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REVIEW ARTICLE

Epidemiology and pathophysiology of autonomic seizures: a systematic review

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Abstract

Purpose To review the epidemiology and pathophysiology of autonomic symptoms and signs during epileptic seizures.

Methods We performed a systematic literature search on the following autonomic symptoms and signs during epileptic seizures: cardiovascular changes, respiratory manifestations, gastrointestinal symptoms, cutaneous manifestations, sexual and genital manifestations, and urinary symptoms.

Results Autonomic symptoms and signs can represent the predominant symptom at the onset of a focal seizure, which would then lead to the seizure being classified as a focal onset autonomic seizure. Conversely, clinically relevant autonomic symptoms and signs frequently accompany seizures of focal, generalized, and/or unknown onset, but the seizure is regardless classified according to other, more relevant features. Autonomic symptoms and signs do not represent mere reactions to motor activity or other behavioral seizure manifestations, but rather they are generated by epileptic discharges affecting the central autonomic network. We have reviewed the localizing and lateralizing information currently available on the seizure onset zone and on seizure propagation pathways as provided by systematic analysis of specific autonomic seizure symptoms and signs. We present data on how autonomic seizure symptoms and signs are useful for gaining a better understanding of the anatomical and functional organization of the central autonomic network. Finally, we discuss the differential diagnosis of focal autonomic seizures with autonomic symptoms and signs representing the sole seizure manifestation versus various non-epileptic conditions.

Conclusions Autonomic seizure symptoms and signs are relevant in clinical epileptology and open a unique window on the functional organization and pathophysiology of the central autonomic network.

Keywords Epilepsy · Seizures · Autonomic · Semiology

Introduction

Autonomic symptoms and signs are frequent manifestations of epileptic seizures [1–8]. These autonomic symptoms and signs can represent the predominant symptom at the onset of a focal seizure, which would lead to the seizure being classified as a focal onset autonomic seizure [9]. The operational classification of seizure types by the International League Against Epilepsy recognizes autonomic seizures as a new and distinct seizure type of focal seizure with nonmotor onset [9]. According to this classification 'focal autonomic seizures can present with gastrointestinal sensations, a sense of heat or cold, flushing, piloerection (goosebumps), palpitations, sexual arousal, respiratory changes, or other autonomic effects' [9]. Conversely, clinically relevant autonomic symptoms and signs can also accompany seizures of focal, generalized, and/or unknown onset, but the seizures are classified otherwise, according to their earliest prominent motor onset or nonmotor onset feature [9].

Autonomic symptoms and signs during epileptic seizures are primarily caused by a direct activation of the central autonomic network by epileptic discharges, rather than by the motor or behavioral effects of the seizures themselves [6]. The central autonomic network includes the insular cortex, anterior cingulate cortex, amygdala,

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hypothalamus, periaqueductal gray, parabrachial nucleus, nucleus of the solitary tract, ventrolateral reticular formation of the medulla, and medullary raphe. These areas are reciprocally interconnected, receive converging visceral and somatosensory information, generate stimulus-specific patterns of autonomic, endocrine, and motor responses, and are regulated according to the behavioral state, including the sleep–wake cycle [10, 11].

Here we review currently available localizing and lateralizing information on the seizure onset zone and on seizure propagation pathways, based on our systematic analysis of specific autonomic seizure symptoms and signs. We present data on how autonomic seizure symptoms and signs are useful for gaining a better understanding of the anatomical and functional organization of the central autonomic network. Finally, we discuss the differential diagnosis of focal autonomic seizures with autonomic symptoms and signs representing the sole seizure manifestation versus various non-epileptic conditions.

Methods

A systematic search of PubMed, Medline, and Scopus was performed for case reports, case series, reviews, and abstracts using the search terms 'autonomic' with the following prefixes: 'seizure,' 'epilepsy,' 'seizure semiology,' 'seizure symptoms,' 'seizure signs,' 'ictal,' and 'postictal.' The reference lists of the articles subsequently identified were also screened to find additional publications and cases that satisfied the search criteria.

We then refined our search to the following specific autonomic seizure symptoms and signs using the following search terms:

- cardiovascular changes, including ictal sinus tachycardia, ictal bradyarrhythmias (including ictal asystole, postictal asystole, other peri-ictal cardiac arrhythmias [i.e,. ictal and postictal atrial flutter/atrial fibrillation]), postictal ventricular fibrillation;
- respiratory manifestations, including ictal hyperventilation, ictal oxygen desaturations, ictal hypoventilation, ictal central apnea, postictal nosewiping, peri-ictal coughing, other respiratory manifestations (i.e., larnyngospasm, nocturnal choking, laryngeal constriction);
- gastrointestinal symptoms, including epigastric aura, ictal vomiting in adults, ictal retching in adults, ictal vomiting in children, peri-ictal spitting, ictal hypersalivation, ictal flatulence, ictal urge to defecate;
- cutaneous manifestations, including ictal flushing, ictal pallor, ictal sweating, ictal piloerection;
- sexual and genital manifestations, including sexual auras, genital auras, sexual automatisms, genital automatisms;
- urinary symptoms, including ictal urinary urge and urination, peri-ictal water drinking.

These search terms were combined with the following prefixes: 'seizure,' 'epilepsy,' 'seizure semiology,' 'seizure symptoms,' 'seizure signs,' 'ictal,' 'postictal.' Once again, the reference lists of the articles identified were screened to find additional publications and cases.

Cardiovascular changes

Ictal sinus tachycardia

Ictal sinus tachycardia represents the most frequent cardiac concomitant of epileptic seizures and can occur during any seizure type.

According to a recent review of 34 studies, ictal sinus tachycardia can be observed in 82% of patients with epilepsy [12]. The absolute increase in heart rate in this review, reported as a weighted average across several studies, averaged 34.23 bpm per seizure and 33.51 beats per minute (bpm) per patient [12]. In terms of seizure type, ictal sinus tachycardia was observed in 12% of subclinical seizures, 71% of focal onset seizures, 64% of all types of generalized seizures (including generalized tonic–clonic seizures), and 76% of mixed seizure types (weighted average across several studies) [12]. Focal seizures evolving to bilateral tonic–clonic seizures result in a higher ictal heart rate as compared to focal seizures with impaired awareness [13]. Also, the pre-ictal heart rate has been reported to be higher—although the increase was not necessarily clinically significant—in seizures evolving to bilateral tonic–clonic seizures than in those focal seizures which remained localized, while the interictal heart rate was similar in patients with both seizure types [14]. Concerning seizure onset zone, ictal sinus tachycardia was found to be more consistent and prevalent in seizures of temporal origin than in those of extratemporal origin [12].

While stimulation of the left-sided insula in humans causes bradycardia, right-sided stimulation results in tachycardia [15], suggesting that right-sided seizures would be more prone to result in ictal tachycardia. Indeed, some studies have suggested a more pronounced increase in heart rate during seizures arising from the non-dominant hemisphere [16–20]. In a study using invasive electrodes no difference in ictal heart rate increase could be found in seizures arising from the left or right temporal lobe; in contrast, a pre-ictal tachycardia (i.e., increase of heart rate of > 1 standard deviation as compared to interictal heart rate) was observed more often in right temporal seizures as compared to left temporal seizures [21]. However, the correlation between seizure

onset lateralization and ictal tachycardia was inconsistent across most studies [12].

In a study using intracranial electrodes, Hirsch et al. studied the temporal relationship between the onset of ictal tachycardia, seizure onset on intracranial electroencephalography (EEG), seizure onset on scalp-EEG, and clinical seizure onset [22]. While ictal tachycardia occurred after seizure onset on intracranial EEG in all seizures, ictal tachycardia preceded scalp-EEG onset in 61.5% of seizures and occurred before the first clinical signs in 71.8% of seizures [22]. Ictal tachycardia was observed earlier in seizures arising from the hippocampal formation than in those of extrahippocampal onset. Finally, ictal tachycardia occurred earlier in seizures originating from the right temporal lobe than in those originating from the left temporal lobe [22].

Ictal bradyarrhythmias including ictal asystole

Bradyarrhythmias occur much less frequently during epileptic seizures than tachyarrhythmias [1, 2, 6, 8, 23]. Ictal bradycardia can vary from mild asymptomatic sinus bradycardia to more severe symptomatic bradyarrhythmias (pronounced sinus bradycardia, sinus arrest, atrioventricular block) and prolonged asystole [1, 2, 6]. The prevalence of ictal bradycardia ranges from 1.3 to 5.5% [16, 17, 24–26].

In one study ictal asystole was observed in 0.18% of all participants (including those without epilepsy) admitted for video-EEG monitoring and in 0.32% of all people with refractory focal epilepsy admitted for video-EEG monitoring [23]. Two studies have looked at long-term cardiac monitoring using implantable cardiac loop recorders [27, 28]. Rugg-Gunn et al. [27] monitored more than 220,000 patient-hours over a 24-month study period in 19 patients. Electrocardiography (ECG) was captured in 377 of a total of 3377 seizures. Ictal bradycardia (< 40 bpm) occurred in eight seizures (2.1%) in seven patients. Four patients (21%) had bradycardia or periods of asystole requiring subsequent permanent pacemaker insertion, of whom three (16% of total study population) had potentially fatal asystole [27]. Nei et al. [28] applied a loop recorder for an average recording duration of 15 and 1.5 months in 18 patients and one patient, respectively. A median of 37 seizures per patient (range 3–657 seizures/patients) and a total of 1477 seizures were recorded. Only one patient with Lennox–Gastaut syndrome had periods of sinus arrest during sleep (up to 4.8 s), but without reported seizures. The discrepancy between these two studies [27, 28] may be due to the small sample sizes and different patient populations. The seemingly contrasting results between short-recordings during video-EEG monitoring and long-term recordings with implantable cardiac loop recorders suggest that—even in patients with documented ictal systole—ictal asystole does not occur during every seizure and may go unnoticed during short-term monitoring [23]. In a recent review the short-term recurrence risk of ictal asystole was 40% [29]. The authors of this review concluded that in patients with clinically suspected ictal asystole the recording of few seizures is not sufficient to rule out ictal asystole. Furthermore, they suggested that in patients with documented ictal asystole the high short-term recurrence risk may favor pacemaker implantation if seizure freedom cannot be achieved [29].

Ictal asystole represents a feature of focal epilepsy and of focal seizures. Thus, in two recent reviews covering 103 [23] and 157 patients with ictal asystole [30], respectively, all patients suffered from focal epilepsy. Predominantly focal seizures remaining focal resulted in ictal asystole; specifically, of 132 seizures with ictal asystole, 120 seizures (90.9%) remained focal, while only in 12 seizures (9.1%) did ictal asystole appear after seizures which had evolved to bilateral tonic–clonic seizures [30]. Seizure types at onset of ictal asystole were focal onset seizures, with impaired awareness in 99% of 103 cases [23]; 7% of focal seizures evolved to bilateral tonic–clonic seizures after the onset of asystole [23].

Applying cortical stimulation to human subjects, Oppenheimer et al. [15] found depressor responses and bradycardia during stimulation of the left insular cortex, whereas the converse occurred during stimulation of the right insular cortex. This result would suggest that seizures arising from the left hemisphere would be more prone to result in ictal bradycardia and eventually ictal asystole than those arising from the right hemisphere. Van der Lende et al. [23] reported no consistent lateralization of epileptic activity during ictal asystole, but Tenyi et al. [30] found that both the seizure-onset zone (62%) and focal seizure activity at asystole beginning (69%), when lateralized, were more often lateralized to the left hemisphere. However, most of the reviewed cases showed bilateral seizure activity [26, 30]. With respect to localization, the seizure onset zone was temporal in 80–82% of cases reviewed by Tenyi et al. [30] and 90% of cases reported by van der Lende et al. [23].

Tenyi et al. [30] distinguished between 'new-onset' ictal asystole in patients with a delay of < 1 year between epilepsy onset and ictal asystole onset versus 'late-onset' ictal asystole in patients with a delay of ≥ 1 year between epilepsy onset and ictal asystole onset. While 'new-onset' ictal asystole, seen in 27% of patients in their study, was associated with female gender, pre-existing heart condition, focal seizure activity at asystole beginning, normal neuroimaging, normal interictal EEG, auditory auras, and drug-responsive epilepsy, 'late-onset' ictal asystole was observed in males with intractable epilepsy [30].

In the studies of Tenyi et al. [30] and van der Lende et al. [23] asystole duration was reported to average 18 s (range 3–96 s) and 20 s (range 3–96 s), respectively. Ictal asystole was longer in drug-responsive patients than in medically

refractory ones. To the contrary, ictal asystole duration did not differ according to whether the patients received or did not receive sodium channel blockers nor was it influenced by the number of sodium channel blockers administered [30].

Tenyi et al. [30] considered ictal asystole that lasted ≤ 30 s as being 'not very prolonged,' while considering ictal asystole lasting > 30 s as being 'very prolonged.' While 90% of patients showed 'not very prolonged' ictal asystole, 10% of patients suffered from 'very prolonged' ictal asystole with a mean duration of 49 ± 18 s (range 31–96 s). 'Very prolonged' ictal asystole was associated with focal seizures evolving to bilateral tonic–clonic seizures and tended to occur more often in extratemporal lobe seizures.

In terms of therapy and outcome, in patients in whom antiepileptic drug therapy or epilepsy surgery lead to sustained freedom, ictal asystole and associated falls can be successfully prevented without the need for cardiac pacemaker implantation [29–35]. To the contrary, in medically refractory patients cardiac pacemaker implantation should be considered because it effectively prevents continued falls caused by ictal asystole [30, 36]. Moseley et al. [36] reported the mean fall rate in their study population was reduced from 3.28 falls per month before to 0.005 falls per month following pacemaker implantation. Tenyi et al. [30] observed no ictal asystole-related death in any of the 157 cases they reviewed, but van der Lende et al. [23] reported that all ictal asystoles were self-limiting, except in one subject for whom resuscitation was started after 44 s of cardiac arrest. These results suggest that ictal asystole is a self-limiting condition. Ictal asystole parallels centrally mediated cardioinhibition seen in vasovagal syncope, with a tendency for tachycardia to precede the asystolic event, which then evolves into progressive bradycardia leading to asystole [37]. Furthermore, cerebral anoxia–ischemia is a potent inhibitor of cerebral hyperactivity and therefore a potential mechanism of seizure self-termination [38, 39]. Indeed, seizures with syncopal ictal asystole have been reported to be shorter than those with non-syncopal ictal bradycardia, and the latter were in turn shorter than non-bradycardic seizures [39]. Also, seizures with ictal asystole accompanied by cerebral anoxia–ischemia manifested by bilateral hypersynchronous slowing on EEG were shorter than seizures without signs of cerebral anoxia–ischemia [38]; the appearance of bilateral hypersynchronous slowing on EEG ultimately reduced seizure duration [38].

Postictal asystole

Ictal asystole must be contrasted to postictal asystole. In a review of 13 cases of postictal asystole, van der Lende et al. [23] reported that 85% of postictal asystoles were seen after focal seizures evolving to bilateral tonic–clonic seizures, with a mean duration of 30 s; 70% of postictal asystoles were preceded by postictal generalized EEG suppression, and 100% were preceded by apnea. Of the 13 patients in this study, seven died of (probable) sudden unexpected death in epilepsy (SUDEP) [23]. Similar findings were obtained in other studies [40, 41].

Other peri-ictal cardiac arrhythmias

Other peri-ictal cardiac arrhythmias include ictal and postictal atrial flutter/atrial fibrillation and postictal ventricular fibrillation [23, 25, 42, 43]. Three cases of postictal ventricular fibrillation preceded by bilateral tonic–clonic seizures were identified in a recent review article. Cardiopulmonary resuscitation was initiated in all three cases, with two cases being classified as near-SUDEP and one as definite SUDEP [23]. While increased sympathetic activity induced by bilateral tonic–clonic seizures has been implicated as a trigger for postictal ventricular fibrillation, other factors, including a higher prevalence of ECG markers for sudden cardiac arrest, peri-ictal QTc prolongation, ST changes, and increased troponin levels, have been suggested as contributing factors [23, 44]. Nevertheless, cardiovascular disease rather than epilepsy characteristics represent the main determinants of ventricular fibrillation in people with epilepsy [45]. Finally, potentially high-risk cardiac arrhythmias, including nonsustained ventricular tachycardia following focal seizures evolving to bilateral tonic–clonic seizures and generalized tonic–clonic seizures, have been associated with the duration of peri-ictal hypoxemia [46].

Respiratory manifestations

Ictal hyperventilation

Hyperventilation can occur during focal seizures. However, there is a lack of precise prevalence figures for ictal hyperventilation as respiration is not routinely assessed during video-EEG monitoring. In temporal lobe epilepsy hyperventilation occurs more frequently in seizures of mesial temporal lobe origin than in neocortical temporal lobe seizures, a phenomenon which can be explained by projections from mesial temporal lobe structures to the hypothalamus and to brain stem autonomic nuclei [47]. In frontal lobe epilepsy hyperventilation has been reported in seizures arising from the frontopolar and the orbitofrontal regions [48]. Hyperventilation and/or shortness of breath during focal emotional seizures must be differentiated from panic attacks and acute hyperventilation [1, 6].

Ictal oxygen desaturations, ictal hypoventilation, and ictal central apnea

In the study of Bateman et al. [49], ictal oxygen desaturations of < 90, < 80, and < 70% were observed in 33.2, 10.2, and 3.6% of all seizures in patients with intractable epilepsy, respectively. In another study, Lacuey et al. [50] found that ictal hypoxemia was present in 70.8% of 79 seizures, with desaturation being mild (mean 92.5) in 46% of all seizures, moderate in 39% (mean 81.5%), and severe in 14% (mean 64.7%). Desaturations of < 90% in the study of Bateman et al. [49] were significantly correlated with a temporal lobe seizure onset, right hemispheric seizure lateralization, and contralateral spread. The degree of desaturation was significantly correlated with seizure duration and with seizure spread to the contralateral hemisphere. All oxygen desaturations of < 85% were accompanied by an increase in end-tidal carbon dioxide ($ETCO_2$) and therefore were regarded by the authors as a consequence of hypoventilation [49]. Oxygen desaturations lasting longer than 70 s with a nadir of 83% have been reported during focal seizures which did not evolve to bilateral tonic–clonic seizures, which further supports the hypothesis of a centrally mediated hypoventilation during focal epileptic seizures [51].

Based on polygraphic measurements, Lacuey et al. reported that ictal central apnea with a mean duration of 28 s was present in 36.5% of seizures in 40.5% of patients [50]. Prolonged ictal central apnea (≥ 60 s) occurred in 6.3% of patients in association with severe hypoxemia (blood oxygen saturation [SpO_2] < 75%). Ictal central apnea preceded EEG seizure onset by an average of 8 s in 54.3% of seizures and was the only clinical manifestation in 16.5% of seizures. Ictal central apnea occurred significantly more often in temporal lobe epilepsy than in extratemporal epilepsy and was correlated with typical temporal seizure semiologies. The manifestation of ictal central apnea agnosia was typical, and thus ictal central apnea may remain unrecognized without polygraphic measurements, including those of breathing parameters. Seizure-induced inhibition or disruption of brainstem inspiratory neuronal function was considered to be the most likely pathomechanism for ictal central apnea [50].

Postictal nosewiping

Unilateral postictal nosewiping occurring in 40–50% patients with temporal lobe epilepsy was taken to indicate an ipsilateral seizure onset in some studies [52–56], but one study failed to confirm its lateralizing value [7]. Postictal nosewiping occurs more frequently after seizures originating in the non-dominant temporal lobe and can be interpreted as a purposeful reaction to increased upper airway secretion during seizures. The hand ipsilateral to the hemisphere of seizure onset is used most probably due to a mild contralateral postictal weakness or neglect [52]. A depth electrode study showed that involvement of the amygdala is crucial for the induction of postictal nosewiping, while seizures restricted to the hippocampus did not result in nosewiping [53]. Postictal nosewiping occurs less frequently in extratemporal seizures than in temporal lobe seizures [52, 53, 55], and it has also been observed after absence seizures either unilaterally (with different hands after different seizures in the same patient) or bilaterally [8, 53, 57].

Peri-ictal coughing

Peri-ictal coughing can occur either during a seizure (ictal coughing) or after seizure offset (postictal coughing). While ictal coughing represents a rare symptom, occurring only in 0.16% of patients [58], postictal coughing has been reported in up to 40% of patients with temporal lobe epilepsy and is associated with a mesial temporal seizure onset [59–63]. Peri-ictal coughing is not of lateralizing significance [58–63]. It is frequently associated with other vegetative signs, including hypersalivation and retching; in these cases it can be considered to be a reactive phenomenon in response to an excessive autonomic activation of respiratory secretions. To the contrary, peri-ictal coughing without additional autonomic symptoms may be generated by a direct activation of the central autonomic network and therefore by a different pathophysiological mechanism altogether [58, 64]. The differential diagnosis of peri-ictal coughing includes psychogenic non-epileptic seizures and rarely cough syncope [58, 65].

Other respiratory manifestations

Isolated nocturnal acute larnyngospasm can represent an unusual seizure manifestation in children [66]. Nocturnal choking associated with abnormal motor activity and excessive daytime sleepiness can occur in nocturnal frontal lobe epilepsy and can be misdiagnosed as sleep apnea syndrome [67]. Insular lobe seizures typically start in full consciousness with a sensation of laryngeal constriction and often unpleasant paraesthesias that affect large cutaneous territories [68].

Gastrointestinal symptoms

Epigastric aura

Epigastric auras represent the most common visceral symptom in adult epilepsy patients [6–8]. They occur significantly more often in temporal lobe epilepsy than in extratemporal epilepsy, with a probability of 73.6%, and are observed

significantly more often in mesial temporal lobe epilepsy than in neocortical temporal lobe epilepsy. Epigastric auras followed by ictal oral and manual automatisms are highly indicative of temporal lobe epilepsy, with a probability of 98.3% [69]. While some early studies reported epigastric auras significantly more often in patients with seizures arising from the non-dominant temporal lobe than from other temporal lobes [70, 71], the most recent studies are in agreement that epigastric auras have no lateralizing significance [7, 60, 69, 72–74].

Ictal vomiting and ictal retching in adults

Ictal vomiting and ictal retching are rare clinical seizure manifestations during temporal lobe seizures in adults and indicate a seizure onset in the non-dominant hemisphere [6, 75–81]. A lateralization towards the non-dominant hemisphere may be explained by a functional hemispheric asymmetry of gastrointestinal motility control [6, 10]. However, several cases with ictal vomiting in seizures originating from the left (dominant) temporal lobe have been described in the literature [82–84], and some recent studies could not find a lateralizing significance of ictal vomiting at all [8, 85].

Ictal vomiting can be preceded by an feeling of nausea and usually is associated with other symptoms typical of temporal lobe seizures, such as behavioral arrest, staring, eye blinking, oral and bilateral hand automatisms, and facial grimacing [76–79, 81]. The specific cortical areas generating ictal vomiting are still controversial. Some authors have suggested that activation of a complex cortical network that includes medial and lateral parts of the temporal lobe, especially the lateral superior temporal cortex (including the insula and possibly the occipital lobes), is necessary for the generation of ictal vomiting [6, 77, 79, 81]. The involvement of such a network is in accordance with the results of cortical stimulation studies in which abdominal sensations were elucidated from several distinct brain areas, which failed however to induce vomiting during stimulation of a single brain site [86, 87]. Kramer et al. [77] evaluated patients using subdural grid electrodes and found that most of the epileptogenic activity was seen in medial temporal regions while ictal vomiting was associated with the spread of EEG seizure activity to more lateral and superior parts of the temporal lobe. In another study in which a patient presenting with ictal vomiting was investigated with bilateral intracranial electrodes, including insular depth electrodes, the seizure onset zone was localized in the left temporomesial structures, but the occurrence of ictal vomiting correlated in time with ictal activity involving exclusively the anterior part of both insular lobes [88]. Ictal SPECT (single-photon emission computed tomography) in patients with ictal vomiting revealed a hyperperfusion of the medial and lateral aspects of the temporal lobe with concomitant involvement of the lateral superior temporal cortex; these results suggested involvement of the insular cortex [81]. However, a recent stereo-EEG study in a girl presenting with ictal vomiting showed that the episodes of vomiting were strictly related to ictal discharges originating in mesial temporal structures without insular propagation [89].

Ictal vomiting in children

Whereas ictal vomiting is rare in adult epilepsy patients, various idiopathic focal epilepsies of childhood are commonly associated with seizures presenting with vomiting [90].

Panayiotopoulos syndrome is a benign age-related focal seizure disorder with a peak onset at around 2 years of age. Patients present with infrequent autonomic seizures that occur predominantly shortly after the child falls asleep and typically last a few minutes. EEG shows shifting and/or multiple foci, often with occipital predominance. Seizures start with lateral tonic eye deviation as well as with emesis, pallor, or flushing, culminating in vomiting in > 75% of patients. Seizures often evolve to rhythmic muscle contractions on one or both sides of the body. Status epilepticus at onset with neurovegetative symptoms may occur [91–95].

Idiopathic photosensitive occipital lobe epilepsy represents an idiopathic localization-related epileptic syndrome with age-related onset. It has a specific mode of precipitation, and seizures present with visual symptoms, cephalic pain, epigastric discomfort, vomiting, and normal or only mildly impaired responsiveness [96].

Guerrini et al. [78] attributed the mechanism causing vomiting in these idiopathic focal epilepsies of childhood to an infrasylvian spread of epileptic discharges to the non-dominant temporal lobe. An alternative suggestion is that both epileptic cortical manifestations and vomiting are the result of an age-related neurotransmitter-mediated process that is excitatory both for the brainstem vomiting center and/or the chemoreceptor triggering zone as well as the cerebral cortex [97]. Finally, postictal migrainous phenomena triggered by an occipital discharge are suspected of being responsible for vomiting associated with occipital lobe seizures [98].

Peri-ictal spitting

Ictal spitting is an infrequent symptom that occurs in 0.3–2% of temporal lobe seizures [7, 62, 99–101]. While the authors of several studies have reported ictal spitting in seizures arising from the non-dominant temporal lobe [99, 101, 102], ictal spitting has also been found in seizures originating in the dominant temporal lobe [7, 100, 103]. In a patient investigated with bilateral depth and strip electrodes, the epileptogenic area was localized to the left mesial temporal lobe [104]. However, the ictal

symptomatogenic area for ictal spitting turned out to be the right hemisphere based on the observation that spitting automatism occurred only after ictal activity propagated to the right temporal depth electrodes [104]. Ictal spitting can occur with and without preserved awareness. Because patients usually do not report ictal gustatory hallucinations, ictal spitting can be considered to be a pure motor automatism rather than a provoked symptom in response to a gustatory aura [6]. Ictal spitting does not occur in association with ictal vomiting, ictal coughing, or ictal fear, suggesting that different brain sites mediate these symptoms [99]. Kellinghaus et al. [101] suggested that ictal spitting with vehemence, at times aimed at other persons, or spitting on the floor could be interpreted as an expression of emotional disgust. Because the language-nondominant hemisphere seems to control emotional behavior, ictal spitting could be explained by functional disturbances of areas controlling emotional behavior in the nondominant hemisphere [101].

Ictal hypersalivation

Shah et al. studied hypersalivation in temporal lobeepilepsy and observed ictal hypersalivation to be a prominent manifestation of complex partial seizures in ten of the 590 consecutive patients (1.7%) admitted for video-EEG monitoring [105]. All patients suffered from mesial temporal lobe epilepsy with hippocampal atrophy (7 from nondominant temporal lobe epilepsy and 3 from dominant temporal lobe epilepsy). These authors concluded that ictal hypersalivation indicates a seizure onset in the nondominant temporal lobe [105]. However, the lateralizing significance of ictal hypersalivation could not be replicated in another study in 141 patients with medial temporal lobe epilepsy [7]. In the latter study, hypersalivation occurred in 12% of patients, but with an equal frequency in seizure arising from the dominant and non-dominant temporal lobe, respectively [7]. Hoffmann et al. [63] observed ictal hypersalivation exclusively in 9.4% of seizures of mesial temporal lobe origin, but in none in those of lateral temporal origin. However, their comparison between left (11.4%) and right (6.9%) mesial temporal origin was statistically insignificant [63]. To summarize, ictal hypersalivation seems to indicate a mesial temporal seizure onset without lateralizing significance.

Ictal flatulence

Ictal flatulence is a rare symptom that occurs in 0.6% of patients. Ictal flatulence points towards temporal or/and insular involvement during seizure evolution, but it seems to have no lateralizing value [106–108].

Ictal urge to defecate

An ictal urge to defecate was reported in few case reports and seems to indicate a seizure onset in the non-dominant temporal lobe [109, 110].

Cutaneous manifestations

Ictal flushing

Seizures with flushing have been reported by several authors [87, 111]. On rare occasions temporal lobe seizures are characterized by transient paroxysms of flushing, hypertension, tachycardia, and increased plasma catecholamine levels at the peak of the spells [112]. Seizures with flushing associated with emesis and/or pallor are almost always among the first symptoms in Panayiotopoulos syndrome, and these usually culminate in ictal vomiting [95]. Seizures with flushing have to be differentiated from pheochromocytoma, carcinoid syndrome, mastocytosis, ‘hot flushes’ in postmenopausal women, and asymmetric face flushing, known as harlequin syndrome [113, 114].

Ictal pallor

Ictal pallor, originally described by Mulder et al. [111] and Van Buren [87], is observed as a cardinal feature in various epileptic conditions, including temporal lobe epilepsy with ictal fear due to atrophy of the amygdala [115], bilateral perisylvian polymicrogyria associated with abdominal epilepsy [116], and Panayiotopoulos syndrome [93, 95]. Pallor is an extremely common prodromal sign in reflex syncope, occurring in up to 93% of seizures [117], and it may also occur in isolation, i.e., without loss of consciousness.

Ictal sweating

Ictal sweating was first reported by Penfield and Kristiansen [118] and later by Mulder [111] and Van Buren [87]. In some patients ictal sweating occurs in association with focal sensory or motor symptoms in an identical spatial distribution [118]. Seizures with sweating have also been described in a patient with a basal forebrain malformation who experienced attacks of episodic sweating and shivering with reduced core temperature as a variant of Shapiro’s syndrome [119], in a patient with abdominal epilepsy in whom sweating occurred in conjunction with abdominal pain, headache, and vertigo [120], in a tumor patient who suffered from attacks of generalized coldness and sweating [121], and recently in a male patient with anti-Ma2-positive autoimmune encephalitis associated with testicular teratoma [122]. This latter patient developed seizures with profuse sweating strictly of the left

face, with thick beads of sweat developing within seconds, ipsilateral to a left most likely temporo-parietal or posterior insular seizure onset zone [122].

Ictal piloerection

Ictal piloerection occurs in 0.4–1.2% of patients with medically refractory seizures, predominantly in patients with temporal lobe epilepsy [123–125]. Ictal piloerection may be distributed unilaterally or bilaterally, occasionally with a somatotopical spread pattern. While unilateral piloerection is most frequently associated with an ipsilateral seizure onset zone, bilateral ictal piloerection has no lateralizing significance [125]. The exact location of the driver underlying ictal piloerection remains unclear, but the insula or amygdala are regarded as key structures [125]. Ictal piloerection frequently occurs in patients with autoimmune encephalitis, especially limbic encephalitis [124, 126], or in patients with malignant brain tumors [127, 128].

Sexual and genital manifestations

Sexual and genital seizure manifestations can occur during or after focal and generalized seizures and can be subdivided into (1) sexual auras, (2) genital auras, (3) sexual automatisms and (4) genital automatisms where 'sexual' refers to symptoms/signs with erotic content while 'genital' refers to symptoms/signs involving the genitals without erotic components [6, 129, 130]. According to the new seizure classification sexual auras can be classified as focal emotional seizures, genital auras as focal sensory seizures, sexual automatisms as focal emotional or focal hyperkinetic seizures, and genital automatisms as focal seizures with automatisms [9].

Sexual auras

Sexual auras consist of erotic thoughts and feelings, sexual arousal, and orgasm, and they may be accompanied by appropriate viscerosensory and autonomic changes of sexual excitement, including vulvovaginal secretory activity, as well as by olfactory hallucinations. Sexual auras occur predominantly in women, suggesting a gender-specific organization of sexual functions within the limbic system [131–135]. Although orgasmic auras occur more frequently in seizures arising from the non-dominant temporal lobe, they have no absolute lateralizing significance [136, 137]. In a depth electrode study, Chaton et al. [138] documented an association between a spontaneous orgasmic aura and ictal discharges limited to the right amygdala without involvement of orbitofrontal, temporobasal, and insular cortices, thereby confirming the central role of the amygdala in human sexuality.

Genital auras

Genital auras are characterized by unpleasant, sometimes painful, frightening or emotionally neutral somatosensory sensations in the genitals and can be accompanied by ictal orgasm. Genital auras are generated by epileptic discharges in the parasagittal postcentral gyrus where the cortical representation of genital sensation resides [139–142].

Sexual automatisms

Sexual automatisms are characterized by hypermotor movements consisting of writhing, thrusting, and rhythmic movements of the pelvis, arms, and legs, sometimes in association with picking and rhythmic manipulation of the groin or genitalia, exhibitionism, and masturbation. These movements are considered to be typical for frontal lobe seizures [143–145].

Genital automatisms

Genital automatisms, defined as repeated grabbing, fondling, or scratching the genitals, can be accompanied by masturbatory activity and exhibitionistic behavior and occur in 3–11.4% of patients with refractory epilepsy [129, 130, 132–134, 146–148]. Genital automatisms are more prevalent in men than in women and occur more frequently in temporal lobe seizures [148]. This temporal preponderance explains why the most frequent genital automatisms consist of subtle phenomena [130]. Genital automatisms have a high lateralizing value to the ipsilateral hemisphere and are mostly concordant with other unilateral hand automatisms [148]. In rare instances genital automatisms have been reported in generalized epilepsies [147, 149].

Sexual and genital automatisms need to be differentiated from self-stimulatory behavior which occurs particularly in young children and which can be mistaken for seizure activity given the repetitive nature of the behavior. The difference is that self-stimulatory behavior can be distracted, while sexual/genital automatisms cannot [150].

Urinary symptoms

Ictal urinary urge and urination

Ictal urinary urge, defined as an ictal 'desire to void,' was first described by Feindel and Penfield in 1954 [151]. Ictal urinary urge is a rare symptom, occurring in 0.4–3% of temporal lobe epilepsy patients [7, 152–156]. While some patients remember ictal urinary urge when interviewed

postictally, others are amnesic regarding this symptom [152]. In most patients reported to date, including two patients investigated with intracranial electrodes [154, 156], the seizure onset could be lateralized to the non-dominant temporal lobe [152, 153, 155]. Only one study did not find a consistent lateralization in patients with ictal urinary urge [7]. The lateralization to the non-dominant hemisphere can be explained by a hemisphere-specific representation of central bladder control. With respect to the specific brain areas responsible for the generation of ictal urinary urge, involvement of the insular cortex has been proposed. Baumgartner et al. [152] performed Ictal SPECT during ictal urinary urge, revealing hyperperfusion of the superior temporal gyrus containing the insular cortex. This hypothesis is further supported by positron emission tomography studies in normal subjects in whom urination was associated with an activation of the right dorsal pontine tegmentum and the right inferior frontal gyrus, whereas during the filled bladder condition the right frontal operculum and/or the right anterior insula were activated [157]. Thus, epileptic activity within the insular cortex could be responsible for a filled bladder sensation resulting in ictal urinary urge. Alternatively, ictal urinary urge could be mediated by the propagation of epileptic activity to frontal lobe structures, i.e., the right inferior frontal gyrus, which are responsible for suprapontine bladder control [6].

Peri-ictal water drinking

Peri-ictal water drinking, defined as the action of drinking during or within 2 min after an electroclinical seizure, was first described in 1933 by Lennox and Cobb [158]. Peri-ictal water drinking has been reported in 7–15% of patients with focal epilepsy [7, 8, 62, 159–164]. Water drinking was observed to occur in the majority of patients during the seizure as an ictal symptom and was observed less frequently postictally. In the study of Pietrafusa et al. [164], all patients had temporal lobe epilepsy, and all but one suffered from symptomatic epilepsy; 75% of patients had involvement of the right hemisphere. In terms of pathophysiology, propagation of epileptiform discharges from mesial temporal structures to the hypothalamus may cause thirst-activating water-seeking. Lateralization to the non-dominant hemisphere could be explained by asymmetric representation of the central autonomic network responsible for fluid control, thirst, and water-seeking behavior [160]. Alternatively, patients with non-dominant temporal lobe epilepsy are more often able to react to external stimuli and could therefore react to the unpleasant feeling of thirst by water-seeking [62]. Finally, peri-ictal water drinking may be considered a rare automatic behavior, similar to other automatisms [164].

Conclusions

Systematic analysis of autonomic seizure symptoms and signs provides important localizing and lateralizing information on the seizure onset zone and on seizure propagation pathways. Conversely, autonomic seizure symptoms open a unique window on the central autonomic network and are useful for gaining a better understanding of the latter's functional anatomy and pathophysiology.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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