Paroxysmal Nonepileptic Events

34

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Paroxysmal nonepileptic events are, like epileptic seizures, time-limited behavioral, cognitive, motor, sensory, and/or vegetative alterations. Unlike epilepsy, however, they do not depend on excessive cortical activity, but either are the result of neurological or systemic disturbances affecting the cerebral functions or have a psychogenic origin. The diagnostic work-up and the differentiation from epileptic seizures rely on the clinical description or visual analysis of the episodes and on several tests and exams.

This chapter will deal with the two conditions that pose the major issues of differential diagnosis with epilepsy: syncope and psychogenic seizures.

34.1 Syncope

34.1.1 Definition and Classification

Syncope is a transient self-limited episode of loss of consciousness resulting from cerebral hypoperfusion [1]. The classification is mainly based on the underlying mechanisms that lead to the transient global hypoperfusion, including the following.

34.1.1.1 Neurally Mediated Syncope (Reflex Syncope)

This is a group of conditions in which the cardiovascular effector mechanisms controlling circulation become overactive, resulting in vasodilatation and/or bradycardia causing a fall of blood pressure and consequently cerebral perfusion [2].

Vasovagal syncope is the most common type of this category and is also the most common cause of non-traumatic transient loss of consciousness, with an estimated 30–40% of people experiencing at least one episode in their lifetime [3]. It is typically triggered by emotional distress or prolonged orthostatism.

Carotid sinus syndrome results from an extreme reflex response to carotid sinus stimulation and may be elicited by rotation or turning of the head or pressure on the carotid sinus (i.e., shaving, tight collars or neckwear, or tumor compression). It is more common in the elderly and primarily in men [4].

Situational syncope may be triggered by different activities such as micturition, defecation, coughing, or swallowing.

34.1.1.2 Orthostatic Hypotension Syncope

Syncope occurs as a consequence of the body's inability to maintain an adequate blood pressure for cerebral perfusion on assuming the upright position [3, 5]. It may be drug-induced (alcohol, vasodilators, diuretics, beta-adrenergic blockers), due to volume depletion (inadequate fluid intake, diarrhea, vomiting, etc.) or caused by a primary autonomic failure (pure autonomic failure, multiple system atrophy, Parkinson's disease with autonomic failure, Lewy body dementia) or secondary autonomic failure (diabetes, amyloidosis, spinal cord injuries).

34.1.1.3 Cardiac Syncope

This category includes mainly syncope due to cardiac arrhythmias. Bradycardia and asystole are the commonest causes. However, supraventricular and ventricular tachyarrhythmias may also trigger syncope.

Less frequently syncope may be caused by valvular or structural heart disease (e.g., severe aortic stenosis, severe mitral stenosis, large left atrial myxoma, acute myocardial infarction) or pulmonary embolism.

34.1.1.4 Syncope Secondary to Cerebrovascular Causes

A transient ischemic attack in the vertebrobasilar distribution is a rare cause of syncope, often accompanied by posterior circulation symptoms (i.e., dizziness and loss of balance).

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Steal syndrome due to subclavian stenosis is a rare condition that may provoke a syncope in case of intense or prolonged use of ipsilateral arm muscles. However, syncope as a solitary manifestation of these conditions is extremely rare [6].

34.1.2 Clinical Features

Syncope semiology includes the rapid onset of loss of consciousness with or without warning symptoms. Symptoms and signs generally fall into two groups [7].

Symptoms of the first group depend on the cause of syncope and include palpitations in arrhythmia or sensation of forthcoming swoon, pallor, and sweating in vasovagal syncope. Carotid sinus syncope generally has no prodromes.

Symptoms of the second group are the consequence of cerebral and retinal hypoperfusion and are therefore less specific, including visual disturbances, loss of consciousness and postural control, stiffness, and myoclonic jerks. When the latter are present, syncope is called "convulsive," and differential diagnosis with epilepsy may be more difficult. As in epileptic seizures, eyes usually remain open in the course of syncope; the most consistent ocular motor sign accompanying syncope is an upward turning of the eyes which can be preceded by a few seconds of downbeat nystagmus [8].

The circumstances in which the episode has occurred together with associated symptoms and signs are important to reach a correct diagnosis.

Several clinical features are useful for differential diagnosis of syncope and epilepsy.

In syncope, stiffness and jerks typically last for a shorter time, and jerks do not present with the typical frequency and amplitude evolution pattern observed in tonic-clonic seizures. Shmuely et al. reported that the number of myoclonic jerks has a strong diagnostic potential in differentiating syncope from convulsive seizures, fewer than 10 jerks indicating syncope and more than 20 seizures.

Loss of tone during transient loss of consciousness strongly favors syncope and argues against a convulsive seizure [9].

Automatisms, such as lip-licking, chewing, fumbling, and reaching for the head as well as growling or moaning vocalizations, are more frequent in epileptic seizures; however they were reported in case series of induced reflex syncope [1, 10].

Recovery from syncope is typically prompt and complete without residual neurologic findings. Enuresis may be observed, while fecal incontinence and tongue biting are very rare [11].

34.1.3 Diagnostic Work-Up

A thorough clinical history taking, together with physical examination, including orthostatic blood pressure measurement, and a basal ECG represent the basic diagnostic work-up, to be performed in all patients presenting with suspected syncope.

Additional exams may be required, depending on the suspected etiology, including cardiac evaluation, echocardiogram, ECG monitoring, and exercise stress testing in the suspicion of a cardiac syncope or provocation tests in reflex syncope [3].

The best-known provocation technique to induce neurally mediated or orthostatic hypotension syncope is tilt-table testing. The most used tilt-table method consists in relatively long duration (20–45 min) passive head-up position on a table with a footboard. A positive test is characterized by the onset of a syncope associated with a documented cardioinhibitory and/or a vasodepressor response causing hypotension. If the passive tilt is nondiagnostic, a pharmacological provocation with nitroglycerine or isoproterenol may be performed [12].

When carotid sinus hyperexcitability is suspected, a carotid sinus massage under ECG monitoring is useful for diagnostic confirmation.

34.1.4 EEG Findings

Although EEG is usually not recommended in the work-up of syncope, several specific features were described and may be of value for shading light on the pathophysiology of the typical clinical signs. The first EEG pattern of syncope to be reported was the "slow-flat-slow" pattern [13, 14], shown in Fig. 34.1. In the first slow phase, the background alpha rhythm is supplanted by a slow activity, decreasing in frequency from theta to delta waves while wave amplitude increases. This slow phase may last for up to 10 s; then the slow activity disappears abruptly, leaving a "flat" EEG whose duration depends on the duration of insufficient flow. The third phase consists of slow activity, in which frequency and amplitude evolve in the reverse order than the first slow phase, and hypersynchronous delta activity may be observed. This pattern is generally thought to denote more severe cerebral hypoperfusion. Accordingly, a second pattern consisting of slow activity only, corresponding to the first "slow" phase of the "slow-flat-slow" pattern, is thought to be associated with shorter or less severe hypoperfusion [7]. Flat EEG is invariably associated with loss of consciousness and postural control; conversely the relation between the level of consciousness and the degree of EEG slowing at the beginning of the episode remains unclear [7]. Motor phenomena occur at various phases and seem to be related to cortical ischemia, with slow EEG resulting from a reduced cortical function and flat EEG periods depending on suppression of cortical activity. Myoclonic jerks are mostly seen during the slow EEG phase, both at the onset of syncope and during its

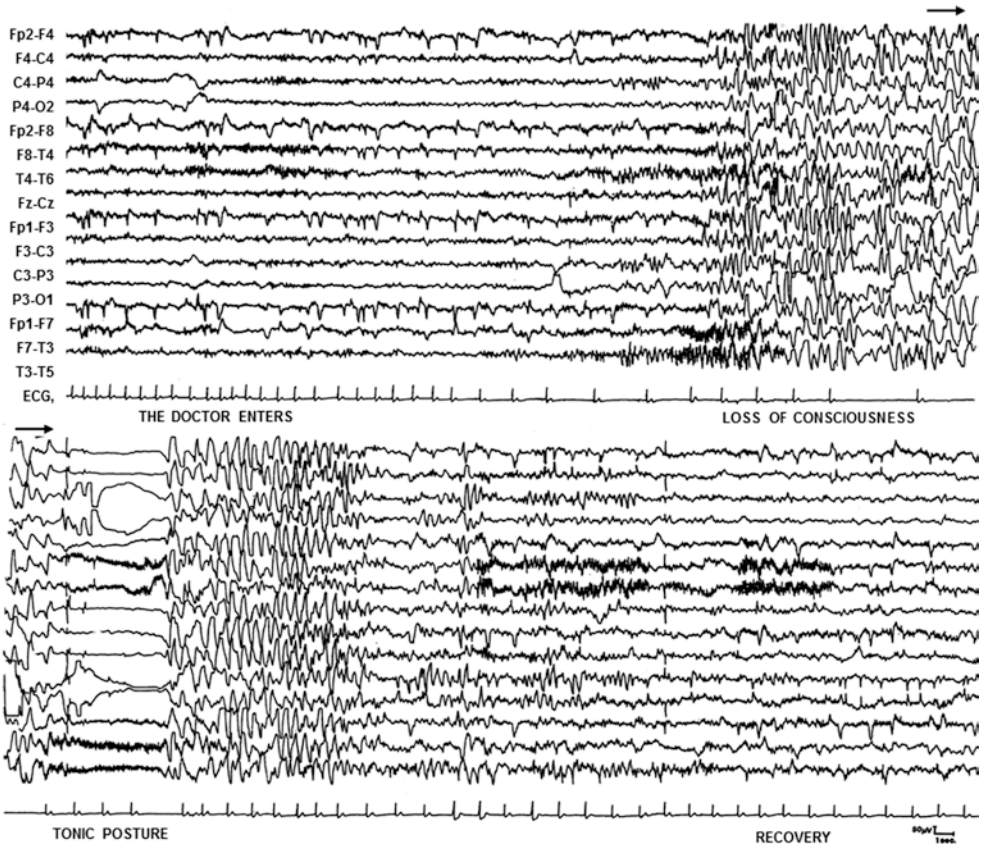

Fig. 34.1 A typical "slow-flat-slow" pattern in an 8-year-old child. After the onset of bradycardia, observable in the ECG trace, the background rhythm is supplanted by a slow, ample, theta-delta activity. During this phase a brief pause on ECG may be noticed and loss of consciousness supervenes. The slow activity then disappears abruptly, leaving a brief flattening on the EEG during which a tonic posture appears. ECG then returns to a normal rhythm, while a second slow phase activity appears on the EEG, evolving from hypersynchronous ample delta waves to theta activity. Subsequently the normal background activity resumes and so does consciousness

conclusion [9, 15]. They are likely of cortical origin and are thought to result from cortical hyperexcitability related to impaired cortical function and cortical disinhibition. Their abolition with electroencephalographic flattening suggests dependence on cortical activity.

Flaccidity invariably occurs during slowing of the EEG, while tonic postures, stertorous breathing, and roving eye movements mostly occur during the flat EEG as a result of brainstem disinhibition related to loss of cortical function.

Interictal EEG is usually normal, but a specific EEG slowing, either focal or diffuse, can be observed.

Mecarelli et al. compared the EEG performed in basal condition and during hyperventilation (HV) in patients with neurally mediated syncope versus healthy controls. They found that syncope subjects presented more abundant and pronounced delta-theta activities and alpha slowing. In particular, the patients presented more frequently with slow activities and a peculiar intermittent rhythmic delta activity during prolonged HV (Fig. 34.2). These "pseudoparoxysmal" EEG changes are distinct, both from the common slowing observed during HV in adult subjects and from epileptiform activity [16]. Simultaneous transcranial Doppler and EEG recording performed in patients presenting this EEG pattern suggested that changes in the sympathetic modulation of cerebral vasoconstriction may explain both the pathophysiology of vasovagal syncope and the typical EEG findings [17].

34.1.5 Syncope in Epilepsy

As a rare and challenging condition, syncope may be the expression of an epileptic seizure. Epileptic syncope is typically preceded (and sometimes accompanied) by signs suggesting a temporal seizure, such as psychic or visceral aura, behavioral arrest, unresponsiveness, staring, gestural and oral automatisms, unilateral hypertonia, head turning, and more rarely clonic lateralized movements. It is usually secondary to ictal asystole or sudden bradycardia with concomitant severe hypotension [18–20].

In a series of 26 patients with a video-EEG-ECG recording of an ictal asystole, a typical electroclinical sequence was described. Ictal bradycardia and subsequent asystole arise after more than 30 s after the onset of seizures, and, clinically, seizure symptoms either continue or are replaced by syncope symptoms, while EEG discharges either continue or are supplanted by diffuse slowing (and, clinically, atonia supervenes) and/or EEG suppression (with hypertonia). On average normal EEG activity resumes after 10 s from the recovery of cardiac activity, while skin flushing and late

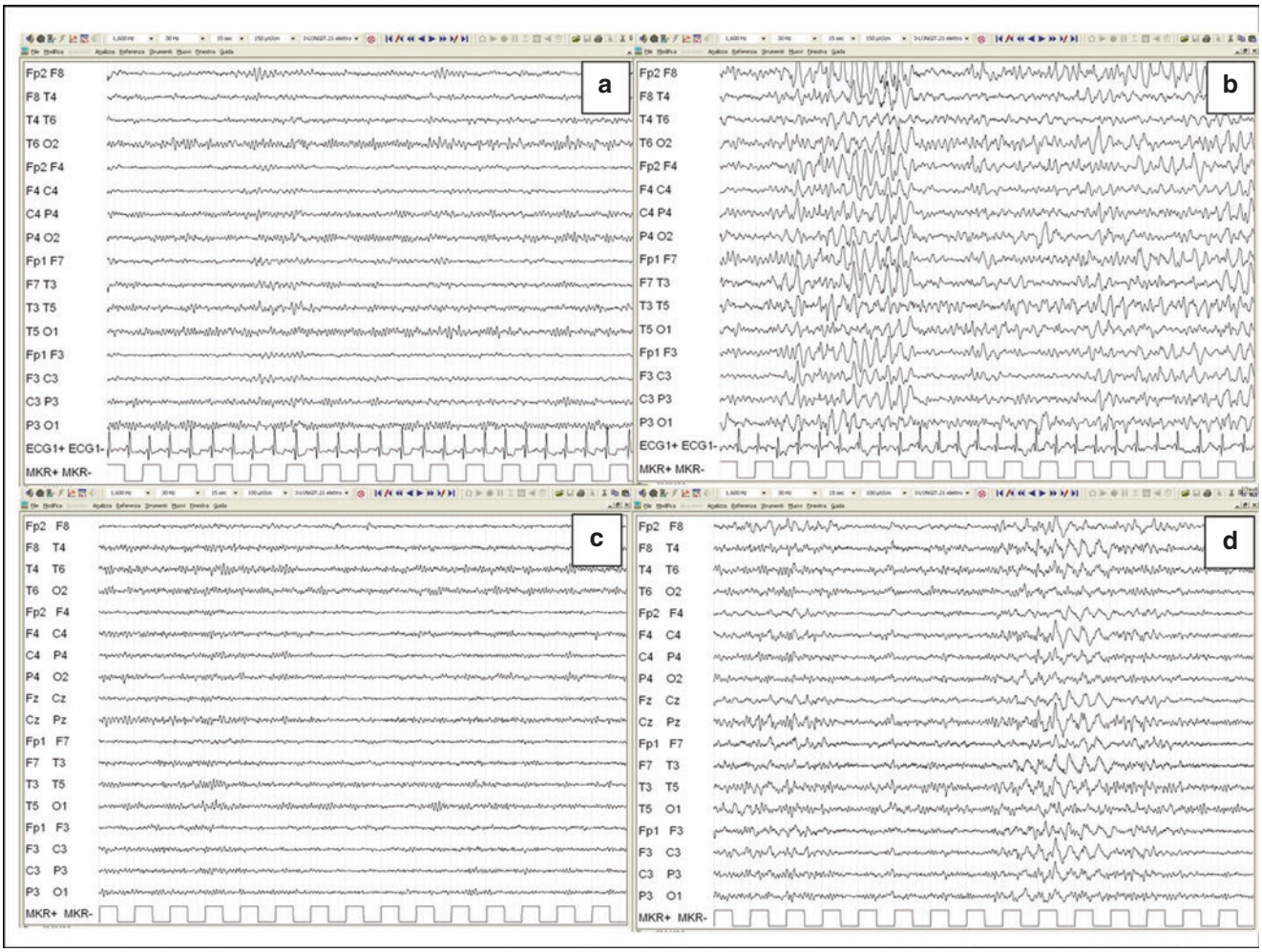

Fig. 34.2 EEG slow "pseudoparoxysmal" patterns after 3 min of hyperpnea in a 16-year-old (**a**, **b**) and in a 21-year-old (**c**, **d**) patients with recurrent vasovagal syncopes (Reproduced from Mecarelli and Zarabla 2009 [21])

myoclonic jerks may appear [20]. Figure 34.3 shows an example of ictal asystole with syncope.

It has also been reported that a syncope can occasionally trigger a seizure in patients with epilepsy [22], probably as a result of the transient cerebral hypoxia.

34.2 Psychogenic Nonepileptic Seizures

34.2.1 Definition and Overview

Psychogenic nonepileptic seizures (PNES) are paroxysmal attacks assumed to be the physical manifestation of a psychic disturbance, the most accredited interpretation being a dissociative response to potentially distressing stimuli. They are not a distinct nosological entity, and most of them are classified as a subtype of conversion disorders in the DSM V [23].

They have been variously named, including functional seizures, hysterical seizures, nonepileptic attack disorder, and pseudoseizures. It is currently advised that the latter should be avoided, as it implies deceit.

PNES are frequently seen in epilepsy centers, where these disturbances represent the final diagnoses for 9 to 50% of patients referred for refractory epilepsy [24–28].

The most common age at presentation is between the third and the fourth decade, and the female to male ratio is 3–4:1 [29, 30].

Patients with intellectual and learning disability and with mild traumatic injury are at greater risk for developing PNES [31, 32]. Although data from different studies are not consistent

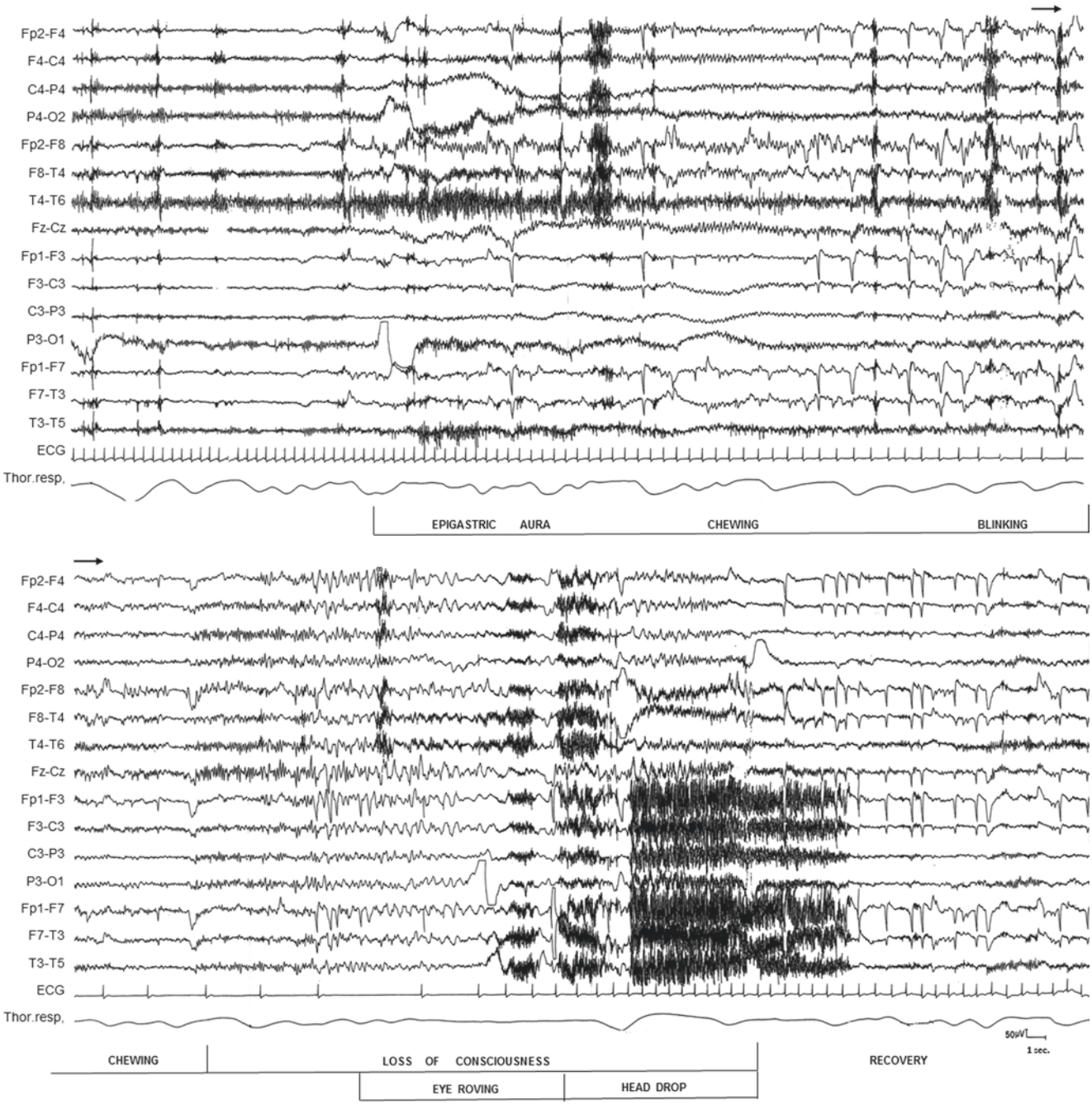

Fig. 34.3 Ictal EEG tracing of a 35-year-old woman with temporal lobe epilepsy. The patient reports an epigastric aura; then she starts chewing, swallowing, and then blinking. She answers correctly to the questions. The automatisms continue for several seconds; then loss of consciousness, accompanied by pallor, eye roving, and head drop, supervenes. During the first part of the seizure, the EEG shows a recruiting discharge over the right temporal lobe, rapidly spreading to the homolateral hemisphere and then contralaterally. Concomitantly to the loss of consciousness, diffuse sharp slow waves appear, followed by a brief EEG flattening and then by a theta activity over the right hemisphere, while the left hemisphere tracing is covered by artifacts. At the beginning of the seizure, the ECG shows tachycardia followed by a progressive decrease of the heart rate, leading to asystole lasting more than 5 s associated with apnea. The bradycardia and subsequent asystole are concomitant to the loss of consciousness, while recovery occurs several seconds after restoring the normal heart rate

concerning specific abnormalities or brain regions, there is evidence of a higher frequency of structural and functional abnormalities in patients with PNES compared to healthy controls [33].

A history of psychological trauma, particularly physical and sexual abuse, and a major life event in the year preceding diagnosis are very common [31, 32, 34–40].

Up to 10% of patients with PNES have comorbid epilepsy [29]. This comorbidity poses peculiar diagnostic difficulties as PNES onset typically follows epilepsy, presenting as a "pseudoresistance" to AEDs, particularly when the new seizures resemble the epileptic ones. Similar diagnostic dilemmas may arise when PNES occur de novo after epilepsy surgery, mimicking a surgical failure [41, 42]. Conversely, preexisting PNES may disappear after epilepsy surgery [42].

Several models were proposed and will not be discussed here, in which biological factors, comorbidities, experiences, and major life events could interact resulting in recurrent PNES [23, 29].

34.2.2 Clinical Ictal Features

Situational features more common in PNES than epileptic seizures (ES) are stressor events as seizure precipitants, habitual presence of "significant" witnesses (including physicians) [27, 30], and gradual onset (contrasting with the abrupt onset of ES) [27, 43]. The occurrence predominantly or exclusively during sleep is very specific of ES. However, PNES may occur during behavioral sleep, with EEG revealing normal waking activity ("pseudosleep") which, on the contrary, is very specific to PNES [26, 44–46].

Prodromal feelings are frequent and may suggest hyperventilation (light headedness, acral paresthesias, and palpitations) [25].

PNES usually last for more than 2 min and may be longer than 30 min, which is very unusual for ES [27, 46].

A motor behavior is frequently reported as part of the attack. The features most often reported in seizures mimicking generalized tonic-clonic seizures (GTCS) are asynchronous out-of-phase limb movements, or absence of in-phase limb movements, side-to-side head/body turning, forward pelvic thrusting, thrashing, and grabbing behavior [27, 46]. The retention of verbal responsiveness during PNES resembling GTCS is pathognomonic for PNES [47].

An important caveat is the resemblance of several motor features with seizures arising from (or propagating to) the frontal mesial structures, during which, to add complexity, awareness is often maintained during bilateral motor attacks, postictal signs are lacking, and ictal EEG may be normal [48–52]. However, movements in PNES generally involve the head and neck, whereas in mesial frontal lobe seizures, they are generalized or mainly involve the lower limbs and trunk. Features strongly pointing toward epilepsy include also turning to a prone position [49], tonic posturing in abduction of upper extremities [48], short-duration, highly stereotyped pattern and frequent or exclusive occurrence during sleep [48, 53].

Tonic posturing and opisthotonus can occur in PNES [26, 47, 48, 54] and the "arc de cercle" described by Charcot and Richer in 1887 is highly specific to these attacks. PNES often exhibit a discontinuous pattern with motor activity alternating with brief periods of rest, in contrast to the epileptic pattern [46, 47, 53, 55]. Motor activity resumes at the same frequency after pauses, and this leads to peculiar artifacts on the ECG and EEG as described later in this chapter [56].

PNES may present also as prolonged limpness without motor symptoms, possibly associated with apparent atonia [26, 35, 47, 54, 55]. Some minimal movements, such as intermittent eye blinking, swallowing, or mouthing movements, may be present [47] and so may be slumping forward [47], staring [26], or avoidance behavior [54]. Eyes are frequently closed, sometimes forcefully. This PNES pattern, which is also sometimes referred to as "pseudosyncope," does not really resemble any seizure type with the exception of very rare conditions like absence status or focal frontal status, in which, however, atonia is rare and the eyes are usually open. As a general rule, episodes with loss of consciousness lasting more than 5 min with immediate and complete recovery are not evocative of organic disturbances [53, 55]. However, caution is needed if the event has not been observed since the beginning, as a long-lasting unresponsiveness can represent the postictal phase following a GTCS [30].

Eye closure is a very important tool for differential diagnosis [27, 43, 46]. Eyes are closed during 55–96% of PNES. Conversely, eyes are open at the beginning or throughout 92–100% of ES, including episodes arising from sleep [27]. A forceful closure with active opposition to opening is very specific to PNES [26, 57].

Although individual ictal features may suggest a specific epileptic seizure type, the temporal sequence of events in PNES is often variable or not congruent with an epileptic discharge spreading [54, 55, 58].

Self-injury is uncommon in PNES. Tongue biting is rare and, when it occurs, is typically on the tip and not in the lateral or anterolateral tongue as in GTCS [27].

Postictal stertorous breathing, agitation, confusion, headache, and fatigue are uncommon after PNES [43, 46, 51, 59]. Ability to recall the seizure, in episodes with apparent impaired awareness, is specific to PNES [46, 53, 58, 60].

Though the absence of stereotypy is specific to PNES, stereotyped events were recorded in 67–90% of video-EEG studies recording multiple PNES in the same patient [27].

A fearsome and frequent complication of PNES (occurring in up to 78% of patients) [61] is psychogenic status, a

condition in which the seizure lasts for a long time. It may rarely constitute the onset of a PNES disorder and can lead to intubation, use of anesthetics, and tracheostomy, if misdiagnosed for status epilepticus.

34.2.3 EEG Findings

34.2.3.1 Interictal EEG

Since interictal EEG alone does not allow the physician to make nor exclude the diagnosis of epilepsy, it cannot result in the diagnosis of PNES. Furthermore, some potential confounders should be kept in mind. EEG abnormalities, which can be found in up to 15% of the general population (less than 1% with an epileptiform appearance), are more common in patients with PNES independent of comorbid epilepsy [62, 63], as well as in borderline personality disorder and in relatives of patients with epilepsy, common conditions in PNES [25, 53, 54, 64]. Generalized epileptiform discharges can occur during drug withdrawal even in patients without epilepsy.

Furthermore, up to 37% of patients with PNES had a report of "epileptiform" abnormalities [65, 66] which, when re-evaluated at an epilepsy center, revealed to be normal variants [65], as in the case reported in Fig. 34.4.

Diagnostic levels of certainty of PNES were proposed by an International League Against Epilepsy (ILAE) Task Force, and an interictal EEG with no epileptiform activity is required, together with history or clinician-witnessed attack, to formulate a possible or probable diagnosis [30].

34.2.3.2 Ictal EEG

By definition, EEG has no alteration before, during, and after a psychogenic attack. However, this might be insufficient for a diagnosis because of two reasons. First, ictal scalp EEG may be normal even during seizures with sensory or very subtle behavioral symptoms and retained awareness [26, 57] and in mesial frontal lobe seizures [52], two types of seizures in which even semiology is often uninformative or challenging.

Second, motion artifacts can obscure the EEG or even be mistaken for ictal discharges. Figure 34.5 shows typical rhythmic artifacts in a PNES resembling GTCS.

However, an EEG, not obscured by artifacts, showing no ictal epileptiform activity in an attack in which it should be expectable if it were epileptic, together with a compatible history and an epileptologist-witnessed event, allows a diagnosis of clinically established PNES according to the diagnostic criteria of the ILAE Task Force [30].

A highly specific and sensitive rhythmic artifactual pattern on the EEG in PNES resembling GTCS has been described in a study. It consists of rhythmic movements with a stable frequency contrasting with the typical patterns observed during GTCS, characterized by rhythms in

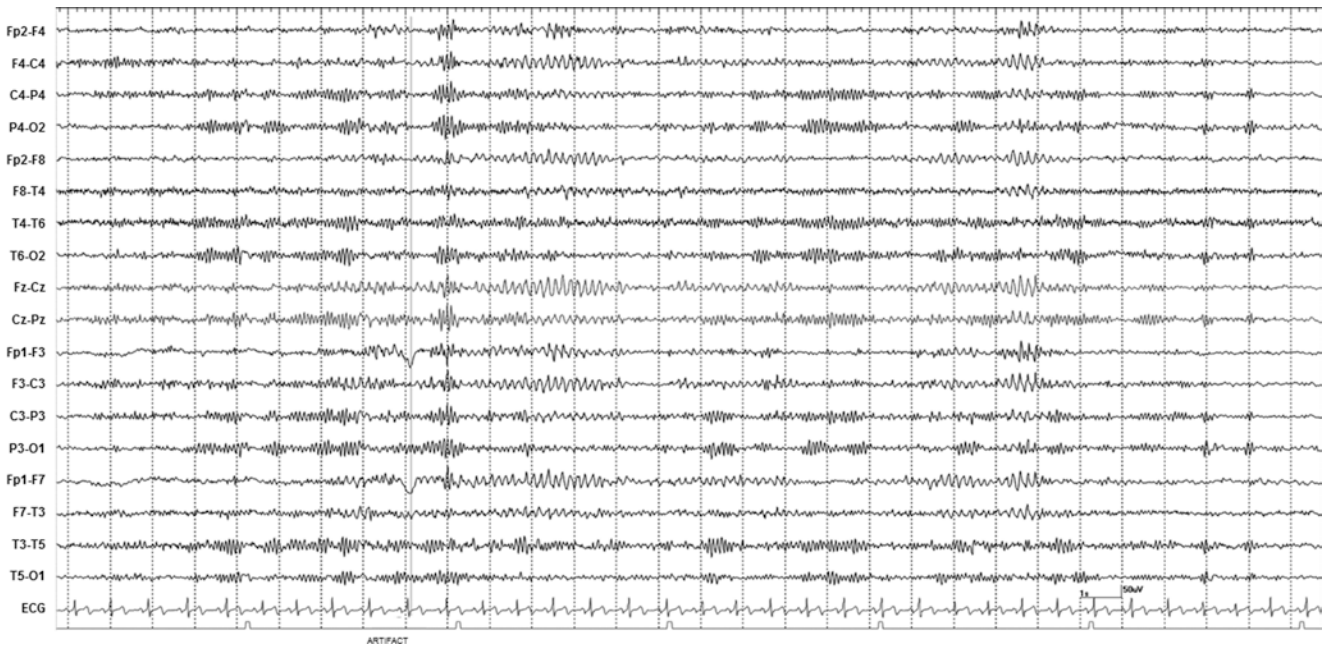

Fig. 34.4 EEG tracing of a 28-year-old woman who experienced three unclear episodes of brief "blackouts" followed by panic, breathing difficulties, and palpitations. A theta activity at 5–6 Hz, most prominent on the central vertex and diffuse over both frontal regions, may be observed, with a sinusoidal and, occasionally, spiky, appearance with a wax and wane pattern, mostly presenting on wakefulness fluctuations. This pattern was previously misinterpreted as "generalized epileptiform discharges" by neurologists who were not epileptologists, and antiepileptic treatment was started. When re-evaluated at our center, it was identified as a "midline theta rhythm," a normal variant

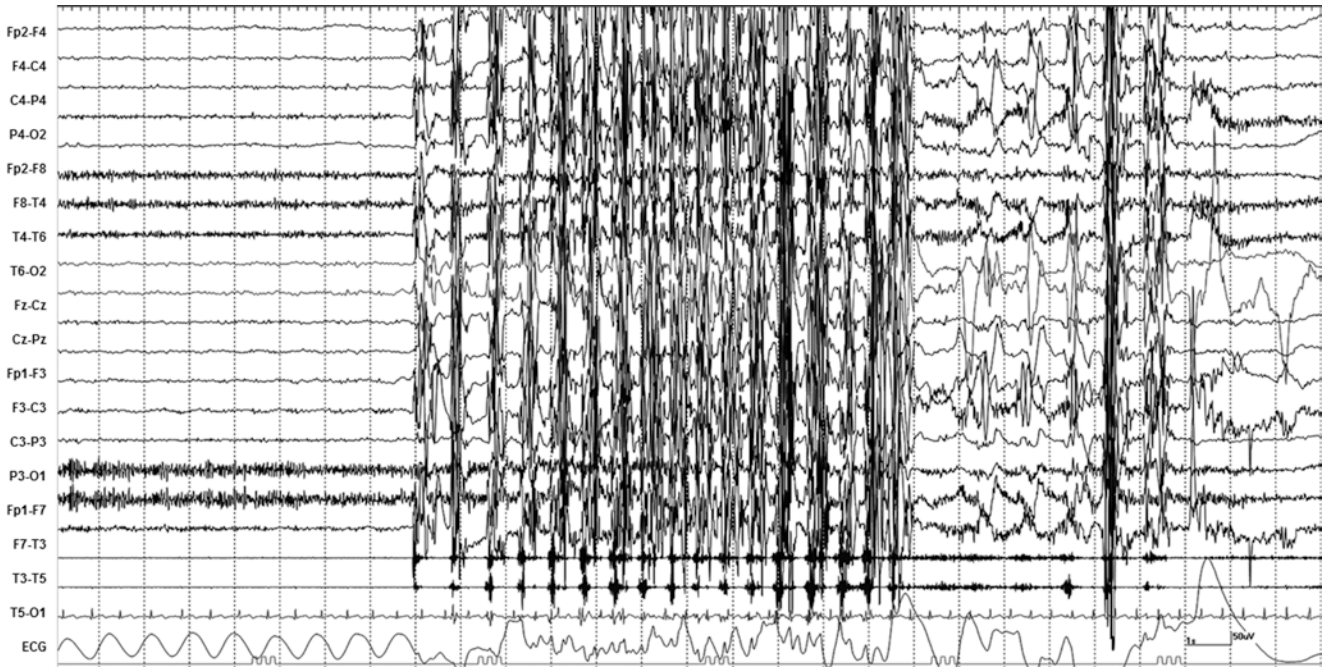

Fig. 34.5 Polygraphic tracing of a 13-year-old girl during an episode of intermittent "generalized" hypertonia with trunk and limb rhythmic jerks, induced by hyperpnea. The EEG shows rhythmic motor artifacts, corresponding to the muscle jerks on the EMG trace, with a rather constant frequency and a discontinuous pattern with a period of rest and subsequent brief resumption in the course of the event

the frequency of delta and beta range evolving from one to the other during the course of the seizure. This was documented with a time-frequency mapping of the EEG artifacts. The PNES pattern reflects a relative stability of limb movement frequency throughout the seizures, which differs from what happens in a GTCS. As a further very specific element, the authors described brief pauses in rhythmic movement, followed by resumption of movement at the same frequency ("on-off-on" pattern), which can be observed clinically and confirmed by analysis of the artifacts [56].

The concomitant recording of an ECG, which should be routinely performed, may offer additional information. Although in PNES an increase in heart rate might be observed, this is generally less significant, rapid, and sustained than the one typically observed during focal seizures with impaired awareness (temporal lobe seizures) and after a GTCS. Relative heart rate during and after a "staring seizure" proved to be a good diagnostic tool in one study: the increase by 30% of baseline rate during the spell had a 97% positive predictive value for epilepsy. Relative heart rate measurement showed a sensitivity of 83% and specificity of 96% [67]. The importance of ictal heart rate was not confirmed by another study in which, however, heart rate increases in the pre-ictal phase and decreases in the postictal phase significantly in PNES compared with ES [68].

34.2.3.3 Video-EEG Telemetry

Video-EEG monitoring is considered the "gold standard" investigation for PNES. Provided the abovementioned limitations on type of seizures and artifacts, in the presence of a suggestive history, the video recording of a typical event and the simultaneous recording of an EEG trace which does not show alteration, but instead keeps being a normal awake EEG before, during, or after the event, allow the diagnosis of documented PNES, according to the ILAE criteria [30]. It is paramount to record the habitual attack, and, when different kinds of seizures are reported, ideally all the types should be recorded, in order not to miss possible comorbid ES [30]. Figures 34.6 and 34.7 show examples of diagnostic tracings.

A typical event occurs within the first hours of video-EEG monitoring in the majority of patients according to several authors [30, 63, 66]. Indeed, according to a large study, more than 90% of the events are recorded in the first 30 min [69]. The presence of additional professional personnel in the EEG lab increases the chance to record an event [69].

Therefore, outpatient monitoring can be cost-effective, especially when provoking techniques are used [69]. Besides verbal suggestion, which should always accompany the others, they include prolonged photic stimulation and hyperventilation, compression of body parts, placing a tuning fork or moistened patches on the skin, intravenous administration of saline or other placebo, and hypnosis [29]. Induction may be used to start or stop the seizure. Although

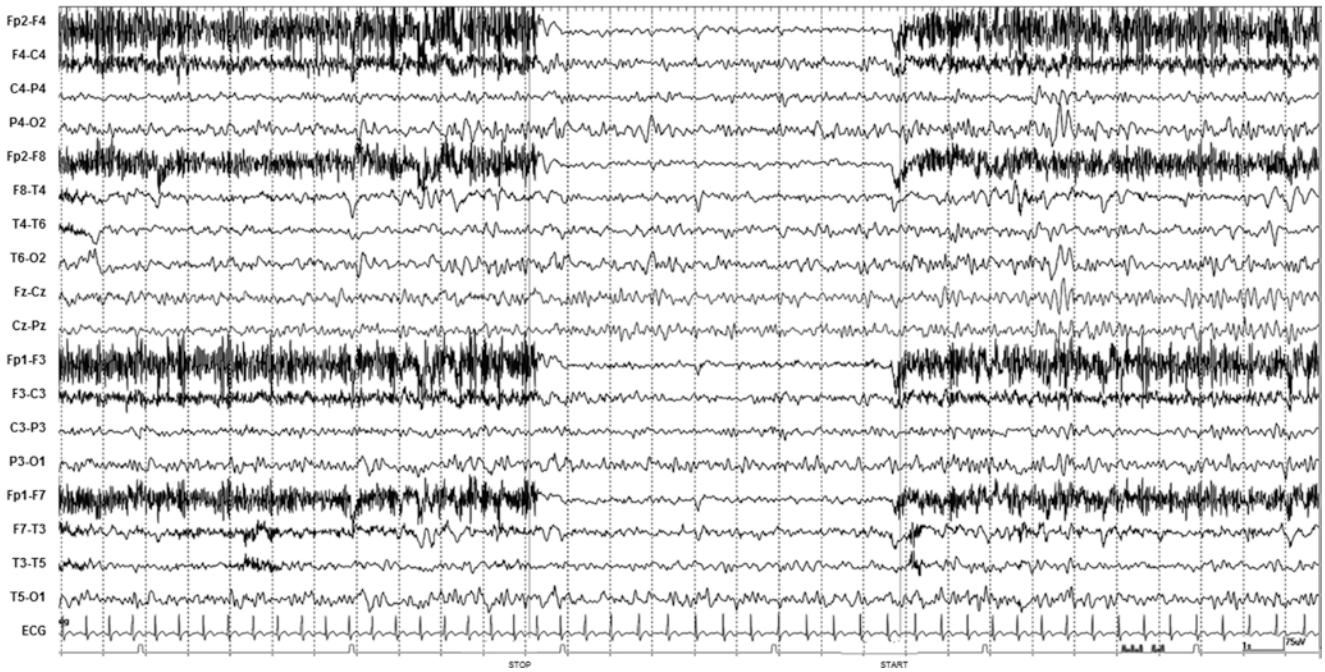

Fig. 34.6 EEG recording of a 6-year-old girl during which numerous episodes of bilateral rapid eyelid movements, with inconstant upward revulsion, were recorded. The EEG shows motor artifacts on the frontal regions during two episodes, while the background activity does not modify during and in between the events persisting as a normal wakefulness activity

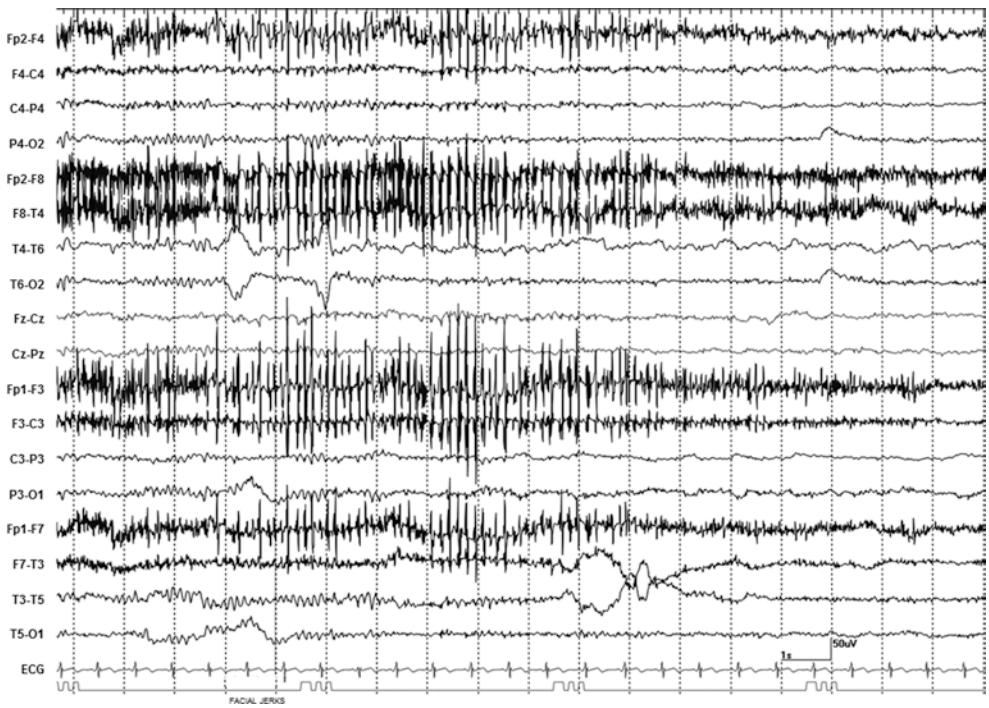

Fig. 34.7 EEG tracing of a 38-year-old woman with a history of focal temporal lobe epilepsy and PNES, recorded during a status of apparent persistent impaired consciousness, subcontinuous facial jerks, and upper arms' "tremor," increasing during the medical visit. The EEG tracing shows a normal wakefulness activity with superimposed rhythmic muscular artifacts on the anterior derivations

the use of placebo/nocebo poses ethical concerns, and some have questioned their specificity [70, 71], according to the majority of studies, induction maneuvers are highly sensitive and their specificity approaches 100% [29], significantly reducing time to diagnosis. Figure 34.8 shows the usefulness of nocebo/placebo in inducing and stopping a seizure in a challenging case.

Clinicians should review videos of the events with patients and families, in particular in cases of comorbidity with epilepsy, teaching them to differentiate PNES from ES in order

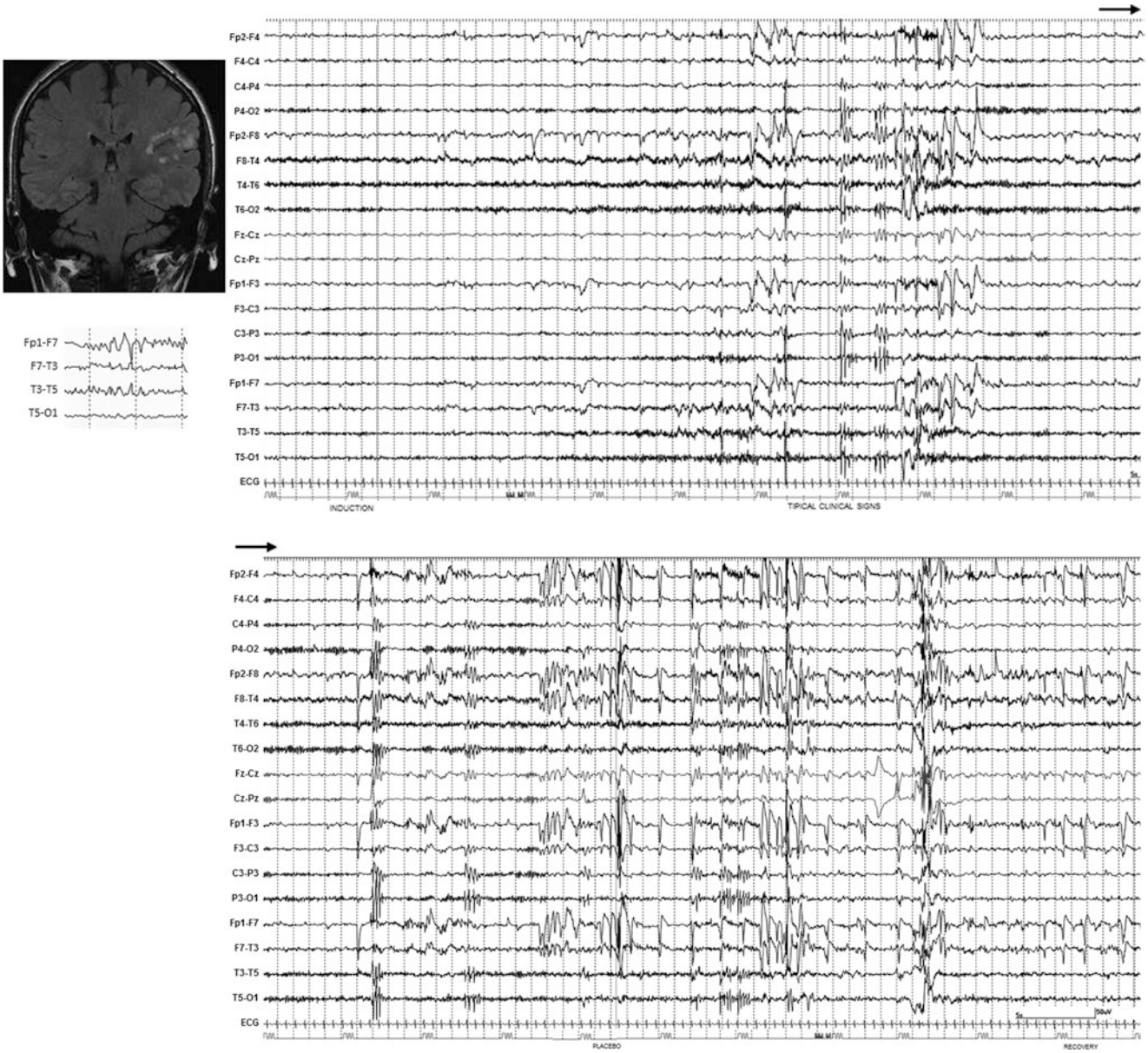

Fig. 34.8 Interictal focal spikes and MRI showing a parietal dysplasia in a 35-year-old woman who had a diagnosis of refractory epilepsy until a typical episode was recorded following intravenous saline nocebo administration. The episode consists of paroxysmal strabismus, aphasia, and lip protrusion and regresses after intravenous placebo

to correctly classify the subsequent events and guide management decisions, avoiding overtreatment [29].

34.2.4 Other Exams

Home video recording alone may have a high accuracy in selected patients when reviewed by experienced observer and in conjunction with a clear clinical history [30, 72]. An important caveat is that the onset of the seizure is frequently missed, and, therefore, the postictal phase of an ES may be mistaken for a PNES. Motor attacks seem more accurately recognized with this technique [30, 72]. In any case home video recording is a good screening tool before video-EEG recording.

Serum prolactin and CPK assays demonstrate with a good level of accuracy the absence of a postictal rise contrasting with the large majority of GTCS and most focal seizures with impaired awareness [30].

A validated linguistic approach proved useful to distinguish PNES from ES on the ground of the communication style patients used to describe their own seizures in German, English, and Italian [73–75].

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