pressler RM, Sen A, et al. Current practice and recommendations in UK epilepsy monitoring units. Report of a national survey and workshop. Seizure 2017;50:92–8.
[10] Beniczky S, Neufeld M, Diehl B, Doberberger J, Trinka E, Mameniskiene R, et al. Testing patients during seizures: a European consensus procedure developed by a joint taskforce of the ILAE – commission on European Affairs and the European Epilepsy Monitoring Unit Association. Epilepsia 2016;57(9):1363–8.
[11] Kandler R, Lai M, Ponnusamy A, Bland J, Pang C. The safety of UK video telemetry units: results of a national service evaluation. Seizure 2013;22(10):872–6.
[12] Buelow JM, Noe K, Shinnar R, Dewar S, Lebisohn PM, Dean P, et al. A consensusbased approach to patient safety in epilepsy monitoring units: recommendations for preferred practices. Epilepsy Behav 2012;25(3):449–56.
[13] Dobesberger J, Ristic AJ, Walser G, Juchukhidze G, Unterberger I, Hofler J, et al. Duration of focal complex, secondarily generalized tonic-clonic, and primarily generalized tonic-clonic seizures – a video-EEG analysis. Epilepsy Behav 2015;49:111–7.
[14] Malloy K, Cardenas D, Blackburn A, Whitmire L, Cavazos JE. Time to response and patient visibility during tonic–clonic seizures in the epilepsy monitoring unit. Epilepsy Behav 2018;89:84–8.
[15] Gloor P. Consciousness as a neurological concept in epileptology: a critical review. Epilepsia 1986;27(Suppl 2):S14–26.

[16] Blume WT, Lüders HO, Mizrahi E, Tassinari C, van Emde Boas W, Engel J. Glossary of descriptive terminology for ictal semiology: report of the ILAE task force on classification and terminology. Epilepsia 2001;42(9):1212–8.
[17] Hanoğlu L, Özkara Ç, Yalçiner B, Nani A, Cavanna AE. Epileptic qualia and selfawareness: a third dimension for consciousness. Epilepsy Behav 2014;30:62–5.
[18] Willems LM, Watermann N, Richter S, Kay L, Hermsen AM, Knake S, et al. Incidence, risk factors and consequences of epilepsy-related injuries and accidents: a retrospective, single center study. Front Neurol 2018;9:414.
[19] Blumenfeld H, Meador KJ. Consciousness as a useful concept in epilepsy classification. Epilepsia 2014;55(8):1145–50.
[20] Hoppe C, Poepel A, Elger CE. Epilepsy: accuracy of patient seizure counts. Arch Neurol 2007;64(11):1595–9.
[21] Inoue Y, Mihara T. Awareness and responsiveness during partial seizures. Epilepsia 1998;39 Suppl 5:7–10.
[22] Detyniecki K, Blumenfeld H. Consciousness of seizures and consciousness during seizures: are they related? Epilepsy Behav 2014;30:6–9.
[23] Schulz R, Lüders HO, Noachtar S, May T, Sakamoto A, Holthausen H, et al. Amnesia of the epileptic aura. Neurology 1995;45(2):231–5.
[24] Blumenfeld H. Impaired consciousness in epilepsy. Lancet Neurol 2012;11(9):814–26.
[25] Monaco F, Mula M, Cavanna AE. Consciousness, epilepsy, and emotional qualia. Epilepsy Behav 2005;7(2):150–60.
[26] Cavanna AE, Rickards H, Ali F. What makes a simple partial seizure complex? Epilepsy Behav 2011;22:651–8.
[27] Blumenfeld H. Epilepsy and the Consciousness System: transient vegetative state? Neurol Clin 2011;29(4):801–23.
[28] Bartolomei F, McGonigal A, Naccache L. Alteration of consciousness in focal epilepsy: the global workspace alteration theory. Epilepsy Behav 2014;30:17–23.
[29] Lüders HO, Amina S, Bailey C, Baumgartner C, Benbadis S, Bermeo A, et al. Proposal: different types of alteration and loss of consciousness in epilepsy. Epilepsia 2014;55(8):1140–4.
[30] Cavanna AE, Mula M, Servo S, Strigaro G, Tota G, Barbagli D, et al. Measuring the level and content of consciousness during epileptic seizures: the Ictal Consciousness Inventory. Epilepsy Behav 2008;13(1):184–8.
[31] Arthuis M, Valton L, Régis J, Chauvel P, Wendling F, Naccache L, et al. Impaired consciousness during temporal lobe seizures is related to increased long-distance cortical–subcortical synchronization. Brain 2009;132:2091–101.
[32] Yang L, Shklyar I, Lee HW, Ezeani CC, Anaya J, Balakirsky S, et al. Impaired consciousness in epilepsy investigated by a prospective responsiveness in epilepsy scale (RES). Epilepsia 2012;53(3):437–47.
[33] Bauerschmidt A, Koshkelashvili N, Ezeani CC, Yoo JY, Zhang Y, Manganas LN, et al. Prospective assessment of ictal behavior using the revised Responsiveness in Epilepsy Scale (RES-II). Epilepsy Behav 2013;26:25–8.
[34] Nani A, Cavanna AE. The quantitative measurement of consciosuness during epileptic seizures. Epilepsy Behav 2014;30:2–5.
[35] Baykan B, Altindag E, Feddersen B, Ozel S, Noachtar S. Does semiology tell us the origin of seizures consisting mainly in an alteration in consciousness? Epilepsia 2011;52(8):1459–66.
[36] Lux S, Kurthen M, Helmstaedter C, Hartje W, Reuber M, Elger CE. The localizing value of ictal consciousness and its constituent functions: a video-EEG study in patients with focal epilepsy. Brain 2002;125(12):2691–8.
[37] Ali F, Rickards H, Cavanna AE. The assessment of consciosuness during partial seizures. Epilepsy Behav 2012;23:98–102.
[38] Peer M, Lyon R, Arzy S. Orientation and disorientation: lessons from patients with epilepsy. Epilepsy Behav 2014;41:149–57.

[39] Kovac S, Scott CA, Maglajlija V, Rodionov R, McEvoy AW, Diehl B. Extraoperative electrical cortical stimulation: characteristics of motor responses and correlation with precentral gyrus. Clin Neurophysiol 2011;28(6):618–24.
[40] Kovac S, Kahane P, Diehl B. Seizures induced by direct electrical cortical stimulation - Mechanisms and clinical considerations. Clin Neurophysiol 2016;127(1):31–9.
[41] Nachev P, Kennard C, Husain M. Functional role of the supplementary and presupplementary motor areas. Nat Rev Neurosci 2008;9(11):856–69.
[42] Kovac S, Diehl B. Atonic phenomena in focal seizures: nomenclature, clinical findings and pathophysiological concepts. Seizure 2012;21(8):561–7.
[43] Lim SH, Dinner DS, Pillay PK, Lüders HO, Morris HH, Klem G, et al. Functional anatomy of the human supplementary sensorimotor area: results of extraoperative electrical stimulation. Electroencephalogr Clin Neurophysiol 1994;91(3):179–93.
[44] Oestreich LJ, Berg MJ, Bachmann DL, Burchfiel J, Erba G. Ictal contralateral paresis in complex partial seizures. Epilepsia 1995;36(7):671–5.
[45] Noachtar S, Luders HO. Focal akinetic seizures as documented by electroencephalography and video recordings. Neurology 1999;53(2):427–9.
[46] Kelemen A, Fogarasi A, Borbély C, Szucs A, Fabo D, Jakus R, et al. Nonmanipulative proximal upper extremity automatisms lateralize contralaterally in temporal lobe epilepsy. Epilepsia 2010;51(2):214–20.
[47] Maillard L, Vignal JP, Gavaret M, Guye M, Biraben A, McGonigal A, et al. Semiologic and electrophysiologic correlations in temporal lobe seizure subtypes. Epilepsia 2004;45(2):1590–9.
[48] Werhahn KJ. Weakness and focal sensory deficits in the postictal state. Epilepsy Behav 2010;19(2):138–9.
[49] Subota A, Khan S, Josephson CB, Manji S, Lukmanji S, Roach P, et al. Signs and symptoms of the postictal period in epilepsy: a systematic review and meta-analysis. Epilepsy Behav 2019;94:243–51.
[50] Kellinghaus C, Kotagal P. Lateralizing value of Todd's palsy in patients with epilepsy. Neurology 2004;62(2):289–91.
[51] Bergen DC, Rayman L, Heydemann P. Bilateral Todd's paralysis after focal seizures. Epilepsia 1992;33(6):1101–5.
[52] Matthews MS, Smith WS, Wintermark M, Dillon WP, Binder DK. Local cortical hypoperfusion imaged with CT perfusion during postictal Todd's paresis. Neuroradiology 2008;50(5):397–401.
[53] Farrell JS, Gaxiola-Valdez I, Wolff MD, David LS, Hi D, Geeraert BL, et al. Postictal behavioural impairments are due to a severe prolonged hypoperfusion/hypoxia event that is COX-2 dependent. Elife 2016;22(5). pii:e19352.
[54] Knecht S. Handedness and hemispheric language dominance in healthy humans. Brain 2002;123(12):2512–8.
[55] Hamberger MJ, Cole J. Language organization and reorganization in epilepsy. Neuropsychol Rev 2011;21(3):240–51.
[56] Möddel G, Lineweaver T, Schuele SU, Reinholz J, Loddenkemper T. Atypical language lateralization in epilepsy patients. Epilepsia 2009;50(6):1505–16.
[57] Rashid RM, Eder K, Rosenow J, Macken MP, Schuele SU. Ictal kissing: a release phenomenon in non-dominant temporal lobe epilepsy. Epileptic Disord 2010;12(4):262–9.
[58] Kaplan PW. Nonconvulsive status epilepticus in the emergency room. Epilepsia 1996;37(7):643–50.
[59] Privitera M, Kohler C, Cahill W, Yeh HS. Postictal language dysfunction in patients with right or bilateral hemispheric language localization. Epilepsia 1996;73(10):936–41.
[60] Wyllie E, Lüders H, Murphy D, Morris 3rd H, Dinner D, Lesser R, et al. Intracarotid amobarbital (Wada) test for language dominance: correlation with results of cortical stimulation. Epilepsia 1990;31(2):156–61.
[61] Tremblay P, Dick AS. Broca and Wernicke are dead, or moving past the classic model of language neurobiology. Brain Lang 2016;162:60–71.

[62] Drane DL, Pedersen NP. Knowledge of language function and underlying neural networks gained from focal seizures and epilepsy surgery. Brain Lang 2019;189:20–33.
[63] Lüders H, Lesser RP, Hahn J, Dinner DS, Morria HH, Wyllie E, et al. Basal temporal language area. Brain 2007;114(2):743–54.
[64] Kececi H, Degirmenci Y, Gumus H. Two foreign language automatisms in complex partial seizures. Epilepsy Behav Case Reports 2013;1(1):7–9.
[65] Loesch AM, Steger H, Losher C, Hartl E, Remi J, Vollmar C, et al. Seizure-associated aphasia has good lateralizing but poor localizing significance. Epilepsia 2017;58(9):1551–5.
[66] Gabr M, Lüders H, Dinner D, Morris H, Wyllie E. Speech manifestations in lateralization of temporal lobe seizures. Ann Neurol 1989;25(1):82–7.
[67] Adam C, Rouleau I, Saint-Hilaire JM. Postictal aphasia and paresis: a clinical and intracerebral EEG study. Can J Neurol Sci 2000;27(1):49–54.
[68] Privitera MD, Morris GL, Gilliam F. Postictal language assessment and lateralization of complex partial seizures. Ann Neurol 1991;30(3):391–6.
[69] Ramirez MJ, Schefft BK, Howe S, Yeh H-S, Privitera MD. Interictal and postictal language testing accurately lateralizes language dominant temporal lobe complex partial seizures. Epilepsia 2008;49(1):22–32.
[70] Fargo JD, Schefft BK, Dulay MF, Privitera MD, Yeh HS. Confrontation naming in individuals with temporal lobe epilepsy: a quantitative analysis of paraphasic error subtypes. Neuropsychology 2005;19(5):603–11.
[71] Trebuchon A, Lambert I, Guisiano B, McGonigal A, Perot C, Bonini F, et al. The different patterns of seizure-induced aphasia in temporal lobe epilepsies. Epilepsy Behav 2018;78:256–64.
[72] Zöllner JP, Haag A, Hermsen A, Bauer S, Stahl F, Wulf K, et al. Ictal conduction aphasia and ictal angular gyrus syndrome as rare manifestations of epilepsy: the importance of ictal testing during video-EEG monitoring. Epilepsy Behav Case Reports 2017;8:55–62.
[73] Hamberger MJ. Cortical language mapping in epilepsy: a critical review. Neuropsychol Rev 2007;17(4):477–89.
[74] Yen DJ, Su MS, Yiu CH, Shih YH, Kwah SY, Tsai CP, et al. Ictal speech manifestations in temporal lobe epilepsy: a video-EEG study. Epilepsia 1996;37(1):45–9.
[75] Asadi-Pooya AA, Asadollahi M, Bujarski K, Rabiei AH, Aminian N, Wyeth D, et al. Ictal verbal help-seeking: occurrence and the underlying etiology. Epilepsy Behav 2016;64(A):15–7.
[76] Wong AM, Sharpe JA. Representation of the visual field in the human occipital cortex: a magnetic resonance imaging and perimetric correlation. Arch Ophthalmol 1999;117(2):208–17.
[77] Salanova V, Andermann F, Oliver A, Rasmussen T, Quesney LF. Occipital lobe epilepsy: electroclinical manifestations, electrocorticography, cortical stimulation and outcome in 42 patients treated between 1930 and 1991: Surgery of occipital lobe epilepsy. Brain 1992;115(6):1655–80.
[78] Taylor I, Scheffer IE, Berkovic SF. Occipital epilepsies: identification of specific and newly recognized syndromes. Brain 2003;126(4):753–69.
[79] Panayiotopoulos CP. Elementary visual hallucinations, blindness, and headache in idiopathic occipital epilepsy: differentiation from migraine. J Neurol Neurosurg Psychiatry 1999;66(4):536–40.
[80] Jaffe SJ, Roach ES. Transient cortical blindness with occipital lobe epilepsy. J Clin Neuroophthalmol 1988;8(4):221–4.
[81] Kölmel HW. Complex visual hallucinations in the hemianopic field. J Neurol Neurosurg Psychiatry 1985;48(1):29–38.
[82] Bien CG, Benninger FO, Urbach H, Schramm J, Kurthen M, Elger CE. Localizing value of epileptic visual auras. Brain 2000;123(2):244–53.
[83] Gloor P. Experiential phenomena of temporal lobe epilepsy: facts and hypotheses. Brain 1990;113(6):1673–94.
[84] Lee HW, Hong SB, Seo DW, Tae WS, Hong SC. Mapping of functional organization in human visual cortex:electrical cortical stimulation. Neurology 2000;54(4):849–54.

[85] Blanke O, Landis T, Seeck M. Electrical cortical stimulation of the human prefrontal cortex evokes complex visual hallucinations. Epilepsy Behav 2000;1(5):356–61.
[86] Vignal JP, Maillard L, McGonigal A, Chauvel P. The dreamy state: hallucinations of autobiographic memory evoked by temporal lobe stimulations and seizures. Brain 2007;130(1):88–99.
[87] Perumal MB, Chinnasami S, Shah A, Rodionov R, Maglajlija V, Miserocchi A, et al. Epileptic kinetopsia localizes to the superior parietal lobule and intraparietal sulcus. Neurology 2014;83(8):768–70.
[88] Laff R, Mesad S, Devinsky O. Epileptic kinetopsia: ictal illusory motion perception. Neurology 2003;61(9):1262–4.
[89] Jacobs L. Visual allesthesia. Neurology 1980;30(10):1059.
[90] Nelson AE, Gilliam F, Acharya J, Miranda S. A unique patient with epilepsy with cinematographic visual hallucinations. Epilepsy Behav Case Rep 2016;5:78–9.
[91] Prilipko O, Seeck M, Mermillod B, Landis T, Pegna AJ. Postictal but not interictal hemispatial neglect in patients with seizures of lateralized onset. Epilepsia 2006;47(12):2046–51.
[92] Mort DJ, Malhotra P, Mannan SK, Rorden C, Pambakian A, Kennard C, et al. The anatomy of visual neglect. Brain 2003;126(9):1986–97.
[93] Penfield W, Boldrey E. Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. Brain 1937;60(4):389–443.
[94] Lüders H, Lesser RP, Dinner DS, Hahn JF, Salanga V, Morris HH. The second sensory area in humans: evoked potential and electrical stimulation studies. Ann Neurol 1985;17(2):177–84.
[95] Tuxhorn IE. Somatosensory auras in focal epilepsy: a clinical, video EEG and MRI study. Seizure 2005;14(4):262–8.
[96] Whitehead K, Kane N, Wardrope A, Kandler R, Reuber M. Proposal for best practice in the use of video-EEG when psychogenic non-epileptic seizures are a possible diagnosis. Clin Neurophysiol Pract 2017;2:130–9.
[97] Brown RJ, Reuber M. Towards an integrative theory of psychogenic non-epileptic seizures (PNES). Clin Psychol Rev 2016;47:55–70.
[98] Seneviratne U, Reutens D, D'Souza W. Stereotypy of psychogenic nonepileptic seizures: insights from video-EEG monitoring. Epilepsia 2010;51(7):1159–68.
[99] Bodde NM, Brooks JL, Baker GA, Boon PA, Hendriksen JG, Aldenkamp AP. Psychogenic non-epileptic seizures-diagnostic issues: a critical review. Clin Neurol Neurosurg 2009;111(1):1–9.
[100] Baslet G, Tolchin B, Dworetzky BA. Altered responsiveness in psychogenic nonepileptic seizures and its implication to underlying psychopathology. Seizure 2017;52:162–8.
[101] Roberts NA, Reuber M. Alterations of consciousness in psychogenic nonepileptic seizures: emotion, emotion regulation and dissoication. Epilepsy Behav 2014;30:43–9.
[102] Kutlubaev MA, Xu Y, Hackett ML, Stone J. Dual diagnosis of epilepsy and psychogenic nonepileptic seizures: systematic review and meta-analysis of frequency, correlates, and outcomes. Epilepsy Behav 2018;89:70–8.
[103] Bendadis SR, O'Neill E, Tatum WO, Heriaud L. Outcome of prolonged video-EEG monitoring at a typical referral epilepsy center. Epilepsia 2004;45(9):1150–3.
[104] Smith D, Defalla BA, Chadwick DW. The misdiagnosis of epilepsy and the management of refractory epilepsy in a specialist clinic. QJM 1999;92(1):15–23.
[105] Lafrance Jr WC, Baker GA, Duncan R, Goldstein LH, Reuber M. Minimum requirements for the diagnosis of psychogenic nonepileptic seizures: a staged approach: a report from the International League Against Epilepsy Nonepileptic Seizures Task Force. Epilepsia 2013;54(11):2005–18.
[106] Devinsky O, Gordon E. Epileptic seizures progressing into nonepileptic conversion seizures. Neurology 1998;51(5):1293–6.
[107] Indranada AM, Mullen SA, Wong MJ, D'Souza WJ, Kanaan RAA. Preictal autonomic dynamics in psychogenic nonepileptic seizures. Epilepsy Behav 2019;92:206–12.

[108] Adamolekun B, Foreman A. Post-ictal alpha activity in supplementary motor seizures mimics nonepileptic seizures. Epilepsy Behav 2010;18(3):317–21.
[109] Whitehead K, Gollwitzer S, Millward H, Wehner T, Scott C, Diehl B. The additional lateralizing and localizing value of the postictal EEG in frontal lobe epilepsy. Clin Neurophysiol 2016;127(3):1774–80.
[110] Ali F, Rickards H, Bagary M, Greenhill L, McCorry D, Cavanna AE. Ictal Consciousness in epilepsy and nonepileptic attack disorder. Epilepsy Behav 2010;19:522–5.
[111] Ulac K, Albakir M, Saygi S. The tendency to have psychogenic non-epileptic attacks out of camera view during long-term video EEG monitoring. Seizure 2002;11:384–5.
[112] Hoepner R, Labudda K, Schoendienst M, May TW, Bien CG, Brandt C. Informing patients about the impact of provocation methods increases the rate of psychogenic nonepileptic seizures during EEG recording. Epilepsy Behav 2013;28(3):457–9.
[113] Eddy CM, Cavanna AE. Video-electroencephalography investigation of ictal alteration of consciosuness in epilepsy and nonepileptic attack disorder: practcal considerations. Epilepsy Behav 2014;30:24–7.
[114] Popkirov S, Grönheit W, Wellmer J. A systematic review of suggestive seizure induction for the diagnosis of psychogenic nonepileptic seizures. Seizure 2015;31:124–32.
[115] Bell WL, Park YD, Thompson EA, Radtke RA. Ictal cognitive assessment of partial seizures and pseudoseizures. Arch Neurol 1998;55(11):1456–9.

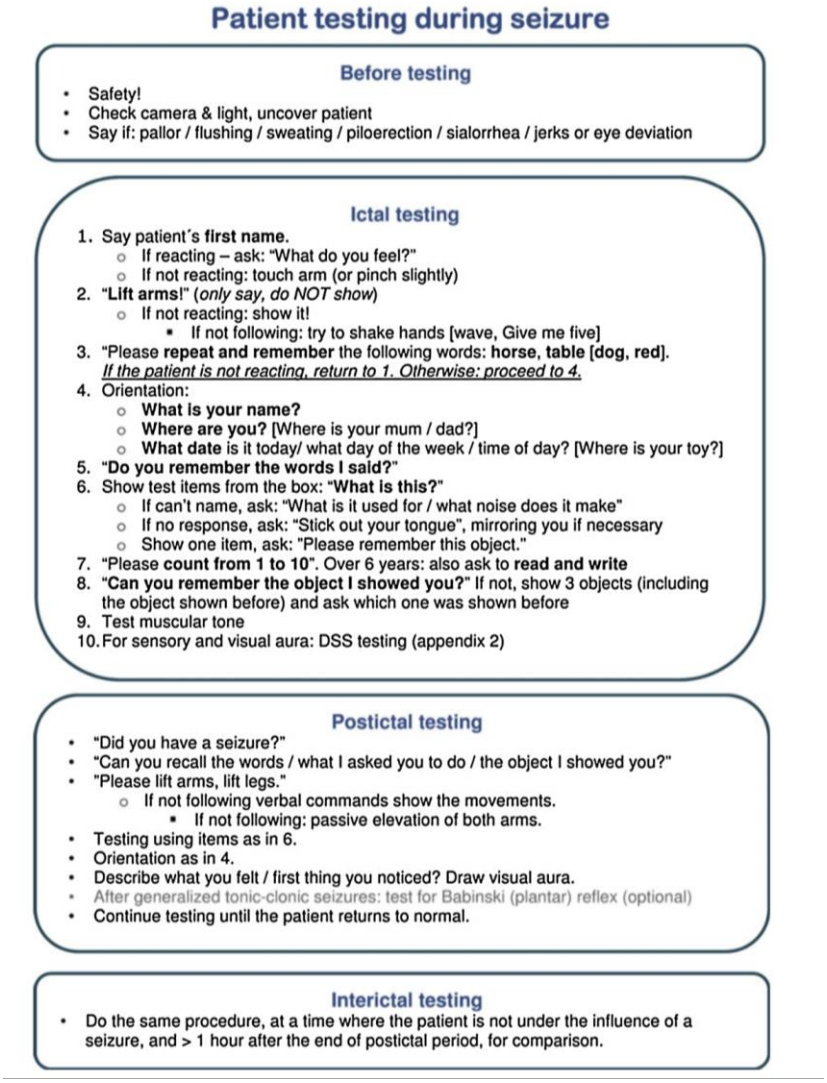


Fig. 1. Summary of the testing battery as proposed by the ILAE-CEA Taskforce with an overview of the domains tested. (Reproduced with kind permission from John Wiley and Sons; see reference [10].

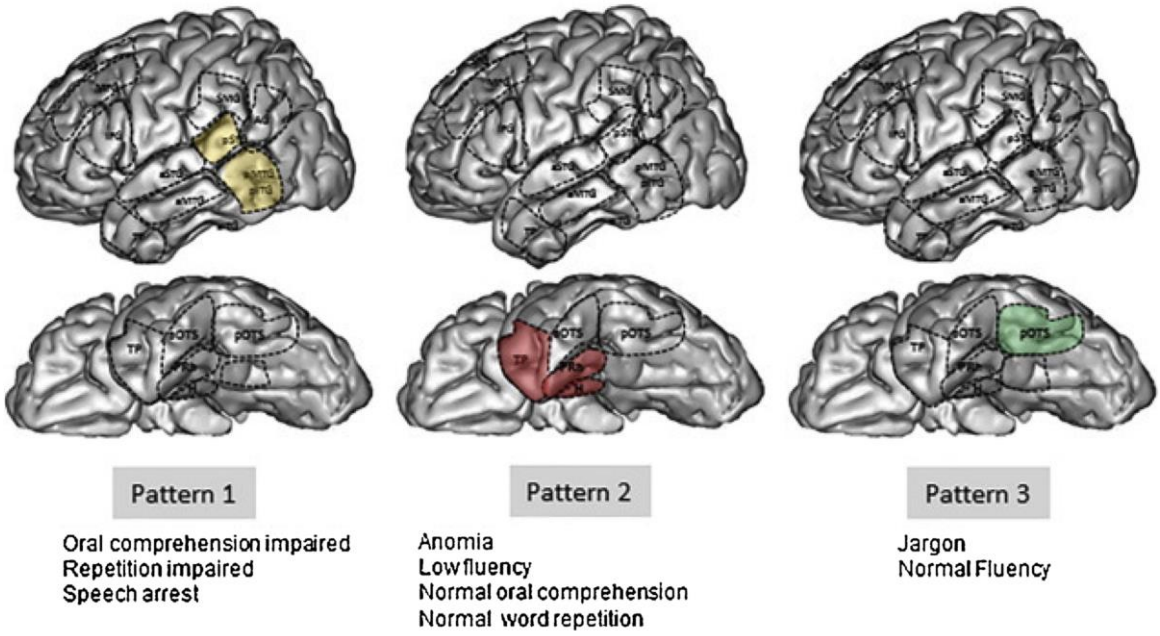


Fig. 2. Sub-lobar localisations of language function in seizures. (Reproduced with kind permission from Elsevier; see reference [63].

Firstly, speech comprehension (Wernicke's pattern) was impaired with posterior-lateral temporal involvement, to include additional speech arrest, reduced fluency, and impaired word repetition. Thus indicating that Broca's area (posterior inferior frontal gyrus) does not have exclusive control over language output. Comprehension was also linked to the posterior part of the superior temporal gyrus, superior temporal sulcus, and middle temporal gyrus. The second pattern seen was anomia, low fluency, with normal comprehension and repetition, in anterior-medio-basal (perirhinal cortex, temporal pole, and hippocampus) temporal lobe, resembling "transcortical motor aphasia". The third pattern; jargon-aphasia (neologisms) with normal fluency was seen with basal temporal onset (indicating fusiform gyrus and posterior aspect of the occipital temporal sulcus involvement).

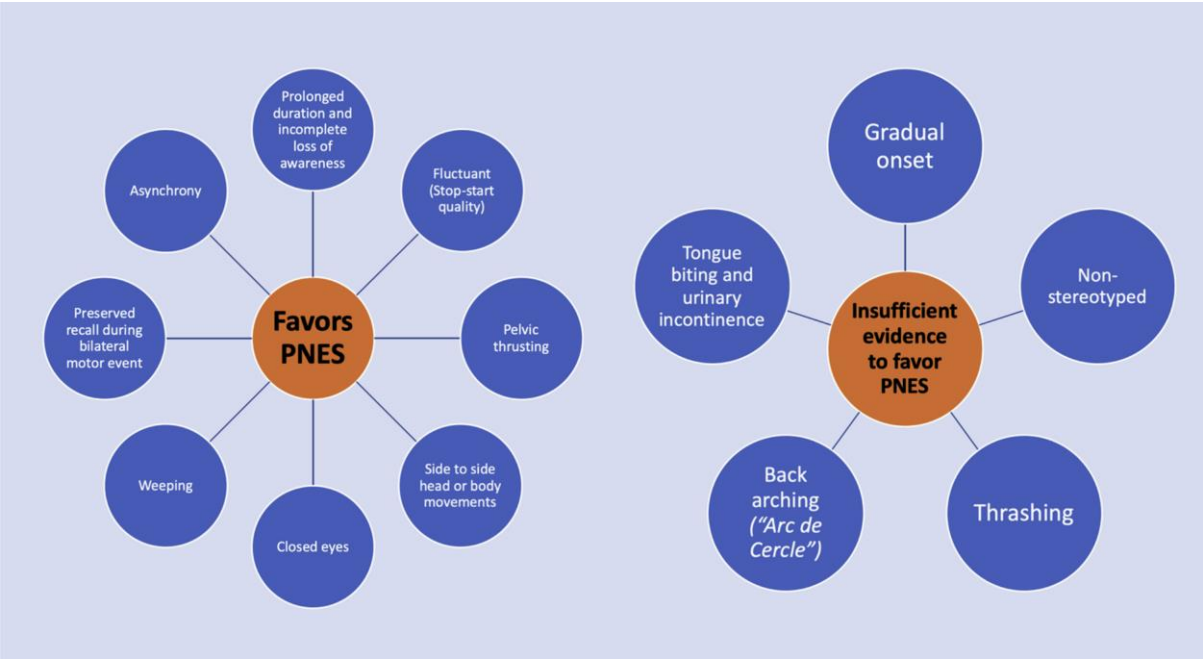


Fig. 3. Key semiological features which favour psychogenic nonepileptic seizures (PNES) or have insufficient evidence to favour PNES (Based on reference [97]. The figure indicates the key semiological features associated with PNES with strong evidence and those with insufficient value to favour PNES.

Table 1: Initial approach at the patient's bedside (adapted and based on [10,12,14]).

• Consider seizure first aid and safety.

• Remain at the bedside, call for help if required.

• Monitor vital signs during and after the seizure.

• Place the patient on their side as soon as possible, and support their head to maintain airway patency and reduce injury (generalised seizures).

• Employ suction where required, and ensure oxygen applied rapidly where required.

• Side rails (ideally padded) should be placed up.

• Follow local departmental intravenous or rescue drug protocol as indicated.

• Turn on the room lights if the room is not well lit.

• Ask for cameras to be repositioned so the patient is central, and avoid obscuring the view of the patient.

• Pull back bed sheets to allow a view of the limbs (always maintain patient dignity, with appropriate exposure). One study [14] found that in 20% of cases no exposure was attempted, and it took 38 seconds from EEG generalisation in tonic clonic seizure before the covers were pulled back.

• Avoid excessively large hospital emergency teams in the room for any non-life threatening situations, as too many people will obscure the camera view.

• Push the event button, to register the event on the EEG recording.

• Locate the ictal-postictal testing protocol (ideally for ease of use, it should be available in each patient room) and commence testing.

• Describe in a loud voice for the camera any subtle features which may be missed on camera (due to poor recording resolution) such as; pallor, flushing, goosebumps, sialorrhoea, subtle jerks around the eyes or face, nystagmus or blinking

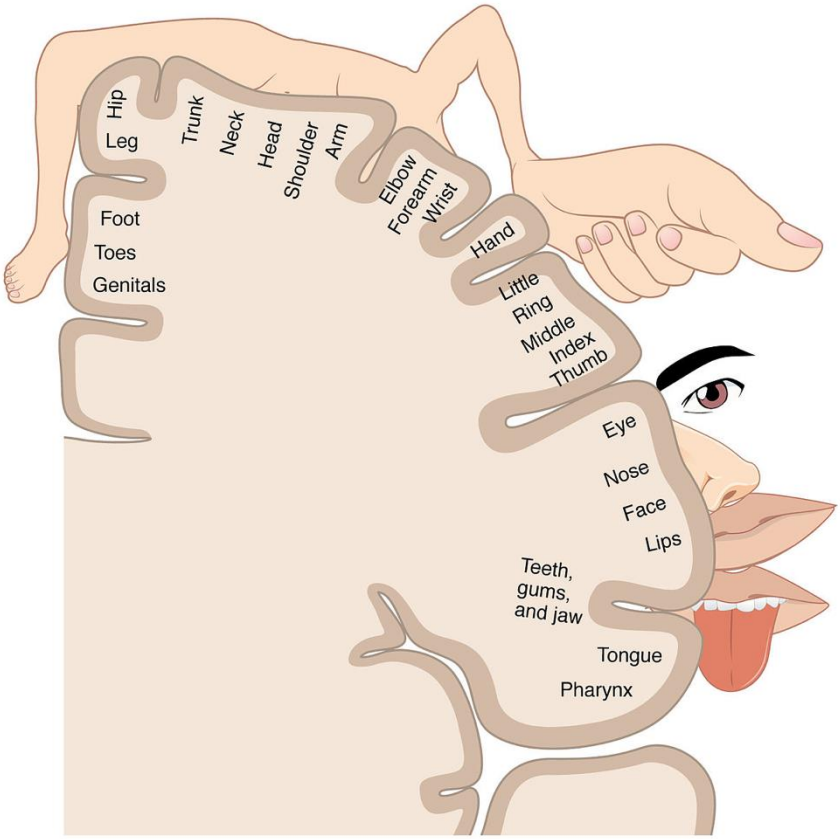
Table 2: Summary of seizure semiology: lateralizing signs during ictal and postictal testing (Reproduced with kind permission from Elsevier with adaptations see references [1,4].

Seizure pattern	Subtype	Symptomatogenic zone	Lateralisation
Aura	Elementary visual	Primary visual cortex (BA 17, 18, 19)	Contralateral
	Complex visual	Temporo-parietal-occipital junction, basal temporal (limbic and neocortical)	Contralateral if unilateral
	Simple auditory	Primary auditory cortex (BA 41) Illusions; lateral temporal, insula	Contralateral if unilateral
	Complex auditory	Auditory association cortex (BA 42, 22)	Contralateral if unilateral
	Vertiginous	Temporo-parieto-occipital junction (superior temporal, and inferior parietal)	Not-lateralising (often right)
	Olfactory	Orbitofrontal, amygdala (uncinate), insula	Non-lateralising
	Gustatory	Rolandic and parietal operculum, basal/mesial temporal and insula	Non-lateralising
	Abdominal	Anterior insula, frontal operculum, mesial temporal lobe, SSMA	Non-Lateralising
	Autonomic	Insula, amygdala, anterior cingulum, and SSMA	Non-Lateralising
	Fear	Amygdala, hippocampus, mesial frontal lobe (anterior cingulate)	Non-lateralising
	Déjà vu/jamais vu	Uncus, entorhinal/perirhinal cortex, and temporal neocortex	Non-lateralising
	Cephalic/whole body	Amygdala, entorhinal cortex, and temporal neocortex/SSII and SSMA	Non-lateralising
	Somatosensory	Primary somatosensory cortex (BA 1, 2, 3b), secondary somatosensory areas (parietal operculum/SSII), SSMA.	Contralateral to primary, ipsilateral if unilateral for secondary somatosensory, and contralateral for SSMA
Dialeptic		Limbic temporal structures, cingulum, intermediate frontal (BA 8), orbitofrontal areas	Non-lateralising
Autonomic	Tachycardia/ hyperventilation	Amygdala, insula, anterior cingulum, and ventro-medial prefrontal cortex, hippocampus.	Non-lateraling (often right)
	Goosebumps	Mesial frontal, anterior cingulate	Ipsilateral
	Mydriasis	Mesial frontal and mesial temporal/insular cortex	Contralateral and ipsilateral respectively
Simple Motor	Myoclonic/negative myoclonus	Primary motor cortex (BA 4) and premotor cortex (BA 6)/primary somatosensory area	Contralateral (if unilateral)
	Clonic	Primary motor cortex, premotor cortex, and SSMA	Contralateral
	Tonic	Primary motor cortex and SSMA	Contralateral (If unilateral)
Complex motor	Hypermotor	Anterior cingulum, orbitofrontal region, frontopolar region, opercular–insular cortex, and medial intermediate frontal area	Non-lateralising
	Automotor (focal onset seizure with automatisms)	Mesial temporal and anterior cingulum, SSMA	Non-lateralising
	Gelastic	Hypothalamus, anteromesial frontal region, and basal temporal area	Non-lateralising

Table 3: Motor semiology: lateralisation and localisation (Reproduced with kind permission from Elsevier; See references [1,4].

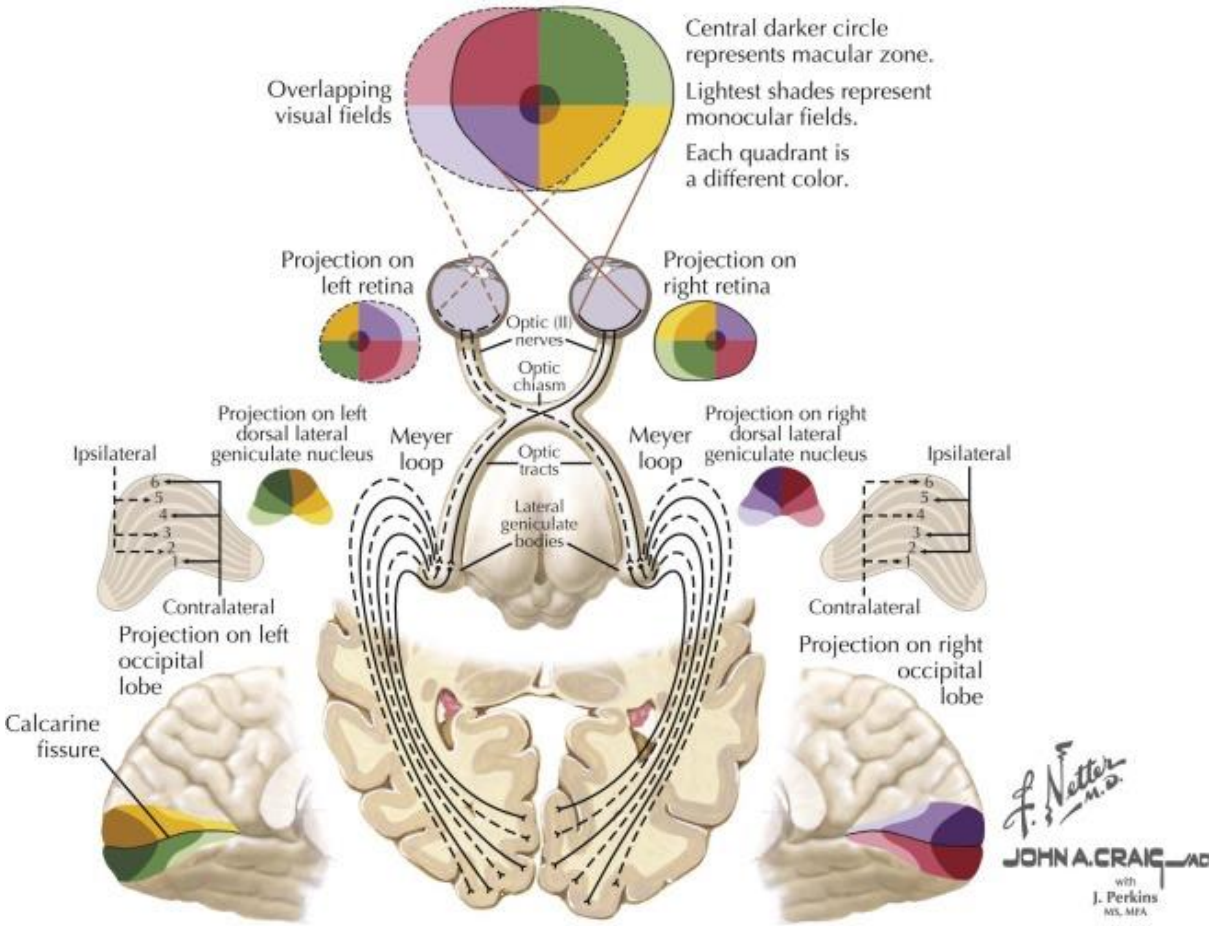
Seizure subtype	Semiology	Symptomatogenic zone or process resulting in semiology	Lateralization
Complex motor signs in motor seizures	**Dystonic posturing**	Basal ganglia involvement	Contralateral
	Tonic posturing	SSMA, basal ganglia, cingulum, primary motor cortex (M1)	Contralateral
	Head turning	Exhaustion of epileptogenic hemisphere, seizures propagate to basal ganglia, or neglect of contralateral space	Ipsilateral
	Eye version	Frontal eye fields (BA 8) and extra-striate cortex (BA 19)	Contralateral
	Unilateral eye blinking	Mesial temporal	Ipsilateral
	Facial alterations	Activation of emotional network (amygdala, prefrontal cortex, hypothalamus, orbitofrontal region, insula) or emotional facial movements in cingulum	Contralateral (if facial weakness)
	Paresis of limb	M1 or premotor cortex exhaustion, or activation of negative motor areas including primary motor, SMA, cingulum.	Contralateral
Signs during secondary generalized tonic clonic seizures	**Asymmetric tonic limb posturing**	SSMA and precentral area	Contralateral
	Asymmetric termination of clonic jerks	Exhaustion of hemisphere of seizure onset	Ipsilateral
	Head version	Premotor area (BA 6 and 8) during secondary generalized tonic–clonic seizures	Contralateral

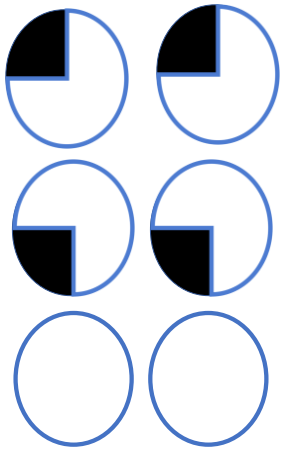
Suppl. Fig. 1: Somatotopic organization in the sensory cortex



Suppl. Fig. 2: Anatomy of the visual pathways and examples of potential visual field defects.

(Reproduced with kind permission from Elsevier; Netter's Neurology, Royden-Jones H, Jr. Srinivasan J, Allam G, Baker R, Saunders 2011)





Contralateral upper quadrantanopia: involvement of Meyer's loop - temporal lobe optic radiation

Contralateral lower quadrantanopia: parietal lobe or optic radiation.

Contralateral homonymous hemianopia: retrochiasmal location; occipital cortex or temporo-parietal optic radiations

*The black colour indicates the area of the visual field that is not visible. Thus it is documented as the patient perceives the visual field.