

¹ Department of Clinical Neurophysiology, Aarhus University Hospital, Aarhus, Denmark, member of the European Reference Network EpiCare

² Danish Epilepsy Centre, Dianalund, Denmark, member of the European Reference Network EpiCare

³ Department of Clinical Medicine, Aarhus University, Aarhus Denmark

⁴ Department of Neurology, Mayo Clinic, Jacksonville, FL, USA

⁵ Departments of Neurology, Neuroscience, Neurosurgery, Yale University School of Medicine, New Haven, Connecticut, USA

⁶ Department of Neurology - Biomagnetism, Epilepsy Center, University Hospital Erlangen, Germany

⁷ Department of Brain and Nervous System, Kokilaben Dhirubhai Ambani Hospital, Mumbai, India

⁸ Department of Neurology and National Reference Centre for Rare Epilepsies, University Hospital of Nancy, Nancy, France

⁹ Research Centre for Automatic Control of Nancy (CRAN), Lorraine University, Vandoeuvre, France

¹⁰ Neurological Institute, Tel Aviv Sourasky Medical Centre and Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

¹¹ Department of Pediatric Neurology, Amrita Institute of Medical Sciences, Cochin, Kerala, India

¹² Department of Neurology, Epilepsy Clinic, Fundacion Santa Fe de Bogotá University Hospital, Bogotá, Colombia

¹³ EEG and Epilepsy Unit, University Hospitals and Faculty of Medicine, University of Geneva, Geneva, Switzerland

¹⁴ Department of Clinical Neurosciences, Lausanne University Hospital, Lausanne, Switzerland

¹⁵ Neurology Department, CHU Grenoble Alpes, Univ. Grenoble Alpes and GIN Inserm 1216, 38000 Grenoble, France

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• **Correspondence:**

Sándor Beniczky
Aarhus University Hospital
and Danish Epilepsy Center,
Visby Allé 5, 4293,
Dianalund, Denmark
<sbz@filadelfia.dk>
<sandor.beniczky@clin.au.dk>

Seizure semiology: ILAE glossary of terms and their significance

Sándor Beniczky^{1,2,3}, William O. Tatum⁴, Hal Blumenfeld⁵, Hermann Stefan⁶, Jayanti Mani⁷, Louis Maillard^{8,9}, Firas Fahoum¹⁰, Kollencheri Puthenveetil Vinayan¹¹, Luis Carlos Mayor¹², Maria Vlachou¹, Margitta Seeck¹³, Philippe Ryvlin¹⁴, Philippe Kahane¹⁵

ABSTRACT

This educational topical review and Task Force report aims to address learning objectives of the International League Against Epilepsy (ILAE) curriculum. We sought to extract detailed features involving semiology from video recordings and interpret semiological signs and symptoms that reflect the likely localization for focal seizures in patients with epilepsy. This glossary was developed by a working group of the ILAE Commission on Diagnostic Methods incorporating the EEG Task Force. This paper identifies commonly used terms to describe seizure semiology, provides definitions, signs and symptoms, and summarizes their clinical value in localizing and lateralizing focal seizures based on consensus in the published literature. Video-EEG examples are included to illustrate important features of semiology in patients with epilepsy.

Key words: glossary, lateralization, localization, epilepsy, seizure semiology, video-EEG

Seizure semiology comprises objective signs and subjective symptoms reported by patients during epileptic seizures. Accurate delineation of seizure semiology is essential for properly diagnosing and classifying seizures and epilepsy syndromes. Although valuable information can be obtained from the patients, and from people who have witnessed seizures [1], more detailed feature-extraction is possible by reviewing video recordings of the typical events [2]. Recent technological advances with high-resolution video cameras allow recording of subtle

details, such as goosebumps, flushing and pallor. Combining EEG that is synchronized with the video allows detailed analysis and sequential electroclinical correlation from seizure onset to seizure termination. Information obtained from patients concerning subjective seizure symptoms (not visible on the video recordings) are essential for correct characterization of the seizures, and for correct localization and lateralization, as described in detail in a previous seminar in epileptology [1]. The term aura denotes subjective symptoms at

seizure onset that are perceived by the patient. It has been described in focal seizures (i.e., focal aware seizures and at the onset of focal impaired awareness seizures, before impairment of consciousness). Aura has also been reported in generalized seizures. Epileptic aura must be differentiated from aura occurring in non-epileptic paroxysmal events (migraine, syncope, dissociative seizures).

Our goal is to provide a list of terms that are used to describe seizure semiology, and to update the glossary initially developed in the ILAE Commission report published 20 years ago [3]. Semiology features were subdivided to reflect similar signs and their corresponding location. We acknowledge that other categories are possible. However, our focus was not to classify the seizure semiology, but rather, to describe the individual characteristics according to a commonly accepted terminology, that may be used individually or in combination to aid in defining the ictogenic network. For further information regarding definitions and classification of seizures and epilepsy, the reader is referred to the ILAE position statements [4-6].

We also included phenomena that occur immediately after seizure termination to distinguish postictal semiology and emphasize their clinical relevance. We used modifiers in accordance with what was previously proposed [7]. For some features, the terminology used to describe semiology applies to the body part that is involved (for example “epigastric aura”), and in other cases somatotopic descriptors have been added (for example “left hand gestural automatisms”). Other descriptors of seizures including duration, timing, and provoking or facilitating factors are also listed and defined.

This glossary of terms was designed to address learning objectives outlined by the ILAE curriculum and encompass an education review. Section 1.3.3 of the ILAE curriculum includes “Extract semiology information from video recordings” and section 1.3.4, “Interpret semiological signs and symptoms allowing hypotheses on the localization of focal seizures” [8]. This report has been developed by a working group of the ILAE Commission on Diagnostic Methods involving the EEG Task Force. For each semiology feature we used the following search strategy in PubMed: (((*name of the semiology feature* [Title/Abstract]) OR (*synonym widely used* [Title/Abstract])) AND ((localization [Title/Abstract]) OR (lateralization [Title/Abstract]))) AND (seizure [Title/Abstract]). Date for last search was 1/11/2021. In addition, we included papers from citations found in reference lists and review articles. *Table 1* summarizes the most plausible localization and lateralization accepted for the most important semiological features. *Table 2* lists video examples.

1. Electrographic seizures

Electrographic seizures are subclinical and do not have any observable signs or reported symptoms despite an unequivocal ictal pattern on EEG: 1) sustained spike-wave discharges with a frequency ≥ 2.5 Hz; 2) rhythmic – evolving ictal activity (with a dynamic evolution in time and space). Electrographic seizures (10 seconds and longer) and electrographic status epilepticus (10 minutes and longer) are often recorded in critically ill patients [9-12], and also in genetic or developmental / epileptic encephalopathies (such as patients with ring chromosome 20, Angelman syndrome and related genetic syndromes), in neonatal epilepsies [13] and in acute symptomatic neonatal seizures.

2. Consciousness

Impaired consciousness is of major clinical importance in epilepsy, affecting the ability of patients to safely drive, effectively work, and maintain the ability to function in society [14-17]. However, because the term “consciousness” is broad, the 2017 ILAE classification adopted the narrower term “awareness” in the classification, as a “simple surrogate marker for consciousness” [5]. Another related feature broadly used in video-EEG monitoring is assessment of the responsiveness, which requires behavioral testing of the patient [18]. It is well-recognized that each individual method for assessing consciousness has its limitations, and care must be taken to interpret results in the context of potential deficits such as motor impairment, aphasia or amnesia [19]. However, with these caveats in mind, all information should be included and is of great value in assessing this clinically important aspect of seizures. Impaired consciousness typically occurs in generalized seizures [20-23]. However, impaired consciousness is also very frequently noted in focal seizures when a large volume of cortex is involved in the seizure network [22, 24-30]. It should be differentiated from amnesic seizures, i.e. while the patient may be unable to recall events during the seizure, she/he is also unable to remember the seizure itself. These features can be dissociated.

2.1. Awareness

Awareness is commonly used as a surrogate marker for assessing consciousness during a seizure [5]. It is defined in the 2017 ILAE classification as “knowledge of self or environment” and assessment depends on “the ability of the person having the seizure to later verify retained awareness” based on “whether

▼ **Table 1.** Key points showing the most accepted localization and lateralization signs for the most important semiological features.

Semiology features		Lateralizing value		Localizing value		
Consciousness	Awareness	D	When complete impairment in TLE			
	Responsiveness	ND	When partial responsiveness with automatisms			
Sensory phenomena	Auditory			T	Posterior - superior temporal neocortex	
	Body-perception illusion			T, P, O		
	Depersonalization					
	Forced thinking					
	Gustatory			Ins	Posterior-dorsal part	
	Olfactory			T, F, Ins	Amygdala, orbito-frontal cortex, postero-dorsal insula	
	Somatosensory	Non painful	CON	When unilateral	P	Sensory cortex (S1)
		Painful & thermal	CON	When unilateral	Ins	Posterior-superior insula
	Vestibular				T, P	Posterior temporo-perisylvian cortex, including insula
	Visual		CON	When unilateral, mainly elementary hallucinations	O	Visual cortex (V1)
Affective (emotional) phenomena	Anger			T, F	Amygdala, prefrontal (orbito-frontal)	
	Anxiety			T	Amygdala	
	Ecstatic/bliss			Ins	Anterior dorsal Insula	
	Fear			T, F	Amygdala, orbito-frontal cortex, anterior cingulate	
	Guilt					
	Mirth					
	Mystic			T		
	Sadness					
Sexual						
Cognitive phenomena	Aphasia			T, P, F	Broca, Wernicke, basal temporal language area	
	Dysmnnesia					
	Dysmnnesia	Amnesia				

▼ **Table 1.** Key points showing the most accepted localization and lateralization signs for the most important semiological features (*continued*).

Semiology features		Lateralizing value		Localizing value	
Déjà (jamais)-vu/ dreamy state	Déjà-vu/dreamy state/ reminiscence		T	Mesio-temporal	
Akinetic			F	Mesial premotor cortex, inferior F gyrus	
Astatic					
Atonic					
Dystonic		CON	When unilateral, mainly in TLE		
Epileptic nystagmus		CON	When considering the fast phase	O	
Epileptic spasm		CON	When unilateral		
Eye blinking		IPSI			
Gyratory		CON	When preceded by head version of the same direction		
Head orientation					
Elementary motor phenomena	Ictal paresis	CON		F	Motor cortex (M1)
	Myoclonic/clonic	CON		F	Motor cortex (M1)
	Myoclonic- atonic				
	Unilateral tonic	CON		F	
	Chapeau de gendarme			F	Anterior prefrontal (mainly anterior cingulate cortex)
Tonic	Fencing posture	CON	When considering the elevated arm	F	Supplementary motor area
	Unilateral tonic- clonic	CON		F	Premotor and motor (M1) cortices
	Asymmetric clonic ending	IPSI	When considering the last clonic jerks		
	Figure-of-four	CON	When considering the extended arm		
	Versive	CON	When followed by FBTC		
Complex motor phenomena	Automatisms	Gestural automatisms- distal	IPSI	When unilateral, mainly in context of TLE	
		Gestural automatisms- genital			

▼ **Table 1.** Key points showing the most accepted localization and lateralization signs for the most important semiological features (*continued*).

Semiology features		Lateralizing value		Localizing value	
	Gestural automatisms-proximal				
	Ictal grasping		F	Anterior cingulate	
	Mimic automatisms Gelastic/ dacrystic		Hypoth	Hypothalamic hamartoma when isolated and in clusters	
	Oro-alimentary automatisms	ND	When awareness is preserved		
	Verbal automatisms		T	Related to insulo-opercular involvement	
	Vocal automatisms	D	In context of TLE and FLE		
	Hyperkinetic behaviour				
Autonomic phenomena	Cardiovascular	Bradycardia + ictal asystole		T, F	Bilateral temporal, orbito-frontal
		Tachycardia		T	
	Cutaneous	Flushing			
		Pallor			
		Piloerection			
		Sweating			
	Gastrointestinal	Epigastric		T	Mainly when followed by automatisms
		Flatulence			
		Hypersalivation			Insulo-opercular
		Spitting		T	
	Pupillary	Vomiting		Ins	
		Miosis			
	Respiratory	Mydriasis			
		Apnea		T	Mainly when contralateral spread
Urinary	Chocking			Insulo-opercular	
	Hyperventilation				
	Hypoventilation				
		ND	For ictal urinary urge	T	

▼ **Table 1.** Key points showing the most accepted localization and lateralization signs for the most important semiological features (*continued*).

	Semiology features	Lateralizing value	Localizing value		
	Postictal autonomic signs				
	Postictal blindness	CON	When unilateral		
	Postictal headache	IPSI	In the context of TLE		
	Postictal language dysfunction	D			
Postictal phenomena	Postictal nose-wiping	IPSI	In the context of TLE	T, F	Temporal more than frontal
	Postictal pallinacousis				
	Postictal paresis (Todd's paresis)	CON			
	Postictal psychiatric signs				
	Postictal unresponsiveness				

D: dominant; ND: non-dominant; CON: contralateral; IPSI: ipsilateral; TLE: temporal lobe epilepsy; FLE: frontal lobe epilepsy; F: frontal; T: temporal; P: parietal; O: occipital, Ins: insula; Operc: opercular; Ins: insula; Hypoth: hypothalamus.

awareness for events occurring during the seizure was retained or impaired". However, the postictal recall is also affected by the ictal involvement of language and memory, not only awareness per se. Even with these limitations, awareness is used as a core descriptor to stratify focal seizures in the recent ILAE classification of seizures [5]. Patient awareness about the occurrence of a seizure is another completely different attribute, which might be clinically relevant for planning diagnostic and management strategies [31]. Impaired awareness (inability of the person having the seizure to later verify retained awareness) occurs in generalized or bilateral tonic-clonic seizures with uncommon exceptions [23, 32-34]. Most absence seizures cause impaired awareness [20-23]. However, in some cases during bursts of generalized spike-wave discharges, the patient does not follow commands and is unable to repeat the test words (impaired responsiveness), yet after the spike-wave bursts terminate, the patient is able to recall the commands and test words given during the seizure, demonstrating retained awareness during the electroclinical absence seizure (non-responsive patient with ictal EEG correlate) [21, 22, 35-41]. In temporal lobe

epilepsies, impaired awareness may be more common in dominant hemispheric onset [29, 42, 43], although this could be biased by mainly verbal tests used for assessment. In most of the temporal lobe epilepsies, the awareness becomes impaired when the seizure involves neocortical and subcortical structures [25, 29].

2.2. Responsiveness

Responsiveness during the seizure to questions, commands or other stimuli is used as a descriptor, but not as a classifier, because "some people are immobilized and consequently unresponsive during a seizure, but still able to observe and recall their environment" [5]. Despite not being used as a classifier, responsiveness during seizures is highly clinically relevant, for example in evaluating driving safety [5, 17, 44-46]. Responsiveness is used as a standard means of assessment in other neurological disorders of consciousness [47-49], and is commonly used in epilepsy monitoring units because it is relatively objective and does not rely on patient subjective report. Testing batteries should be used for

▼ **Table 2.** Seizure semiology: videos.

Semiology	Seizure onset and propagation	Video link
MOTOR PHENOMENA		
Akinetic	Aura (presses the alarm button) → R manual automatisms + L arm & leg immobile + facial automatisms → non-responsive → R head & eyes orientation (not forced) → oroalimentary automatisms + bilateral eye blinking → proximal head & leg automatisms → R manual automatisms + L arm & leg still immobile	https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289505 [403]
Atonic	Oro-alimentary automatisms + non-responsiveness → atonic seizure with head drop and slight R deviation + arms atonia	https://www.jle.com/en/revues/epd/e-docs/intracranial_video_eeg_and_surgery_for_focal_atonic_seizures_296712/article.phtml?tab=videos&cle_video_une=296336 [404]
	Muscle atonia of neck (head drop) and trunk	Supplementary video 1 Supplementary figure 1
	Seizures with negative myoclonus of arms +/- head atonia (head drop)	https://www.jle.com/en/revues/epd/e-docs/induction_of_epileptic_negative_myoclonus_by_oxcarbazepine_in_symptomatic_epilepsy_265696/article.phtml?tab=videos&cle_video_une=285271 [405]
Negative myoclonus	Focal aware seizures: L arm negative myoclonus	(case 6, sec 7.17-7.43) https://www.jle.com/en/revues/epd/e-docs/semiology_of_typical_and_atypical_rolandic_epilepsy_a_video_eeg_analysis_110047/article.phtml?tab=videos&cle_video_une=285253 [406]
	Follows command to raise both arms → R arm repetitive negative myoclonus	https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289032 [403]
Unilateral eye blinking	Left eye blinking while patient describes difficulties in speaking (speaking slowly but coherently)	https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289029 [403]
Myoclonic	Myoclonic jerks in arms	https://www.jle.com/en/revues/epd/e-docs/crossing_the_lines_between_epilepsy_syndromes_a_myoclonic_epilepsy_variant_with_prominent_eyelid_myoclonia_and_atonic_components_310815/article.phtml?tab=videos&cle_video_une=311030 [407]
	Myoclonic jerks of facial muscles	https://www.jle.com/en/revues/epd/e-docs/myoclonic_jerks_are_commonly_associated_with_absence_seizures_in_early_onset_absence_epilepsy_309712/article.phtml?tab=videos&cle_video_une=309981 [408]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
	Myoclonic jerks of facial muscles, eyelids, trunk and arms	https://www.jle.com/en/revues/epd/e-docs/myoclonic_status_epilepticus_as_a_presentation_of_caspr2_antibody_associated_autoimmune_encephalitis_303162/article.phtml?tab=videos&cle_video_une=304023 [409]
	Bilateral myoclonic jerks, symmetric (<i>Note the surface EMG with myogenic artifact during brief, bilateral muscle activation</i>)	Supplementary video 2 Supplementary figure 2
Clonic	Tonic (asymmetric: R arm elevation, L arm flexion) → L head version → bilateral asymmetric clonic jerks → R version → terminal L asymmetric clonic jerks → hypoventilation	https://www.jle.com/en/revues/epd/e-docs/classification_of_paroxysmal_events_and_the_four_dimensional_epilepsy_classification_system_313798/article.phtml?tab=videos&cle_video_une=313814 [410]
	Bilateral symmetric clonic jerks (<i>Note the surface EMG channels with myogenic artifact during repetitive, bilateral, rhythmic muscle activation</i>)	Supplementary video 3 Supplementary figure 3
Myoclonic-atonic	Bilateral myoclonic jerks of shoulders → atonic component involving head drop	https://www.jle.com/en/revues/epd/e-docs/diaphragm_myoclonus_followed_by_generalised_atonia_in_a_patient_with_trisomy_4p_unusual_semiology_in_an_unusual_condition_305890/article.phtml?tab=videos&cle_video_une=306153 [411]
	Brief bilateral myoclonic jerks in shoulders → atonia and fall (<i>Note the surface EMG during myoclonic jerks and upon cessation</i>)	Supplementary video 4 Supplementary figure 4
Dystonic	Oroalimentary automatisms → bilateral leg proximal automatisms → hyperventilation → vocal automatisms + R arm dystonic → non-responsive + L arm gestural automatisms → L arm exploratory movements → L postictal nose wiping, coughing, and unresponsiveness (see <i>Postictal</i>)	https://www.jle.com/en/revues/epd/e-docs/autonomic_symptoms_during_epileptic_seizures_110095/article.phtml?tab=videos&cle_video_une=285222 [295]
	Ictal: L head version → L gestural automatisms → R gestural automatisms + R arm dystonic. Postictal: L nose wiping, R (Todd's) paresis, and non-fluent aphasia. (<i>Sequence 1, up to Section 5.05</i>) (<i>see also Distal Automatisms and Postictal</i>)	https://www.jle.com/en/revues/epd/e-docs/postictal_signs_of_lateralizing_and_localizing_significance_published_with_videosequences_110069/article.phtml?tab=videos&cle_video_une=285233 [374]
	Aura (presses the alarm button) → L arm automatisms → non responsive → R arm dystonia+ L head orientation → bilat arm and leg automatisms + oroalimentary automatisms → R arm akinesia → L nose wiping + responsive + postictal anomia	https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289024 [403]
	Head orientation L → R arm dystonia → non-responsive → ictal cry → R face tonic → R arm tonic extension → L arm tonic flexion (figure-of-four) → bilateral tonic → bilateral tonic-clonic → Asymmetric clonic termination L leg (<i>See Figure-of-4 & Asymmetric Ictal Termination</i>)	https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289022 [403]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
Gyratory	Non-responsive → R head version → R gyratory → ictal cry → bilateral tonic – clonic (lateralizing features of GTCS in IGE)	https://www.jle.com/en/revues/epd/e-docs/videoeeg_evidence_of_lateralized_clinical_features_in_primary_generalized_epilepsy_with_tonicclonic_seizures__261669/article.phtml?tab=videos&cle_video_une=285334 [412]
	Focal impaired awareness seizure: fulminant, hyperkinetic behavior → R gyratory (see <i>Hyperkinetic</i>)	Supplementary video 5
Epileptic nystagmus	pt. indicating aura (according to video description) → brief R face myoclonus → R epileptic nystagmus (fast phase)	https://www.jle.com/en/revues/epd/e-docs/epileptic_nystagmus_due_to_a_large_parieto_temporo_occipital_multilobar_dysplasia_318835/article.phtml?tab=videos&cle_video_une=318847 [413]
Ictal paresis	(Case 1) Several seizures with L ictal hemiparesis → tetraparesis. In two seizures accompanying eye blinking + L eye version OR seizures with accompanying unconjugated eye movements + oroalimentary automatism	https://www.jle.com/en/revues/epd/e-docs/alternating_hemiplegia_of_childhood_presentation_of_two_cases_regarding_the_extent_of_variability__262373/article.phtml?tab=videos&cle_video_une=285256 (Case 1: 0.28-2.28 sec) [414]
	Clusters of epileptic spasms	https://www.jle.com/en/revues/epd/e-docs/epileptic_spasms_in_clusters_without_hypsarrhythmia_in_infancy__260543/article.phtml?tab=videos&cle_video_une=285303 [415]
Epileptic spasm	Clusters of epileptic spasms – One seizure (section 1.26) starting with epileptic spasms → bilateral tonic → bilateral tonic with vibratory component → clonic → epileptic spasms	https://www.jle.com/en/revues/epd/e-docs/epileptic_spasms_in_clusters_without_hypsarrhythmia_in_infancy__260543/article.phtml?tab=videos&cle_video_une=285302 + tonic (descrip) [415]
	Clusters of epileptic spasms (Note the surface EMG channels at the bottom line and the “diamond shaped” myogenic artifact)	Supplementary video 6 Supplementary figure 5
Tonic	Bilateral symmetric tonic	https://www.jle.com/en/revues/epd/e-docs/bilateral_symmetric_tonic_posturing_suggesting_propagation_to_the_supplementary_motor_area_in_a_patient_with_precuneate_cortica_276426/article.phtml?tab=videos&cle_video_une=285442 [416]
	Recurrent, bilateral tonic (tonic status epilepticus)	https://www.jle.com/en/revues/epd/e-docs/tonic_status_epilepticus_in_a_centenarian_woman_313796/article.phtml?tab=videos&cle_video_une=313818 [417]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
Chapeau de gendarme	Ictal pouting (chapeau de gendarme) + partially responsive → postictal agitation + responsive	https://www.jle.com/en/revues/epd/e-docs/chapeau_de_gendarme_in_a_toddler_points_to_focal_epilepsy_originating_from_the_subcentral_gyrus_319960/article.phtml?tab=videos&cle_video_une=319980 [93]
a. Tonic-clonic (GTCS with lateralizing features in IGE)	Oroalimentary automatisms → L head version → L arm clonic jerks → bilateral tonic asymmetric + R arm clonic jerks → bilateral tonic clonic → asymmetric clonic ending with last jerks on the R → stridor	https://www.jle.com/en/revues/epd/e-docs/videoeeg_evidence_of_lateralized_clinical_features_in_primary_generalized_epilepsy_with_tonicclonic_seizures_261669/article.phtml?tab=videos&cle_video_une=285336 [412]
b. Tonic-clonic (GTCS in IGE)	Ictal cry → Bilateral tonic symmetric → Bilateral tonic-clonic symmetric <i>(Note the surface EMG channels showing the gradual “build up” of the muscle artifact during the tonic phase, followed by gradually longer “silent” periods corresponding to the clonic phase of a GTCS)</i>	Supplementary video 7 Supplementary figure 6A, B
Figure-of-four (FBTCS)	Aura → R head version → tonic → figure-of-four → bilat tonic-clonic → postictal unresponsiveness <i>Refer to dystonic and asymmetric clonic termination</i>	https://www.jle.com/en/revues/epd/e-docs/classification_of_paroxysmal_events_and_the_four_dimensional_epilepsy_classification_system_313798/article.phtml?tab=videos&cle_video_une=313811 [410] https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289022 [403]
Asymmetric clonic ending	<i>Refer to tonic-clonic (GTCS with lateralizing features in IGE)</i> <i>Refer to dystonic and figure-of-four</i>	https://www.jle.com/en/revues/epd/e-docs/videoeeg_evidence_of_lateralized_clinical_features_in_primary_generalized_epilepsy_with_tonicclonic_seizures_261669/article.phtml?tab=videos&cle_video_une=285336 [412] https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289022 [403]
Versive	Upward eye deviation → R eye and head version → R Tonic arm flexion → clonic jerks in R side of face and R arm	https://www.jle.com/en/revues/epd/e-docs/adversive_seizures_associated_with_periodic_lateralised_epileptiform_discharges_pleds_after_left_orbital_contusion_294991/article.phtml?tab=videos&cle_video_une=295286 [418]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
COMPLEX MOTOR PHENOMENA		
Automatisms		
Distal	Oroalimentary automatisms → L gestural automatisms → R head orientation → Bilateral gestural automatisms → L head orientation → oro-alimentary automatisms (See <i>Oroalimentary</i>)	https://www.jle.com/en/revues/epd/e-docs/seizure_ending_signs_in_patients_with_dyscognitive_focal_seizures_305235/article.phtml?tab=videos&cle_video_une=305311 [419]
	(Seizure from sleep) opening eyes → oroalimentary automatisms → R gestural automatisms → responsive → R postictal nose wiping (see <i>Postictal</i>)	https://www.jle.com/en/revues/epd/e-docs/seizure_ending_signs_in_patients_with_dyscognitive_focal_seizures_305235/article.phtml?tab=videos&cle_video_une=305312 [419]
	Ictal: L head version → L gestural automatisms → R gestural automatisms + R arm dystonic. Postictal: L nose wiping, R (Todd's) paresis, and non-fluent aphasia. (<i>Sequence 1, up to Section 5.05</i>) (see <i>Dystonic and Postictal</i>)	https://www.jle.com/en/revues/epd/e-docs/postictal_signs_of_lateralizing_and_localizing_significance_published_with_videosequences__110069/article.phtml?tab=videos&cle_video_une=285233 [374]
Genital	Fearful facial expression + staring → L head version → tonic posture L arm → Bilateral tonic posture arms → tonic-clonic L face → R arm tonic elevation → Bilateral gestural automatisms → nonresponsive → R hand genital automatisms → ictal coughing → Bilateral proximal leg automatisms (i.e., pedaling/bicycling)	https://www.jle.com/en/revues/epd/e-docs/autonomic_symptoms_during_epileptic_seizures_110095/article.phtml?tab=videos&cle_video_une=285227 [295]
Proximal	R arm proximal automatisms	https://www.jle.com/en/revues/epd/e-docs/lgi_1_antibody_encephalitis_in_a_seven_year_old_girl_315902/article.phtml?tab=videos&cle_video_une=315925 [420]
Mimetic-dacrystic	Dacrystic automatisms → oroalimentary automatisms	https://www.jle.com/en/revues/epd/e-docs/giant_hypothalamic_hamartoma_and_dacrystic_seizures_273133/article.phtml?tab=videos&cle_video_une=285409 [421]
Mimetic- gelastic	Staring → gelastic automatisms → Bilateral arm proximal automatisms	https://www.jle.com/en/revues/epd/e-docs/gelastic_seizures_in_ring_chromosome_20_syndrome_a_case_report_with_video_illustration_293185/article.phtml?tab=videos&cle_video_une=294858 [422]
	L arm gestural self-directed automatisms → ictal vocalization → gelastic automatisms+ nonresponsive → distal Bilateral feet automatisms → L postictal nose wiping	https://www.jle.com/en/revues/epd/e-docs/hypothalamic_hamartoma_and_epilepsy_in_children_illustrative_cases_of_possible_evolution_262012/article.phtml?tab=videos&cle_video_une=285374 [423]
Oroalimentary	Oroalimentary automatisms → L gestural automatisms → head orientation R → Bilateral gestural automatisms → head orientation L → oro-alimentary automatisms (see <i>Distal Automatisms</i>)	https://www.jle.com/en/revues/epd/e-docs/seizure_ending_signs_in_patients_with_dyscognitive_focal_seizures_305235/article.phtml?tab=videos&cle_video_une=305311 [419]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
Verbal	S1: verbal automatisms (repeating religious passages) → ictal kissing → L gestural automatisms → orolimentary automatisms (see <i>Mystic</i>)	https://www.jle.com/en/revues/epd/e-docs/ictal_kissing_and_religious_speech_in_a_patient_with_right_temporal_lobe_epilepsy__265692/article.phtml?tab=videos&cle_video_une=285267 [424]
Vocal	S1: R gestural + orolimentary automatisms → verbal automatism + affective (anger) → vocal automatisms (see <i>Affective</i>)	https://www.jle.com/en/revues/epd/e-docs/seizure_semiology_of_anti_lgi1_antibody_encephalitis_310814/article.phtml?tab=videos&cle_video_une=311024 [425]
Hyperkinetic behaviours		
	Fearful facial expression → hyperkinetic → vocal automatisms → non-responsive + gestural + orolimentary automatisms	https://www.jle.com/en/revues/epd/e-docs/seizure_semiology_reflects_spread_from_frontal_to_temporal_lobe_evolution_of_hyperkinetic_to_automotor_seizures_as_documented_by_invasive_eeg_video_recordings_297991/article.phtml?tab=videos&cle_video_une=298011 [426]
Hyperkinetic Seizure	Focal impaired awareness seizure: fulminant, hyperkinetic behavior → R gyratory	Supplementary video 5
	Focal impaired awareness seizure: ictal fear → hyperkinetic behavior (See <i>Affective</i>)	Supplementary video 8 Supplementary figure 7
	Hyperventilation → L gestural automatisms → hyperkinetic → ictal vocalizations → R head version → tonic (see <i>Hyperventilation</i>)	https://www.jle.com/en/revues/epd/e-docs/autonomic_symptoms_during_epileptic_seizures_110095/article.phtml?tab=videos&cle_video_une=285223 [295]
SENSORY PHENOMENA		
Auditory	Ictal fear + auditory hallucinations (Note: it is not possible to evaluate hearing in this video, but the patient is heard verbalizing "they are coming" due to auditory hallucinations of someone approaching from her left)	https://www.jle.com/en/revues/epd/e-docs/auditory_hallucinations_as_ictal_phenomena_in_a_patient_with_voltage_gated_potassium_channel_antibody_associated_limbic_encephalitis_298909/article.phtml?tab=videos&cle_video_une=299012 [427]
Visual	Several seizures starting as focal aware seizures with L blurred vision, flashes, and visual hallucinations, (as described by the patient) → R eye and head version → eye blinking → non-responsive (in some of the seizures)	https://www.jle.com/en/revues/epd/e-docs/benign_idiopathic_occipital_epilepsy_report_of_a_case_of_the_early_benign_type_published_with_video_sequences__110057/article.phtml?tab=texte [428]
	Focal aware NCSE: oculomotor apraxia → ataxia → simultanagnosia	https://www.jle.com/en/revues/epd/e-docs/blint_like_syndrome_as_an_unusual_representation_of_non_convulsive_status_epilepticus_292166/article.phtml?tab=videos [429]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
	L trunk painful sensation	https://www.jle.com/en/revues/epd/e-docs/focal_epilepsy_with_paroxyssmal_pain_due_to_somatic_injury_309906/article.phtml?tab=videos&cle_video_une=309985 [430]
Somatosensory	Focal aware seizure: brief abnormal sensation R side of neck & R arm → R arm clonic → R neck tonic → R abdominal clonic	https://www.jle.com/en/revues/epd/e-docs/unilateral_abdominal_clonic_seizures_of_parietal_lobe_origin_eeg_findings_311765/article.phtml?tab=videos&cle_video_une=311824 [431]
AFFECTIVE (EMOTIONAL) PHENOMENA		
Anger	S1: R gestural + oroalimentary automatisms → ictal speech + affective (anger) → ictal vocalization. S2: L face grimacing → non-fluent aphasia (anomia) S3: R gestural automatisms → affective S4: R gestural automatisms → ictal speech + affective (anger)	https://www.jle.com/en/revues/epd/e-docs/seizure_semiology_of_anti_lgi1_antibody_encephalitis_310814/article.phtml?tab=videos&cle_video_une=311024 [425]
Anxiety	Anxiety + hyperventilation → L somatosensory aura → vestibular symptoms → dysarthria → ictal cry	https://www.jle.com/en/revues/epd/e-docs/long-lasting_seizure_related_anxiety_in_patients_with_temporal_lobe_epilepsy_and_comorbid_psychiatric_disorders_305196/article.phtml?tab=videos&cle_video_une=305316 [432]
Fear	Ictal fear + ictal vocalization → L head version + dystonic posture → L hand automatisms + non-responsive Focal impaired awareness seizure: ictal fear → hyperkinetic behavior (See <i>Hyperkinetic</i>)	https://www.jle.com/en/revues/epd/e-docs/semiological_study_of_ictal_affective_behaviour_in_epilepsy_and_mental_retardation_limited_to_females_efmr_293200/article.phtml?tab=videos&cle_video_une=295278 [433] Supplementary video 8 Supplementary figure 7
Mystic	S1: verbal automatisms (repeating religious statement) → ictal kissing → L gestural automatisms → oroalimentary automatisms S2: hyperventilation → Bilateral gestural automatisms → verbal automatisms (repeating religious passages) → musical automatism (whistling) + Bilateral gestural automatisms → ictal kissing → oroalimentary automatisms → L gestural automatisms → oroalimentary → R gestural automatisms → non-responsive	https://www.jle.com/en/revues/epd/e-docs/ictal_kissing_and_religious_speech_in_a_patient_with_right_temporal_lobe_epilepsy_265692/article.phtml?tab=videos&cle_video_une=285267 [424]
COGNITIVE PHENOMENA		
Aphasia	Focal impaired awareness seizure: R gestural automatisms → ictal aphasia Epigastric aura (presses the alarm button) → responsive (follows command to raise R arm) → ictal aphasia/anomia (unable to name objects)+ R hand ictal dystonia	Supplementary video 9 Supplementary figure 8 https://www.jle.com/en/revues/epd/e-docs/video_atlas_of_lateralising_and_localising_seizure_phenomena_288739/article.phtml?tab=videos&cle_video_une=289023 [403]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
	L face grimacing → non-fluent aphasia (anomia) (see <i>Affective</i>)	(seizure 2: 1.02-1.25 sec) https://www.jle.com/en/revues/epd/e-docs/seizure_semiology_of_anti_lgi1_antibody_encephalitis_310814/article.phtml?tab=videos&cle_video_une=311024 [425]
AUTONOMIC PHENOMENA		
Cardiovascular		
Tachycardia	Ictal apnea → tachycardia → ictal coughing (Note the respiratory and ECG signal and video description) (see <i>Hypoventilation</i>)	https://www.jle.com/en/revues/epd/e-docs/intractable_apnoeic_seizures_in_a_child_with_a_deletion_typically_associated_with_williams_syndrome_313319/article.phtml?tab=videos&cle_video_une=313364 [434]
Ictal bradycardia and asystole	Ictal asystole → syncope (Note interruption of the ECG signal)	https://www.jle.com/en/revues/epd/e-docs/ictal_asystole_with_intercurrent_cardiopathy_a_complex_combination_leading_to_delayed_diagnosis_315811/article.phtml?tab=videos&cle_video_une=315867 [435]
	Bradycardia → ictal asystole → syncope (Note interruption of the ECG signal)	https://www.jle.com/en/revues/epd/e-docs/syncope_due_to_ictal_asystole_a_challenging_semiology_318836/article.phtml?tab=videos&cle_video_une=318848 [436]
Cutaneous		
Piloerection	Ictal piloerection	https://www.jle.com/en/revues/epd/e-docs/autonomic_symptoms_during_epileptic_seizures_110095/article.phtml?tab=videos&cle_video_une=285229 [295]
Gastrointestinal		
Ictal spitting	Focal aware seizure: aura (feeling that she wants to spit) → ictal spitting	https://www.jle.com/en/revues/epd/e-docs/ictal_spitting_in_non_dominant_temporal_lobe_epilepsy_an_anatomo_electrophysiological_correlation_311737/article.phtml?tab=videos&cle_video_une=311820 [437]
Ictal vomiting	Ictal vomiting (Panayiotopoulos syndrome): Brief tonic posture of legs → non-responsiveness + eye deviation upwards → eye version L → ictal vomiting (see <i>Section 3.09</i>)	https://www.jle.com/en/revues/epd/e-docs/panayiotopoulos_syndrome_videoeeg_illustration_of_a_typical_seizure_263027/article.phtml?tab=videos&cle_video_une=285259 [438]
	Focal aware seizure: aura (patient pressing the alarm button) ictal retching → ictal vomiting → R arm automatisms → slight dysarthria	https://www.jle.com/en/revues/epd/e-docs/autonomic_symptoms_during_epileptic_seizures_110095/article.phtml?tab=videos&cle_video_une=285225 [295]

▼ **Table 2.** Seizure semiology: videos (*continued*).

Semiology	Seizure onset and propagation	Video link
Respiratory Dysfunction		
Hyperventilation	Hyperventilation → L gestural automatisms → hyperkinetic → ictal vocalizations → R head version → tonic (see <i>Hyperkinetic</i>)	https://www.jle.com/en/revues/epd/e-docs/autonomic_symptoms_during_epileptic_seizures_110095/article.phtml?tab=videos&cle_video_une=285223 [295]
Hypoventilation	Apnea → tachycardia → ictal coughing (Note the respiratory and ECG signals; see <i>Tachycardia</i>)	https://www.jle.com/en/revues/epd/e-docs/intractable_apnoeic_seizures_in_a_child_with_a_deletion_typically_associated_with_williams_syndrome_313319/article.phtml?tab=videos&cle_video_une=313364 [434]
POSTICTAL PHENOMENA		
Postictal nose wiping	(Seizure from sleep) opening eyes → oroalimentary automatisms → R gestural automatisms → responsive → R postictal nose wiping (see <i>Distal Automatisms</i>)	https://www.jle.com/en/revues/epd/e-docs/seizure_ending_signs_in_patients_with_dyscognitive_focal_seizures_305235/article.phtml?tab=videos&cle_video_une=305312 [419]
Postictal paresis and aphasia	Ictal: L head version → L gestural automatisms → R gestural automatisms + R arm dystonic. Postictal: L nose wiping, R (Todd's) paresis, and non-fluent aphasia. (Sequence 1, up to Section 5.05) (see also under <i>Dystonic and Distal Automatisms</i>)	https://www.jle.com/en/revues/epd/e-docs/postictal_signs_of_lateralizing_and_localizing_significance_published_with_videosequences_-_110069/article.phtml?tab=videos&cle_video_une=285233 [374]
Postictal coughing, nose wiping and unresponsiveness	Oroalimentary automatisms → Bilateral leg proximal automatisms → hyperventilation → vocal automatisms +R arm dystonic → nonresponsive L arm gestural automatisms → L arm exploratory movements → Postictal: L nose wiping + coughing, + unresponsiveness	https://www.jle.com/en/revues/epd/e-docs/autonomic_symptoms_during_epileptic_seizures_110095/article.phtml?tab=videos&cle_video_une=285222 [295]

standardized behavioral testing of the patients in epilepsy monitoring units [18]. An appropriate response is a composite end-result of functional activities in complex neurological networks, which might include proper sensory perception and processing along with prompt motor execution. These responses might be context and culture specific, affected by the intellectual and emotional level of the patient [19, 24].

Non-responsive implies lack of response to repeated questions commands or other external stimuli. Spinal reflex withdrawal in response to a noxious stimulus without an appropriate emotional response might still occur in this non-responsive phase [14, 19, 23]. *Partially responsive* implies inconsistent,

inappropriate responses and/or an unusually prolonged reaction time.

Responsiveness is lost in generalized tonic-clonic seizures (GTCS), but not necessarily during generalized myoclonic seizures. In most absence seizures, patients are non-responsive, but the responsiveness might be partial [21, 22, 40]. In focal seizures, partial or complete absence of responsiveness might be seen. Verbal responses might be more affected in focal epilepsies with dominant hemispheric involvement, while partial responsiveness during automatisms suggests involvement of the non-dominant hemisphere [20, 50, 51].

Motor or behavioral arrest is related but not identical to impaired responsiveness. Behavioral arrest is

common in absence seizures and in focal seizures with impaired awareness or impaired responsiveness. However, behavioral arrest may be observed based on interruption of ongoing motor activity even if responsiveness to external stimuli is not assessed.

3. Elementary motor signs

Elementary or simple motor signs involve the skeletal musculature during seizures. They consist of an increase (positive) or decrease (negative) in contraction of a muscle or group of muscles that is unnatural, stereotyped and not decomposable into phases. There can be synchronous (or asynchronous) involvement of motor signs. They can be isolated events or occur in a repetitive fashion. When they do occur, they do so at the same rate of contraction, and in the various muscle groups in the body. The commonly used term tonic-clonic is an elementary sequence of motor signs.

3.1. Akinetic

Akinetic motor signs are those characterized by the inability to perform voluntary movements with preserved postural tone and awareness. It is a rare ictal phenomenon and must be distinguished from motor and behavioral arrest with staring, impaired awareness, and motor arrest. Akinetic motor signs are without strong localizing or lateralizing value. Akinetic motor signs are often associated with dystonic phenomena (see 3.8), but it can also evolve to include progressive loss of muscle tone (see 3.3). Muscle tone may be lost, but akinesia dominates the semiology. The same phenomenon can be observed for speech production (*aphemia* or speech inhibition). Ictal akinesia can manifest by an abrupt freezing of one's gait initiation and may be precipitated by startle [52]. Akinetic motor signs and *aphemia* must be distinguished from ictal paresis and aphasia respectively (see 3.11 and 7.2). *Aphemia* is characterized by the inability of articulation, including other movements of the oro-laryngofacial muscles (i.e., inability to purse the lips, stick out the tongue open the mouth or swallow voluntary in the presence of preserved comprehension). Akinetic signs are likely to occur during seizures that involve "negative motor" areas (i.e., mesial premotor cortex and inferior frontal gyrus) [53-55], and can be produced by direct electrical stimulation of the same regions [55, 56].

3.2. Astatic

Astatic seizures (a.k.a. "epileptic drop attack") refer to a sudden loss of maintaining an erect posture. This

leads to falling without specificity for identifying the underlying mechanism (i.e., from atonic, myoclonic, or tonic seizures) which is most often ill-defined in the absence of polygraphic video-EEG monitoring that involves recording surface electromyography.

3.3. Atonic

Atonic refers to a sudden loss or decrease in muscle tone involving the head, trunk, jaw, and limbs. Atonic seizures typically last 1-2 seconds and can result in a loss of postural tone, without tonic, clonic, or myoclonic manifestations. Consequences of atonic seizures frequently involve falls and injury. Atonic seizures occur in both generalized and focal epilepsies and are a cardinal semiology in Lennox Gastaut syndrome. Their occurrence in focal seizures suggest motor and premotor cortex involvement [57].

• 3.3.1. Negative myoclonus

Negative myoclonus is used to describe a brief interruption of muscle tone (<500 msec), like atonic seizures. This results in a sudden brief lapse in movement that may grossly appear like a "positive" myoclonic jerk which is manifest as a single lightning-like jerk (see 3.5). However, the pathogenesis is different [58]. It can occur in a heterogeneous group of epilepsy syndromes disorders, ranging from self-limited syndromes, atypical self-limited childhood epilepsy with centrotemporal spikes and electrographic status epilepticus in slow wave sleep, to focal epilepsies involving lesions of the mesial frontal structures (as in neuronal migration disorders), and severe static or progressive myoclonic developmental and epileptic encephalopathies.

3.4. Eye blinking

Ictal eye blinking consists of a series of repetitive tonic contraction of the eyelids during a seizure. This occurs without any other facial muscle contraction. Its pathogenesis suggests activation of the trigeminal nerve fibers associated with the blink reflex [59]. Ictal eye blinking must be separated from repetitive facial clonic jerks that are observed during motor seizures, as well as from eyelid myoclonia (often associated with a sustained upward gaze – see next sub-section). The localizing significance of eye blinking remains uncertain – though it seems over-represented in patients with posterior quadrant epilepsies [60]. Its lateralizing value, when unilateral, suggests an ipsilateral epileptogenic hemisphere [61]. Unilateral ictal eye blinking is believed to result from cortical blink inhibition of the contralateral eye, due to seizures in the frontotemporal region [61].

3.5. Myoclonic / 3.6. Clonic

Myoclonic jerks (a.k.a. *myoclonus*) are a sudden, brief (<100-msec) involuntary, single or multiple contraction(s) of muscle(s) or muscle groups of variable topography. The term *clonic* (synonym rhythmic myoclonic) should be preferred to account for myoclonic jerks that are regular and repetitive, at a low frequency that is typically 0.2-5 Hz. They involve the same muscle groups and vary in duration but are commonly prolonged. These elementary motor phenomena can occur both in generalized and in focal seizures. In generalized seizures, they are usually bilateral, but not always synchronous and symmetric. The traditional term “Jacksonian march” indicates spread of unilateral focal clonic movements through contiguous areas on the body. This approximates seizure propagation within the motor homunculus of the brain. In focal motor seizures, unilateral clonic manifestations have a high lateralizing value to the contralateral hemisphere (approximately 90%) [62-64] and strongly reflect involvement of the motor cortex. *Eyelid myoclonia* refers to rapid (3.5-6-Hz) eye blinking, flickering, trembling, fluttering, twitching, or jerking. It is often associated with sustained upward gaze deviation, with and without absences, in patients with Jeavons syndrome [65]. The characteristic EEG correlate is generalized polyspike-wave or generalized spike-wave discharges at 3 to 6 Hz. These seizures are typically seen after eye closure (eye closure sensitivity) and the patients are photosensitive. It should be distinguished from the repetitive, 3-4-Hz eye blink seen during absence seizures in patients with idiopathic / genetic generalized epilepsy. fMRI studies suggested that eyelid myoclonia with absences involve a circuit encompassing the occipital cortex and the cortical-subcortical systems physiologically involved in the motor control of eye closure and eye movements [66].

3.7. Myoclonic-atonic

The term myoclonic-atonic, often replaced by the more imprecise term myoclonic-astatic, refers to a type of generalized seizure associated with a myoclonic jerk followed by an atonic motor component [5]. This seizure type is the hallmark semiology in patients with Doose syndrome [67].

3.8. Dystonic

Dystonia associated with a seizure (ictal dystonia) consists of sustained contractions involving both agonist and antagonist muscles. This produces athetoid or twisting movements which, when prolonged, may produce unnatural postures of an arm or

leg. The dystonic posture can manifest as flexion or extension and be proximal or distal, frequently with a rotatory component. In the context of patients with temporal lobe epilepsy, unilateral ictal dystonia of the upper limb almost always lateralizes to the hemisphere contralateral to the seizure onset zone [68-70]. This sign, however, may have a weak interrater reliability when temporal and extratemporal lobe epilepsies are mixed [64] and may be related to ictal propagation and involvement of the basal ganglia following seizure onset [71].

3.9. Gyrotory

Gyrotory seizures (a.k.a. rotatory, circling, volvular) refer to seizures where the patient rotates around their long body axis. This involves at least 180 degrees and occasionally produces one or even more than one turn involving 360 degrees. This phenomenon may be initiated by a versive head movement in the same direction (see 7.13) and may culminate in a focal-to-bilateral tonic-clonic (FBTC) seizure. Gyrotory manifestations have been reported during focal seizures and rarely in patients with generalized epilepsies [72]. During focal seizures, the direction of the rotation lateralizes the seizure onset zone to the contralateral hemisphere when head version initiates the gyration [73]. In gyrotory seizures without a preceding forced head version, the direction of rotation may be ipsilateral or contralateral to the side of seizure onset [73]. The mechanism remains unknown, though basal ganglia involvement [74] and interhemispheric imbalance of cortical excitability [75] have been suggested.

3.10. Epileptic nystagmus

Epileptic nystagmus, sometimes described as oculo-clonic jerks, is a rapid, repetitive movement of the eyeballs probably caused by the epileptiform activity involved in activation of the cortical region governing saccadic or pursuit eye movements. It is binocular in most cases and may be preceded or accompanied by simple visual hallucinations. It is often associated with rotation of the head and eyes that is often in the same direction as the fast phase of nystagmoid eye movement which is most prominent [76]. Epileptic nystagmus is mainly seen during seizures of occipital lobe origin, and the fast phase is most often contralateral to the site of seizure onset [77].

3.11. Ictal paresis

Ictal paresis is a rare phenomenon. It is characterized by weakness or paralysis of a part of one's body or hemibody [78-80]. Ictal paresis must be differentiated from ictal akinesia (see 7.1), ictal immobile limb in

patients with temporal lobe epilepsy [81], and postictal Todd's paresis (see 9.2). A proper diagnosis therefore necessitates video-EEG to test the patient [18] and confirm the ictal origin of the motor deficit. Ictal paresis is almost always contralateral to the seizure onset zone and is likely to be produced by seizures that occur in close vicinity to the primary motor cortex [82].

3.12. Epileptic spasm

An epileptic spasm describes a seizure comprised of sudden flexion, extension, or mixed extension-flexion. Spasms involve predominantly the proximal head, appendicular and truncal muscles and are more sustained than brief clonic and myoclonic movements, but not as sustained as tonic posturing which may last 0.5-2 seconds. Typically, spasms consist of a series of episodes with abduction and extension of both arms. Subtle forms of spasms, with minimal / discrete manifestations may occur, including head nodding, grimacing, smiling, or chin movement. Epileptic spasms usually occur in clusters, often upon awakening [83]. They have been classified as generalized seizures, focal seizures and unclassified seizures in the current ILAE seizure classification [5]. When unilateral or asymmetric, they point towards a lateralized onset in the contralateral hemisphere but do not have localizing value [84].

3.13. Tonic

A tonic seizure consists of a sudden posture involving increased tone resulting in stiffness or tense posture due to sustained muscular contraction that usually causes an extension. Seizures with tonic features may also affect the flexor muscles. Each event ranges from a few seconds to minutes in duration. Tonic contraction may involve one or more limbs, the body axis and face. When tonic seizures are bilateral, they may be symmetric or asymmetric. They can occur in focal and generalized seizures. Tonic seizures can be precipitated by startle, in which case the tonic contraction is bilateral. They may be symmetrical or asymmetrical and frequently lead to falls [85]. Unilateral tonic seizures lateralize to the contralateral hemisphere in approximately 90% of patients with focal epilepsy. However, its localizing value is poor [62-64, 86]. Direct electrical cortical stimulation has shown that contralateral tonic contractions can be elicited by stimulating the mesial premotor and primary motor cortices [87].

• 3.13.1. *Chapeau de gendarme*

In the original description, the "*chapeau de gendarme*" sign (a.k.a. ictal pouting) consists of a

"symmetrical and sustained (>3-sec) lowering of labial commissures with contraction of the chin, mimicking an expression of fear, disgust, or menace" [88]. This produces a facial expression that is recognized by a turned down mouth that appears as an inverted smile. Seizures involving this semiology have been termed *chapeau de gendarme* due to the shape of the hat used by French police (gendarme) during the reign of Napoleon. When it occurs early during seizures, it has a strong localizing value to the frontal lobe [89], especially the anterior prefrontal and anterior cingulate cortices [88, 90]. However, other localizations have also been described [91-93].

• 3.13.2. *Fencing posture*

The fencing posture (a.k.a. fencer's posture) is a motor sign with tonic extension at the elbow and elevation of one arm and strongly lateralizes to the contralateral hemisphere. In addition, there is associated ipsilateral flexion of the elbow in the ipsilateral upper limb and elevation at the shoulder to approximate the posture held during fencing. This typically occurs late in the course of a seizure. A similar unilateral posture, M2e, is a similar semiology beginning with flexion of the elbow to about 90 degrees and followed by an abduction of the shoulder to approximately 90 degrees, associated with external rotation with or without head turning towards the affected arm [94]. The hand may be clenched or open, and the head is typically deviated towards the hand. The initial description emphasized that a M2e motor posture should be used as a descriptor only if the contralateral arm is initially uninvolved or becomes involved in tonic activity later during the seizure. This sign has a strong lateralizing value and points to the contralateral hemisphere, with an excellent interrater reliability [64]. It is classically ascribed to the mesial frontal lobe, and more specifically to the supplementary motor area.

• 3.14. *Tonic-clonic*

This is an important ictal motor sequence of events consisting of a tonic phase followed by a clonic phase. In GTCS, it may be preceded by sporadic or irregular myoclonic jerks (myoclonic-tonic-clonic), by an initial clonic phase (clonic-tonic-clonic) or by an absence seizure (absence-to-bilateral-tonic-clonic seizure) [95]. In focal seizures, the bilateral tonic-clonic phases are typically preceded by other focal semiologies (FBTC). However, when they occur at night and are unwitnessed or before its treatment, it may be impossible to identify the presence of focal signs or symptoms. Two types of FBTC have been proposed based on their semiology, suspected pathophysiology, and potential impact on the risk of SUDEP [96]. GTCS type 1 at some point displays the classic

sequence of bilateral and symmetrical tonic posturing with arms extended in supination, as in decerebrate posturing (suggesting involvement of the ictal discharge in the brainstem), followed by bilateral clonic jerking with gradually decreasing frequency, which is generated by progressively longer silent periods interrupting sustained tonic activity from inhibition of muscle activation waning until the seizure stops [97]. GTCS type 2 involves a tonic and clonic sequence that is often superimposed. When the two phases are sequential, the clonic phase has a rhythmic quality that is more sustained, without the exponential dynamic changes observed in GTCS type 1 [97]. The motor features in GTCS type 2 suggest primary involvement of the pre-motor and/or motor cortex, rather than propagation of the ictal discharge to the subcortical and brainstem structures. This is further denoted by awareness that is partially retained in rare patients with GTCS type 2. Furthermore, the same clinical sequence described in patients with GTCS type 2 can be observed unilaterally and asymmetrically typically pointing to a contralateral seizure onset zone. Finally, a GTC type 2 can evolve into a GTCS type 1, in which case the latter is described as the main semiology of the seizure. GTCS type 1 leads to postictal EEG suppression more frequently than GTCS type 2 [96]. This suggests that patients with GTC type 1 might be associated with a higher risk of SUDEP.

• 3.14.1. *Figure-of-4 sign*

The figure-of-4 sign may occur at the onset of the tonic phase prior to the clonic phase in patients with FBTC seizures. This semiology consists of unilateral tonic extension of one arm and tonic flexion of the contralateral arm. The latter may be held in front or behind the extended arm. This sign is considered to have a good lateralizing significance [64, 98] with the extended arm occurring contralateral to the hemisphere involved in seizure onset. The sequence of motor signs is important and can be complex, with tonic extension of one limb first, followed by the figure-of-4 sign pointing to the other hemisphere. In this situation, it is the first manifestation of arm extension that prevails to lateralize the seizure onset zone.

• 3.14.2. *Asymmetric clonic ending*

This sign refers to the terminal expression involving clonic jerks following a focal-to-bilateral-tonic seizure. When convulsive seizures terminate asymmetrically with unilateral clonic jerks that persist on one side of the body, this has good lateralizing significance for seizure onset. The last unilateral clonic activity that persists occurs ipsilateral to the hemisphere of seizure onset, regardless of lobar localization including temporal or frontal origin [64, 99-101].

It is important to differentiate asymmetric clonic ending from asymmetric amplitude of the jerks between the two sides of the body, as this alone should not be considered as asymmetric clonic ending.

3.15. *Versive*

Version must be used only to describe an unnatural, forced and sustained movement of the eyes, head, trunk or whole body to one side. Small lateral saccadic and clonic features can be superimposed during version. During head-turning, the chin may move laterally but also upward or downward, depending on the muscles involved. Occasionally, seizures with version can lead the patient to rotate around the long body axis. In this case, the term *gyratory* is applied (see 3.9). Ictal version does not refer to a gaze preference or to other situations where the patient's eyes, head or trunk deviate to one side (see 3.16). *Ictal body turning* is a term that encompasses different semiological signs. Ictal version can be seen with focal seizures arising from any location. Their lateralizing significance varies depending on the type of version and whether it involves the eyes, head, or body, and whether they are conjugate, sequential, and accompanying signs that occur [102]. Isolated version involving the eyes likely involves the contralateral frontal eye fields. Direct electrocortical stimulation has supported this localization by reproducing the same clinical signs [103]. However, when initial visual illusions / hallucinations are congruent with ocular version, this suggests seizures arise from a contralateral occipital lobe origin. Head version strongly lateralizes to the contralateral hemisphere when it is associated with neck extension and occurs in the first 10 seconds of a FBTC seizure [64, 104, 105]. In the situation where two successive head versions occur prior to a FBTC seizure, it is the movement immediately preceding the tonic-clonic phase that prevails as the lateralizing sign.

3.16. *Head orientation*

Non-versive turning of the head and eyes is a frequent, but not specific sign when determining lateralization and localization. When it occurs early in temporal lobe seizures, it is often ipsilateral to the focus [106].

4. *Complex motor behaviors*

Complex motor behaviors refer to a heterogeneous group of motor signs and sequences. While some may look voluntary, the patient has no capacity to control

them during the seizure and they are inappropriate for the situation. Gestures involving complex motor behavior may or may not resemble natural movements. They can be mixed with elementary motor signs and may be associated with facial expressions and verbalization or vocalization congruent with the motor pattern [107]. The pathophysiology of complex motor behaviors is unclear and possibly depends on the type of motor involvement. Features that occur during seizures with complex motor behaviors could be due to a release phenomenon of stereotyped inborn networks with fixed action patterns (central pattern generators) [108] or due to transient “desynchrony” in emotion-regulation networks [109], rather than to a specific ictal involvement of an individual area in the brain.

4.1. Automatism

The term automatism is used to account for an irrepressible, discrete or excessive and repetitive single motor activity that often resembles a voluntary behavior. They may appear to occur with a semi-purposeful act such as disrobing and manipulating objects or without the appearance of a purposeful behavior such as body rocking. Depending on the clinical situation at seizure onset (i.e., eating, writing, waving etc.), automatisms may consist of an inappropriate continuation of preictal activity. The speed and amplitude of the movements are most often in the context of normal movement but they may also appear to be exaggerated (as in hyperkinetic behaviors; see 4.2), and awareness is usually, but not necessarily, impaired. Patients seldom retain awareness when exhibiting automatisms yet they will typically be unaware of them.

• 4.1.1. Gestural automatisms

Gestural automatisms are rhythmic repetitive movements of the hands and occasionally also of the feet, as distal automatisms. They may also involve the limbs and body axis involving proximal automatisms. Gestural automatisms may be unilateral or bilateral when they occur (see 4.1.2-4.1.5). They need to be distinguished from rhythmic ictal non-clonic hand motions (RINCH), which are usually contralateral to the focus [107, 110]. RINCH are unilateral, rhythmic, non-clonic, non-tremor hand movements (low-amplitude milking, grasping, fist clenching, or pill rolling, or larger-amplitude hand opening-closing movements), usually followed by dystonic posturing, sometimes with overlap.

• 4.1.2 Distal automatisms

Distal automatisms (e.g., tapping, crumbling, fumbling, grasping, finger snapping, exploratory movements,

manipulating movements) can be self-directed (virtually independent of environmental influence) or involve more than self (environmentally influenced). In the context of temporal lobe epilepsy, *unilateral manual automatisms* lateralize to the ipsilateral hemisphere mainly when associated with a contralateral hand dystonia [111].

• 4.1.3. Genital automatisms

Gestural automatisms are directed to involve the genital region. These complex motor behaviors may appear with a sexual appearance including fondling, grabbing, scratching among other behaviors. They are often unilateral and associated with manual automatisms. They are frequently ipsilateral to the hemisphere involving the seizure onset zone [112].

• 4.1.4. Ictal grasping

Ictal grasping is defined as a uni- or bi-manual forced prehensile movement often preceded by reaching. It is either directed towards the patient's body or surrounding one's personal space. It rarely occurs as the main clinical component of the seizure [113] and is frequently part of hyperkinetic behaviors (see 4.2) [114]. It may be reproduced by direct electrocortical stimulation of the anterior cingulate cortex [115]. Ictal grasping is often contralateral, but it is without consistent lateralizing value.

• 4.1.5. Proximal automatisms

Proximal automatisms are described when rhythmic repetitive movements of the proximal extremities occur (e.g., pedaling, kicking, wing flapping, body rolling, rocking, crawling, and copulatory movements). These often result in highly visible movements with high amplitudes and fast speeds. They co-exist as part of hyperkinetic behavior (see 4.2) and their localizing significance is unclear, though often are associated with seizure onset in the frontal lobe involving the anterior cingulate or orbito-frontal cortex, though may also be seen in insular, temporal and parietal lobe seizures.

• 4.1.6. Mimic automatisms

Mimic automatisms refer to a stereotyped mimicry or behavior that resembles the usual way one expresses oneself to reflect an affect that is not accompanied by the corresponding emotion. The most frequent types of mimetic automatisms are laughing (gelastic) [116-118] and crying (dacrystic, quiritarian) [119, 120] behaviors which, when isolated and occurring in clusters, should make one consider the possibility of hypothalamic localization (i.e., hamartoma). Other localizations also exist, mainly involving the frontal and temporal lobes. Laughter can be elicited by direct electrocortical stimulation of the anterior cingulate gyrus, which may be first asymmetric (contralateral to

the stimulation) and then bilateral [121, 122]. A tendency for ictal laugh, though without overt laughter (hence not qualifying as a gelastic seizure), has been described in patients with small hypothalamic hamartomas [123].

Other mimic automatisms are rarely identified but include the expression of negative (e.g., biting, fear, sadness) [124, 125] and positive (i.e., kissing) emotional valences [126], or musical automatisms (e.g., humming and whistling) [127-129].

- **4.1.7. Oroalimentary automatisms**

Oro-alimentary automatisms including chewing, lip smacking, lip pursuing, licking, and swallowing resemble normal routine behaviors and are frequently encountered in patients with temporal lobe seizures, especially those arising from the mesial structures. Recent findings also suggest that insulo-percular involvement is needed to facilitate their occurrence [130, 131]. The appearance of preserved awareness during such automatisms is more likely to occur during temporal lobe seizures that arise from the non-dominant hemisphere for language [132].

- **4.1.8. Verbal automatisms**

Verbal automatisms reflect the iterative and stereotyped production of intelligible words or sentences and must be distinguished from ictal speech, which is the ability of patients to converse during a seizure. The term ictal speech has incorrectly been used as a substitute term for verbal automatisms generating confusion. Verbal automatisms are frequently seen in patients with temporal lobe seizure though lateralization is inconsistent. Foreign ictal speech automatisms are a rare form of verbal automatisms and are more likely to occur in patients with seizures arising from the non-dominant amygdala [133].

- **4.1.9. Vocal automatisms**

Vocal automatisms, or vocalization, refers to single or repetitive sounds (e.g., grunts, shrieks, moaning etc.) that do not have the linguistic qualities of language like verbal automatisms (see 4.1.8) and are unaccompanied by apnea, tonic, clonic, or tonic-clonic motor manifestations [134]. Ictal vocalization by itself has no localizing significance but it occurs more frequently in dominant (left-sided) temporal [135] and frontal [136] lobe seizures.

4.2. Hyperkinetic behaviors

Hyperkinetic or hypermotor seizures refer to the quantitative aspect of motor involvement. Hyperkinetic behavior shows an excessive amount and speed of motor movement. There is an increased rate and acceleration of ictal motor movement and the appearance or inappropriate rapid performance of a

movement. The overall resulting appearance of the motor behavior can be integrated with physiologic movement, though may be exaggerated during a motor sequence and be aligned with a facial expression and vocalization. It may also be non-integrated appearing unnatural and separated from other behavioral features [107]. Hyperkinetic behaviors are often produced by seizures arising from the frontal lobe [137-139]. In frontal lobe epilepsies, the behaviors exhibit a clinical continuum. Seizures with hyperkinetic behavior may originate from the rostral prefrontal regions, where highly integrated behaviors predominate, or arise from the premotor cortex that produces more elementary motor signs [107, 139, 140]. However, hyperkinetic behaviors may also occur in seizures originating in extra-frontal regions of the brain. These include the temporal lobe, the insula or insulo-operculum, and the posterior cortex [138].

5. Sensory phenomena

Sensory phenomena involve paroxysmal perception of positive (or rarely negative) sensory experiences that are not entirely caused by external stimuli, and not due to a non-epileptic etiology (e.g. carpal tunnel syndrome). When these subjective ictal phenomena precede observable signs of a clinical seizure, they are described by patients as a sensory aura. When they occur independently, they constitute a focal aware sensory seizure [3].

5.1. Auditory symptoms

Elementary auditory symptoms are uni- or bilateral buzzing, drumming, ringing, whistling sounds. They can be narrow- (single tone) or broad-band (noise) [141, 142]. Auditory illusions refer to an alteration of unimodal auditory perceptions which can consist of a modification of intensity (more or less intense) of tone (higher or lower pitched) or distance (closer or farther away). They may also occur as an echo of the percept and be perceived monaurally or binaurally [141, 142]. Elaborate auditory hallucinations refer to unimodal auditory hallucinations such as voices or melodies without multimodal or a clear mnemonic component [141, 142].

Auditory phenomena point almost exclusively to the superior temporal gyrus [143, 144], and rarely to the long posterior gyrus or the short anterior gyrus in the insula [145].

Elementary auditory phenomena point to the primary auditory cortex in the superior part of the temporal lobe (Heschl's gyrus). Elaborate auditory features are rare and point to the "interpretive" auditory cortex

comprising the anterior superior temporal gyrus and the planum temporale without clear lateralizing value [143, 146].

5.2. Visual symptoms

Visual symptoms may be composed of either positive or negative phenomena during seizures. Positive elementary phenomena are comprised of static or moving unformed flashes of lights, “spots”, or “blobs”. They may be white, black or include color, remain localized or be non-localized within a visual field (i.e., quadrant, hemi-field, or occupy the whole visual field) [147-149]. Negative elementary phenomena correspond to transient amaurosis of the entire visual field or scotoma when it is confined to part of it (quadrantanopia, hemianopia, tunnel vision) [147, 148, 150, 151]. Intermediary visual hallucinations are more complex. They may appear as geometric forms (i.e., stars, circles, triangles, squares, diamond) [148, 150] and possess a slightly different sub-lobar localizing value [148]. Visual illusions comprise a great variety of visual phenomena affecting an object or its visual background. This spans from visual blurring to an alteration of size (micropsia/macropsia), color (dyschromatopsia), shape (metamorphopsia), distance or movement (kinetopsia), and number (diplopia, polyopia) [147, 148, 151-154]. Palinopsia refers to the persistence or recurrence of a visual object when the stimulus is no longer present. Elaborate visual phenomena refers to unimodal visual hallucinations with detailed and meaningful implications such as faces, people, body parts, animals, and images of scenes that occur without a clear mnemonic or affective component, as well as unimodal visual hallucinations associated with elaborate negative phenomena which is dominated by the presence of prosopagnosia [148, 155-157].

Visual phenomena are a key feature of occipital seizures occurring in 68 - 88% of patients [158, 159]. However, they are not specific for occipital localization and may occur less frequently during parietal, anterior ventral and medial temporal and occipito-temporal lobe seizures. Elementary visual hallucinations may reflect the transient disturbance in the calcarine sulcus (primary visual cortex) caused by seizures, but they may also arise from the lingula, cuneus, and fusiform gyrus. Less often, they arise more anteriorly from the lateral, basal and medial temporal structures [148, 149]. When located in the center of the visual field, they reflect the disturbance of the calcarine sulcus. When elementary visual symptoms are unilateral, they occur contralateral to the site of seizure onset. Intermediate hallucinations have the same localizing and lateralizing value except that they reflect the disturbances of more anterior

lingula and ventral temporal cortex. Ictal visual loss is thought to reflect a disruption of function in the calcarine sulcus. This may be either unilateral or bilateral. Regarding the visual loss, concentric changes of visual fields have a different localizing value and would point towards a more anterior basal or medial temporal disturbance [151]. Illusions such as dyschromatopsia and blurring appears to be more specific for the ventral and medial temporal structures [148] and often reflects a right hemispheric lateralization. Illusions of movement (kinetopsia) are represented by a disturbance of the lateral occipital and lateral occipito-temporal junction [153]. Illusions of size (e.g. micropsia, macropsia and the Alice in Wonderland syndrome), shape (metamorphopsia) or number (polyopia) suggest a disturbance in the posterior medial parietal region (pre-cuneus and occipito-parietal sulcus). Elaborate hallucinations of faces point towards a disturbance of the right cuneus, occipito-parietal sulcus, and the pre-cuneus and fusiform gyrus [148]. Other elaborate hallucinations (places, persons, body parts, scenes) would reflect a disturbance of the basal and medial temporal structures [151, 157]. Prosopagnosia and distortion of facial perception are the only reported elaborate visual deficits described and point to the right temporo-basal/fusiform and inferior occipital gyrus [155, 160, 161].

5.3. Gustatory symptoms

Gustatory symptoms represent abnormal paroxysmal tastes within the mouth or in the throat. These are usually unpleasant, and may be reported as salty, metallic, bitter, sour, acidic or indescribable [162, 163]. Gustatory symptoms are sometimes difficult for patients to separate from olfactory symptoms and may be associated together in the same subjective experience during a seizure.

Gustatory symptoms during seizures are rare (occurring in around 4-8% cases) [162, 164]. They have been reported in seizures from peri-rolandic, insular and opercular regions, and less frequently from the medial temporal structures (amygdalohippocampal complex) without clear lateralizing value [144, 162, 165-167].

5.4. Olfactory symptoms

Olfactory symptoms are defined as a paroxysmal unexplained sense of smell. These are usually, but not always, unpleasant and can be neutral or even pleasant experiences. Olfactory hallucinations can be described as an odor of burning, sulfur, alcohol, gas, garbage, barbecue, flowers, among other descriptions, or be indescribable [168, 169]. These olfactory symptoms are sometimes difficult to differentiate

from gustatory symptoms by the patients and may be associated in the same subjective ictal experience. Olfactory symptoms during seizures are rare, constituting about 0.6% - 16% of all subjective manifestations [168].

Olfactory symptoms have been reported mostly in seizures arising from medial temporal structures involving a range of etiologies including tumor and hippocampal sclerosis though without lateralization value [170, 171]. Electrical cortical stimulation studies refined and extended these correlations and evoked olfactory symptoms in the medial temporal structures, especially the amygdala, piriform cortex and uncus [172], near the olfactory bulb in the medial orbitofrontal cortex [169], mid-dorsal insula, and around the insular central sulcus [163]. Olfactory auras have no lateralizing value.

5.5. Somatosensory symptoms

Somatosensory symptoms may be described as tingling, numbness, electrical, shock-like sensation, pain, or sense of movement [3]. They can be classified according to the localization and the quality of symptoms as unilateral paresthesia or numbness that follows a somatotopic pattern or displays a “Jacksonian march” in a segmental unilateral or bilateral distribution or as a painful somatosensory aura or sensation of heat [147, 166].

Somatosensory auras typically occur in patients with parietal lobe seizures and represent the most frequently described aura arising from this region (in 29-63% cases) [165, 173, 174]. They are mostly unilateral sensations, contralateral to the epileptogenic zone [4] but may also occasionally be bilateral or even uni- and ipsilateral to seizure onset in 5% and 2.5% of people, respectively [165].

Based on cortical electrical stimulation studies, unilateral paresthesia or numbness following a somatotopic pattern typically point towards the contralateral primary somatosensory cortex on the posterior bank of the central sulcus. Face and superior limb paresthesia represent the most common localization (reflecting the large representation of these body parts in the sensory homunculus) and involve lateral neocortex, while lower limb paresthesia point towards medial involvement [175]. If the involvement of the paresthesia occupies one side of the body, then seizures are most likely to arise contralateral to the involved upper and lower limbs. When involvement of the paresthesia is bilateral, seizures are more likely to involve the midline body parts, especially the face and trunk [175].

Unilateral or bilateral paresthesia or numbness of upper and lower limbs with segmental localization without a sensory march typically point towards the

secondary somatosensory area in the parietal operculum. Contralateral or bilateral sensory symptoms have also been reported to originate in the supplementary sensorimotor area, the superior frontal gyrus and cingulate motor area [176], and in the posterior-superior part of the insula [145].

Painful somatosensory auras are described by patients as acute and intense pain (burning sensation, pricking ache, throbbing pain or muscle tearing sensation), affecting the body. Painful auras most often lateralizes the hemisphere involved at seizure onset [177]. Painful sensory auras may secondarily produce the motor behavioral manifestations of pain, involving a corresponding facial expression, verbal complaints including shouting and crying, movements in an effort to avoid a perceived stimulus, or autonomic changes such as facial pallor or flushing. Painful auras strongly suggest involvement of the posterior insula and parietal operculum [178]. Painful auras typically are contralateral to seizure onset but they may be bilateral or even ipsilateral when the painful sensation centers in the face and trunk [179].

Segmental sensory auras involving temperature such as warmth and cold are usually described as unpleasant. They often are unilateral when involving a limb, and seem to be associated with either somatosensory or painful auras [180]. They are believed to point to the same regions as those associated with painful auras. They should be differentiated from a non-localized non-lateralized cold sensation associated with shivering which suggests localization in the amygdala.

Ictal headache should be strictly differentiated from a painful somatosensory aura because they are thought to reflect a vascular mechanism rather than an electrophysiologic disturbance involving a localized cortical somatosensory area responsible for pain [177]. It does not have localizing value [14] when it is encountered, but is more likely to be ipsilateral to the seizure onset when it occurs in patients with temporal lobe seizures [181].

5.6. Vestibular symptoms

Vestibular symptoms may be defined as a sensation of spinning or motion involving the body, with or without a sensation of unsteadiness [182]. This illusion of motion might be described as rotatory symptoms in the up-down or left-right plane, a sense of movement, floating, or undefinable sense of body motion [183]. In practice, it can be difficult to delineate the semiology of vestibular symptoms and differentiate them from the general non-specific term of dizziness that are vague and ill-defined sensations. Vestibular auras are rare, yet important symptoms reported with the highest prevalence in patients with parietal lobe seizures [165, 173]. However, they are not specific and

may also be encountered in patients with temporal [144, 171], occipital [184], insular [167] and occasionally frontal lobe seizures. At a sub-lobar level, focal cortical electrical stimulation studies during SEEG emphasize the importance of the posterior perisylvian region involving the supra-marginal gyrus, inferior parietal lobule, angular gyrus, superior and middle temporal gyri, pre-cuneus, and insula. Vestibular phenomena have no consistent lateralizing value.

5.7. Body-perception illusion

Somatagnosic auras result from a disturbance of unimodal somatosensory (proprioceptive and tactile) self-representation of the body. *Autoscopic symptoms* result from a disturbance of multimodal self-integration of body including visual perception of one's body, either from an internal perspective of the body (autoscopic hallucination), or from an external perspective (out-of-body-experience), or when patients cannot decide whether one's self is located inside the physical properties of the body or reside outside of it (heautosopic hallucination) [185]. Somatagnosic auras comprise non-visual, illusions of the body such that one feels disconnected, dislocated or a sense of movement, experiences phantom limbs or reduplication, or deformity involving a body part [185]. These are rare but suggest a non-dominant (right) temporo-parietal junction as the site of seizure involvement [186].

Autoscopic hallucinations are rare, tend to be more often perceived in the contralateral visual field and suggest right (non-dominant) medial occipitoparietal cortex (pre-cuneus and occipito-parietal sulcus) involvement [148, 187].

An out-of-body-experience typically points to the non-dominant lateral parietal lobe [174, 186], but also may occur with seizures arising from the dominant temporoparietal region [188].

5.8. Depersonalization

Depersonalization is defined as the detachment from oneself, either the mind or body. In addition, this may involve being a detached external observer of oneself during a seizure. Depersonalization is not at all specific to epileptic seizures. It is classified as a symptom according to the DSM-5, involved in a dissociative disorder [189], and is often encountered in anxiety and panic disorders, as well as obsessive-compulsive disorder, severe stress, use of recreational drugs and even in normal people following severe sleep deprivation. There is a phenomenological overlap between autoscopic phenomena (out-of-body experience) and depersonalization. When associated with epilepsy, it is associated with various ictal

symptomatology such as dreamy state ("mental diplopia"), autoscopy, affective signs or symptoms, or conscious automatic behavior. Therefore, it is not specific regarding localization and has been reported in frontal, temporal, insular, parietal and occipital lobe seizures. Depersonalization needs to be differentiated from derealization, which refers to the altered perception of one's surroundings that is experienced as a sensation of unreality.

5.9. Forced thinking

Forced thinking is defined as involving intrusive thoughts, ideas, or words in people and should be differentiated from both a dreamy state and complex sensory hallucinations [190, 191]. According to some authors, the definition also includes an overwhelming impulse to perform a certain act [192]. Due to the low number of cases in the studies, the lateralizing and localizing value of ictal forced thinking remains uncertain. Most likely, the symptomatogenic zone resides in the dominant frontal lobe [190].

6. Affective (emotional) features

Affective semiologies encompass an alteration in mood or an apparent emotional experience or observation that is manifest by patients during seizures. Often, these subjective ictal symptoms are reported at the onset of the seizures and may be *followed* by other semiologies. When motor signs occur simultaneously with them, the expression of an affective (emotional) feature is classified under mimic automatisms (see 4.1.6). The majority of ictal experiences of emotion are unpleasant, such as anxiety and fear (60%), and depression (20%), while ictal joy is only rarely reported [193]. In one study, across all regions of interest, 58% of reported subjective affective experiences were negatively valenced and unpleasant, whereas 15% of experiences were positive and pleasant [194]. Clinical observations have often reported that patients with seizures arising from limbic structures lateralized to the right side of the brain have a higher incidence of emotional disturbances, such as fear and anxiety, than those who have seizures arising from the left side [195]. The emotions with negative valence are most often formed from the left (dominant) hemisphere, and those with a positive or pleasant valence (or neutral valence) [196] are most often lateralized to the right (non-dominant) hemisphere [193, 196]. In 100 children, based on a study to assess the value of focal seizures, emotions with positive (pleasant) character were significantly more frequent during right-sided seizures while negative emotions did not lateralize the seizure onset zone.

Emotional semiologies are typically associated with involvement of the limbic structures. The best-known examples of affective symptoms are fear and anxiety. These are generally considered to reflect involvement of the amygdala during temporal lobe seizures. Video-EEG monitoring identified affective auras in 18% of 184 patients with temporal lobe epilepsy who underwent resective surgery [197]. In this study, affective aura was unable to lateralize or localize the seizures. They were more likely to be negative auras (15%) than positive (3%) while ictal affective behavior was present in 22% of patients (10% positive, 14% negative) [197].

6.1. Anger

Anger may occur in patients with seizures associated with a strong feeling of annoyance, displeasure, or hostility. Focal emotional seizures with anger are distinguished from episodes of dyscontrol such as temper tantrums and rage reactions (sudden, out-of-control bursts of anger) by unorganized, purposeless, non-goal-directed aggression. In addition, stereotyped evolution during each event, and the presence of other features and semiologies are often present. The lateralization of anger is uncertain though possibly left hemispheric in adults [196], whereas it may be the opposite in children; right hemispheric EEG foci were found to be more common in children who exhibited emotional impairments such as anger [198]. Focal seizures with anger localize to the mesial temporal networks [199], especially the amygdala [200]. This was noted in early studies involving direct electrical stimulation [172]. Those symptoms may also arise from the prefrontal and orbitofrontal cortex less frequently [196, 201].

True ictal aggression, defined as intense anger followed by hostile, injurious or destructive behavior, is extremely rare and may be seen extending into the postictal phase. It can manifest as severe agitation with self-injury and destruction of property. In a review of 24 cases, most patients suffered from focal epilepsy and psychiatric co-morbidities such as alcohol dependence or other drug addiction. After careful analysis, no predominant focus side or localization could be identified [202].

6.2. Anxiety

Anxiety during a focal aware seizure may produce a feeling of worry, nervousness, or unease typically about an imminent event, or something with an uncertain outcome. Clinical observations have often reported that patients with focal seizures arising from limbic structures within the right side of the brain have a higher incidence of emotional disturbances including anxiety (and fear) than those who have

seizures lateralized to limbic structures on the left side [195]. However, there have been some inconsistent reports regarding the lateralizing value of ictal anxiety [203-207] making lateralization less distinct. Seizures with anxiety when they arise from the mesial temporal structures involve the limbic network and especially the amygdala, [172, 204, 208-210]. However, they may also be widely distributed within the temporal lobe [211], including the temporal neocortex and any of the limbic structures (i.e., amygdala, hippocampus and parahippocampal gyrus) [212]. Overall, the best examples of ictal anxiety are generally considered to reflect involvement of the amygdala. Other locations include the temporal pole and frontal lobe when they are associated with anxiety/panic during seizures [205]. The insula has also been implicated during SEEG evaluation [145].

6.3. Fear

Fear is an intense, unpleasant emotion caused by the belief that someone or something is dangerous and likely to inflict pain or perform a physical threatening act. Ictal fear or ictal panic is the most frequently reported ictal affective manifestation and is reported in 10-15% of patients with focal seizures [213]. The lateralization is inconsistent, though right hemispheric involvement is more probable than the left [196, 214-217], and either of the hemispheres may be involved. Most patients with epilepsy and ictal fear have a seizure onset zone in the mesial temporal lobe [172, 218], usually associated with involvement of the amygdala [208, 219]. In addition, the hippocampus [171, 218] may be linked to patients with more severe or “burned out” disease [220]. Fear appearing early in a clinical seizure may also implicate the lateral temporal neocortex [221], orbital frontal cortex [222] and anterior cingulate network [219, 223, 224]. Occipital seizure onset assessed by intracranial EEG has rarely been reported and raises the issue of seizure propagation to the mesial temporal networks [214, 224, 225]. Seizures occur in mesial frontal and orbitofrontal epilepsies, and ventromedial frontal lobe epilepsy may be associated with facial expressions of fear [137]. Emotional facial expressions including fear are observed significantly more frequently in hyperkinetic seizures of frontal vs temporal lobe origin when followed by bilateral facial contraction, in contrast to the corners of the mouth dropping in temporal seizures [226]. Affective symptoms manifesting as fearful screaming and terrified expression have been associated with ictal involvement of the amygdala and hippocampus, the orbitofrontal region, and the anterior cingulate gyrus [219].

6.4. Sadness

Although a seizure is often associated with depression, that emotional state is unusual as an ictal experience alone [227]. Sadness during seizures reflects the condition or quality of feeling sad or being unhappy and encompassing a feeling or showing sorrow. The lateralization is ill-defined and uncertain though the left hemisphere has demonstrated symptoms of sadness [120, 196, 228]. Ictal depression or sadness has been reported by 1% of patients with focal epilepsy arising from the temporal lobe [213, 221] (rarely during electrical cortical stimulation). They can also occur in seizures arising from the mesial temporal region [120], orbito-frontal cortex [196], and the anterior insula in a single patient during electrical stimulation studies [228]. Seizures associated with sadness often accompany seizures with laughing in the setting of a hypothalamic hamartoma. Focal seizures associated with sadness may be accompanied by lachrimation, sad facial expressions and sobbing when they arise from a hypothalamic hamartoma [229].

6.5. Guilt

Guilt is an affective symptom involving an emotional feeling of having done wrong or failed in an obligation. No clear data is available regarding possible lateralizing value and therefore the lateralization is uncertain [213], though considering the valence, it possibly points to the left hemisphere [196, 230]. Given the paucity of information regarding guilt and focal seizures, there is insufficient localizing information.

6.6. Ecstatic feeling / bliss

Focal aware seizures with a positive affective component are infrequently characterized by ecstasy or a sense of bliss, and more commonly involve negative sensations such as fear, anxiety, or panic. Ecstatic seizures evoke a feeling of bliss and intense positive emotion, enhanced physical well-being, and clarity with heightened self-awareness or perception of the external world with an overwhelming feeling or expression of happiness or joyful excitement. An ecstatic phenomenon is an altered state of consciousness with a sense of "hyper-reality", and complete present-moment awareness with a feeling of union with the universe [231]. Pleasure has been described as elation, intense pleasure, serenity, gladness, joy, exhilaration, and completeness [227]. Left lateralization was suspected and an overlap with right lateralization with the presence of religious overtones [232], though consistent lateralization has not been

confirmed in larger studies with either left or right lateralization possible [233, 234]. In the prior reports of single or small series of patients with ecstatic seizures, a temporal lobe origin was initially suspected [233], yet no precise localization was demonstrated until recently, when Picard and Craig proposed the anterior dorsal insula as the origin of intense positive feelings experienced during ecstatic epileptic seizures [231, 234-236]. The anterior cingulate has also been noted during electrical stimulation studies to reproduce pleasure [237], euphoria and a sense of well-being [238]. Also, rare cases involving the left frontal [239], right occipital [240] and parietal lobe [241] as well as the hypothalamus [242] in a case report have been reported, though in the latter case, this was felt to be likely due to activation of the insula. Most have been described with focal epilepsies, but a case was reported with generalized epilepsy induced by watching television [243].

6.7. Mystic

Mystic seizures or experiences with altered state of consciousness impart a religious or spiritual meaning to a person during seizures. This experiential affect involves a sensation that the person becomes one with God or believes in the spiritual apprehension of truth that is beyond the intellect. Religious attributions are rarely divulged though may include clairvoyance, telepathy, autoscopy, religious phrases, prayer, being in the presence of God, and auditory or visual hallucinations of religious figures [232]. There is a predominance of right lateralization as the critical site of religious experience due to the emotional nature [244]. Mystical experiences have been most often linked to the temporal lobe [234, 245, 246]. Mystic, religious, and spiritual experiences have also been reported to involve neocortical areas when visual and auditory hallucinations or complex ideation occur [246].

6.8. Sexual

Sexual experiences of desire, arousal or orgasmic aura may infrequently be involved in the ictal features of focal epilepsies. They are more often observed in women [247]. Sexual auras do not have strong lateralizing value though most arise from the right hemisphere [248]. Sexual auras often stem from the mesial temporal structures [248, 249] but also have been noted arising from the anterior cingulate cortex [194]. For seizures marked by sexual automatisms such as masturbatory behavior, pelvic thrusting or similar automatisms, although typically attributed to temporal lobe disease, frontal lobe seizures using depth EEG electrodes have also been described [250].

7. Cognitive ictal semiology

7.1. Dysmnnesia

Dysmnnesia is the disturbance of memory and comprises ictal amnesia, feelings of familiarity (*déjà vu*), unfamiliarity (*jamais vu*), a dreamy state and reminiscence that may be seen in patients with seizures.

• 7.1.1. Amnesia

Ictal amnesia implies that the patient cannot recall events during the seizure, or at least a part of the seizure. It does not have localizing or lateralizing value. Ascertaining this semiology feature is important, as it allows determining awareness of the patient during seizures. This helps classify focal seizures based on the current ILAE seizure classification system [5]. In practical terms, if a patient cannot recall events during a focal seizure, then the seizure is classified as a focal impaired awareness seizure. However, ictal amnesia and awareness are not completely synonymous, because, exceptionally, seizures can present with isolated ictal amnesia, yet with clear awareness [251]. However, detailed observation is required to distinguish and separate these two cognitive semiologies [5].

• 7.1.2. Déjà / jamais vu / dreamy state

These experiential phenomena consist of: (1) *déjà vu* or a feeling that one has lived through the present situation before; (2) *jamais vu* as the feeling one has experienced the situation for the first time, despite having been in the situation before; (3) a dreamy state with vivid memory-like hallucinations. These symptoms suggest a symptomatogenic zone in the temporal lobe, primarily the mesial structures (amygdala and hippocampus) and the sub-hippocampal structures (rhinal cortices), without lateralizing value. The temporal neocortex might also play a significant role in producing these phenomena. Of note, these symptoms are not specific to patients with epilepsy and can occur in other neurologic and psychiatric conditions [172].

• 7.1.3 Derealization

Derealization refers to the altered perception of one's surroundings that are experienced as unreal. It should be differentiated from depersonalization (see section 5.8). Although usually reported in patients with psychiatric conditions (depression, anxiety), case reports and small case series have revealed derealization in epileptic seizures. Due to the low number of cases, the localization is uncertain, but it is most likely associated with temporal lobe epilepsy [252].

7.2. Aphasia

Ictal aphasia is the inability to comprehend or formulate language during focal seizures. It is

important to distinguish an ictal component from aphasia that is more commonly identified in the postictal state. Ictal aphasia can be manifest as Wernicke's (receptive), Broca's (expressive), or global (mixed) aphasia. It may be difficult to differentiate aphasia in patients with impaired consciousness, unless behavioral testing specifically addresses specific components of language [18]. Inability to name objects has been referred to as ictal anomia. Ictal aphasia suggests involvement of the speech and language areas (Wernicke, Broca, basal temporal language area) in the dominant hemisphere. In an SEEG study of patients with temporal lobe epilepsy, different patterns of ictal aphasia were identified: impaired comprehension was associated with posterior lateral involvement, anomia and reduced verbal fluency with the anterior mediobasal structures, whereas Wernicke's aphasia with rapid verbal output ("jargonaphasia") was found with basal temporal involvement [253]. However, in a large study involving ictal aphasia in patients with focal epilepsies, this semiological presentation had limited localizing value within the dominant hemisphere and was most commonly seen in patients with seizures originating from the parieto-occipital regions, and even with seizures spreading from the non-dominant to dominant language areas of the brain [254].

8. Autonomic seizure semiology

Autonomic symptoms and signs are common accompanying features of focal or generalized seizures, and these include cardiovascular (palpitations, tachycardia), respiratory (hyperventilation), cutaneous (feelings of heat or cold, piloerection), gastrointestinal (bloating or nausea, retching and vomiting) and genitourinary (urinary urge, sexual arousal) phenomena. When autonomic signs occur early and prominently in a focal seizure, then the event is recognized as a focal autonomic seizure (a subset of focal aware seizures- non-motor) [5]. Some autonomic phenomena, such as ictal vomiting, ictal urinary urge or coughing, may have lateralizing or localizing value. When ictal symptoms and signs are confined to the autonomic nervous system, these may be mistaken for non-epileptic events. It is also important to distinguish between subjective symptoms (autonomic auras) and objective autonomic phenomena (autonomic seizure) during seizures.

8.1. Cardiovascular manifestations

Common cardiovascular manifestations include *alterations in heart rate and rhythm*, blood pressure, ECG changes and chest pain.

• 8.1.1. Sinus tachycardia

Sinus tachycardia is the most common ictal autonomic sign, occurring in 82% of patients with epilepsy [255]. Ictal sinus tachycardia was observed in 12% of patients with subclinical seizures, 71% of focal seizures, 64% of generalized seizures, and 76% with mixed seizure types. Ictal tachycardia often preceded EEG changes and occurred more frequently in patients with temporal rather than extratemporal seizures [255]. Views on the lateralizing value of ictal tachycardia are conflicting [255-257]. On the other hand, unlike bradycardia, tachycardia does not help differentiate epileptic from non-epileptic paroxysmal events [258].

• 8.1.2. Bradycardia and ictal asystole

Bradycardia and ictal asystole are much less frequent (0-2%) than ictal tachycardia [259-263]. This is usually observed in patients with temporal lobe seizures [264-271], but also rarely arises from other areas, such as the left cingulate [272]. While some authors suggest left lateralization [265, 273], the majority of studies did not confirm a lateralizing value for ictal bradycardia [267, 268, 270, 271]. Bradycardia may occur in the context of respiratory depression [274]. Falls and myoclonus were observed with prolonged ictal asystole (mean: 14.8 ± 7 s) [269, 275]. Thus, loss of muscle tone or bilateral asymmetric jerky limb movements during a seizure suggest the possibility of ictal asystole [275].

8.2. Cutaneous ictal phenomena

• 8.2.1. Flushing

Flushing from skin hyperperfusion has been reported by several authors as part of the autonomic manifestations of seizures [276-278]. Large series evaluating autonomic symptoms found that flushing occurred in patients with both temporal and extratemporal lobe epilepsies, without clear lateralizing value [279]. Ictal flushing should be differentiated from the effects produced by pheochromocytoma, carcinoid syndrome, mastocytosis, "hot flushes" in postmenopausal women, and asymmetric facial flushing (a.k.a., Harlequin syndrome).

• 8.2.2. Pallor

Pallor is extremely common as a prodromal sign in patients with syncope occurring in up to 93% of cases [280]. However, pallor can rarely be an ictal sign [276, 277]. In children, ictal pallor has been associated with focal seizures arising from the mesial temporal lobe and lateralized to the left side in 10 of 11 cases in one study [281]. It has also been reported in association with ictal fear related to amygdala atrophy [282] and bilateral perisylvian polymicrogyria [283], and in patients with Panayiotopoulos syndrome [278, 284].

• 8.2.3. Sweating

Sweating has been described in early reports of patients with seizures [276, 277]. It usually occurs in association with other symptoms including shivering [285], abdominal pain, headache and vertigo [286] or with the sensation of generalized coldness [287]. Most reports are anecdotal, and more recent reports have linked unilateral facial sweating to anti-Ma2-positive autoimmune encephalitis associated with testicular teratoma [288]. However, there are no systematic studies to provide consistent lateralizing or localizing value in patients with ictal sweating.

• 8.2.4. Piloerection

Piloerection is defined as erection of the hairs of the skin due to contraction of the tiny arrectores pilorum muscles that elevates the hair follicles above the rest of the skin, moving the hair vertically and appearing to "stand on end" (gooseflesh or goosebumps). Piloerection is a rare sign that is seen in 0.40-0.65% of patients in large VEM series [289, 290]. Piloerection is usually associated with other autonomic symptoms, such as sweating, flushing, and shivering. There is a strong association with temporal lobe epilepsies including the insula and amygdala. However, lateralization is inconsistent [290-293]. Recent reports reveal a strong representation of autoimmune encephalitis and gliomas as a cause of seizures associated with piloerection [289, 293, 294].

8.3. Gastrointestinal ictal phenomena

• 8.3.1. Epigastric aura

Epigastric aura was the earliest autonomic symptom to be recognized as an aura [276]. It is also the most common visceral symptom in adult epilepsy patients [295, 296] and occurs significantly more often in those with TLE as opposed to ETLE [297] with a probability of 73.6%. It occurs more frequently in patients with mesial temporal epilepsies compared to those with neocortical TLE [297, 298]. Epigastric auras followed by oral and manual automatisms are highly indicative of TLE with a probability of 98.3% [297]. Epigastric aura (mainly without automatisms) has been reported in ETLE (frontal lobe, insula). While some early studies reported epigastric auras more often in patients with seizures arising from the non-dominant temporal lobe [299, 300], more recent studies did not find any lateralizing significance for epigastric auras [69, 296-298, 301, 302].

• 8.3.2. Hypersalivation or sialorrhea

Hypersalivation or sialorrhea is the excessive production of saliva during a seizure. It is a rare occurrence seen in TLE patients, occurring in 2% of patients evaluated for epilepsy surgery [303] and 9.4-12% of TLE in smaller series [296, 304]. Ictal hypersalivation appears to suggest a mesial temporal localization

without clear lateralizing significance [296, 303, 304]. Invasive EEG recordings in isolated cases have implicated the perisylvian frontal operculum [305] and the insula [306].

• 8.3.3. *Spitting*

Spitting is an infrequent symptom reported in 0.3-2% of patients with temporal lobe seizures [296, 307-310], and several studies implicate non-dominant lateralization [296, 310-312]. However, the left temporal lobe has also been involved in case reports [309, 313, 314]. In a case report evaluated with invasive EEG electrodes, although the seizure onset zone was in the left mesial temporal lobe, the ictal spitting coincided with the propagation of the ictal discharge to the right temporal lobe, implicating the latter as the symptomatogenic zone [312]. Direct electrical stimulation studies during invasive EEG monitoring have identified the entorhinal cortex as a site involved in ictal spitting [311]. Ictal spitting does not occur in association with ictal vomiting, ictal coughing, or ictal fear, suggesting there are different sites in the brain that mediate these symptoms with unique specificity [308]. Although ictal spitting has been reported as a gastrointestinal autonomic phenomenon, it has a clear motor component and could also be listed under automatisms.

• 8.3.4. *Vomiting*

Vomiting is a striking feature when it occurs and is not uncommon in self-limited childhood occipital epilepsies. In adult patients evaluated for epilepsy surgery, ictal vomiting has been reported in patients with TLE, especially involving the mesial structures. Seizures commonly originate from the right hemisphere [315-320], but also may involve the left temporal lobe [321-323]. A recent series on ictal vomiting failed to demonstrate consistent lateralization [324]. Intracranial recordings have also suggested that the symptomatogenic zone may reside in the anterior insula when vomiting results from propagation of the ictal activity from the mesial temporal structures to the insula [325, 326].

• 8.3.5. *Flatulence*

Flatulence is a rare symptom that occurs in 0.6% of patients [327]. It suggests temporal and insular involvement during seizure propagation, but is without any lateralizing value [327, 328]. According to anecdotal reports, rare related gastrointestinal symptoms including an ictal urge to defecate [329, 330] and ictal burping [331] may localize to the right temporal lobe.

8.4. Respiratory dysfunction

Respiratory dysfunction, specifically hypoventilation (bradypnea), apnea, and oxygen desaturation, is more

frequently seen in epileptic seizures than in patients with non-epileptic events [258].

• 8.4.1. *Hypoventilation*

Hypoventilation and oxygen desaturation of <90%, <80% and <70% was observed in 33.2%, 10.2% and 3.6% of all seizures recorded in patients with drug-resistant epilepsy, respectively [332]. Another study reported the presence of ictal hypoxemia in 70.8% of patients experiencing 79 seizures. In this cohort, desaturation was mild (mean: 92.5%) in 46% of all seizures, moderate in 39% (mean: 81.5%), and severe in 14% (mean: 64.7%) [333]. Oxygen desaturation lasting longer than 70 seconds with a nadir of 83% has been reported during focal seizures without evolution to bilateral tonic-clonic seizures. This supports the hypothesis of centrally mediated hypoventilation during focal epileptic seizures [258].

• 8.4.2. *Apnea*

Apnea was reported to occur in approximately 40% of seizures recorded in adult series with duration lasting for more than 10 seconds (range: 10-63 seconds; mean: 24 seconds) based on polygraphic measurements [274, 333]. Prolonged central apnea (≥ 60 seconds) occurred in 6.3% of patients in association with severe hypoxemia during seizures with blood oxygen saturation levels (SpO_2) of less than 75%. Similarly, episodes with ictal apnea have been reported in neonates and children with TLE [279, 334]. Central apnea may precede seizure onset recorded on scalp EEG in more than a half of cases, and rarely persists into the postictal period. Patients typically do not recall ictal apneas, and therefore this might remain unrecognized without respiratory monitoring [333]. Oxygen desaturation less than 90% significantly correlated with temporal lobe localization at seizure onset, right hemispheric seizure lateralization, and contralateral hemispheric propagation [332]. The degree of desaturation correlated with seizure duration and propagation and involved the contralateral hemisphere [335]. No significant differences were seen between mesial and neocortical temporal lobe seizure onsets [336]. On intracranial EEG recordings in patients with mesial TLE, 68.4% showed ictal apnea, either accompanied by, or following seizure onset. Apnea may be the principal or sole manifestation in 15% of TLE cases [337].

• 8.4.3. *Choking*

Choking due to acute laryngospasm as an isolated event associated with nocturnal seizures may rarely represent an ictal manifestation [338]. Nocturnal choking can occur in sleep-related hypermotor seizures associated with nocturnal frontal lobe epilepsy, and may be misdiagnosed as sleep apnea syndrome due to abnormal nocturnal motor activity

and excessive daytime somnolence [339]. Localizing information about ictal choking is sparse, however, anecdotally, ictal choking observed during stimulation studies has implicated the frontal operculum [340] and anterior insula [341, 342]. In patients with insular seizures, the semiology typically begins in patients who are fully conscious and experience a sensation of laryngeal constriction that is often associated with unpleasant paresthesia, affecting large cutaneous territories [343].

• 8.4.4. Hyperventilation

Hyperventilation may occur during focal seizures. However, there is a lack of precise prevalence for patients with ictal hyperventilation because respiration is not routinely evaluated during video-EEG monitoring. Tachypnea was found in approximately a half of patients in a series of children with epilepsy [344], especially in those with focal seizures. It was found to be more common in older children, females, individuals with frontal lobe seizures, and seizures with right-sided lateralization [345]. While hyperventilation is more common in children with FLE, adults with seizures and hyperventilation are more likely to have TLE [346] and seizures that arise from mesial rather than neocortical structures [347]. In patients with FLE, hyperventilation was noted to occur in seizures arising from the frontopolar and the orbitofrontal regions [348]. Hyperventilation and shortness of breath that occurs during focal seizures with an emotional content must be differentiated from non-epileptic panic attacks with hyperventilation [295, 349] because right temporal seizures with ictal anxiety and hyperventilation can mimic non-epileptic panic attacks [204].

8.5. Pupillary ictal phenomena

• 8.5.1. Miosis

Miosis is a rare autonomic sign that has been described as a part of generalized photosensitive epilepsies [350] and in patients with focal epilepsies involving the temporal, parietal and occipital lobe. The localizing and lateralizing value for miosis in patients with epilepsy is uncertain [351-354].

• 8.5.2. Mydriasis

Mydriasis is a common sign seen bilaterally when it is associated with seizures in patients with either GTCS or FBTC seizures. This is due to widespread activation of the cortical and subcortical structures of the brain that influence the sympathetic nervous system [295, 349]. Unilateral mydriasis is rarely reported to occur in patients with temporal lobe seizures [256, 355] or occipito-temporal seizures, and appears ipsilateral to the seizure onset zone [356]. It has also been reported to occur in the contralateral pupil of a young boy with left frontal seizures [357].

8.6. Urinary and genital ictal phenomena

• 8.6.1. Urinary sensations

Urinary sensations are rarely an autonomic symptom associated with focal seizures in which the patient develops an intense desire to urinate. The frequency is estimated to range from 0.8 to 8% of patients [358]. It has been reported to arise from the non-dominant hemisphere and more specifically from the temporal lobe [358-360]. There are some reports of ictal single-photon emission computed tomography studies that demonstrate hyperperfusion involving the right temporal lobe (superior temporal gyrus) and insula.

• 8.6.2. Genital signs

Genital signs are rare in patients with focal seizures. They are comprised of various sexual manifestations and genital automatisms. Sexual symptoms in patients with seizures include erotic and genital sensations (see 4.1.3) and signs of sexual arousal, orgasm, and sexual or genital automatism. Genital automatisms consist of grabbing, fondling, and scratching the genitals during or immediately following a seizure [248]. Several reports pointed to a non-dominant mesial temporal lobe as the most frequent localization. Sexual automatisms were reported to be more frequent in men than in women [361]. Sexual behavior with extratemporal seizures is rare. A study of 15 patients implicated three different regions of the brain including the perisylvian region, anterior cingulate gyrus and paracentral lobule [362]. One case report described a patient with a right amygdalar tumor, with ictal symptoms of feeling transformed to another gender, which has been coined "ictal delusional misidentification syndrome" [363].

9. Postictal phenomenology

Postictal neurological deficits and/or psychiatric signs and symptoms are often accompanied by diffuse or focal slowing or postictal generalized EEG suppression after the clinical signs of the seizure have ended [364]. Many of these signs and symptoms are attributed to intrinsic inhibitory mechanisms which contribute to seizure termination. The duration of a postictal state is variable and can be up to several days. However, while some symptoms (headache, muscle aches, postictal psychiatric symptoms) may last longer, most postictal features last 5-30 minutes [365]. Prolonged postictal altered consciousness or prolonged postictal focal neurological deficits should raise the suspicion of ongoing seizures and non-convulsive status epilepticus. In these cases, patients should be expeditiously evaluated with EEG [9, 11]. Rarely, in the peri-ictal and postictal state, patients

may succumb to sudden unexpected death in epilepsy (SUDEP) [366].

9.1. Unresponsiveness

Unresponsiveness was the most common phenomenon (mean frequency of 96%) in a recently published systematic review and meta-analysis of 45 studies on the postictal state [367]. Recovery from postictal unresponsiveness may take seconds to minutes [368].

9.2. Paresis

Paresis (Todd's paresis) may occur after focal motor or FBTC seizures. A large study showed postictal paresis in 6% of FBTC seizures [369]. However, this could be an underestimate, due to often insufficient postictal testing of the patients. Postictal paresis may last from minutes to hours [369]. It lateralizes to the contralateral hemisphere, regardless of whether the seizure spreads to the contralateral hemisphere on surface EEG or not [370-373]. However, postictal paresis can also be bilateral. Postictal paresis develops after ictal involvement of the motor cortex, most often in frontal lobe seizures. However, it has also been documented in seizures originating in temporal, parietal and occipital lobes [374]. Sensory deficits following seizures have been rarely reported and can be missed if sensation is not specifically tested or symptoms addressed [375].

9.3. Blindness.

Following seizures, loss of vision may occur on one side of the visual field (hemianopsia) or both sides (amaurosis) [376, 377]. The duration typically lasts from seconds to hours [367]. It has been reported in two-thirds of patients with childhood occipital epilepsy of Gastaut [378]. Postictal blindness points to involvement of the visual cortex and has been described in patients with occipital and temporal lobe seizures. Ictal homonymous hemianopsia lateralizes to the contralateral hemisphere [379].

9.4. Language dysfunction

Language dysfunction comprises Broca's, Wernicke's and global aphasia, anomia, paraphasia and errors in repetition and writing. Approximately 38% of patients experience language impairment, with duration typically of seconds to minutes [367, 380]. Postictal speech disturbances can be indicative of seizure localization or seizure propagation [380]. Most postictal language disturbances occur in patients with focal seizures associated with either onset or propagation to the dominant temporal lobe [374]. In one study, postictal language delay (time from last ictal EEG to reading a

sentence) occurred in 100% of 62 seizures, and patients took more than 68 seconds to read a test phrase correctly [380]. Overall, postictal speech disturbances correctly lateralized to the dominant hemisphere in 81.3% of all patients, regardless of which kind of dysfunction was documented (*i.e.* no statistically significant difference, although one must recognize the small groups of paraphasias).

9.5. Nose wiping

Nose wiping has been observed significantly more often in patients with temporal rather than extratemporal lobe epilepsy [381-384]. It was shown to be ipsilateral to the hemisphere of seizure onset in 86.5% of patients with TLE, and patients with extratemporal lobe epilepsy performed ipsilateral nose wiping in 54.5% of all seizures [370].

9.6. Headache

Headache occurs in 41.6-66% of patients with seizures with a duration lasting from minutes to hours [367, 385]. Younger age at seizure onset and longer duration of epilepsy, drug-resistant epilepsies, and tonic-clonic seizures are additional risk factors [386]. Postictal headache often manifests with severe migraine-like features. In temporal lobe epilepsy, postictal headache was found to be ipsilateral to the site of seizure onset in 90% of cases [387]. However, no significant lateralization value was found in patients with frontal lobe epilepsies [388]. Peri-ictal headache was more likely to be ipsilateral to the seizure onset in patients with temporal lobe epilepsies (90%) than in those with extratemporal lobe epilepsies (12%) [181].

9.7. Palinacousis

Palinacousis involving preservation of an external auditory stimulus or retained fragment of a previously heard sentence may occur after seizure cessation. These auditory illusions have been described in a few cases, though the localizing and lateralizing value of this symptom is uncertain [389].

9.8. Autonomic signs

Autonomic signs comprise a wide range of phenomena including ictal tachycardia, bradycardia, arrhythmia, hypersalivation, coughing, apnea, tachypnea, changes in blood pressure, hyperthermia, and neurogenic edema [368, 390]. Postictal coughing occurs in both temporal and extratemporal lobe epilepsies and may only be indicative of a temporal lobe seizure onset when seizures are represented by a stereotypical semiology. Coughing may be due to a different

mechanism and occur independent of additional autonomic signs or symptoms [391]. Rarely, postictal autonomic features can be life-threatening. Respiratory depression and cardiac dysfunction, occurring after a GTCS (including FBTC seizures), are critical factors in SUDEP [366]. A postictal breathing pattern may also be a useful clinical sign in terms of differentiating epileptic seizures from non-epileptic seizures; the presence of postictal hyperpnea is more likely to occur after an epileptic seizure than a non-epileptic event [392].

9.9. Psychiatric symptoms and syndromes

These include delirium, psychosis, catatonia, impaired cognition and amnesia [368]. These do not have localizing or lateralizing value. Delirium, changes in perception (hallucinations), thoughts (incoherence, delusions) and catatonia may last for hours to days [368, 393]. Postictal delirium may transition into postictal psychosis, including violent bizarre or sexually inappropriate behavior. The prevalence of postictal psychosis ranges between 2% and 7%, independent of type of epilepsy [368, 394]. It can develop after a “lucid phase” when the patient recovers from initial postictal confusion, only to reappear six hours to a week later [395]. The mean duration of postictal psychosis is variable, lasting up to 9-10 days (range: 12 hours - 3 months) without treatment. Approximately a half of patients with postictal psychotic symptoms also have a history of psychiatric disorders, such as depression, anxiety and attention deficit disorder [396]. Postictal psychosis occurs more often after a cluster of GTCS [395]. Postictal psychosis is a treatable condition, which left untreated, is associated with serious patient-related morbidity and mortality [395]. Homicide during the postictal psychosis has been described in a single case report [397]. Postictal depressive and anxiety symptoms may occur for more than 24 hours and within five days following a seizure [398]. For focal epilepsy, 18% of patients showed postictal depressive symptoms, characterized by anhedonia, helplessness, self-deprecation or suicidal thoughts. Postictal dysphoric symptoms are more likely in temporal plus epilepsies, compared to pure temporal lobe epilepsies [399]. Postictal anxiety lasts from minutes to hours, and is often accompanied by depression.

10. Somatotopic modifiers and time-related features

Some ictal phenomena can occur in different parts of the body, hence the somatotopic modifiers should be added to the respective ictal phenomena [3, 5].

10.1. Unilateral

Exclusive or virtually exclusive involvement of one side as a motor, sensory or autonomic phenomenon.

10.2. Bilateral

Involvement of both left and right sides; bilateral manifestations may be symmetric or asymmetric.

10.3. Centricity

Axial (trunk, including neck), proximal limb, distal limb.

10.4. Incidence

The number of seizures within a time period or the number of seizure days per unit of time (i.e., 3-4 seizures/month).

10.5. Cluster

The incidence of seizures within a given period (usually one or a few days) that exceeds the average incidence over a longer period for the patient (i.e., 3-5 seizures over 8 hours). Note that this definition does not apply to the presence of a cluster of epileptic spasms (which denotes repetitive occurrence / series of epileptic spasms).

10.6. Provocative factor

Endogenous or exogenous elements that are capable of triggering seizures in people with chronic epilepsy and evoking seizures in susceptible individuals without epilepsy. It is important to distinguish facilitating factors that temporarily increase seizure risk from precipitating factors (i.e., the specific triggers in reflex epilepsies). Acute symptomatic seizures are often referred to as “provoked” seizures.

10.7. Prodrome

The subjective constellation of a non-specific, broad spectrum of “warning” symptoms (such as an ill-localized sensation or agitation) that heralds the onset of an epileptic seizure but does not form part of it [3]. The duration is over 30-60 minutes, and it may persist for up to several days prior to seizure onset.

11. Conclusion

Scalp and invasive video-EEG monitoring has been instrumental to update and define terminology, and

provide lateralizing and localizing significance to semiology in patients with epilepsy. Often, several semiological features occur during the same seizure (either simultaneously or in a sequence), due to propagation of the seizure within the network (table 2). Seizure semiology is dynamically produced by interconnected structures, in which specific electrophysiologic interconnections, rather than anatomical location, likely play an important role in clinical expression [400]. Seizure propagation within an epileptic network and the corresponding evolution in time of the seizure semiology have been compared to “words in a meaningful sentence” [401].

Observing and reporting the onset and propagation of the ictal semiology is essential for correct interpretation, as it relates to the clinical significance and seizure network. The timing is important. In general, the earlier a semiology feature occurs in a seizure, the stronger its localizing and lateralizing value. This underscores the importance of signs and symptoms associated with an aura. Auras (focal aware seizures) typically precede other clinical signs. Yet, the epileptogenic zone and seizure onset zone may reside in an area of “silent” cortex that does not generate a clinical sign or symptom, and the first ictal manifestation appears as the consequence of seizure propagation.

Symbols can be used to describe the dynamics and temporal sequence of a seizure. The plus sign (+) between the phenomena signifies that signs occur simultaneously. Arrows to the right (→) indicate the next sign in the sequence over time (table 2). For example: epigastric aura → oro-alimentary automatisms → left gestural automatisms + right hand dystonic posturing + ictal aphasia → versive hand movement to the right + figure-of-4 sign with extension of the right elbow → bilateral tonic-clonic → persistent unilateral hemi-clonic jerks on the left are a classic example of an FBTC seizure depicted by semiology. In this case, the seizure would be suspected to arise from the left mesial temporal lobe. When EEG is co-registered with the video, it is important to specify the time delay between the onset of clinical symptoms or signs and the ictal EEG. When clinical signs or symptoms begin prior to scalp ictal EEG, a propagated pattern from the start of a seizure should be considered.

Some sequences involving semiology have been well-documented and have strong localizing value. Epigastric auras followed by automatism suggest a focus in the temporal lobe. Laryngeal constriction and a choking sensation, followed by focal motor and sensory symptoms involving the face, suggest a seizure onset in the insula. When the sequence of two or more semiologies point towards the same hemisphere (especially involving motor signs), the

lateralization value is higher when more features are present. Ictal fear followed by hyperkinetic behavior suggests seizure involvement of the frontal lobe, while fear followed by distal gestural and oro-alimentary automatisms suggests a focus in the temporal lobe. Seizures starting in the posterior quadrant of the brain (occipital and parietal lobes) often show rapid propagation to anterior structures (frontal and temporal lobes) and may manifest without a visual aura. A detailed interview about the symptoms and possible occurrence of auras (including those that occurred in the past but do not exist anymore) may be important for determining localization. Seizures with motor manifestations suggesting frontal or temporal lobe seizure onset might begin with a sensory aura, indicating onset in the parietal or occipital lobe, propagating to the frontal lobe. Recognition of the combination and sequence of different ictal signs facilitates identification of a diagnostic “gestalt” for seizure dynamics in an individual patient [402]. ■

Supplementary material.

Supplementary data accompanying the manuscript are available at www.epilepticdisorders.com.

Disclaimer.

This report was written by experts selected by the International League Against Epilepsy (ILAE) and was approved for publication by the ILAE. Opinions expressed by the authors, however, do not necessarily represent the policy or position of the ILAE.

Disclosures.

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References

1. Wolf P, Benbadis S, Dimova PS, Vinayan KP, Michaelis R, Reuber M, *et al*. The importance of semiological information based on epileptic seizure history. *Epileptic Disord* 2020; 22 (1): 15-31.
2. Tatum W, Rubboli G, Kaplan P, Mirsatari S, Radhakrishnan K, Gloss D, *et al*. Clinical utility of EEG in diagnosing and monitoring epilepsy in adults. *Clin Neurophysiol* 2018; 129 (5): 1056-82.
3. Blume WT, Lüders HO, Mizrahi E, Tassinari C, van Emde Boas W, Engel Jr J. Glossary of descriptive terminology for ictal semiology: report of the ILAE task force on classification and terminology. *Epilepsia* 2001; 42(9): 1212-8.
4. Fisher RS, Acevedo C, Arzimanoglou A, Bogacz A, Cross JH, Elger CE, *et al*. ILAE official report: a practical clinical definition of epilepsy. *Epilepsia* 2014; 55(4): 475-82.
5. Fisher RS, Cross JH, French JA, Higurashi N, Hirsch E, Jansen FE, *et al*. Operational classification of seizure types by the International League Against Epilepsy: Position Paper of

- the ILAE Commission for Classification and Terminology. *Epilepsia* 2017; 58(4): 522-30.
6. Scheffer IE, Berkovic S, Capovilla G, Conolly MB, French J, Guilhoto L, et al. ILAE classification of the epilepsies: position paper of the ILAE Commission for Classification and Terminology. *Epilepsia* 2017; 58: 512-21.
 7. Lüders H, Acharya J, Baumgartner C, Benbadis S, Bleasel A, Burgess R, et al. Semiological seizure classification. *Epilepsia* 1998; 39(9): 1006-13.
 8. Blümcke I, Arzimanoglou A, Beniczky S, Wiebe S. Roadmap for a competency-based educational curriculum in epileptology: report of the Epilepsy Education Task Force of the International League Against Epilepsy. *Epileptic Disord* 2019; 21(2): 129-40.
 9. Beniczky S, Hirsch LJ, Kaplan PW, Pressler R, Bauer G, Aurlen H, et al. Unified EEG terminology and criteria for nonconvulsive status epilepticus. *Epilepsia* 2013; 54: 28-9.
 10. Trinka E, Cock H, Hesdorffer D, Rossetti AO, Scheffer IE, Shinnar S, et al. A definition and classification of status epilepticus – Report of the ILAE Task Force on Classification of Status Epilepticus. *Epilepsia* 2015; 56(10): 1515-23.
 11. Leitinger M, Trinka E, Gardella E, Rohrachner A, Kalss G, Qerama E, et al. Diagnostic accuracy of the Salzburg EEG criteria for non-convulsive status epilepticus: a retrospective study. *Lancet Neurol* 2016; 15(10): 1054-62.
 12. Hirsch LJ, Fong MW, Leitinger M, LaRoche SM, Beniczky S, Abend NS, et al. American clinical neurophysiology society's standardized critical care EEG terminology: 2021 version. *J Clin Neurophysiol* 2021; 38(1): 1-29.
 13. Pressler RM, Cilio MR, Mizrahi EM, Moshé SL, Nunes ML, Plouin P, et al. The ILAE classification of seizures and the epilepsies: Modification for seizures in the neonate. Position paper by the ILAE Task Force on Neonatal Seizures. *Epilepsia* 2021; 62(3): 615-28.
 14. Blumenfeld H. Impaired consciousness in epilepsy. *Lancet Neurol* 2012; 11(9): 814-26.
 15. Blumenfeld H. *Epilepsy and consciousness. The neurology of consciousness*. Paris: Elsevier, 2016: 255-70.
 16. Charidimou A, Selai C. The effect of alterations in consciousness on quality of life (QoL) in epilepsy: searching for evidence. *Behav Neurol* 2011; 24(1): 83-93.
 17. Chen WC, Chen EY, Gebre RZ, Johnson MR, Li N, Vitkovskiy P, et al. Epilepsy and driving: potential impact of transient impaired consciousness. *Epilepsy Behav* 2014; 30: 50-7.
 18. Beniczky S, Neufeld M, Diehl B, Dobesberger 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.
 19. Gloor P. Consciousness as a neurological concept in epileptology: a critical review. *Epilepsia* 1986; 27: S14-26.
 20. Unterberger I, Trinka E, Kaplan PW, Walser G, Luef G, Bauer G. Generalized nonmotor (absence) seizures – What do absence, generalized, and nonmotor mean? *Epilepsia* 2018; 59(3): 523-9.
 21. Guo JN, Kim R, Chen Y, Negishi M, Jhun S, Weiss S, et al. Impaired consciousness in patients with absence seizures investigated by functional MRI, EEG and behavioural measures: a cross-sectional study. *Lancet Neurol* 2016; 15(13): 1336-45.
 22. Blumenfeld H. Consciousness and epilepsy: why are patients with absence seizures absent? *Progr Brain Res* 2005; 150: 271-603.
 23. McPherson A, Rojas L, Bauerschmidt A, Ezeani CC, Yang L, Motelow JE, et al. Testing for minimal consciousness in complex partial and generalized tonic-clonic seizures. *Epilepsia* 2012; 53(10): e180-3.
 24. Cavanna AE, Rickards H, Ali F. What makes a simple partial seizure complex? *Epilepsy Behav* 2011; 22(4): 651-8.
 25. Arthuis M, Valton L, Regis 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(8): 2091-101.
 26. Blumenfeld H. Arousal and consciousness in focal seizures. *Epilepsy Curr* 2021; 21(5): 353-9.
 27. Blumenfeld H, McNally KA, Vanderhill SD, Paige AL, Chung R, Davis K, et al. Positive and negative network correlations in temporal lobe epilepsy. *Cereb Cortex* 2004; 14(8): 892-902.
 28. Bonini F, Lambert I, Wendling F, McGonigal A, Bartolomei F. Altered synchrony and loss of consciousness during frontal lobe seizures. *Clin Neurophysiol* 2016; 127(2): 1170-5.
 29. Englot DJ, Yang L, Hamid H, Danielson N, Bai X, Marfeo A, et al. Impaired consciousness in temporal lobe seizures: role of cortical slow activity. *Brain* 2010; 133(12): 3764-77.
 30. Lambert I, Arthuis M, McGonigal A, Wendling F, Bartolomei F. Alteration of global workspace during loss of consciousness: a study of parietal seizures. *Epilepsia* 2012; 53(12): 2104-10.
 31. Detyniecki K, Blumenfeld H. Consciousness of seizures and consciousness during seizures: are they related? *Epilepsy Behav* 2014; 30: 6-9.
 32. Botez MI, Serbănescu T, Stoica I. The problem of focal epileptic seizures on both parts of the body without loss of consciousness. *Psychiatr Neurol Neurochir* 1966; 69(6): 431-7.
 33. Weinberger J, Lusins J. Simultaneous bilateral focal seizures without loss of consciousness. *Mt Sinai J Med* 1973; 40(5): 693-6.
 34. Bell WL, Walczak TS, Shin C, Radtke RA. Painful generalised clonic and tonic-clonic seizures with retained consciousness. *J Neurol Neurosurg Psychiatry* 1997; 63(6): 792-5.
 35. Shimazono YHT, Okuma T, Fukuda T, Yamamasu E. Disturbance of consciousness in petit mal epilepsy. *Epilepsia* 1953; (2): 49 55.

36. Jung R. *Correlation of bioelectrical and autonomic phenomena with alterations of consciousness and arousal in man*. Springfield, Illinois: Thomas, 1954.
37. Kooi KA, Hovey HB. Alterations in mental function and paroxysmal cerebral activity. *AMA Arch Neurol Psychiatry* 1957; 78(3): 264-71.
38. Boudin G, Barbizet J, Masson S. Étude de la dissolution de la conscience dans 3 cas de petit mal avec crises prolongées. *Rev Neurol* 1958; 99: 483-7.
39. Tizard B, Margerison J. Psychological functions during wave-spike discharges. *Brit J Soc Clin Psychol* 1963; 3: 6-15.
40. Mirsky AF, Vanburen JM. On the nature of the "absence" in centrencephalic epilepsy: a study of some behavioral electroencephalographic and autonomic factors. *Electroencephalogr Clin Neurophysiol* 1965; 18: 334-48.
41. Panayiotopoulos CP, Obeid T, Waheed G. Differentiation of typical absence seizures in epileptic syndromes. A video EEG study of 224 seizures in 20 patients. *Brain* 1989; 112 (Pt 4): 1039-56.
42. Inoue Y, Mihara T. Awareness and responsiveness during partial seizures. *Epilepsia* 1998; 39(S5): 7-10.
43. Lux S, Kurthen M, Helmstaedter C, Hartje W, Reuber M, Elger C. 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.
44. Cohen E, Antwi P, Banz BC, Vincent P, Saha R, Arencibia CA, et al. Realistic driving simulation during generalized epileptiform discharges to identify electroencephalographic features related to motor vehicle safety: feasibility and pilot study. *Epilepsia* 2020; 61(1): 19-28.
45. Naik PA, Fleming ME, Bhatia P, Harden CL. Do drivers with epilepsy have higher rates of motor vehicle accidents than those without epilepsy? *Epilepsy Behav* 2015; 47: 111-4.
46. Nirikko AC, Bernasconi C, von Allmen A, Liechti C, Mathis J, Krestel H. Virtual car accidents of epilepsy patients, interictal epileptic activity and medication. *Epilepsia* 2016; 57 (5): 832-40.
47. Giacino JT, Kalmar K, Whyte J. The JFK coma recovery scale-revised: measurement characteristics and diagnostic utility. *J Phys Med Rehabil* 2004; 85(12): 2020-9.
48. Giacino JT, Katz DI, Schiff ND, Whyte J, Ashman EJ, Ashwal S, et al. Practice guideline update recommendations summary: disorders of consciousness: report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology; the American Congress of Rehabilitation Medicine; and the National Institute on Disability, Independent Living, and Rehabilitation Research. *J Phys Med Rehabil* 2018; 99(9): 1699-709.
49. Laureys S, Gosseries O, Tononi G. *The neurology of consciousness: cognitive neuroscience and neuropathology*. Academic Press, 2015.
50. Cavanna AE, Monaco F. Brain mechanisms of altered conscious states during epileptic seizures. *Nat Rev Neurol* 2009; 5(5): 267-76.
51. Langston ME, Tatum WO 4th. Focal seizures without awareness. *Epilepsy Res* 2015; 109: 163-8.
52. Toledano R, García-Morales I, Kurtis MM, Pérez-Sempere A, Ciordia R, Gil-Nagel A. Bilateral akinetic seizures: a clinical and electroencephalographic description. *Epilepsia* 2010; 51(10): 2108-15.
53. Meletti S, Rubboli G, Testoni S, Michelucci R, Cantalupo G, Stanzani-Maserati M, et al. Early ictal speech and motor inhibition in fronto-mesial epileptic seizures: a polygraphic study in one patient. *Clin Neurophysiol* 2003; 114(1): 56-62.
54. Noachtar S, Lüders HO. Focal akinetic seizures as documented by electroencephalography and video recordings. *Neurology* 1999; 53(2): 427-9.
55. Lüders H, Dinner DS, Morris H, Wyllie E, Comair YG. Cortical electrical stimulation in humans. The negative motor areas. *Adv Neurol* 1995; 67: 115-29.
56. Lim SH, Dinner DS, Pillay PK, Lüders H, 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.
57. Scholly J, Bartolomei F, Valenti-Hirsch MP, Boulay C, De Saint Martin A, Timofeev A, et al. Atonic seizures in children with surgically remediable epilepsy: a motor system seizure phenotype? *Epileptic Disord* 2017; 19(3): 315-26.
58. Tassinari CA, Rubboli G, Parmeggiani L, Valzania F, Plasmati R, Riguzzi P, et al. Epileptic negative myoclonus. *Adv Neurol* 1995; 67: 181-97.
59. Saporito MAN, Vitaliti G, Pavone P, Di Stefano G, Striano P, Caraballo RH, et al. Ictal blinking, an under-recognized phenomenon: our experience and literature review. *Neuropsychiatr Dis Treat* 2017; 13: 1435.
60. Lagarde S, Dirani M, Trébuchon A, Lepine A, Villeneuve N, Scavarda D, et al. Ictal blinking in focal seizures: Insights from SEEG recordings. *Seizure* 2020; 81: 21-8.
61. Kalss G, Leitingner M, Dobesberger J, Granbichler CA, Kuchukhidze G, Trinkka E. Ictal unilateral eye blinking and contralateral blink inhibition – a video-EEG study and review of the literature. *Epilepsy Behav Case Rep* 2013; 1: 161-5.
62. Bonelli SB, Lurger S, Zimprich F, Stogmann E, Assem-Hilger E, Baumgartner C. Clinical seizure lateralization in frontal lobe epilepsy. *Epilepsia* 2007; 48(3): 517-23.
63. Janszky J, Fogarasi A, Jokeit H, Ebner A. Lateralizing value of unilateral motor and somatosensory manifestations in frontal lobe seizures. *Epilepsy Res* 2001; 43(2): 125-33.
64. Marshly A, Ewida A, Agarwal R, Younes K, Lüders HO. Ictal motor sequences: lateralization and localization values. *Epilepsia* 2016; 57(3): 369-75.
65. Jeavons PM. Nosological problems of myoclonic epilepsies in childhood and adolescence. *Dev Med Child Neurol* 1977; 19(1): 3-8.

66. Vaudano AE, Ruggieri A, Tondelli M, Avanzini P, Benuzzi F, Gessaroli G, et al. The visual system in eyelid myoclonia with absences. *Ann Neurol* 2014; 76(3): 412-27.
67. Doose H. Myoclonic-astatic epilepsy. *Epilepsy Res Suppl* 1992; 6: 163-8.
68. Kotagal P, Lüders H, Morris H, Dinner D, Wyllie E, Godoy J, et al. Dystonic posturing in complex partial seizures of temporal lobe onset: a new lateralizing sign. *Neurology* 1989; 39(2): 196-201.
69. Marks Jr WJ, Laxer KD. Semiology of temporal lobe seizures: value in lateralizing the seizure focus. *Epilepsia* 1998; 39(7): 721-6.
70. Yu H-Y, Yiu C-H, Yen D-J, Chen C, Guo Y-C, Kwan S-Y, et al. Lateralizing value of early head turning and ictal dystonia in temporal lobe seizures: a video-EEG study. *Seizure* 2001; 10(6): 428-32.
71. Dupont S, Semah F, Baulac M, Samson Y. The underlying pathophysiology of ictal dystonia in temporal lobe epilepsy: an FDG-PET study. *Neurology* 1998; 51(5): 1289-92.
72. Aguglia U, Gambardella A, Piane EL, Messina D, Russo C, Oliveri R, et al. Idiopathic generalized epilepsies with versive or circling seizures. *Acta Neurol Scand* 1999; 99(4): 219-24.
73. Dobesberger J, Walser G, Embacher N, Unterberger I, Luef G, Bauer G, et al. Gyrotory seizures revisited: a video-EEG study. *Neurology* 2005; 64(11): 1884-7.
74. Vercueil L, Kahane P, Francois-Joubert A, Hirsch E, Hoffmann D, Depaulis A, et al. Basal ganglia involvement in rotational seizures. *Epileptic Disord* 1999; 1(2): 107-12.
75. Aguglia U, Gambardella A, Quartarone A, Girlanda P, Le Piane E, Messina D, et al. Interhemispheric threshold differences in idiopathic generalized epilepsies with versive or circling seizures determined with focal magnetic transcranial stimulation. *Epilepsy Res* 2000; 40(1): 1-6.
76. Gire C, Somma-Mauvais H, Niçaise C, Roussel M, Garnier J-M, Farnarier G. Epileptic nystagmus: electroclinical study of a case. *Epileptic Disord* 2001; 3(1): 33-8.
77. Ma Y, Wang J, Li D, Lang S. Two types of isolated epileptic nystagmus: case report. *Int J Clin Exp Med* 2015; 8(8): 13500.
78. Globus M, Lavi E, Fich A, Abramsky O. Ictal hemiparesis. *Eur Neurol* 1982; 21(3): 165-8.
79. Dale RC, Cross JH. Ictal hemiparesis. *Dev Med Child Neurol* 1999; 41(5): 344-7.
80. Oono M, Uno H, Umesaki A, Nagatsuka K, Kinoshita M, Naritomi H. Severe and prolonged ictal paresis in an elderly patient. *Epilepsy Behav Case Rep* 2014; 2: 105-7.
81. Kuba R, Krízová J, Brázdil M, Tyrlíková I, Rektor I. Lateralized ictal immobility of the upper limb in patients with temporal lobe epilepsy. *Eur J Neurol* 2005; 12(11): 886-90.
82. Maillard L, Gavaret M, Régis J, Wendling F, Bartolomei F. Fast epileptic discharges associated with ictal negative motor phenomena. *Clin Neurophysiol* 2014; 125(12): 2344-8.
83. Pavone P, Polizzi A, Marino SD, Corsello G, Falsaperla R, Marino S, et al. West syndrome: a comprehensive review. *Neurol Sci* 2020; 41(12): 3547-62.
84. Loddenkemper T, Kotagal P. Lateralizing signs during seizures in focal epilepsy. *Epilepsy Behav* 2005; 7(1): 1-17.
85. Job AS, De Palma L, Principe A, Hoffmann D, Minotti L, Chabardès S, et al. The pivotal role of the supplementary motor area in startle epilepsy as demonstrated by SEEG epileptogenicity maps. *Epilepsia* 2014; 55(8): e85-8.
86. Werhahn KJ, Noachtar S, Arnold S, Pfänder M, Henkel A, Winkler PA, et al. Tonic seizures: their significance for lateralization and frequency in different focal epileptic syndromes. *Epilepsia* 2000; 41(9): 1153-61.
87. Chassagnon S, Minotti L, Kremer S, Hoffmann D, Kahane P. Somatosensory, motor, and reaching/grasping responses to direct electrical stimulation of the human cingulate motor areas. *J Neurosurg* 2008; 109(4): 593-604.
88. Souirti Z, Landré E, Mellerio C, Devaux B, Chassoux F. Neural network underlying ictal pouting ("chapeau de gendarme") in frontal lobe epilepsy. *Epilepsy Behav* 2014; 37: 249-57.
89. Tan Y-L, Muhlhofer W, Knowlton R. Pearls and Oysters: The chapeau de gendarme sign and other localizing gems in frontal lobe epilepsy. *Neurology* 2016; 87(10): e103-5.
90. Lu H, Zhang W, Liu S, Sun W. The chapeau de gendarme sign can be evoked by lateral prefrontal epileptic seizures. *Seizure* 2021; 87: 17-20.
91. Wiwchar LD, Hader W, Pauranik A, Joseph JT, Appendino JP. Focal seizures associated with the chapeau de gendarme sign or ictal pouting of insular origin. *Epilepsy Behav Rep* 2019; 12: 100347.
92. Cebeci D, Arhan E, Hirfanoglu T, Karalok ZS, Ercelebi H, Dedeoğlu Ö, et al. Ictal pouting ("Chapeau de gendarme") in three pediatric cases with cortical dysplasia. *Eur J Paediatr Neurol* 2020; 26: 82-8.
93. Rüscher CT, Bölsterli BK, Carosio C, Ramantani G. Chapeau de gendarme in a toddler points to focal epilepsy originating from the subcentral gyrus. *Epileptic Disord* 2021; 23(2): 412-8.
94. Ajmone-Marsan CRB. *The epileptic seizure: a clinical-electrographic analysis of Metrazol attacks*. Springfield, Illinois: Thomas, 1957.
95. Beniczky S, Rubboli G, Covanis A, Sperling MR. Absence-to-bilateral-tonic-clonic seizure: a generalized seizure type. *Neurology* 2020; 95(14): e2009-15.
96. Alexandre V, Mercedes B, Valton L, Maillard L, Bartolomei F, Szurhaj W, et al. Risk factors of postictal generalized EEG suppression in generalized convulsive seizures. *Neurology* 2015; 85(18): 1598-603.
97. Conradsen I, Wolf P, Sams T, Sorensen HB, Beniczky S. Patterns of muscle activation during generalized tonic and tonic-clonic epileptic seizures. *Epilepsia* 2011; 52(11): 2125-32.
98. Kotagal P, Bleasel A, Geller E, Kankirawatana P, Moorjani BI, Rybicki L. Lateralizing value of asymmetric tonic limb posturing observed in secondarily generalized tonic-clonic seizures. *Epilepsia* 2000; 41(4): 457-62.

99. Leutmezer F, Wöginger S, Antoni E, Seidl B, Baumgartner C. Asymmetric ending of secondarily generalized seizures. A lateralizing sign in TLE. *Neurology* 2002; 59(8): 1252-4.
100. Trinka E, Walser G, Unterberger I, Luef G, Benke T, Bartha L, et al. Asymmetric termination of secondarily generalized tonic-clonic seizures in temporal lobe epilepsy. *Neurology* 2002; 59(8): 1254-6.
101. Walser G, Unterberger I, Dobesberger J, Embacher N, Falkenstetter T, Larch J, et al. Asymmetric seizure termination in primary and secondary generalized tonic-clonic seizures. *Epilepsia* 2009; 50(9): 2035-9.
102. Wyllie E, Lüders H, Morris HH, Lesser RP, Dinner DS. The lateralizing significance of versive head and eye movements during epileptic seizures. *Neurology* 1986; 36(5): 606-11.
103. Lobel E, Kahane P, Leonards U, Grosbras M-H, Lehericy S, Le Bihan D, et al. Localization of human frontal eye fields: anatomical and functional findings of functional magnetic resonance imaging and intracerebral electrical stimulation. *J Neurosurg* 2001; 95(5): 804-15.
104. Kernan J, Devinsky O, Luciano D, Vazquez B, Perrine K. Lateralizing significance of head and eye deviation in secondary generalized tonic-clonic seizures. *Neurology* 1993; 43(7): 1308-10.
105. O'Dwyer R, Silva Cunha JP, Vollmar C, Mauerer C, Feddersen B, Burgess RC, et al. Lateralizing significance of quantitative analysis of head movements before secondary generalization of seizures of patients with temporal lobe epilepsy. *Epilepsia* 2007; 48(3): 524-30.
106. Abarrategui B, Mai R, Sartori I, Francione S, Pelliccia V, Cossu M, et al. Temporal lobe epilepsy: a never-ending story. *Epilepsy Behav* 2021; 122: 108122.
107. Bonini F, McGonigal A, Trébuchon A, Gavaret M, Bartolomei F, Giusiano B, et al. Frontal lobe seizures: from clinical semiology to localization. *Epilepsia* 2014; 55(2): 264-77.
108. Tassinari CA, Rubboli G, Gardella E, Cantalupo G, Calandra-Buonaura G, Vedovello M, et al. Central pattern generators for a common semiology in fronto-limbic seizures and in parasomnias. A neuroethologic approach. *Neurol Sci* 2005; 26(Suppl 3): s225-32.
109. Bartolomei F, Trébuchon A, Gavaret M, Régis J, Wendling F, Chauvel P. Acute alteration of emotional behaviour in epileptic seizures is related to transient desynchrony in emotion-regulation networks. *Clin Neurophysiol* 2005; 116(10): 2473-9.
110. Lee GR, Arain A, Lim N, Lagrange A, Singh P, Abou-Khalil B. Rhythmic ictal nonclonic hand (RINCH) motions: a distinct contralateral sign in temporal lobe epilepsy. *Epilepsia* 2006; 47(12): 2189-92.
111. Janszky J, Fogarasi A, Magalova V, Gyimesi C, Kovács N, Schulz R, et al. Unilateral hand automatisms in temporal lobe epilepsy. *Seizure* 2006; 15(6): 393-6.
112. Dede H, Bebek N, Gürses C, Baysal-Kıraç L, Baykan B, Gökyiğit A. Genital automatisms: reappraisal of a remarkable but ignored symptom of focal seizures. *Epilepsy Behav* 2018; 80: 84-9.
113. Horinouchi T, Sakurai K, Kurita T, Takeda Y, Yoshida Y, Akiyama H, et al. Seizure manifesting as a reaching/grasping movement in a patient with post-traumatic epilepsy. *Clin Case Rep* 2018; 6(11): 2271-5.
114. Gardella E, Rubboli G, Tassinari CA. Video-EEG analysis of ictal repetitive grasping in "frontal-hyperkinetic" seizures. *Epileptic Disord* 2006; 8(4): 267-73.
115. Caruana F, Gerbella M, Avanzini P, Gozzo F, Pelliccia V, Mai R, et al. Motor and emotional behaviours elicited by electrical stimulation of the human cingulate cortex. *Brain* 2018; 141(10): 3035-51.
116. Arroyo S, Lesser RP, Gordon B, Uematsu S, Hart J, Schwerdt P, et al. Mirth, laughter and gelastic seizures. *Brain* 1993; 116(Pt 4): 757-80.
117. Iapadre G, Zagaroli L, Cimini N, Belcastro V, Concolino D, Coppola G, et al. Gelastic seizures not associated with hypothalamic hamartoma: a long-term follow-up study. *Epilepsy Behav* 2020; 103(Pt A): 106578.
118. Oehl B, Biethahn S, Schulze-Bonhage A. Mirthful gelastic seizures with ictal involvement of temporobasal regions. *Epileptic Disord* 2009; 11(1): 82-6.
119. Asadi-Pooya AA, Wyeth D, Sperling MR. Ictal crying. *Epilepsy Behav* 2016; 59: 1-3.
120. Blumberg J, Fernández IS, Vendrame M, Oehl B, Tatum WO, Schuele S, et al. Dacrystic seizures: demographic, semiologic, and etiologic insights from a multicenter study in long-term video-EEG monitoring units. *Epilepsia* 2012; 53(10): 1810-9.
121. Caruana F, Avanzini P, Gozzo F, Francione S, Cardinale F, Rizzolatti G. Mirth and laughter elicited by electrical stimulation of the human anterior cingulate cortex. *Cortex* 2015; 71: 323-31.
122. Sperli F, Spinelli L, Pollo C, Seeck M. Contralateral smile and laughter, but no mirth, induced by electrical stimulation of the cingulate cortex. *Epilepsia* 2006; 47(2): 440-3.
123. Sturm JW, Andermann F, Berkovic SF. "Pressure to laugh": an unusual epileptic symptom associated with small hypothalamic hamartomas. *Neurology* 2000; 54(4): 971-3.
124. Hays RS, Lal N, Rosenow J, Macken MP, Schuele SU. Mimetic automatisms expressing a negative affect in two patients with temporal lobe epilepsy. *Epilepsy Behav* 2011; 20(3): 572-8.
125. Tassinari CA, Tassi L, Calandra-Buonaura G, Stanzani-Maserati M, Fini N, Pizza F, et al. Biting behavior, aggression, and seizures. *Epilepsia* 2005; 46(5): 654-63.
126. Özkara Ç, Taşkıran E, Çarpraz İY, Bilir E, Demir AB, Bora İ, et al. Ictal kissing: electroclinical features of an unusual ictal phenomenon. *Seizure* 2016; 42: 44-8.
127. Guedj E, Guye M, de Laforte C, Chauvel P, Liegeois-Chauvel C, Mandler O, et al. Neural network underlying ictal humming demonstrated by very early SPECT: a case report. *Epilepsia* 2006; 47(11): 1968-70.

128. Raghavendra S, Mirsattari S, McLachlan RS. Ictal whistling: a rare automatism during temporal lobe seizures. *Epileptic Disord* 2010; 12(2): 133-5.
129. Rini J, Ochoa J. Mapping musical automatism: further insights from epileptic high-frequency oscillation analysis. *Neurol Clin Neurosci* 2020; 8(4): 177-82.
130. Aupy J, Noviawaty I, Krishnan B, Suwankpakdee P, Bulacio J, Gonzalez-Martinez J, et al. Insulo-opercular cortex generates oroalimentary automatisms in temporal seizures. *Epilepsia* 2018; 59(3): 583-94.
131. Wang Y, Wang X, Mo JJ, Sang L, Zhao BT, Zhang C, et al. Symptomatogenic zone and network of oroalimentary automatisms in mesial temporal lobe epilepsy. *Epilepsia* 2019; 60(6): 1150-9.
132. Ebner A, Dinner DS, Noachtar S, Lüders H. Automatisms with preserved responsiveness: a lateralizing sign in psychomotor seizures. *Neurology* 1995; 45(1): 61-4.
133. Montavont A, Kahane P, Guenot M, Ryvlin P. Foreign language ictal speech automatisms in nondominant temporal lobe epilepsy. *Neurology* 2008; 71(20): 1579-85.
134. Hartl E, Knoche T, Choupina HMP, Rémi J, Vollmar C, Cunha JPS, et al. Quantitative and qualitative analysis of ictal vocalization in focal epilepsy syndromes. *Seizure* 2018; 60: 178-83.
135. Horvath RA, Fogarasi A, Schulz R, Perlaki G, Kalmar Z, Tóth V, et al. Ictal vocalizations occur more often in temporal lobe epilepsy with dominant (left-sided) epileptogenic zone. *Epilepsia* 2009; 50(6): 1542-6.
136. Janszky J, Fogarasi A, Jokeit H, Ebner A. Are ictal vocalisations related to the lateralisation of frontal lobe epilepsy? *J Neurol Neurosurg Psychiatry* 2000; 69(2): 244-7.
137. Rheims S, Ryvlin P, Scherer C, Minotti L, Hoffmann D, Guenot M, et al. Analysis of clinical patterns and underlying epileptogenic zones of hypermotor seizures. *Epilepsia* 2008; 49(12): 2030-40.
138. Fayerstein J, McGonigal A, Pizzo F, Bonini F, Lagarde S, Braquet A, et al. Quantitative analysis of hyperkinetic seizures and correlation with seizure onset zone. *Epilepsia* 2020; 61(5): 1019-26.
139. Zhang C, Zhao BT, McGonigal A, Hu WH, Wang X, Shao XQ, et al. Superior frontal sulcus focal cortical dysplasia type II: an MRI, PET and quantified SEEG study. *Front Neurol* 2019; 10: 1253.
140. Gibbs SA, Proserpio P, Terzaghi M, Pigorini A, Sarasso S, Lo Russo G, et al. Sleep-related epileptic behaviors and non-REM-related parasomnias: insights from stereo-EEG. *Sleep Med Rev* 2016; 25: 4-20.
141. De Graaf JB L-CC, Vignal JP, Chauvel P. Electrical stimulation of the auditory cortex. *Epileptic Seizures Pathophysiol Clin Semiology*. NY: Churchill Livingstone, 2000: 228-36.
142. Lüders H. *Textbook of epilepsy surgery*. CRC Press, 2008.
143. Penfield W, Perot P. The brain's record of auditory and visual experience. a final summary and discussion. *Brain* 1963; 86: 595-696.
144. 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(12): 1590-9.
145. Afif A, Minotti L, Kahane P, Hoffmann D. Anatomofunctional organization of the insular cortex: a study using intracerebral electrical stimulation in epileptic patients. *Epilepsia* 2010; 51(11): 2305-15.
146. Florindo I, Bisulli F, Pittau F, Naldi I, Striano P, Striano S, et al. Lateralizing value of the auditory aura in partial seizures. *Epilepsia* 2006; 47(Suppl 5): 68-72.
147. Rona SLH. *Auras: localizing and lateralizing value*. In: Textbook of epilepsy surgery. CRC Press, 2008.
148. Jonas J, Frismand S, Vignal JP, Colnat-Coulbois S, Koessler L, Vespignani H, et al. Right hemispheric dominance of visual phenomena evoked by intracerebral stimulation of the human visual cortex. *Hum Brain Mapp* 2014; 35(7): 3360-71.
149. Murphey DK, Maunsell JH, Beauchamp MS, Yoshor D. Perceiving electrical stimulation of identified human visual areas. *Proc Natl Acad Sci U S A* 2009; 106(13): 5389-93.
150. Salanova V, Andermann F, Olivier 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(Pt 6): 1655-80.
151. Bien CG, Benninger FO, Urbach H, Schramm J, Kurthen M, Elger CE. Localizing value of epileptic visual auras. *Brain* 2000; 123(2): 244-53.
152. Laff R, Mesad S, Devinsky O. Epileptic kinetopsia: ictal illusory motion perception. *Neurology* 2003; 61(9): 1262-4.
153. Rauschecker AM, Dastjerdi M, Weiner KS, Witthoft N, Chen J, Selimbeyoglu A, et al. Illusions of visual motion elicited by electrical stimulation of human MT complex. *PLoS One* 2011; 6(7): e21798.
154. Richer F, Martinez M, Cohen H, Saint-Hilaire JM. Visual motion perception from stimulation of the human medial parieto-occipital cortex. *Exp Brain Res* 1991; 87(3): 649-52.
155. Jonas J, Descoins M, Koessler L, Colnat-Coulbois S, Sauvé M, Guye M, et al. Focal electrical intracerebral stimulation of a face-sensitive area causes transient prosopagnosia. *Neuroscience* 2012; 222: 281-8.
156. Rangarajan V, Hermes D, Foster BL, Weiner KS, Jacques C, Grill-Spector K, et al. Electrical stimulation of the left and right human fusiform gyrus causes different effects in conscious face perception. *J Neurosci* 2014; 34(38): 12828-36.
157. Mégevand P, Groppe DM, Goldfinger MS, Hwang ST, Kingsley PB, Davidesco I, et al. Seeing scenes: topographic visual hallucinations evoked by direct electrical stimulation of the parahippocampal place area. *J Neurosci* 2014; 34(16): 5399-405.
158. Blume WT, Wiebe S, Tapsell LM. Occipital epilepsy: lateral versus mesial. *Brain* 2005; 128(Pt 5): 1209-25.
159. Williamson PD, Thadani VM, Darcey TM, Spencer DD, Spencer SS, Mattson RH. Occipital lobe epilepsy: clinical

- characteristics, seizure spread patterns and results of surgery. *Ann Neurol* 1992; 31(1): 3-13.
160. Jonas J, Rössion B, Brissart H, Frismand S, Jacques C, Hossu G, *et al.* Beyond the core face-processing network: Intracerebral stimulation of a face-selective area in the right anterior fusiform gyrus elicits transient prosopagnosia. *Cortex* 2015; 72: 140-55.
161. Parvizi J, Jacques C, Foster BL, Witthoft N, Rangarajan V, Weiner KS, *et al.* Electrical stimulation of human fusiform face-selective regions distorts face perception. *J Neurosci* 2012; 32(43): 14915-20.
162. Hausser-Hauw C, Bancaud J. Gustatory hallucinations in epileptic seizures. Electrophysiological, clinical and anatomical correlates. *Brain* 1987; 110(Pt 2): 339-59.
163. Mazzola L, Royet JP, Catenoux H, Montavont A, Isnard J, Mauguière F. Gustatory and olfactory responses to stimulation of the human insula. *Ann Neurol* 2017; 82(3): 360-70.
164. Kanemoto K, Janz D. The temporal sequence of aura-sensations in patients with complex focal seizures with particular attention to ictal aphasia. *J Neurol Neurosurg Psychiatry* 1989; 52(1): 52-6.
165. Kim DW, Lee SK, Yun CH, Kim KK, Lee DS, Chung CK, *et al.* Parietal lobe epilepsy: the semiology, yield of diagnostic workup and surgical outcome. *Epilepsia* 2004; 45(6): 641-9.
166. Perven G, So NK. Epileptic auras: phenomenology and neurophysiology. *Epileptic Disord* 2015; 17(4): 349-62.
167. Isnard J, Hagiwara K, Montavont A, Catenoux H, Mazzola L, Ostrowsky-Coste K, *et al.* Semiology of insular lobe seizures. *Rev Neurol* 2019; 175(3): 144-9.
168. Acharya V, Acharya J, Lüders H. Olfactory epileptic auras. *Neurology* 1998; 51(1): 56-61.
169. Kumar G, Juhász C, Sood S, Asano E. Olfactory hallucinations elicited by electrical stimulation *via* subdural electrodes: effects of direct stimulation of olfactory bulb and tract. *Epilepsy Behav* 2012; 24(2): 264-8.
170. Binder DK, Garcia PA, Elangovan GK, Barbaro NM. Characteristics of auras in patients undergoing temporal lobectomy. *J Neurosurg* 2009; 111(6): 1283-9.
171. Ferrari-Marinho T, Caboclo LO, Marinho MM, Centeno RS, Neves RS, Santana MT, *et al.* Auras in temporal lobe epilepsy with hippocampal sclerosis: relation to seizure focus laterality and post surgical outcome. *Epilepsy Behav* 2012; 24(1): 120-5.
172. Gloor P, Olivier A, Quesney LF, Andermann F, Horowitz S. The role of the limbic system in experiential phenomena of temporal lobe epilepsy. *Ann Neurol* 1982; 12(2): 129-44.
173. Salanova V, Andermann F, Rasmussen T, Olivier A, Quesney LF. Parietal lobe epilepsy. Clinical manifestations and outcome in 82 patients treated surgically between 1929 and 1988. *Brain* 1995; 118(Pt 3): 607-27.
174. Bartolomei F, Gavaret M, Hewett R, Valton L, Aubert S, Régis J, *et al.* Neural networks underlying parietal lobe seizures: a quantified study from intracerebral recordings. *Epilepsy Res* 2011; 93(2-3): 164-76.
175. Mazzola L, Isnard J, Mauguière F. Somatosensory and pain responses to stimulation of the second somatosensory area (SII) in humans. A comparison with S1 and insular responses. *Cereb Cortex* 2006; 16(7): 960-8.
176. Lim S, Dinner D, Pillay P, Lüders H, Morris H, 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.
177. Young GB, Blume WT. Painful epileptic seizures. *Brain* 1983; 106(Pt 3): 537-54.
178. Mazzola L, Isnard J, Peyron R, Mauguière F. Stimulation of the human cortex and the experience of pain: wilder Penfield's observations revisited. *Brain* 2012; 135(Pt 2): 631-40.
179. Montavont A, Mauguière F, Mazzola L, Garcia-Larrea L, Catenoux H, Ryvlin P, *et al.* On the origin of painful somatosensory seizures. *Neurology* 2015; 84(6): 594-601.
180. Mauguière F, Courjon J. Somatosensory epilepsy. A review of 127 cases. *Brain* 1978; 101(2): 307-32.
181. Bernasconi A, Andermann F, Bernasconi N, Reutens DC, Dubeau F. Lateralizing value of peri-ictal headache: a study of 100 patients with partial epilepsy. *Neurology* 2001; 56(1): 130-2.
182. Hewett R, Bartolomei F. Epilepsy and the cortical vestibular system: tales of dizziness and recent concepts. *Front Integr Neurosci* 2013; 7: 73.
183. Kahane P, Hoffmann D, Minotti L, Berthoz A. Reappraisal of the human vestibular cortex by cortical electrical stimulation study. *Ann Neurol* 2003; 54(5): 615-24.
184. Kun Lee S, Young Lee S, Kim DW, Soo Lee D, Chung CK. Occipital lobe epilepsy: clinical characteristics, surgical outcome, and role of diagnostic modalities. *Epilepsia* 2005; 46(5): 688-95.
185. Blanke O, Landis T, Spinelli L, Seeck M. Out-of-body experience and autoscopia of neurological origin. *Brain* 2004; 127(2): 243-58.
186. Maillard L, Vignal JP, Anxionnat R, Taillandier-Vespignani L. Semiologic value of ictal autoscopia. *Epilepsia* 2004; 45(4): 391-4.
187. Jonas J, Maillard L, Frismand S, Colnat-Coulbois S, Vespignani H, Rössion B, *et al.* Self-face hallucination evoked by electrical stimulation of the human brain. *Neurology* 2014; 83(4): 336-8.
188. Arzy S, Seeck M, Ortigue S, Spinelli L, Blanke O. Induction of an illusory shadow person. *Nature* 2006; 443(7109): 287.
189. American Psychiatric Association. . *Diagnostic and statistical manual of mental disorders (DSM-5[®])*. American Psychiatric Pub, 2013.
190. Stephani C, Koubeissi M. Hypercognitive seizures – Proposal of a new term for the phenomenon forced thinking in epilepsy. *Epilepsy Res* 2017; 134: 63-71.
191. Mendez MF, Cherrier MM, Perryman KM. Epileptic forced thinking from left frontal lesions. *Neurology* 1996; 47(1): 79-83.

192. McGonigal A, Chauvel P. Frontal lobe epilepsy: seizure semiology and presurgical evaluation. *Pract Neurol* 2004; 4(5): 260-73.
193. Johanson M, Valli K, Revonsuo A, Wedlund JE. Content analysis of subjective experiences in partial epileptic seizures. *Epilepsy Behav* 2008; 12(1): 170-82.
194. Yih J, Beam DE, Fox KC, Parvizi J. Intensity of affective experience is modulated by magnitude of intracranial electrical stimulation in human orbitofrontal, cingulate and insular cortices. *Soc Cogn Affect Neurosci* 2019; 14(4): 339-51.
195. Fournier NM, Brandt LE, Kalynchuk LE. The effect of left and right long-term amygdala kindling on interictal emotionality and Fos expression. *Epilepsy Behav* 2020; 104(Pt A): 106910.
196. Fox KCR, Yih J, Raccah O, Pendekanti SL, Limbach LE, Maydan DD, et al. Changes in subjective experience elicited by direct stimulation of the human orbitofrontal cortex. *Neurology* 2018; 91(16): e1519-27.
197. Toth V, Fogarasi A, Karadi K, Kovacs N, Ebner A, Janszky J. Ictal affective symptoms in temporal lobe epilepsy are related to gender and age. *Epilepsia* 2010; 51(7): 1126-32.
198. Mathiak KA, Mathiak K, Wolańczyk T, Ostaszewski P. Psychosocial impairments in children with epilepsy depend on the side of the focus. *Epilepsy Behav* 2009; 16(4): 603-8.
199. LaBar KS, LeDoux JE, Spencer DD, Phelps EA. Impaired fear conditioning following unilateral temporal lobectomy in humans. *J Neurosci* 1995; 15(10): 6846-55.
200. van Elst LT, Woermann FG, Lemieux L, Thompson PJ, Trimble MR. Affective aggression in patients with temporal lobe epilepsy: a quantitative MRI study of the amygdala. *Brain* 2000; 123(Pt 2): 234-43.
201. Davidson RJ, Putnam KM, Larson CL. Dysfunction in the neural circuitry of emotion regulation – a possible prelude to violence. *Science* 2000; 289(5479): 591-4.
202. Saleh C, Reuber M, Beyenburg S. Epileptic seizures and criminal acts: is there a relationship? *Epilepsy Behav* 2019; 97: 15-21.
203. Saegusa S, Takahashi T, Moriya J, Yamakawa J, Itoh T, Kusaka K, et al. Panic attack symptoms in a patient with left temporal lobe epilepsy. *J Int Med Res* 2004; 32(1): 94-6.
204. Sazgar M, Carlen PL, Wennberg R. Panic attack semiology in right temporal lobe epilepsy. *Epileptic Disord* 2003; 5(2): 93-100.
205. Bartolomeil F, Guye M, Wendling F, Gavaret M, Régis J, Chauvel P. Fear, anger and compulsive behavior during seizure: involvement of large scale fronto-temporal neural networks. *Epileptic Disord* 2002; 4(4): 235-41.
206. Altshuler LL, Devinsky O, Post RM, Theodore W. Depression, anxiety, and temporal lobe epilepsy. Laterality of focus and symptoms. *Arch Neurol* 1990; 47(3): 284-8.
207. Devinsky O, Barr WB, Vickrey BG, Berg AT, Bazil CW, Pacia SV, et al. Changes in depression and anxiety after resective surgery for epilepsy. *Neurology* 2005; 65(11): 1744-9.
208. LeDoux JE. Emotion circuits in the brain. *Annu Rev Neurosci* 2000; 23: 155-84.
209. Young GB, Chandarana PC, Blume WT, McLachlan RS, Muñoz DG, Girvin JP. Mesial temporal lobe seizures presenting as anxiety disorders. *J Neuropsychiatry Clin Neurosci* 1995; 7(3): 352-7.
210. Hingray C, McGonigal A, Kotwas I, Micoulaud-Franchi JA. The relationship between epilepsy and anxiety disorders. *Curr Psychiatry Rep* 2019; 21(6): 40.
211. Hurley RA, Fisher R, Taber KH. Sudden onset panic: epileptic aura or panic disorder? *J Neuropsychiatry Clin Neurosci* 2006; 18(4): 436-43.
212. Gloor P. Experiential phenomena of temporal lobe epilepsy. Facts and hypotheses. *Brain* 1990; 113(Pt 6): 1673-94.
213. Mula M. Epilepsy-induced behavioral changes during the ictal phase. *Epilepsy Behav* 2014; 30: 14-6.
214. Guimond A, Braun CM, Bélanger E, Rouleau I. Ictal fear depends on the cerebral laterality of the epileptic activity. *Epileptic Disord* 2008; 10(2): 101-12.
215. Labudda K, Mertens M, Steinkroeger C, Bien CG, Woermann FG. Lesion side matters – an fMRI study on the association between neural correlates of watching dynamic fearful faces and their evaluation in patients with temporal lobe epilepsy. *Epilepsy Behav* 2014; 31: 321-8.
216. Urbanic PT, Zaar K, Eder H, Gruber-Cichocky L, Feichtinger M. Ictal fear auras after selective amygdalohypocampectomy: the use of ictal SPECT and scalp EEG in the presurgical reevaluation. *Epilepsy Behav* 2011; 22(3): 577-80.
217. Gläscher J, Adolphs R. Processing of the arousal of subliminal and supraliminal emotional stimuli by the human amygdala. *J Neurosci* 2003; 23(32): 10274-82.
218. Manfred M, Fish DR, Shorvon SD. An analysis of clinical seizure patterns and their localizing value in frontal and temporal lobe epilepsies. *Brain* 1996; 119(Pt 1): 17-40.
219. Biraben A, Taussig D, Thomas P, Even C, Vignal JP, Scarabin JM, et al. Fear as the main feature of epileptic seizures. *J Neurol Neurosurg Psychiatry* 2001; 70(2): 186-91.
220. Nair PP, Menon RN, Radhakrishnan A, Cherian A, Abraham M, Vilanilam G, et al. Is 'burned-out hippocampus' syndrome a distinct electro-clinical variant of MTL-HS syndrome? *Epilepsy Behav* 2017; 69: 53-8.
221. Meletti S, Tassi L, Mai R, Fini N, Tassinari CA, Russo GL. Emotions induced by intracerebral electrical stimulation of the temporal lobe. *Epilepsia* 2006; 47(Suppl 5): 47-51.
222. Chang CN, Ojemann LM, Ojemann GA, Lettich E. Seizures of fronto-orbital origin: a proven case. *Epilepsia* 1991; 32(4): 487-91.
223. Alkawadri R, So NK, Van Ness PC, Alexopoulos AV. Cingulate epilepsy: report of 3 electroclinical subtypes with surgical outcomes. *JAMA Neurol* 2013; 70(8): 995-1002.
224. Wang J, Wang Q, Wang M, Luan G, Zhou J, Guan Y, et al. Occipital lobe epilepsy with ictal fear: evidence from a stereo-electroencephalography (sEEG) case. *Front Neurol* 2018; 9: 644.

225. Oehl B, Schulze-Bonhage A, Lanz M, Brandt A, Altenmüller DM. Occipital lobe epilepsy with fear as leading ictal symptom. *Epilepsy Behav* 2012; 23(3): 379-83.
226. Nitta N, Usui N, Kondo A, Tottori T, Terada K, Araki Y, et al. Semiology of hyperkinetic seizures of frontal versus temporal lobe origin. *Epileptic Disord* 2019; 21(2): 154-65.
227. Williams D. The structure of emotions reflected in epileptic experiences. *Brain* 1956; 79(1): 29-67.
228. Singh TD, Sabsevitz DS, Desai NN, Middlebrooks EH, Feyissa AM, Grewal S, et al. Crying with depressed affect induced by electrical stimulation of the anterior insula: a stereo EEG case study. *Epilepsy Behav Rep* 2021; 15: 100421.
229. ILAE. *Emotional seizures*. www.epilepsydiagnosis.org [6/30/2021].
230. Perini G, Mendius R. Depression and anxiety in complex partial seizures. *J Nerv Ment Dis* 1984; 172(5): 287-90.
231. Bartolomei F, Lagarde S, Scavarda D, Carron R, Bénar CG, Picard F. The role of the dorsal anterior insula in ecstatic sensation revealed by direct electrical brain stimulation. *Brain Stimul* 2019; 12(5): 1121-6.
232. Carrazana E, Cheng J. St Theresa's dart and a case of religious ecstatic epilepsy. *Cogn Behav Neurol* 2011; 24(3): 152-5.
233. Asheim Hansen B, Brodtkorb E. Partial epilepsy with "ecstatic" seizures. *Epilepsy Behav* 2003; 4(6): 667-73.
234. Gschwind M, Picard F. Ecstatic epileptic seizures: a glimpse into the multiple roles of the insula. *Front Behav Neurosci* 2016; 10: 21.
235. Picard F, Craig AD. Ecstatic epileptic seizures: a potential window on the neural basis for human self-awareness. *Epilepsy Behav* 2009; 16(3): 539-46.
236. Picard F, Kurth F. Ictal alterations of consciousness during ecstatic seizures. *Epilepsy Behav* 2014; 30: 58-61.
237. Meyer GMM, Martin W, McGraw CP. Stereotactic cingulotomy with results of acute stimulation and serial psychological testing. In : Laitinen LV, ed. *Surgical approaches in psychiatry*. Lancaster (UK): Baltimore: 39-58.
238. Talairach J, Bancaud J, Geier S, Bordas-Ferrer M, Bonis A, Szikla G, et al. The cingulate gyrus and human behaviour. *Electroencephalogr Clin Neurophysiol* 1973; 34(1): 45-52.
239. Penfield WKK. *Epileptic seizure patterns: a study of the localizing value of initial phenomena in focal cortical seizures*. Springfield, Illinois: Thomas, 1951.
240. Vera CL, Patel SJ, Naso W. "Dual pathology" and the significance of surgical outcome in "Dostoevsky's epilepsy". *Epileptic Disord* 2000; 2(1): 21-5.
241. Stefan H, Schulze-Bonhage A, Pauli E, Platsch G, Quiske A, Buchfelder M, et al. Ictal pleasant sensations: cerebral localization and lateralization. *Epilepsia* 2004; 45(1): 35-40.
242. Roodakker KR, Ezra B, Gauffin H, Latini F, Zetterling M, Berntsson S, et al. Ecstatic and gelastic seizures relate to the hypothalamus. *Epilepsy Behav Rep* 2020; 14: 100358.
243. Cabrera-Valdivia F, Jiménez-Jiménez FJ, Tejeiro J, Ayuso-Peralta L, Vaquero A, Garcia-Albea E. Dostoevsky's epilepsy induced by television. *J Neurol Neurosurg Psychiatry* 1996; 61(6): 653.
244. Dolgoff-Kaspar R, Ettinger AB, Golub SA, Perrine K, Harden C, Croll SD. Numinous-like auras and spirituality in persons with partial seizures. *Epilepsia* 2011; 52(3): 640-4.
245. Saver JL, Rabin J. The neural substrates of religious experience. *J Neuropsychiatry Clin Neurosci* 1997; 9(3): 498-510.
246. Devinsky O, Lai G. Spirituality and religion in epilepsy. *Epilepsy Behav* 2008; 12(4): 636-43.
247. Janszky J, Ebner A, Szupera Z, Schulz R, Hollo A, Szücs A, et al. Orgasmic aura – a report of seven cases. *Seizure* 2004; 13(6): 441-4.
248. Aull-Watschinger S, Pataria E, Baumgartner C. Sexual auras: predominance of epileptic activity within the mesial temporal lobe. *Epilepsy Behav* 2008; 12(1): 124-7.
249. Remillard GM, Andermann F, Testa GF, Gloor P, Aube M, Martin JB, et al. Sexual ictal manifestations predominate in women with temporal lobe epilepsy: a finding suggesting sexual dimorphism in the human brain. *Neurology* 1983; 33(3): 323-30.
250. Spencer SS, Spencer DD, Williamson PD, Mattson RH. Sexual automatisms in complex partial seizures. *Neurology* 1983; 33(5): 527-33.
251. Felician O, Tramoni E, Bartolomei F. Transient epileptic amnesia: update on a slowly emerging epileptic syndrome. *Rev Neurol (Paris)* 2015; 171(3): 289-97.
252. Heydrich L, Marillier G, Evans N, Seeck M, Blanke O. Depersonalization- and derealization-like phenomena of epileptic origin. *Ann Clin Transl Neurol* 2019; 6(9): 1739-47.
253. 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.
254. Loesch AM, Steger H, Losher C, Hartl E, Rémi J, Vollmar C, et al. Seizure-associated aphasia has good lateralizing but poor localizing significance. *Epilepsia* 2017; 58(9): 1551-5.
255. Eggleston KS, Olin BD, Fisher RS. Ictal tachycardia: the head-heart connection. *Seizure* 2014; 23(7): 496-505.
256. Garcia M, D'Giano C, Estellés S, Leiguarda R, Rabonowicz A. Ictal tachycardia: its discriminating potential between temporal and extratemporal seizure foci. *Seizure* 2001; 10(6): 415-9.
257. Weil S, Arnold S, Eisensehr I, Noachtar S. Heart rate increase in otherwise subclinical seizures is different in temporal versus extratemporal seizure onset: support for temporal lobe autonomic influence. *Epileptic Disord* 2005; 7(3): 199-204.
258. Pavlova M, Abdennadher M, Singh K, Katz E, Llewellyn N, Zarowsky M, et al. Advantages of respiratory monitoring during video-EEG evaluation to differentiate epileptic seizures from other events. *Epilepsy Behav* 2014; 32: 142-4.

259. Moseley BD, Wirrell EC, Nickels K, Johnson JN, Ackerman MJ, Britton J. Electrocardiographic and oximetric changes during partial complex and generalized seizures. *Epilepsy Res* 2011; 95(3): 237-45.
260. Jaychandran R, Chaitanya G, Satishchandra P, Bharath RD, Thennarasu K, Sinha S. Monitoring peri-ictal changes in heart rate variability, oxygen saturation and blood pressure in epilepsy monitoring unit. *Epilepsy Res* 2016; 125: 10-8.
261. van der Lende M, Arends JB, Lamberts RJ, Tan HL, de Lange FJ, Sander JW, et al. The yield of long-term electrocardiographic recordings in refractory focal epilepsy. *Epilepsia* 2019; 60(11): 2215-23.
262. Walker F, Fish DR. Recording respiratory parameters in patients with epilepsy. *Epilepsia* 1997; 38(11 Suppl): S41-2.
263. Leutmezer F, Scherthner C, Lurger S, Pötzelberger K, Baumgartner C. Electrocardiographic changes at the onset of epileptic seizures. *Epilepsia* 2003; 44(3): 348-54.
264. Bartlam R, Mohanraj R. Ictal bradyarrhythmias and asystole requiring pacemaker implantation: combined EEG-ECG analysis of 5 cases. *Epilepsy Behav* 2016; 64(Pt A): 212-5.
265. Monté CP, Monté CJ, Boon P, Arends J. Epileptic seizures associated with syncope: ictal bradycardia and ictal asystole. *Epilepsy Behav* 2019; 90: 168-71.
266. Strzelczyk A, Cenusa M, Bauer S, Hamer HM, Mothersill IW, Grunwald T, et al. Management and long-term outcome in patients presenting with ictal asystole or bradycardia. *Epilepsia* 2011; 52(6): 1160-7.
267. Tinuper P, Bisulli F, Cerullo A, Carcangiu R, Marini C, Pierangeli G, et al. Ictal bradycardia in partial epileptic seizures: autonomic investigation in three cases and literature review. *Brain* 2001; 124(Pt 12): 2361-71.
268. Reeves AL, Nolle KE, Klass DW, Sharbrough FW, So EL. The ictal bradycardia syndrome. *Epilepsia* 1996; 37(10): 983-7.
269. Schuele SU, Bermeo AC, Alexopoulos AV, Locatelli ER, Burgess RC, Dinner DS, et al. Video-electrographic and clinical features in patients with ictal asystole. *Neurology* 2007; 69(5): 434-41.
270. Britton JW, Ghearing GR, Benarroch EE, Cascino GD. The ictal bradycardia syndrome: localization and lateralization. *Epilepsia* 2006; 47(4): 737-44.
271. van der Lende M, Surges R, Sander JW, Thijs RD. Cardiac arrhythmias during or after epileptic seizures. *J Neurol Neurosurg Psychiatry* 2016; 87(1): 69-74.
272. Leung H, Schindler K, Kwan P, Elger C. Asystole induced by electrical stimulation of the left cingulate gyrus. *Epileptic Disord* 2007; 9(1): 77-81.
273. Behbahani S, Dabanloo NJ, Nasrabadi AM, Dourado A. Classification of ictal and seizure-free HRV signals with focus on lateralization of epilepsy. *Technol Health Care* 2016; 24(1): 43-56.
274. Nashef L, Walker F, Allen P, Sander JW, Shorvon SD, Fish DR. Apnoea and bradycardia during epileptic seizures: relation to sudden death in epilepsy. *J Neurol Neurosurg Psychiatry* 1996; 60(3): 297-300.
275. Ghearing GR, Munger TM, Jaffe AS, Benarroch EE, Britton JW. Clinical cues for detecting ictal asystole. *Clin Auton Res* 2007; 17(4): 221-6.
276. Van Buren JM. The abdominal aura. A study of abdominal sensations occurring in epilepsy and produced by depth stimulation. *Electroencephalogr Clin Neurophysiol* 1963; 15: 1-19.
277. Mulder DW, Daly D, Bailey AA. Visceral epilepsy. *AMA Arch Intern Med* 1954; 93(4): 481-93.
278. Specchio N, Trivisano M, Di Ciommo V, Cappelletti S, Masciarelli G, Volkov J, et al. Panayiotopoulos syndrome: a clinical, EEG, and neuropsychological study of 93 consecutive patients. *Epilepsia* 2010; 51(10): 2098-107.
279. Fogarasi A, Janszky J, Tuxhorn I. Autonomic symptoms during childhood partial epileptic seizures. *Epilepsia* 2006; 47(3): 584-8.
280. van Dijk JG, Thijs RD, van Zwet E, Tannemaat MR, van Niekerk J, Benditt DG, et al. The semiology of tilt-induced reflex syncope in relation to electroencephalographic changes. *Brain* 2014; 137(Pt 2): 576-85.
281. Fogarasi A, Janszky J, Tuxhorn I. Ictal pallor is associated with left temporal seizure onset zone in children. *Epilepsy Res* 2005; 67(3): 117-21.
282. Cendes F, Andermann F, Gloor P, Gambardella A, Lopes-Cendes I, Watson C, et al. Relationship between atrophy of the amygdala and ictal fear in temporal lobe epilepsy. *Brain* 1994; 117(Pt 4): 739-46.
283. García-Herrero D, Fernández-Torre JL, Barrasa J, Calleja J, Pascual J. Abdominal epilepsy in an adolescent with bilateral perisylvian polymicrogyria. *Epilepsia* 1998; 39(12): 1370-4.
284. Yalçın AD, Ertaşoğlu Toydemir H. Panayiotopoulos syndrome with a special emphasis of ictal semiologic features. *Epilepsy Res* 2017; 131: 37-43.
285. Klein CJ, Silber MH, Halliwill JR, Schreiner SA, Suarez GA, Low PA. Basal forebrain malformation with hyperhidrosis and hypothermia: variant of Shapiro's syndrome. *Neurology* 2001; 56(2): 254-6.
286. Mendler MH, Sautereau D, Pillegand B, Ravon R. A case of digestive epilepsy with late diagnosis: a disease not to be disregarded. *Gastroenterol Clin Biol* 1998; 22(2): 235-9.
287. Cortés V, Landete L, Gómez E, Blasco R. Partial simple vegetative crisis: importance of electroencephalographic findings. *Rev Neurol* 1997; 25(148): 1931-3.
288. Franco AC, Noachtar S, Rémi J. Ictal ipsilateral sweating in focal epilepsy. *Seizure* 2017; 50: 4-5.
289. Rocamora R, Becerra JL, Fossas P, Gomez M, Vivanco-Hidalgo RM, Mauri JA, et al. Pilomotor seizures: an autonomic semiology of limbic encephalitis? *Seizure* 2014; 23(8): 670-3.
290. Loddenkemper T, Kellinghaus C, Gandjour J, Nair DR, Najm IM, Bingaman W, et al. Localising and lateralising value of ictal piloerection. *J Neurol Neurosurg Psychiatry* 2004; 75(6): 879-83.

291. Stefan H, Feichtinger M, Black A. Autonomic phenomena of temperature regulation in temporal lobe epilepsy. *Epilepsy Behav* 2003; 4(1): 65-9.
292. Stefan H, Pauli E, Kerling F, Schwarz A, Koebnick C. Autonomic auras: left hemispheric predominance of epileptic generators of cold shivers and goose bumps? *Epilepsia* 2002; 43(1): 41-5.
293. Tényi D, Bóné B, Horváth R, Komoly S, Illés Z, Beier CP, et al. Ictal piloerection is associated with high-grade glioma and autoimmune encephalitis – Results from a systematic review. *Seizure* 2019; 64: 1-5.
294. Baysal-Kirac L, Tuzun E, Erdag E, Ulusoy C, Vanli-Yavuz EN, Ekizoglu E, et al. Neuronal autoantibodies in epilepsy patients with peri-ictal autonomic findings. *J Neurology* 2016; 263(3): 455-66.
295. Baumgartner C, Lurger S, Leutmezer F. Autonomic symptoms during epileptic seizures. *Epileptic Disord* 2001; 3(3): 103-16.
296. Janszky J, Fogarasi A, Toth V, Magalova V, Gyimesi C, Kovacs N, et al. Peri-ictal vegetative symptoms in temporal lobe epilepsy. *Epilepsy Behav* 2007; 11(1): 125-9.
297. Henkel A, Noachtar S, Pfänder M, Lüders HO. The localizing value of the abdominal aura and its evolution: a study in focal epilepsies. *Neurology* 2002; 58(2): 271-6.
298. Gil-Nagel A, Risinger MW. Ictal semiology in hippocampal versus extrahippocampal temporal lobe epilepsy. *Brain* 1997; 120(1): 183-92.
299. Gupta A, Jeavons P, Hughes R, Covanis A. Aura in temporal lobe epilepsy: clinical and electroencephalographic correlation. *J Neurol Neurosurg Psychiatry* 1983; 46(12): 1079-83.
300. Taylor DC, Lochery M. Temporal lobe epilepsy: origin and significance of simple and complex auras. *J Neurol Neurosurg Psychiatry* 1987; 50(6): 673-81.
301. Palmi A, Gloor P. The localizing value of auras in partial seizures: a prospective and retrospective study. *Neurology* 1992; 42(4): 801-8.
302. Fried I, Spencer DD, Spencer SS. The anatomy of epileptic auras: focal pathology and surgical outcome. *J Neurosurg* 1995; 83(1): 60-2.
303. Shah J, Zhai H, Fuerst D, Watson C. Hypersalivation in temporal lobe epilepsy. *Epilepsia* 2006; 47(3): 644-51.
304. Hoffmann JM, Elger CE, Kleefuss-Lie AA. The localizing value of hypersalivation and postictal coughing in temporal lobe epilepsy. *Epilepsy Res* 2009; 87(2-3): 144-7.
305. Satow T, Ikeda A, Hayashi N, Yamamoto J, Takayama M, Matsuhashi M, et al. Surgical treatment of seizures from the peri-Sylvian area by perinatal insult: a case report of ictal hypersalivation. *Acta Neurochir* 2004; 146(9): 1021-6.
306. Proserpio P, Cossu M, Francione S, Tassi L, Mai R, Didato G, et al. Insular-opercular seizures manifesting with sleep-related paroxysmal motor behaviors: a stereo-EEG study. *Epilepsia* 2011; 52(10): 1781-91.
307. Musilová K, Kuba R, Brázdil M, Tyrliková I, Rektor I. Occurrence and lateralizing value of “rare” peri-ictal vegetative symptoms in temporal lobe epilepsy. *Epilepsy Behav* 2010; 19(3): 372-5.
308. Voss NF, Davies KG, Boop FA, Montouris GD, Hermann BP. Spitting automatism in complex partial seizures: a nondominant temporal localizing sign? *Epilepsia* 1999; 40(1): 114-6.
309. Vojvodic N, Ristic AJ, Bascarevic V, Popovic L, Parojcic A, Koprivsek K, et al. Ictal spitting in left temporal lobe epilepsy and fMRI speech lateralization. *Clin Neurol Neurosurg* 2013; 115(4): 495-7.
310. Kellinghaus C, Loddenkemper T, Kotagal P. Ictal spitting: clinical and electroencephalographic features. *Epilepsia* 2003; 44(8): 1064-9.
311. Quevedo-Diaz M, Campo AT, Vila-Vidal M, Principe A, Ley M, Rocamora R. Ictal spitting in non-dominant temporal lobe epilepsy: an anatomo-electrophysiological correlation. *Epileptic Disord* 2018; 20(2): 139-45.
312. Park S-M, Lee S-A, Kim JH, Kang JK. Ictal spitting in a patient with dominant temporal lobe epilepsy: supporting evidence of ictal spitting from the nondominant hemisphere. *Eur Neurol* 2007; 57(1): 47.
313. Özkara C, Hanoglu L, Eskazan E, Kulaksizoglu IB, Özyurt E. Ictal spitting during a left temporal lobe-originated complex partial seizure: a case report. *Epileptic Disord* 2000; 2(3): 169-72.
314. Caboclo LOSF, Miyashira FS, Hamad APA, Lin K, Carrete Jr H, Sakamoto AC, et al. Ictal spitting in left temporal lobe epilepsy: report of three cases. *Seizure* 2006; 15(6): 462-7.
315. Fiol ME, Leppik IE, Mireles R, Maxwell R. Ictus emeticus and the insular cortex. *Epilepsy Res* 1988; 2(2): 127-31.
316. Kramer RE, Lüders H, Goldstick L, Dinner D, Morris H, Lesser R, et al. Ictus emeticus: an electroclinical analysis. *Neurology* 1988; 38(7): 1048-52.
317. Guerrini R, Ferrari A, Battaglia A, Salvadori P, Bonanni P. Occipitotemporal seizures with ictus emeticus induced by intermittent photic stimulation. *Neurology* 1994; 44(2): 253-9.
318. Devinsky O, Frasca J, Pacia S, Luciano D, Paraiso J, Doyle W. Ictus emeticus: further evidence of nondominant temporal involvement. *Neurology* 1995; 45(6): 1158-60.
319. Kotagal P, Lüders HO, Williams G, Nichols TR, McPherson J. Psychomotor seizures of temporal lobe onset: analysis of symptom clusters and sequences. *Epilepsy Res* 1995; 20(1): 49-67.
320. Baumgartner C, Olbrich A, Lindinger G, Patarai E, Gröppel G, Bacher J, et al. Regional cerebral blood flow during temporal lobe seizures associated with ictal vomiting: an ictal SPECT study in two patients. *Epilepsia* 1999; 40(8): 1085-91.
321. Chen C, Yen D-J, Yiu C-H, Shih Y-H, Yu H-Y, Su M-S. Ictal vomiting in partial seizures of temporal lobe origin. *Eur Neurol* 1999; 42(4): 235-9.
322. Schäuble B, Britton JW, Mullan BP, Watson J, Sharrow FB, Marsh WR. Ictal vomiting in association with left

- temporal lobe seizures in a left hemisphere language-dominant patient. *Epilepsia* 2002; 43(11): 1432-5.
323. Schindler K, Wieser HG. Ictal vomiting in a left hemisphere language-dominant patient with left-sided temporal lobe epilepsy. *Epilepsy Behav* 2006; 8(1): 323-7.
324. Tarnutzer AA, Mothersill I, Imbach LL. Ictal nausea and vomiting – Is it left or right? *Seizure* 2018; 61: 83-8.
325. Pietrafusa N, de Palma L, De Benedictis A, Trivisano M, Marras CE, Vigevano F, et al. Ictal vomiting as a sign of temporal lobe epilepsy confirmed by stereo-EEG and surgical outcome. *Epilepsy Behav* 2015; 53: 112-6.
326. Catenox H, Isnard J, Guénot M, Petit J, Remy C, Mauguière F. The role of the anterior insular cortex in ictal vomiting: a stereotactic electroencephalography study. *Epilepsy Behav* 2008; 13(3): 560-3.
327. Strzelczyk A, Nowak M, Bauer S, Reif PS, Oertel WH, Knake S, et al. Localizing and lateralizing value of ictal flatulence. *Epilepsy Behav* 2010; 17(2): 278-82.
328. Vittal NB, Singh P, Azar NJ. Ictal flatulence: seizure onset in the nondominant hemisphere. *Epilepsy Behav* 2009; 16(4): 663-5.
329. Koubeissi MZ, Crone NE, Lesser RP. Seizures manifesting as an urge to defecate, with an ictal discharge in the right hemisphere. *Epilepsia* 2005; 46(8): 1330-2.
330. Taieb G, Renard D, Labauge P, Janicot F, Briere C. Ictal urge to defecate associated with a right-sided mesial temporal cavernoma. *Epilepsy Behav* 2012; 24(2): 272-3.
331. Mestre TA, Bentes C, Pimentel J. Ictal eructation: a case report. *Epileptic Disord* 2008; 10(2): 170-2.
332. Bateman LM, Li CS, Seyal M. Ictal hypoxemia in localization-related epilepsy: analysis of incidence, severity and risk factors. *Brain* 2008; 131(Pt 12): 3239-45.
333. Lacuey N, Zonjy B, Hampson JP, Rani MRS, Zaremba A, Sainju RK, et al. The incidence and significance of periictal apnea in epileptic seizures. *Epilepsia* 2018; 59(3): 573-82.
334. Singh B, Al Shahwan A, Al Deeb SM. Partial seizures presenting as life-threatening apnea. *Epilepsia* 1993; 34(5): 901-3.
335. Seyal M, Bateman LM. Ictal apnea linked to contralateral spread of temporal lobe seizures: intracranial EEG recordings in refractory temporal lobe epilepsy. *Epilepsia* 2009; 50(12): 2557-62.
336. Tio E, Culler GW, Bachman EM, Schuele S. Ictal central apneas in temporal lobe epilepsies. *Epilepsy Behav* 2020; 112: 107434.
337. Lacuey N, Hupp NJ, Hampson J, Lhatoo S. Ictal Central Apnea (ICA) may be a useful semiological sign in invasive epilepsy surgery evaluations. *Epilepsy Res* 2019; 156: 106164.
338. Cohen HA, Ashkenazi A, Barzilai A, Lahat E. Nocturnal acute laryngospasm in children: a possible epileptic phenomenon. *J Child Neurol* 2000; 15(3): 202-4.
339. Oldani A, Zucconi M, Castronovo C, Ferini-Strambi L. Nocturnal frontal lobe epilepsy misdiagnosed as sleep apnea syndrome. *Acta Neurol Scand* 1998; 98(1): 67-71.
340. Rathore G, Larsen P, Parakh M, Fernandez C. Choking at night: a case of opercular nocturnal frontal lobe epilepsy. *Case Rep Pediatr* 2013; 2013: 606385.
341. Davis KA, Cantor C, Maus D, Herman ST. A neurological cause of recurrent choking during sleep. *J Clin Sleep Med* 2008; 4(6): 586-7.
342. Geevasinga N, Archer JS, Ng K. Choking, asphyxiation and the insular seizure. *J Clin Neurosci* 2014; 21(4): 688-9.
343. Isnard J, Guénot M, Sindou M, Mauguière F. Clinical manifestations of insular lobe seizures: a stereo-electroencephalographic study. *Epilepsia* 2004; 45(9): 1079-90.
344. O'Regan ME, Brown JK. Abnormalities in cardiac and respiratory function observed during seizures in childhood. *Dev Med Child Neurol* 2005; 47(1): 4-9.
345. Singh K, Katz ES, Zarowski M, Loddenkemper T, Llewellyn N, Manganaro S, et al. Cardiopulmonary complications during pediatric seizures: a prelude to understanding SUDEP. *Epilepsia* 2013; 54(6): 1083-91.
346. Pavlova M, Singh K, Abdennadher M, Katz ES, Dworetzky BA, White DP, et al. Comparison of cardiorespiratory and EEG abnormalities with seizures in adults and children. *Epilepsy Behav* 2013; 29(3): 537-41.
347. Foldvary N, Lee N, Thwaites G, Mascha E, Hammel J, Kim H, et al. Clinical and electrographic manifestations of lesional neocortical temporal lobe epilepsy. *Neurology* 1997; 49(3): 757-63.
348. Harvey AS, Hopkins IJ, Bowe JM, Cook DJ, Shield LK, Berkovic SF. Frontal lobe epilepsy: clinical seizure characteristics and localization with ictal 99mTc-HMPAO SPECT. *Neurology* 1993; 43(10): 1966-80.
349. Freeman R, Schachter SC. Autonomic epilepsy. *Semin Neurol* 1995; 15(2): 158-66.
350. Shahar E, Andraus J. Near reflex accommodation spasm: unusual presentation of generalized photosensitive epilepsy. *J Clin Neurosci* 2002; 9(5): 605-7.
351. Rosenberg ML, Jabbari B. Miosis and internal ophthalmoplegia as a manifestation of partial seizures. *Neurology* 1991; 41(5): 737-9.
352. Sadek AR, Kirkham F, Barker S, Gray WP, Allen D. Seizure-induced miosis. *Epilepsia* 2011; 52(12): e199-203.
353. Afifi AK, Corbett JJ, Thompson HS, Wells KK. Seizure-induced miosis and ptosis: association with temporal lobe magnetic resonance imaging abnormalities. *J Child Neurol* 1990; 5(2): 142-6.
354. Lance JW, Smee RI. Partial seizures with visual disturbance treated by radiotherapy of cavernous hemangioma. *Ann Neurol* 1989; 26(6): 782-5.
355. Tamburin S, Turri G, Kuhdari P, Fiaschi A, Manganotti P. Unilateral fixed mydriasis: an uncommon presentation of temporal lobe epilepsy. *J Neurol* 2012; 259(2): 355-7.
356. Masjuan J, García-Segovia J, Barón M, Alvarez-Cermeño JC. Ipsilateral mydriasis in focal occipitotemporal seizures. *J Neurol Neurosurg Psychiatry* 1997; 63(6): 810-1.

357. Gadoth N, Margalith D, Bechar M. Unilateral pupillary dilatation during focal seizures. *J Neurol* 1981; 225(3): 227-30.
358. Loddenkemper T, Foldvary N, Raja S, Neme S, Lüders HO. Ictal urinary urge: further evidence for lateralization to the nondominant hemisphere. *Epilepsia* 2003; 44(1): 124-6.
359. Gurgenshvili K, Massey SL, Grant M, Piatt Jr J, Legido A, Valencia I. Intracranial localisation of ictal urinary urge epileptogenic zone to the non-dominant temporal lobe. *Epileptic Disord* 2011; 13(4): 430-4.
360. Yilmaz S, Gokben S, Turhan T, Serdaroglu G, Tekgul H. Ictal urinary urge: localization and lateralization value in a pediatric case. *Childs Nerv Syst* 2015; 31(12): 2383-5.
361. Dobesberger J, Walser G, Unterberger I, Embacher N, Luef G, Bauer G, et al. Genital automatisms: a video-EEG study in patients with medically refractory seizures. *Epilepsia* 2004; 45(7): 777-80.
362. Stoffels C, Munari C, Bonis A, Bancaud J, Talairach J. Genital and sexual manifestations occurring in the course of partial seizures in man (author's transl.). *Rev Electroencephalogr Neurophysiol Clin* 1980; 10(4): 386-92.
363. Kasper BS, Kerling F, Graf W, Stefan H, Pauli E. Ictal delusion of sexual transformation. *Epilepsy Behav* 2009; 16(2): 356-9.
364. Fisher RS, Schachter SC. The postictal state: a neglected entity in the management of epilepsy. *Epilepsy Behav* 2000; 1(1): 52-9.
365. Engel JJ. *Seizures and epilepsy*. Philadelphia: F.A. Davis Company, 1989.
366. Ryvlin P, Nashef L, Lhatoo SD, Bateman LM, Bird J, Bleasel A, et al. Incidence and mechanisms of cardiorespiratory arrests in epilepsy monitoring units (MORTEMUS): a retrospective study. *Lancet Neurol* 2013; 12(10): 966-77.
367. 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.
368. Pottkämper JCM, Hofmeijer J, van Waarde JA, van Putten M. The postictal state – What do we know? *Epilepsia* 2020; 61(6): 1045-61.
369. Rolak LA, Rutecki P, Ashizawa T, Harati Y. Clinical features of Todd's post-epileptic paralysis. *J Neurol Neurosurg Psychiatry* 1992; 55(1): 63-4.
370. Leutmezer F, Serles W, Patariaia E, Olbrich A, Bacher J, Aull S, et al. The postictal state. A clinically oriented observation of patients with epilepsies. *Wien Klin Wochenschr* 1998; 110(11): 401-7.
371. Adam C, 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.
372. Gallmetzer P, Leutmezer F, Serles W, Assem-Hilger E, Spatt J, Baumgartner C. Postictal paresis in focal epilepsies – incidence, duration, and causes: a video-EEG monitoring study. *Neurology* 2004; 62(12): 2160-4.
373. Kellinghaus C, Kotagal P. Lateralizing value of Todd's palsy in patients with epilepsy. *Neurology* 2004; 62(2): 289-91.
374. Leutmezer F, Baumgartner C. Postictal signs of lateralizing and localizing significance. *Epileptic Disord* 2002; 4(1): 43-8.
375. Werhahn KJ. Weakness and focal sensory deficits in the postictal state. *Epilepsy Behav* 2010; 19(2): 138-9.
376. Kosnik E, Paulson GW, Laguna JF. Postictal blindness. *Neurology* 1976; 26(3): 248-50.
377. Hadjikitoutis S, Sawhney IM. Occipital seizures presenting with bilateral visual loss. *Neurol India* 2003; 51(1): 115-6.
378. Caraballo R, Koutroumanidis M, Panayiotopoulos CP, Fejerman N. Idiopathic childhood occipital epilepsy of Gastaut: a review and differentiation from migraine and other epilepsies. *J Child Neurol* 2009; 24(12): 1536-42.
379. Salmon JH. Transient postictal hemianopsia. *Arch Ophthalmol* 1968; 79(5): 523-5.
380. Privitera M, Kim KK. Postictal language function. *Epilepsy Behav* 2010; 19(2): 140-5.
381. Leutmezer F, Serles W, Lehrner J, Patariaia E, Zeiler K, Baumgartner C. Postictal nose wiping: a lateralizing sign in temporal lobe complex partial seizures. *Neurology* 1998; 51(4): 1175-7.
382. Geyer JD, Payne TA, Faught E, Drury I. Postictal nose-rubbing in the diagnosis, lateralization, and localization of seizures. *Neurology* 1999; 52(4): 743-5.
383. Hirsch LJ, Lain AH, Walczak TS. Postictal nosewiping lateralizes and localizes to the ipsilateral temporal lobe. *Epilepsia* 1998; 39(9): 991-7.
384. Wennberg R. Electroclinical analysis of postictal noserubbing. *Can J Neurol Sci* 2000; 27(2): 131-6.
385. Çilliler AE, Güven H, Çomoğlu SS. Epilepsy and headaches: further evidence of a link. *Epilepsy Behav* 2017; 70(Pt A): 161-5.
386. Ekstein D, Schachter SC. Postictal headache. *Epilepsy Behav* 2010; 19(2): 151-5.
387. Yankovsky AE, Andermann F, Bernasconi A. Characteristics of headache associated with intractable partial epilepsy. *Epilepsia* 2005; 46(8): 1241-5.
388. Ito M, Schachter SC. Frequency and characteristics of interictal headaches in patients with epilepsy. *J Epilepsy* 1996; 9(2): 83-6.
389. Mohamed W, Ahuja N, Shah A. Palinacousis – evidence to suggest a post-ictal phenomenon. *J Neurol Sci* 2012; 317(1-2): 6-12.
390. Simon RP. Heart and lung in the postictal state. *Epilepsy Behav* 2010; 19(2): 167-71.
391. Fauser S, Wuwer Y, Gierschner C, Schulze-Bonhage A. The localizing and lateralizing value of ictal/postictal coughing in patients with focal epilepsies. *Seizure* 2004; 13(6): 403-10.

392. Azar NJ, Tayah TF, Wang L, Song Y, Abou-Khalil BW. Postictal breathing pattern distinguishes epileptic from nonepileptic convulsive seizures. *Epilepsia* 2008; 49(1): 132-7.
393. Oueslati B, Fekih-Romdhane F, Ridha R. Postictal delirium and violent behavior in patients with post-neurosurgical epilepsy. *World Neurosurg* 2018; 115: 193-5.
394. Clancy MJ, Clarke MC, Connor DJ, Cannon M, Cotter DR. The prevalence of psychosis in epilepsy; a systematic review and meta-analysis. *BMC Psychiatry* 2014; 14: 75.
395. Devinsky O. Postictal psychosis: common, dangerous and treatable. *Epilepsy Curr* 2008; 8(2): 31-4.
396. Kanner AM, Soto A, Gross-Kanner H. Prevalence and clinical characteristics of postictal psychiatric symptoms in partial epilepsy. *Neurology* 2004; 62(5): 708-13.
397. Eisenschenk S, Krop H, Devinsky O. Homicide during postictal psychosis. *Epilepsy Behav Case Rep* 2014; 2: 118-20.
398. Kanner AM, Trimble M, Schmitz B. Postictal affective episodes. *Epilepsy Behav* 2010; 19(2): 156-8.
399. Barba C, Barbati G, Minotti L, Hoffmann D, Kahane P. Ictal clinical and scalp-EEG findings differentiating temporal lobe epilepsies from temporal 'plus' epilepsies. *Brain* 2007; 130(Pt 7): 1957-67.
400. McGonigal A, Bartolomei F, Chauvel P. On seizure semiology. *Epilepsia* 2021; 62(9): 2019-35.
401. Chauvel P. *Contributions of Jean Talairach and Jean Bancaud to epilepsy surgery. Epilepsy surgery*. Philadelphia: Lippincott Williams & Wilkins, 2001.
402. Stefan H. *The challenge epilepsy treatment – New epileptic drugs*. Oxford: Blackwell science, 1998.
403. Stoyke C, Bilgin O, Noachtar S. Video atlas of lateralising and localising seizure phenomena. *Epileptic Disord* 2011; 13(2): 113-24.
404. Donadío M, Ugarnes G, Segalovich M, Arakaki N, Sanchez Gonzalez F, Petre C, et al. Intracranial video-EEG and surgery for focal atonic seizures. *Epileptic Disord* 2013; 15(1): 62-6.
405. Hahn A, Fischenbeck A, Stephani U. Induction of epileptic negative myoclonus by oxcarbazepine in symptomatic epilepsy. *Epileptic Disord* 2004; 6(4): 271-4.
406. Saint-Martin AD, Carcangiu R, Arzimanoglou A, Massa R, Thomas P, Motte J, et al. Semiology of typical and atypical Rolandic epilepsy: a video-EEG analysis. *Epileptic Disord* 2001; 3(4): 173-82.
407. Dragoumi P, Emery J, Chivers F, Brady M, Desurkar A, Cross JH, et al. Crossing the lines between epilepsy syndromes: a myoclonic epilepsy variant with prominent eyelid myoclonia and atonic components. *Epileptic Disord* 2018; 20(1): 35-41.
408. Nasser H, Lopez-Hernandez E, Ilea A, Le Morvan N, Bellavoine V, Delanoë C, et al. Myoclonic jerks are commonly associated with absence seizures in early-onset absence epilepsy. *Epileptic Disord* 2017; 19(2): 137-46.
409. Ramanathan S, Wong CH, Rahman Z, Dale RC, Fulcher D, Bleasel AF. Myoclonic status epilepticus as a presentation of caspr2 antibody-associated autoimmune encephalitis. *Epileptic Disord* 2014; 16(4): 477-81.
410. Lüders H, Vaca GF, Akamatsu N, Amina S, Arzimanoglou A, Baumgartner C, et al. Classification of paroxysmal events and the four-dimensional epilepsy classification system. *Epileptic Disord* 2019; 21(1): 1-29.
411. Varley J, Wehner T, Sisodiya S. Diaphragm myoclonus followed by generalised atonia in a patient with trisomy 4p: unusual semiology in an unusual condition. *Epileptic Disord* 2015; 17(4): 473-7.
412. Casaubon L, Pohlmann-Eden B, Khosravani H, Carlen PL, Wennberg R. Video-EEG evidence of lateralized clinical features in primary generalized epilepsy with tonic-clonic seizures. *Epileptic Disord* 2003; 5(3): 149-56.
413. Jayakumar H, Gopinath S, Pillai A, Kumar A. Epileptic nystagmus due to a large parieto-temporo-occipital multilobar dysplasia. *Epileptic Disord* 2020; 22(5): 691-2.
414. Saltik S, Cokar O, Uslu T, Uludüz D, Dervent A. Alternating hemiplegia of childhood: presentation of two cases regarding the extent of variability. *Epileptic Disord* 2004; 6(1): 45-8.
415. Caraballo RH, Fejerman N, Bernardina BD, Ruggieri V, Cersósimo R, Medina C, et al. Epileptic spasms in clusters without hypsarrhythmia in infancy. *Epileptic Disord* 2003; 5(2): 109-13.
416. Umeoka S, Baba K, Terada K, Matsuda K, Tottori T, Usui N, et al. Bilateral symmetric tonic posturing suggesting propagation to the supplementary motor area in a patient with precuneate cortical dysplasia. *Epileptic Disord* 2007; 9(4): 443-8.
417. Fernández-Torre JL, Riancho J, Martín-García M, Martínez-de Las Cuevas G, Bosque-Varela P. Tonic status epilepticus in a centenarian woman. *Epileptic Disord* 2019; 21(1): 92-6.
418. Takeda T, Osawa M, Toi S, Mizuno S, Shimizu Y, Uchiyama S. Adversive seizures associated with periodic lateralised epileptiform discharges (PLEDs) after left orbital contusion. *Epileptic Disord* 2012; 14(4): 422-5.
419. Gavvala JR, Gerard EE, Macken M, Schuele SU. Seizure ending signs in patients with dyscognitive focal seizures. *Epileptic Disord* 2015; 17(3): 255-62.
420. Mir A, Thani Z, Bashir S, Ayed H, Albaradie R. LGI-1 antibody encephalitis in a seven-year-old girl. *Epileptic Disord* 2019; 21(6): 591-7.
421. López-Laso E, Mateos González ME, Camino León R, Jiménez González MD, Esparza Rodríguez J. Giant hypothalamic hamartoma and dacrycystic seizures. *Epileptic Disord* 2007; 9(1): 90-3.
422. Dimova P, Boneva I, Todorova A, Minotti L, Kahane P. Gelastic seizures in ring chromosome 20 syndrome: a case report with video illustration. *Epileptic Disord* 2012; 14(2): 181-6.
423. Arzimanoglou AA, Hirsch E, Aicardi J. Hypothalamic hamartoma and epilepsy in children: illustrative cases of possible evolutions. *Epileptic Disord* 2003; 5(4): 187-99.

424. Ozkara C, Sarý H, Hanođlu L, Yeni N, Aydođdu I, Ozyurt E. Ictal kissing and religious speech in a patient with right temporal lobe epilepsy. *Epileptic Disord* 2004; 6(4): 241-5.
425. Beimer NJ, Selwa LM. Seizure semiology of anti-LGI1 antibody encephalitis. *Epileptic Disord* 2017; 19(4): 461-4.
426. Tezer FI, Agan K, Borggraefe I, Noachtar S. Seizure semiology reflects spread from frontal to temporal lobe: evolution of hyperkinetic to automotor seizures as documented by invasive EEG video recordings. *Epileptic Disord* 2013; 15(3): 338-41.
427. Boyd M, Attarian H, Raizer J, Kumthekar P, Macken MP, Schuele SU, *et al.* Auditory hallucinations as ictal phenomena in a patient with voltage-gated potassium channel antibody-associated limbic encephalitis. *Epileptic Disord* 2013; 15(4): 433-6.
428. Thomas P, Arzimanoglou A, Aicardi J. Benign idiopathic occipital epilepsy: report of a case of the late (Gastaut) type [corrected]. *Epileptic Disord* 2003; 5(1): 57-9.
429. Ristić AJ, Marjanović I, Brajković L, Wolgamuth BR, Odalović S, Lavrnić S, *et al.* Bálint-like syndrome as an unusual representation of non-convulsive status epilepticus. *Epileptic Disord* 2012; 14(1): 80-4.
430. Yu T, Liu C, Zhang G, Qiao L, Ni D, Wang Y, *et al.* Focal epilepsy with paroxysmal pain due to somatic injury. *Epileptic Disord* 2017; 19(2): 207-11.
431. Aljaafari D, Nascimento FA, Abraham A, Andrade DM, Wennberg RA. Unilateral abdominal clonic seizures of parietal lobe origin: EEG findings. *Epileptic Disord* 2018; 20(2): 158-63.
432. Boulogne S, Catenoux H, Ryvlin P, Rheims S. Long-lasting seizure-related anxiety in patients with temporal lobe epilepsy and comorbid psychiatric disorders. *Epileptic Disord* 2015; 17(3): 340-4.
433. Antelmi E, Mastrangelo M, Bisulli F, Spaccini L, Stipa C, Mostacci B, *et al.* Semiological study of ictal affective behaviour in epilepsy and mental retardation limited to females (EFMR). *Epileptic Disord* 2012; 14(3): 304-9.
434. Jan S, Ochi A, Kagawa K, Gill P, Guttmann A, Otsubo H, *et al.* Intractable apnoeic seizures in a child with a deletion typically associated with Williams syndrome. *Epileptic Disord* 2018; 20(6): 530-4.
435. Toffa DH, Pana R, Nguyen DK. Ictal asystole with intercurrent cardiopathy: a complex combination leading to delayed diagnosis. *Epileptic Disord* 2019; 21(6): 598-602.
436. Leal Rato M, Ionel C, Baduro Y, Oliveira R, Franco AC, Peralta AR, *et al.* Syncope due to ictal asystole: a challenging semiology. *Epileptic Disord* 2020; 22(5): 693-4.
437. Quevedo-Diaz M, Campo AT, Vila-Vidal M, Principe A, Ley M, Rocamora R. Ictal spitting in non-dominant temporal lobe epilepsy: an anatomo-electrophysiological correlation. *Epileptic Disord* 2018; 20(2): 139-45.
438. Demirbilek V, Dervent A. Panayiotopoulos syndrome: video-EEG illustration of a typical seizure. *Epileptic Disord* 2004; 6(2): 121-4.

Legend for video sequences

Supplementary video 1

A patient with an atonic seizure, presenting as atonia of the neck and trunk musculature.

Key words for video research on www.epilepticdisorders.com

Phenomenology: atonic seizure (drop attack)

Localization: generalized

Syndrome: epileptic encephalopathy not otherwise classified

Aetiology: unknown

Supplementary video 2

An infant with a myoclonic seizure showing a sudden, very brief bilateral muscle jerk of the upper limbs. The second movement is a reactive movement, as the spontaneous seizure jerk scares the child.

Key words for video research on www.epilepticdisorders.com

Phenomenology: myoclonic seizure, generalized

Localization: generalized

Syndrome: early myoclonic encephalopathy

Aetiology: genetic disorder

Supplementary video 3

A patient with a bilateral synchronous clonic seizure.

Key words for video research on www.epilepticdisorders.com

Phenomenology: clonic seizure

Localization: generalized

Syndrome: unknown

Aetiology: unknown

Supplementary video 4

A young child with a generalized seizure, starting with a bilateral muscle jerk in the shoulders (myoclonic), followed by muscle activity cessation (atonia), resulting in a fall.

Key words for video research on www.epilepticdisorders.com

Phenomenology: myoclonic atonic seizure

Localization: generalized

Syndrome: Doose syndrome

Aetiology: idiopathic

Supplementary video 5

A focal seizure with impaired awareness arising from the frontal lobe during sleep. Note that the patient is sleeping on a mattress on the floor with protective mattresses around him. The seizure starts with fulminant hyperkinetic behavior, followed by a gyratory movement of the body to the right. The patient's awareness is impaired and he is not responding to the nurse's commands. In the immediate postictal phase, the patient becomes responsive, and is able to point to the window, in response to the verbal command.

Key words for video research on www.epilepticdisorders.com

Phenomenology: hypermotor seizure, gyratory (not from the existing list)

Localization: frontal prefrontal mesiolateral

Syndrome: focal, non-idiopathic frontal (FLE)

Aetiology: unknown

Supplementary video 6

An infant with clusters of epileptic spasms. Note the typical gestalt of the spasms with flexion in the neck and simultaneous abduction in the shoulders and flexion in the hips.

Key words for video research on www.epilepticdisorders.com

Phenomenology: spasm (epileptic)

Localization: generalized

Syndrome: west syndrome

Aetiology: genetic disorder

Supplementary video 7

Generalized tonic-clonic seizure occurring during sleep. The patient lies in the bed in prone position. The seizure starts with ictal crying -the characteristic sound produced by the forced push of air through the airways, due to the tonic contraction of the diaphragm. Symmetric tonic contraction of all four limbs is then observed, followed by symmetric clonic jerks which gradually decrease in frequency.

Key words for video research on www.epilepticdisorders.com

Phenomenology: generalized, tonic-clonic seizure

Localization: generalized

Syndrome: idiopathic generalized not specified

Aetiology: idiopathic/genetic predisposition

Supplementary video 8

The patient is sitting in bed and watching TV. The seizure starts abruptly with fearful behavior, followed by integrated automatisms. During the seizure, there is impaired awareness. After the seizure ends, the patient answers the nurse's questions and follows commands.

Key words for video research on www.epilepticdisorders.com

Phenomenology: fear, hypermotor seizure

Localization: frontal, prefrontal mesial

Syndrome: focal non-idiopathic frontal (FLE)

Aetiology: focal cortical dysplasia

Supplementary video 9

A focal impaired awareness seizure that starts with gestural automatisms and continues with ictal aphasia. The patient does not follow verbal commands, but follows visual clues.

Key words for video research on www.epilepticdisorders.com

Phenomenology: aphasic seizure, cognitive (not in the list), automatisms

Localization: left temporal lobe

Syndrome: focal non-idiopathic mesiotemporal (MTLE with or without HS)

Aetiology: hippocampal sclerosis