



Review

Semiology of epileptic seizures: A critical review

Soheyl Noachtar*, Astrid S. Peters

Epilepsy Center, Department of Neurology, University of Munich, Marchioninstrasse 15, 81377 Munich, Germany

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ABSTRACT

Epileptic seizures are characterized by a variety of symptoms. Their typical semiology served for a long time as the major tool to classify epilepsy syndromes. The signs and symptoms of epileptic seizures include the following spheres: sensorial sphere, consciousness, motor and autonomic spheres. Most seizures involve more than one sphere, however, some like for instance aura (sensorial sphere) or dialeptic seizures (consciousness) involve only one sphere. The predominant clinical features of a seizure determines the seizure classification. The following review gives an introduction into the semiological seizure classification. This approach enables us to better identify the epileptogenic zone of our patients and to choose the most effective medical or surgical treatment.

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1. Introduction

Careful clinical observations and detailed reports of seizure semiology by the patient or other observers have been used for centuries to classify epileptic seizures and epileptic syndromes. A detailed analysis of seizure semiology is still essential for the proper management of patients with epilepsy. Seizures constitute the main symptomatology of patients with epilepsy, and their control is the target of all treatments. A clear definition of seizure type is important for classification of the epilepsy syndrome. The syndrome, together with the etiology of the epilepsy, is the essential factor determining the prognosis, as well as the most effective pharmacological treatment [1]. Seizure semiology also plays an important role in the presurgical workup, particularly when analyzed independently of other presurgical tests (EEG monitoring, neuroradiology, etc.) [2]. In addition, seizure semiology can be used effectively to differentiate between epileptic and nonepileptic seizures.

Modern video techniques allow a detailed look at a given seizure to investigate its features. Such analyses of epileptic seizures documented by means of simultaneous video and EEG recordings have dramatically improved our knowledge of epileptic seizure semiology. However, interobserver reliability based on qualitative visual analysis is poor for most semiological features [3]. Quantitative analysis of movements during video-recorded seizures may help to develop objective criteria for the analysis of seizure semiology [4–6]. Several lateralizing and localizing seizure types and seizure evolutions have been described.

2. How to classify epileptic seizures

In 2001 a new diagnostic scheme for people with epileptic seizures and epilepsy was proposed by the International League Against Epilepsy (ILAE) to aid clinicians in daily routine and overcome the shortcomings of the seizure classification introduced in 1981 [7,8]. However, this seizure classification system uses an a priori distinction between focal and generalized seizures and semiological aspects in parallel. The clinical decision between a focal seizure and a generalized seizure is often possible only on the basis of additional information from EEG or imaging studies rather than semiological criteria. Another approach to seizure classification is based on strictly semiological criteria to achieve clearness in seizure classification [9]. The distinction between focal and generalized is used in classification of the epileptogenic zone/epilepsy syndrome and not on the level of the epileptic seizure [10].

Epileptic seizures are characterized by a variety of signs and symptoms. Conventional seizure analysis is based on visual expert opinion and focuses mainly on the predominant ictal features. The following four categories are involved in the ictal phenomena of epileptic seizures [11]:

- Sensorial sphere
- Consciousness
- Motor sphere
- Autonomic sphere

Although symptoms corresponding to several of the above-listed spheres occur simultaneously during most epileptic seizures, ictal phenomena of one or the other sphere almost always predominate (Table 1). Only few seizure types involve only one category. An aura, for instance, affects exclusively the sensorial sphere. Be-

* Corresponding author. Fax: +49 (0) 89 70 95 6691.

E-mail address: noa@med.uni-muenchen.de (S. Noachtar).

cause of the absence of objective signs, the examiner has to rely on the patient's depiction of the aura.

Many seizures are associated with a disturbance of consciousness. However, only for some seizures is the disturbance considered the predominant feature of the seizure. The ascertainment of a disturbed consciousness is not proof of a certain type of seizure and therefore cannot be used exclusively for seizure classification. For example, generalized tonic–clonic seizures are typically associated with impairment of consciousness. As the predominant seizure symptomatology involves the motor sphere, the seizure is classified as generalized tonic–clonic seizure.

So far, several terms are currently used for seizures whose predominant semiology is disturbed consciousness. According to the EEG results and the underlying epilepsy syndrome, for example, the episodic alteration of consciousness is labeled *absence seizure* if the patient has generalized 3-Hz spike–wave complexes on the EEG, whereas the same seizure semiology would be called *complex partial seizure* if the patient had a focal epilepsy or the EEG showed focal epileptiform discharges [7]. Terms like *pseudo-absence* have been proposed for these seizures in patients with focal epilepsy [12], because the term *absence* is used exclusively for patients with generalized epilepsy in the seizure classification of the ILAE [7]. Thus, different terms (*absence*, *complex partial seizure*) were used, although the seizure semiology may be clinically indistinguishable. To avoid this confusion the term *dialeptic seizure* was introduced for ictal episodes with an alteration of consciousness as the main manifestation, disregarding the underlying type of epilepsy [13,14]. The term *dialeptic* is derived from the Greek verb *διαλείπω πειν* (*dialeipein*), which means “to stand still,” “to interrupt,” or “to pass out.”

Seizures with motor phenomena as their main manifestations are called motor seizures.

Autonomic seizures are extremely rare. The predominant symptomatology for this type of seizure is an objectively documented alteration of the autonomic system (i.e., tachycardia documented by ECG recording) regardless of whether the patient is aware of the seizure. In contrast, an autonomic alteration (i.e., tachycardia) noticed by the patient without being objectively tested (i.e., ECG recording) or observed is considered an autonomic aura.

Seizures that cannot be assigned to any of the four groups outlined above are included in the group “special seizures.” This category includes primarily seizures characterized as “negative” ictal phenomena (atonic seizure, negative myoclonic seizure, etc.).

The semiological seizure classification [13,14] (Table 1) enables clinicians to classify seizures as precise as possible according to the available information. For example, an epileptic seizure of a patient would be classified as an “epileptic seizure” if no further information were available. If the seizure were characterized primarily by motor phenomena, it would be classified as a “motor seizure.” If the motor seizure affected the right arm, but further specification was not possible, it would be classified as a “right arm motor seizure.” If clonic jerking of the right arm occurred during the seizure, the best seizure classification would be “right arm clonic seizure.” This is the most precise way to label the seizure according to the semiological seizure classification. If little information is available, it still leads to a clinically meaningful classification (motor seizure) rather than classifying the seizures as not-classified seizures.

In the following, the localizing and lateralizing significance of the different seizure types based on the semiological seizure classification system is discussed in detail [13,15].

3. Status epilepticus

Essentially any seizure type discussed above can manifest as status epilepticus, although this clinical experience has not been

evaluated in detail. Typical examples are clonic status of one extremity (“epilepsia partialis continua”), generalized tonic–clonic status (“grand mal status”), and dialeptic status in patients with absence epilepsy (“absence status”). In generalized tonic–clonic status, patients do not regain consciousness between seizures. Clonic status of one limb can present as continuous twitching, as jerking, or, per definition, as a series of seizures longer than 30 minutes. The epileptogenic zone is typically contralateral to the jerking.

Consciousness may fluctuate in patients with “absence status,” particularly if the condition lasts days. The term *nonconvulsive status* is broad and theoretically includes a variety of status forms such as dialeptic status, automotor status, and also rare examples such as aphasic status. In daily practice, the term *nonconvulsive status* is usually used as a synonym for dialeptic or automotor status. Dialeptic or automotor status can be localized only with EEG and imaging studies.

The classification of status epilepticus (SE) has been a subject of discussion for many years, yet no satisfactory agreement has been reached. Because of their complexity, status episodes often defy classification according to the current international classification scheme. The semiological seizure classification (SSC) has been in use in several epilepsy centers for about two decades, and has proven to be a valid approach to the classification of epileptic seizures. Based on the detailed analysis of more than 100 episodes of SE documented with video/EEG recordings, a proposal for a semiological classification of status epilepticus (SCSE) was presented recently [16]. The SCSE reflects the assumption implied by all modern definitions of SE that “there are as many types of status as there are types of seizures” and relies on the same principles as the SSC, focusing on the main clinical manifestations and the evolution of the status episode. The clinical manifestations of SE are subdivided into semiological components and classified along three axes: the type of brain function predominantly compromised by the seizure activity, the body part involved, and the evolution over time. Each axis contains several subcategories, so that many different levels of accuracy are possible. The SCSE, just like the SSC, is meant to be part of a comprehensive epilepsy classification that classifies as independent variables (epileptogenic zone, ictal semiology, etiology, related medical conditions) the main features of the patient's epilepsy, allowing for each variable maximum flexibility [16].

4. The localizing significance of different seizure types

4.1. Auras

Auras as part of epileptic seizures are defined as exclusively subjective symptoms without objective signs that can be documented by an observer. Auras usually occur at the beginning of a seizure (“warning symptoms”) for seconds up to minutes, although they may be seen in isolation as well. Usually they evolve into other seizure types as a result of the spread of the epileptiform discharge. Several different forms of auras have been described (Table 1). As auras are the first clinical expression of a seizure, they frequently provide extremely useful localizing information about the seizure onset zone [17].

Somatosensory auras include somatosensory sensations limited to a clearly defined region of the body, such as tingling of one arm or one side of the face. The typical “march” of a somatosensory aura reflects the spreading activation of the epileptiform discharge on the contralateral somatosensory cortex. Ipsilateral or bilateral, poorly described sensations of the trunk or distal extremities have rarely been observed in patients with seizures arising from the supplementary sensorimotor area or the secondary sensory area [18,19]. Some of these symptoms may simply be an awareness of

Table 1
Semiological seizure classification

| |
|---|
| Epileptic seizures |
| Auras |
| Somatosensory aura ^a |
| Visual aura ^a |
| Auditory aura ^a |
| Olfactory aura |
| Gustatory aura |
| Autonomic aura |
| Abdominal aura |
| Psychic aura |
| Autonomic seizures ^a |
| Dialeptic seizures ^b |
| Typical dialeptic seizure ^b |
| Motor seizures ^a |
| Simple motor seizures ^a |
| Myoclonic seizure ^a |
| Epileptic spasm ^a |
| Tonic-clonic seizure |
| Tonic seizure ^a |
| Clonic seizure ^a |
| Versive seizure ^a |
| Complex motor seizures ^b |
| Hypermotor seizure ^b |
| Automotor seizure ^b |
| Gelastic seizure |
| Special seizures |
| Atonic seizure ^a |
| Astatic seizure |
| Akinetic seizure ^a |
| Negative myoclonic seizure ^a |
| Hypomotor seizure ^b |
| Aphasic seizure ^b |
| Paroxysmal events |

^a Left/right/axial/generalized/bilateral asymmetric.

^b Lateralizing signs occurring during this seizure type are listed separately.

the tension developing in muscle groups involved in the tonic contraction.

Visual auras usually appear as “bright spots” or sometimes as “dark spots,” and result from epileptic activation of the visual cortex. Ictal blindness may reflect a form of visual aura, but it can also occur as a postictal phenomenon after a visual aura. Visual auras are caused by epileptic activation of the striate and most likely also parts of the parastriate cortex.

Auditory auras consist of auditory hallucinations such as sounds, which originate from epileptic activation of Heschl's gyrus. In addition complex auditory hallucinations, such as hearing voices or tunes, can also occur occasionally. They are attributed to an activation of the temporal association cortex [20].

Olfactory auras are relatively rare and an expression of epileptic activation of the amygdala [21] or, less frequently, of the orbito-frontal part of the gyrus rectus. The patients usually have difficulty describing the characteristics of the odor they perceive.

Gustatory auras are characterized by the perception of taste and are likewise rare. Like olfactory auras, the patients usually cannot identify the taste, except that it is unpleasant. They are most common in patients with temporal epilepsy and less frequent with extratemporal, particularly frontal lobe, epilepsy [22].

Psychic auras consist of a “strange feeling” that arises when the internal or external world is perceived in a distorted manner. Besides emotions like fear and anxiety, phenomena like “*déjà vu*” and “*jamais vu*” can be reported as auras and go along with the feeling that objects, a situation, or a constellation is experienced as familiar or foreign. Patients consistently sense that the feeling is unreal and strange. Psychic auras most probably arise from epileptic activation of the temporal association cortex. Psychic auras are more common in neocortical temporal than mesial temporal epilepsy [23].

Abdominal auras typically consist of a vague, unpleasant feeling in the stomach, as if something were rising. They are often accompanied by autonomic symptoms such as nausea [19]. This aura is frequently described by patients with temporal lobe epilepsy [17,24,25] most likely as a result of epileptic activation of the insula.

Autonomic auras include tachycardia, altered breathing, and sweating. As most of the time these events are recognized subjectively by the patients and cannot be verified without special recordings, these symptoms should be classified as an aura. Epileptic activation of the basal frontal region and the anterior cingulate gyrus can evoke autonomic symptoms without the occurrence of other aura experiences or motor phenomena [19]. Pure ictal tachycardia without any other clinical symptoms is highly correlated with temporal rather than extratemporal epileptogenic activity [26]. However, most occurrences of tachycardia or other autonomic symptoms like sweating at seizure onset are reactive and often an emotional response to the first signs of a seizure. Similar autonomic reactions occur very often in patients with other auras or focal motor seizures, as a result of their fear that the seizure will evolve further. Such autonomic reactions that seem to depend on other seizure symptoms should not be classified as autonomic auras.

4.2. Seizures characterized by a disturbance of the autonomic sphere

These rare seizures show, for example, clear EEG seizure patterns but affect only the autonomic system, such as tachycardia or ictal pallor, without the patient consciously experiencing them; such seizures are documented by polygraphic recordings. Here, strictly speaking, there is no aura, and the seizure would be classified as an autonomic seizure. Pure ictal tachycardia has been shown to have a high localizing value, as the majority of seizures arise from the temporal lobe rather than from extratemporal regions [27]. Ictal heart rate can increase as a result of epileptic activation of autonomic cortex, reflecting a temporal lobe autonomic influence. Ictal apnea has been described rarely, but well-documented examples indicate that it may represent the only seizure symptomatology in patients with focal epilepsies [28].

4.3. Seizures characterized by a disturbance of consciousness

Dialeptic seizures are defined as linked to a disturbed consciousness, during which patients cannot react to external stimuli at all or only to a limited extent, and which cannot be recalled later. To classify a seizure as dialeptic, it should not be associated with any significant motor activity. As consciousness is difficult to define [29], altered consciousness (as does the ILAE classification of electroclinical complexes) [7] was determined as episodes of unresponsiveness or decreased responsiveness that are not caused by motor alterations.

Dialeptic seizures occur in several generalized and focal epilepsies [30]. They frequently occur in generalized epilepsies and typically they are highly associated with generalized spike and wave complexes on the EEG (“absence seizure”). Dialeptic seizures in patients with generalized epilepsies usually consist of short (5–20 seconds) episodes of loss of consciousness, which abruptly occur and also suddenly cease. These dialeptic seizures (“typical absence”) are easily precipitated by hyperventilation (particularly at school age) and, to a lesser degree, by photic stimulation. The main manifestation of this seizure type is loss of consciousness, but minor motor phenomena occur not infrequently, such as eyelid fluttering at a rate of 3 Hz and mild oral and manual automatisms [31]. Based on their associated motor phenomena, “absence” seizures have been divided into six subtypes [31], which have been integrated into the seizure classification of the ILAE [7].

Patients with Lennox–Gastaut syndrome also have dialeptic seizures, but they usually last longer and are of less acute onset and cessation than those in patients with absence epilepsy. These seizures are also called “atypical absence” seizures, and the EEG shows characteristic slow spike–wave complexes, which repeat at a frequency of less than 2.5 Hz. Hyperventilation and photic stimulation do not precipitate dialeptic seizures or the characteristic EEG changes in patients with Lennox–Gastaut syndrome. The distinction between “atypical absences” and “typical absences” of the ILAE is better defined by EEG criteria than clinically [32] according to the repetition rate of the spike–wave complexes.

The pathogenesis of dialeptic seizures (“absence”) in the setting of a generalized epilepsy is still not well established [33,34]. These seizures may arise from diffuse inactivation of the cortex as a result of generalized epileptiform discharges. The initial defect in absences is thought to occur in the corticothalamic neuronal mechanisms responsible for selective attention and/or planning of action, rather than in mechanisms that establish either states or the contents of consciousness [35]. A functional MRI study showed that activation predominated over deactivation in the thalamus, whereas the opposite was seen in the cerebral cortex [36].

Dialeptic seizures also occur in patients with focal epilepsies [12,30,37]; however, there has been no systematic study of the frequency and specific semiological differences of dialeptic seizures occurring in different epilepsy syndromes. Patients with frontal epilepsies have dialeptic seizures termed *frontal absence* or *pseudo-absence* because of their resemblance to the dialeptic seizures seen in patients with generalized epilepsies (“absence”) [12,38]. These “frontal absences” have a tendency to last longer (about 30 seconds) than the typical dialeptic seizures in patients with absence epilepsy. Patients with temporal lobe epilepsy frequently exhibit motionless staring and loss of contact at seizure onset. This dialeptic episode can then be followed by oral and manual automatisms [39]. Isolated examples of dialeptic seizures in patients with extratemporal lesions (parietal) have also been documented [30]. The pathogenesis of dialeptic seizures in patients with focal epilepsies is poorly understood [30]. A SPECT study demonstrated that the thalamus and upper brain stem are involved in disturbance of consciousness in different types of focal epileptic seizures: impairment of consciousness showed a strong association with secondary hyperperfusion in the thalamic/upper brainstem region [40].

4.4. Seizures characterized by motor phenomena

Motor phenomena during epileptic seizures are very characteristic of specific seizure types. Thus, these seizures are divided into simple and complex motor seizures on the basis of the type of motor symptomatology.

4.4.1. Seizures characterized by simple motor phenomena

These seizures are characterized by unnatural, relatively simple movements that can be reproduced by electrical stimulation of the primary and supplementary sensorimotor areas. Simple motor seizures can be divided into the following subtypes depending on the duration of the muscle contraction, the rhythmicity of movement repetition, and the muscles involved: myoclonic seizures, clonic seizures, tonic seizures, epileptic spasms, versive seizures, and tonic–clonic seizures.

4.4.1.1. Myoclonic seizures. Myoclonic seizures consist of sudden muscle jerks of short duration (less than 400 ms), which do not recur in a rhythmic fashion. They can be either bilateral (generalized) or unilateral. Generalized myoclonic seizures, which affect predominantly the shoulders and proximal arms, are typical of patients with juvenile myoclonic epilepsy [41]. They are also often

seen in patients with Lennox–Gastaut syndrome [42]. Myoclonic seizures are frequently associated with generalized polyspikes that have a frontal maximum. The primary motor cortex or premotor areas are most likely involved in the generation of this seizure type.

4.4.1.2. Clonic seizures. Clonic seizures consist of more or less regular, repeated, short contractions of various muscle groups. Focal clonic seizures affect mostly the distal segments of the extremities (e.g., the hand) or the face. Generally they are an expression of epileptic activation of the primary motor or the premotor areas [43]. Clonic activity may show a “march” from the distal to the proximal parts of the extremities, reflecting the spreading activation of the primary motor cortex. Electrical stimulation of the supplementary sensorimotor area can elicit distal clonic movements, but only very rarely [44]. Typically, clonic seizures start with a tonic phase, which frequently is not clinically detected unless polygraphic recordings reveal that the frequency of muscle contraction is higher in the beginning of the seizure and gradually slows, thus leading to recognizable clonic jerks [45].

Unilateral clonic seizures are present in several focal epilepsies. In patients with frontal lobe epilepsy, clonic seizures tend to occur early in the seizure evolution and the patient is usually conscious at the time the clonic activity begins [43,46]. When the seizure is the result of spread of epileptiform activity from the occipital or temporal lobe into the frontal lobe, consciousness is usually altered at the onset of unilateral clonic seizures. Generalized clonic seizures only very rarely occur with preserved consciousness [47].

4.4.1.3. Tonic seizures. Tonic seizures consist of a sustained contraction of one or more muscle groups usually lasting >3 seconds and leading to “positioning.” Tonic seizures in patients with focal epilepsy preferentially affect proximal muscle groups on both sides of the body; however, they predominate most often in the contralateral musculature, leading to an asymmetric posture [48]. In most patients with focal epilepsy, consciousness is unclouded, at least at the onset of such unilateral or asymmetric seizures [49–51]. Strictly unilateral tonic seizures have a high lateralizing significance, pointing to a contralateral seizure onset [51]. Consciousness is disturbed from the beginning of generalized tonic seizures, which are common in patients with Lennox–Gastaut syndrome [42]. Focal tonic seizures most probably originate in the cortical motor areas, that is, the primary motor and supplementary sensorimotor areas. However, the reticular formation of the brain stem and the thalamus were reported to be involved in the generation of tonic seizures in patients with Lennox–Gastaut syndrome [52].

4.4.1.4. Epileptic spasms. Epileptic spasms typically occur between 3 and 12 months of age. They are frequently observed in children with West syndrome and in this context they have also been called *infantile spasms* [53]. Epileptic spasms consist of relatively symmetric tonic and myoclonic features, which may vary in the same patient from one seizure to another. The muscle contractions affect predominantly the proximal and axial muscles and typically lead to flexion of the neck (and legs) and abduction of both arms. Less frequently, myoclonic or tonic extension may lead to an opisthotonic posture. Epileptic spasms usually last 2–10 seconds and frequently occur in clusters where short myoclonic contractions may mix with tonic contractions.

This seizure type is relatively age specific and also occurs in focal epilepsies with different epileptogenic zones. Consequently, it does not allow localization. Children with epileptic spasms typically develop other seizure types after 3–5 years of age [54].

4.4.1.5. Versive seizures. Versive seizures consist of a sustained, unnatural turning of the eyes and head to one side. The version usually consists of a smooth, tonic lateral deviation of the eyes with, not infrequently, a clonic superimposed component. These seizures are the expression of epileptic activation of the frontal eye field that is contralateral to the side to which the eyes turn [19]. Epileptic activity from the temporal lobe or other structures distant from the frontal eye field may spread into this area and cause versive seizures [55]. In this case the version occurs when the patient has already lost consciousness. The lateralizing value of versive seizures in temporal lobe epilepsy was controversially discussed [56,57] until a new definition of the version as a forced, sustained, and unnatural movement was stated [58]. With the use of this definition versive seizures have a high lateralizing significance, particularly when they occur immediately before a generalized tonic–clonic seizure [3].

4.4.1.6. Tonic–clonic seizures. Tonic–clonic seizures are characterized by a typical sequence of a generalized tonic contraction followed by clonic contractions. *Grand mal* (= “the great evil”) is a synonym for generalized tonic–clonic seizure, which is the only seizure type in grand mal epilepsies (i.e., epilepsy with grand mal [generalized tonic–clonic] seizures on awakening) [59]. The seizures have a typical evolution, initially occurring with tonic posturing, adduction, and extension of all four extremities and flexion of the wrist and fingers. This phase lasts approximately 5 to 12 seconds and then evolves into a “tremor-like” twitching [60]. The repetition rate of the twitches gradually decreases and the amplitude increases, giving rise to the clonic phase. The clonic phase consists predominantly of flexion myoclonic jerks of the elbow, hip, and knee. The duration of the tonic–clonic seizures varies between 1 and 2 minutes [60]. Consciousness is always disturbed with the beginning of the tonic phase. Generalized tonic–clonic seizures are always followed by a prolonged postictal coma and confusion.

Generalized tonic–clonic seizures may occur in generalized and focal epilepsy syndromes. Occasionally, other generalized seizure types may evolve into generalized tonic–clonic seizures (i.e., generalized myoclonic seizure → generalized tonic–clonic seizure or dialeptic seizure → generalized tonic–clonic seizure). The evolution of generalized myoclonic seizures into generalized tonic–clonic seizures is typical of juvenile myoclonic epilepsy. In focal epilepsies, generalized tonic–clonic seizures usually constitute the end of a seizure evolution. The focal seizure types preceding a generalized tonic–clonic seizure depend on the cortical region where epileptogenic activity originates. Secondarily generalized tonic–clonic seizures may infrequently evolve into a short (2–10 seconds) focal motor seizure that may be generated by persisting epileptiform discharges in the hemisphere of origin or may involve the contralateral hemisphere (paradoxical version)[58]. The clonic phase of generalized tonic–clonic seizures may end asymmetrically, with clonic jerks persisting in the limbs ipsilateral to the hemisphere of seizure onset [61].

The pathophysiological considerations on the origin of generalized tonic–clonic seizures are the same as discussed above for generalized tonic and generalized clonic seizures.

4.4.2. Seizures characterized by complex motor phenomena

Complex motor seizures consist of motor seizures during which the patient performs movements that imitate natural movements (“automatisms”), and which are relatively complex and tend to involve different body segments moving in different planes. Complex motor seizures are subdivided into three types depending on the characteristics of the automatisms.

4.4.2.1. Hypermotor seizures. Hypermotor seizures are characterized by complex sequences of movement that affect primarily

the proximal body segments and result in large movements that appear violent when executed rapidly. Preserved consciousness is common. The seizure duration is usually less than a minute. This seizure type is frequently seen in patients with epilepsies arising from mesial frontal or supplementary sensorimotor area (SSMA) cortex [18,62,63]. However, spread of epileptic activity into the frontal lobe or SSMA from a remote seizure onset zone is also a frequent cause of hypermotor seizures [64]. Interictal and even ictal EEG recordings may not be revealing in these patients. This and the bizarre appearance of the seizure with preserved consciousness frequently lead to the erroneous diagnosis of nonepileptic (psychogenic) pseudo-seizures [65].

4.4.2.2. Automotor seizures. Seizures characterized by manual and oral automatisms have been identified for a long time, and their relation to temporal lobe epilepsy has been well established [66–68]. Typical examples are orolimentary automatisms such as chewing, swallowing, and smacking the lips or hand automatisms such as fumbling [39,69]. Generally, consciousness is impaired during these automatisms, although there are well-documented exceptions to this rule in patients with temporal lobe epilepsy of the non-speech-dominant hemisphere [70,71]. The term *psychomotor seizure* refers to seizures characterized by automatisms and lapse of consciousness [66]. An almost motionless “arrest behavior” (dialeptic phase) is often observed at the onset of typical automotor seizures [72].

Automotor seizures occur most frequently in patients with temporal lobe epilepsies, but they may also occur in patients with frontal lobe epilepsies [46], especially of orbitofrontal origin [12]. When automotor seizures result from spread into one of the temporal lobes, they are often preceded by other seizure types.

The symptomatogenic zone of automotor seizure onset is not clearly defined, but there is some evidence that epileptic activation of the anterior cingulate gyrus leads to distal automatisms [73].

4.4.2.3. Gelastic seizures. The main feature of this seizure type is ictal “laughing.” The laughter produced in gelastic seizures is usually a stereotyped caricature of normal laughter [74]. Gelastic seizures may be preceded or followed by other seizure types and frequently occur in patients with hypothalamic hamartomas [75].

4.4.3. Special seizures

This group includes all seizures that cannot be classified in one of the four types described earlier (auras, autonomic, dialeptic, or motor seizures). Most of these seizures characteristically have a “negative” influence on motor (atonic, akinetic) or cognitive (aphasic) activity.

4.4.3.1. Atonic seizures. Atonic seizures are characterized by a sudden reduction of postural tone that results in a loss of posture (head drop, falls, etc.). These seizures are often preceded by a brief myoclonic seizure with propulsion or retropulsion. The loss of balance before the fall makes the patients especially prone to injuries, even more so than in an “atonic collapse.” Atonic seizures are frequently seen in patients who also have generalized tonic seizures (Lennox–Gastaut syndrome).

This type of seizure is usually generalized and primarily affects the axial muscles. Generalized atonic seizures are most probably the result of activation, mediated by cortical epileptic discharges, of the inhibitory centers in the brain stem (e.g., nucleus reticularis gigantocellularis) via fast corticoreticulospinal systems [76,77]. However, focal atonia involving only distal parts of the body has been reported and was labeled *ictal paresis* or *inhibitory seizure* [78]. These seizures are classified as akinetic seizures if consciousness is preserved. It is sometimes difficult to distinguish these patients, in whom focal atonia occurs and consciousness is disturbed,

from those with postictal (“Todd”) paralysis and nonepileptic mechanisms, such as migraine and transient ischemic attacks. The pathogenesis of these seizures has not yet been sufficiently clarified. It is possible that some of these seizures reflect ictal activation of the negative motor areas [79]. Electrical stimulation of these areas causes an inability to perform voluntary movements, and in addition, very frequently various degrees of atonia are seen in distal muscle groups (fingers, hands, face, tongue) [80].

4.4.3.2. Astatic seizures. Astatic seizures are characterized by epileptic falls, which can be due to atonic, myoclonic, or tonic seizures. Polygraphic studies show that tonic activity frequently causes the patient to fall, but in a few cases, the fall is caused by a loss of muscle tone. Frequently, a myoclonic seizure leads to a loss of balance and the fall is produced by an atonia, which occurs immediately after the initial myoclonic jerk. In the majority of the patients, however, no polygraphic studies are available, and the pathogenesis of the fall remains uncertain. In such cases it seems useful to classify the epileptic fall as an astatic seizure. If the pathogenesis of the epileptic fall is established, the seizure should be termed accordingly (tonic seizure, atonic seizure, generalized myoclonic seizure → generalized atonic seizure, etc.).

4.4.3.3. Negative myoclonic seizures. A negative myoclonic seizure, which is also called epileptic negative myoclonus, consists of short (ca. 30–400 ms) phases of muscle atonia. The seizure is expressed clinically only during muscle innervation; that is, it does not occur when the patient is at rest [81]. Generalized as well as focal negative myoclonic seizures have been described [82]. Polygraphic recordings have shown that these seizures are frequently preceded by epileptiform discharges in the central region (20–30 ms before the atonia). There is much evidence for a causal sudden inhibition of tonic innervation of the motor neurons, which is reflected in the silent period of the EMG. The cortex regions giving rise to negative myoclonic seizures have not yet been defined. Primary somatosensory motor cortex [83] and premotor cortex have been suggested to act as a generator [84]. A postcentral generator, which inhibits tonic motor activity, was identified in a patient with postcentral focal cortical dysplasia and hyperexcitability of the postcentral cortex, as documented by giant somatosensory evoked potentials and frequent spikes [85].

4.4.3.4. Akinetic seizures. Akinetic seizures are characterized by an inability to perform voluntary movements. By definition, consciousness is not disturbed during this seizure type [86,87]. The inability to initiate and maintain voluntary movements may involve the entire body or only parts of it. Such seizures probably arise from epileptic activation of the so-called negative motor areas, which are identified in the frontal lobe by electrical stimulation of the cortex [79,80].

4.4.3.5. Aphasic seizures. This seizure is characterized by an inability to speak or to comprehend language. Consciousness is preserved by definition. Aphasic seizures most probably reflect epileptic activation of cortical language areas in the speech-dominant hemisphere [88].

4.4.3.6. Hypomotor seizures. The extent of motor activity is reduced or totally absent in these seizures. By definition, the seizures refer only to patients where the state of consciousness cannot be assessed during or after the seizure, that is, newborns, infants, and severely mentally retarded patients. Most likely consciousness is affected in a considerable number of patients, although this cannot be tested in these patients [89]. However, the pathogenetic mechanisms involved in the arrest of motor activity may be different in some hypomotor seizures. For example, consciousness may be pre-

served, and the absence of movements may be the reaction to an aura or an expression of the inability to move, as in akinetic seizures. Hypomotor seizures can occur in focal (mostly temporal) and generalized epilepsies.

5. The localizing significance of seizure evolution

Epileptic seizures frequently evolve from one seizure type into another. It is a well-established fact that the initial seizure symptoms provide information on the location of the seizure onset zone. The initial symptoms such as auras reflect activation of the symptomatogenic cortex, which is likely to be close to the seizure onset zone [17]. However, seizures may arise in silent cortical regions that do not express any clinical symptoms, and in such cases the epileptic activation remains unnoticed. There are typical seizure sequences that point to different epilepsy syndromes. For example, generalized myoclonic seizures not infrequently occur in clusters, particularly if precipitated by sleep deprivation, and evolve into a generalized tonic-clonic seizure [41]. Another example is an abdominal aura that is followed by an automotor seizure. This sequence is frequently, although not exclusively, seen in patients with temporal lobe epilepsy [24]. Early clonic seizures following manual hand automatisms occur significantly more frequently in patients with neocortical temporal lobe epilepsy than in patients with mesial temporal lobe epilepsy. In contrast, patients with mesial temporal lobe epilepsy showed hand dystonia significantly more often in the course of their seizures than patients with neocortical temporal lobe epilepsy [90]. The combination of ipsilateral hand automatism and contralateral hand dystonia was seen only in patients with neocortical temporal epilepsy [91]. Responsiveness can be preserved during automatisms if the seizure origin is in the nondominant hemisphere [70]. Hypermotor or bilateral tonic seizures with preserved responsiveness point to a seizure origin near the supplementary sensorimotor area [92]. In some patients, particularly those with generalized epilepsies, the transition between interictal and ictal events is gradual. Cognitive disturbances such as dialeptic seizures (“absences”) can be documented by reaction time tasks, with stimulus and response times recorded on an event marker channel.

The termination of seizures also provides lateralizing information. The end of generalized tonic-clonic seizures was asymmetric in 65% of the patients of a recent study. The unilateral clonic jerks at the end of the seizures were ipsilateral to the hemisphere of seizure onset in 80% of the patients [61,93]. The postictal period also provides localizing and lateralizing information. Postictal aphasia and Todd’s paralysis are examples that are discussed in the next section.

6. Ictal lateralizing phenomena

Most patients with medically intractable focal epilepsy who are considered for epilepsy surgery show ictal lateralizing phenomena such as dystonic hand posturing, version, ictal vomiting, unilateral clonic seizures, postictal aphasia, and preserved responsiveness during automatisms (Table 2). Dystonic hand posturing and forced head version reliably indicate a seizure onset in the contralateral hemisphere [55,94,95]. Unilateral manual automatisms have been associated with ipsilateral seizure onset in temporal lobe epilepsy [96]. This observation probably reflects, at least partly, dystonia of the contralateral hand and seems to be of lateralizing significance only if there is dystonia of the contralateral hand [39]. The hand used to perform postictal nose wiping was ipsilateral to the side of seizure origin in 97% of patients with temporal lobe epilepsy [97,98], a finding that might be due to neglect of the contralateral arm.

Table 2
Lateralizing seizure phenomena.

| Lateralizing seizure phenomenon | Hemisphere | Authors |
|---|---------------|--|
| Head and eye deviation | Contralateral | Wyllie et al., 1986 [58] O'Dwyer et al., 2007 [4] |
| Dystonic hand posturing | Contralateral | Bleasel et al., 1997 [3] Kotagal et al., 1989 [95] |
| Figure-4 sign | Contralateral | Kotagal et al., 2000 [108] |
| Automatisms with preserved responsiveness | Non-dominant | Ebner et al., 1995 [70] Noachtar et al., 1992 [71] |
| Ictal speech | Nondominant | Gabr et al., 1989 [99] |
| Postictal aphasia | Dominant | Gabr et al., 1989 [99] |
| Ictal vomiting | Nondominant | Kramer et al., 1988 [100] |
| Ictal spitting | Nondominant | Voss et al., 1999 [102] |
| Peri-ictal urinary urge | Nondominant | Baumgartner et al., 2000 [101] |
| Postictal nose rubbing | Ipsilateral | Leutmezer et al., 1998 [97] |
| Postictal coughing | Nondominant | Wennberg, 2001 [103] |
| Unilateral clonic seizure | Contralateral | Jackson, 1898 [67] |
| Unilateral tonic seizure | Contralateral | Werhahn et al., 2000 [51] |
| Unilateral eye blinking | Ipsilateral | Benbadis et al., 1996 [104] Henkel et al., 1999 [105] Wada, 1980 [106] |
| Asymmetric ending | Ipsilateral | Trinka et al., 2002 [61] |

Postictal aphasia is highly suggestive of a seizure onset in the speech-dominant hemisphere [99]. Preserved responsiveness during ictal automatisms was seen only in patients with temporal epilepsy of the non-speech-dominant hemisphere [70,71].

Several seizure phenomena have been described that point to a seizure onset in the typically right nondominant hemisphere (Table 2). Ictal vomiting lateralizes the seizure onset to the right hemisphere in patients with temporal lobe epilepsy [100]. Ictal urinary urge was associated with nondominant temporal seizure onset in a series of six patients [101]. Ictal spitting points to a seizure onset in the nondominant temporal lobe [102]. Postictal coughing occurs with seizure onset in the nondominant hemisphere [103]. Ipsilateral eye blinking is a rare phenomenon (1.5%) that is observed mostly in patients with temporal lobe epilepsy, but is highly suggestive of an ipsilateral seizure onset [104–106].

The positive predictive value of the above-mentioned lateralizing seizure phenomena is correct in 80–100% of such patients. This important lateralizing information should be considered in the classification of epileptic seizures [15,107].

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