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Review

The differential diagnosis of epilepsy: A critical review

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ABSTRACT

The wrong diagnosis of epilepsy is common. At referral epilepsy centers, psychogenic non-epileptic attacks are by far the most common condition found to have been misdiagnosed as epilepsy, with an average delay of 7–10 years. There are many "red flags" that can raise the suspicion of psychogenic non-epileptic attacks. Syncope is the second most common condition misdiagnosed as epilepsy, and it is probably more common in outpatient populations. Other conditions more rarely misdiagnosed as epilepsy include hypoglycemia, panic attacks, paroxysmal movement disorders, paroxysmal sleep disorders, TIAs, migraines, and TGA. Conditions specific to children include nonepileptic staring spells, breath-holding spells, and shudder attacks. At all ages, the over-interpretation of EEGs plays an important part in the misdiagnosis of epilepsy.

1. Introduction

The wrong diagnosis of epilepsy is unfortunately common. Of patients diagnosed with epilepsy who are seen at epilepsy centers, 20% to 30% are found to have been misdiagnosed [1–3]. This percentage is astonishingly consistent across centers, countries, and continents. Psychogenic nonepileptic attacks (PNEAs) are by far the most common condition found at referral epilepsy centers and epilepsy monitoring units, though syncope may be more common in a general neurology practice setting. Other paroxysmal conditions can also occasionally be misdiagnosed as epilepsy. As is true of other chronic conditions (e.g., multiple sclerosis), when a wrong diagnosis of epilepsy has been given, it is easily perpetuated without being questioned, which explains the usual diagnostic delay and its consequences [4–6]. Amazingly, despite the ability to make a diagnosis of epilepsy (and its main mimic PNEA) with near certainty, the delay in diagnosis remains long at about 7 to 10 years [7,8]. This suggests that neurologists may not have a high enough index of suspicion to question the diagnosis of "seizures" when drugs fail. This article reviews the main conditions that can mimic and be misdiagnosed as epilepsy.

2. Psychogenic nonepileptic attacks

PNEAs constitute by far the most common (>90%) condition misdiagnosed as epilepsy, at least at referral epilepsy centers [9,10]. They are probably common in the general population, with an estimated prevalence of 2 to 33 per 100,000 [11]. The majority of patients with PNEAs are young women, but no group is spared and PNEAs also affect men and elderly patients relatively often [12,13]. In addition to being common, PNEAs represent a challenge in both diagnosis and in management. The terminology on the topic has been variable and continues to be confusing. Various terms have been used, including pseudoseizures, nonepileptic seizures, and psychogenic seizures. Strictly speaking, terms such as *pseudoseizures* and *nonepileptic seizures* include both psychogenic and nonpsychogenic (i.e., organic) epilepsy mimics. On the other hand, a term such as *psychogenic nonepileptic seizures* should be preferred because it adds the important connotation of a psychological origin. Lastly, the word *seizures* is confusing to patients, and for those reasons, *psychogenic nonepileptic attacks* will be used here.

2.1. Suspecting the diagnosis

PNEAs are initially suspected in the clinic on the basis of the history and examination. A number of "red flags" are useful in raising a suspicion that "seizures" may be psychogenic rather than epileptic.

2.2. History

The most helpful historical features are the following:

- Resistance to AEDs: This is usually the reason for referral to the epilepsy center, and most (about 80%) patients with PNEAs have received AEDs for some time before the correct diagnosis is made [14].
- A very high frequency of seizures (multiple daily episodes) that is completely unaffected by AEDs.
- Specific triggers that are unusual for epilepsy (e.g., stress, getting upset, pain, certain movements, sounds), especially if they are alleged to *consistently* trigger a seizure.

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- The circumstances in which attacks occur: PNEAs tend to occur in the presence of an audience, and occurrence in the physician's office or waiting room is particularly suggestive of PNEAs [15]. Similarly, PNEAs tend not to occur in sleep, although they may seem to and be reported as doing so [16].

- The presence of "fashionable" diagnoses, such as fibromyalgia and chronic pain [15] (and probably others such as chronic fatigue syndrome and Lyme disease). Similarly, a florid review of systems, especially written lengthy lists of symptoms or diagnoses suggesting somatization [17], should raise the suspicion.

- The psychosocial history, including associated psychiatric diagnoses.

2.3. Detailed description of the spells

This often includes characteristics that are inconsistent with epileptic seizures. In particular, some characteristics of the motor (convulsive) phenomena are associated with PNEAs, but this is better assessed with video/EEG monitoring (see below). However, witnesses' accounts are rarely detailed enough to describe these accurately, and in fact, even seizures witnessed by physicians are often wrongly diagnosed.

2.4. Examination

The examiner should pay particular attention to mental status evaluation, general demeanor, affect, level of concern, overdramatization, and histrionic features. Specifically, the examination may uncover histrionic behaviors such as give-way weakness and tight roping. Performing the examination can in itself act as an induction in suggestible patients, making an attack more likely to occur during the history taking or examination.

In contrast to the above, certain symptoms when present argue in favor of epileptic seizures. These include significant postictal confusion, incontinence, occurrence out of sleep, and, most important, significant injury, although injuries may be *reported* by patients with PNEAs. In particular, tongue biting is highly specific to generalized tonic–clonic seizures [18] and, thus, is a very helpful sign when present.

2.5. Confirming the diagnosis

2.5.1. EEG

Because of its low sensitivity, routine EEG is not very helpful in making a diagnosis of PNEA. However, the presence of repeated normal EEGs, especially in light of frequent attacks and resistance to medications, certainly can be viewed as a "mild" red flag [19]. Ambulatory EEGs can contribute to the diagnosis by recording the habitual episode and documenting the absence of EEG changes. However, because the lack of EEG changes needs to be interpreted in light of the clinical attack and of the difficulties in conveying this diagnosis (see Section 2.6), it should always be confirmed by video/EEG monitoring. (The use of *prolactin* is often discussed in the context of suspecting PNEAs versus seizures [20], but in the age of video/EEG monitoring, it has little value unless resources are very limited.)

2.5.2. Video/EEG monitoring

The "gold standard" for diagnosis [1,9,10,21], video/EEG monitoring is indicated in all patients who continue to have frequent seizures despite medications. In the hands of experienced epileptologists, the combined electroclinical analysis of both the clinical semiology of the ictus and the ictal EEG findings allows a definitive diagnosis in nearly all cases. If an attack is recorded, the diagnosis is usually not difficult, and it is very rare that the simple question of PNEA versus epilepsy cannot be answered.

The principle of video/EEG monitoring is to record an episode and demonstrate that: (1) no change occurs in the EEG during the clinical event, and (2) the clinical attack is inconsistent with seizure types that can be unaccompanied by EEG changes. Ictal EEG has limitations because it may be negative in some partial seizures [22]. Ictal EEG may also be uninterpretable if movements generate excessive artifact [23]. Analysis of the ictal semiology (i.e., video) is as important as (and sometimes more important than) the ictal EEG, as it often shows behaviors that are obviously nonorganic and incompatible with epileptic seizures or other organic conditions, although this can be difficult to put into words.

Certain characteristics of the motor phenomena are strongly associated with PNEAs. These include very gradual onset or termination; pseudosleep; discontinuous (stop and go), irregular, or asynchronous (out of phase) activity; side-to-side head movements; pelvic thrusting; opisthotonic posturing; stuttering; weeping; preserved awareness during bilateral motor activity; and persistent eye closure [1,10,24–26]. It should be pointed out that although some of these behaviors are highly specific for PNEAs, none is in itself pathognomonic and establishes the diagnosis in isolation. False positives do exist, and for example, preserved awareness during bilateral motor activity can be seen in some frontal lobe seizures [27]. Provocative techniques, activation procedures, or inductions can be very useful for the diagnosis of PNEAs, particularly when the diagnosis remains uncertain and no spontaneous attacks occur during monitoring [21,28–31]. Further, in difficult situations where the combination of semiology and EEG does not allow the conclusion that an episode is psychogenic in origin (e.g., uninterpretable EEG due to movement-related artifacts or symptoms consistent with a simple partial seizure), the very presence of suggestibility (i.e., suggestion triggers the episode in question) is the strongest argument to support a psychogenic mechanism [32].

2.5.2.1. Short-term outpatient video/EEG monitoring with activation. An extension of the use of inductions is that, when patients are strongly suspected to have PNEAs on clinical grounds, they can undergo outpatient video/EEG monitoring with activation. This can be very cost effective while retaining the same specificity and a reasonably high sensitivity, thus obviating the need for prolonged video/EEG monitoring [33,34].

2.6. Psychopathology

PNEAs are by definition a psychiatric disorder. According to the *Diagnostic and Statistical Manual of Mental Disorders* classification, physical symptoms caused by psychological causes can fall into three categories: (1) somatoform disorders, (2) factitious disorders, and (3) malingering. Somatoform disorders involve the unconscious production of physical symptoms because of psychological factors, which means that symptoms are not under voluntary control; that is, the patient is not intentionally trying to deceive. Somatoform disorders are subdivided into several disorders depending on the characteristics of the physical symptoms and their time course, and the two most relevant to PNEAs are conversion disorder and somatization disorder. In contrast to the unconscious (unintentional) production of symptoms of somatoform disorders, factitious disorder and malingering imply that the patient is purposely deceiving the physician, that is, faking the symptoms. The difference between these two conditions is that in malingering, the reason for doing so is tangible and rationally understandable, whereas in factitious disorder, the motivation is

a pathological need for the sick role. It is generally accepted that most patients with PNEAs fall into the somatoform category (unconscious production of symptoms), rather than the intentional faking type (malingering and factitious).

The literature often implies that PNEAs represent a unique disorder, but in reality PNEAs are but one type of somatoform disorder (or malingering or factitious disorder). Only the expression of the psychopathology is different. Fundamentally, the underlying psychopathology, the prognosis, and the management are no different for PNEAs than they are for other psychogenic symptoms. Whatever the manifestations, psychogenic symptoms represent a challenge both in diagnosis and in management. Every medical specialty deals with symptoms that can be psychogenic [35]. Among such symptoms, PNEAs are unique in one principal characteristic. With video/EEG monitoring, they can be diagnosed with near certainty. This is in sharp contrast to other psychogenic symptoms, which are almost always a diagnosis of exclusion.

2.7. Management

The role of the neurologist or epileptologist is to determine whether organic disease is present. Once the attacks have been shown to be psychogenic, the exact psychiatric diagnosis and its treatment should be best handled by the mental health professionals (psychiatrist, psychologist, counselor). However, implementation of this in practice is plagued by many difficulties [35]. The role of the neurologist should not end when the diagnosis of PNEAs is made. In fact, arguably the most important step in initiating treatment is the delivery of the diagnosis to patients and families. Patients' reactions can include disbelief, denial, and anger. Another obstacle is that psychiatrists tend to be skeptical about the diagnosis of psychogenic symptoms, even for PNEAs where video/EEG monitoring allows a near-certain diagnosis [35,36].

3. Syncope

Syncope is common, and although it is a distant second to PNEAs in terms of conditions misdiagnosed as epilepsy at referral epilepsy centers, it may be more common in general neurology practices. The first reason that syncope is misdiagnosed as seizures is the erroneous belief that seizures can cause a flaccid motionless episode of loss of consciousness (LOC) for seconds to minutes. In reality, no seizure type does this. Generalized tonic–clonic seizures have obvious motor manifestations, myoclonic seizures are very short jerks with no detectable LOC, atonic seizures may cause abrupt falls but no prolonged LOC, and complex partial seizures or absence seizures cause alteration of awareness but not limp motionless LOC. In general, episodes of LOC with eyes closed for several seconds to minutes are either psychogenic or syncopal, but not epileptic (nor transient ischemic attacks!). The second reason for the misdiagnosis is the frequency with which syncopal events are "convulsive." Although the conventional teaching is that syncopal episodes are limp motionless events, they in fact frequently involve brief body jerks. In a study of patients with an implantable defibrillator in whom syncope was deliberately induced, 45% of episodes included tonic or clonic motor activity [37]. In another study of patients diagnosed with epilepsy who underwent tilt-table testing, 63% of induced episodes of syncope were convulsive [38]. Lastly and most impressively, when syncope was induced in healthy volunteers (using hyperventilation, squatting, and Valsalva maneuver), 38 [90%] of 42 episodes had some clonic-like jerking activity [39]. Motor symptoms associated with syncope are clonic- or myoclonic-like, tend to last only a few seconds, and terminate once the patient is horizontal, in sharp contrast to the typical generalized tonic–clonic seizure duration of 30 to 90 seconds. EEGs are very sensitive to decreased cerebral flow, and by the time LOC occurs, EEG changes are present. When syncope (convulsive or not) is recorded on video/EEG monitoring, the EEG proceeds through a very stereotyped pattern of changes (delta slowing and suppression) [40,41]. When an accurate description is missing (e.g., unwitnessed event), the distinction between syncope and seizures can at times be difficult, as it is based on history alone. However, several symptoms are helpful in pointing one way or the other [42,43]. Among these are the circumstances of the attacks, as the most common mechanism for syncope (vasovagal response) is typically triggered by clear precipitants (e.g., pain such as inflicted by medical procedures, emotions, cough, micturition, hot environment, prolonged standing, exercise). Other historical features that favor syncope include presyncopal prodromes (malaise, sweating, dizziness, lightheadedness, nausea, chest pain, palpitations), as well as age and a history of cardiovascular disease. Historical features that favor seizures include tongue biting, head turning, posturing, urinary incontinence, cyanosis, prodromal déjà-vu, and postictal confusion [42,43]. A point system using most of these features has been designed and reportedly has 94% sensitivity and specificity for the diagnosis of seizures [43]. Management of syncope depends entirely on its cause. The majority of syncopal episodes are benign vasovagal episodes, but the concerning etiologies are cardiac related. Even with extensive evaluations, a large proportion of syncopal episodes remain unexplained. Many patients with "unexplained syncope" (or presyncope) probably have psychogenic pseudo-syncope, and when red flags are present (identical to those for PNEAs), video/EEG monitoring should be performed as it can easily be used to make the diagnosis [40].

4. Other organic conditions

4.1. Hypoglycemia

Hypoglycemia rarely causes complete LOC. When it does, it resembles syncope and is also preceded by florid prodromes of hunger, weakness, tremulousness, malaise, and abnormal behaviors. Hypoglycemia typically occurs in reasonably obvious settings (e.g., diabetic patients, insulin or oral antihyperglycemics, fasting).

4.2. Panic attacks

Panic attacks are paroxysmal manifestations of anxiety or panic disorder and may be mistaken for seizures [44]. The border between panic attacks and PNEAs may be imprecise and some overlap may exist [45]. Conversely, fear is a relatively common (psychic) aura in patients with mesiotemporal epilepsy. The identification of fear as an epileptic aura is easy when it evolves into a clear seizure, but can be difficult in the absence of other seizure types [46]. Panic attacks include intense autonomic, especially cardiovascular and respiratory, symptoms. Abrupt and intense fear is accompanied by at least four of the following symptoms: palpitations, diaphoresis, tremulousness or shaking, shortness of breath or sensation of choking, chest discomfort, nausea or abdominal discomfort, dizziness or lightheadedness, derealization or depersonalization, fear of losing control, fear of dying, paresthesias, and chills or hot flashes. The symptoms typically peak within 10 minutes. Panic disorder often coexists with other manifestations of anxiety such as agoraphobia and social phobia and also with depressive disorders. Thus, the diagnosis is not usually difficult and rarely requires video/EEG recordings.

4.3. Paroxysmal movement disorders

4.3.1. Acute dystonic reactions

Acute dystonic reactions are caused by dopamine receptor blockers such as antipsychotics (including atypical ones) and antiemetics, although other dugs can be involved (e.g., carbamazepine,

lithium, trazodone, illicit drugs). They typically occur within 1 to 4 days of beginning the medication and are characterized by twisting movements affecting the cranial, pharyngeal, and cervical muscles. The oculogyric crisis is a dramatic subtype characterized by acute conjugate eye deviation, usually in an upward direction. The typical attack lasts 1 to 2 hours, during which the abnormal movement occurs repetitively for seconds to minutes. These dystonic reactions respond very well and rapidly to anticholinergics (trihexyphenidyl, benztropine, diphenhydramine) and levodopa [47].

4.3.2. Hemifacial spasm

Hemifacial spasm (HFS) may superficially resemble a simple partial seizure with motor symptoms of the face, or more simply a facial clonic seizure, but clear differences make the differentiation easy. HFS is a chronic progressive (rather than paroxysmal) disorder. While facial motor seizures typically involve the perioral area (because of a large representation on the motor homunculus), the unilateral facial twitching of HFS typically affects the periorbital muscles first and then spreads to other (ipsilateral) facial muscles over a period of months to years. Over time or with exacerbations, the clonic movements can result in a sustained tonic contraction causing forceful (unilateral) eyelid closure (blepharospasm) [48].

4.3.3. Nonepileptic myoclonus

Nonepileptic myoclonus is defined as myoclonus that is not of cortical origin, that is, not visible on EEG. Hiccups and hypnic jerks are examples of normal nonepileptic myoclonus, but abnormal nonepileptic myoclonus can be seen in metabolic or toxic encephalopathies and neurodegenerative diseases.

4.4. Sleep disorders

4.4.1. Parasomnias

Parasomnias are the most likely sleep disorders to present a diagnostic challenge because they are, by definition, short-lived paroxysmal behaviors that occur out of sleep. In particular, the non-REM parasomnias (night terrors, sleepwalking, and confusional arousals) can superficially resemble seizures, as they include complex behaviors and some degree of unresponsiveness and amnesia for the event. The non-REM parasomnias are most common between ages 4 and 12, and night terrors are particularly common. They are often familial and may be worsened by stress, sleep deprivation, and intercurrent illnesses. Similarly, rhythmic movement disorder is a parasomnia typically seen at transition or stage 1 sleep, and can also resemble partial seizures. One common example is head banging (*jactatio capitis*). Among REM sleep parasomnias, nightmares rarely present a diagnostic challenge, but REM behavior disorder may with violent and injurious behaviors during REM sleep. The diagnosis of REM behavior disorder is usually easy as it affects older men and the description of acting out a dream is quite typical.

Several historical features can help in differentiating parasomnias from seizures [49], but occasionally video/EEG monitoring may be necessary, provided that the episodes are frequent enough. Video/EEG monitoring will usually confirm the absence of an EEG seizure and usually shows that the behavior arises from a specific stage of sleep [50]. Occasionally, in the absence of ictal EEG changes, the differentiation between seizure and parasomnia can be difficult.

4.4.2. Cataplexy

Cataplexy is part of the narcolepsy tetrad and consists of an abrupt loss of tone. As such, it could theoretically be mistaken for atonic seizures or "drop attacks," but there are several distinguishing features: The most characteristic feature of cataplexy is that it is typically triggered by emotions, most commonly laughter [51,52]. Severe daytime sleepiness characteristic of narcolepsy is almost always present. Lastly, atonic seizures usually occur in a completely different context of symptomatic generalized epilepsies of the Lennox–Gastaut type.

4.4.3. Hypnic jerks

Hypnic jerks or sleep starts are benign myoclonic jerks that everyone experiences on occasion. Although they resemble the jerks of myoclonic seizures, their occurrence only on falling asleep stamps them as benign nonepileptic phenomena. They occur at all ages and can lead to evaluations for seizures, especially when the jerks are unusually violent. They are easily identified on video/EEG monitoring by the fact that they occur in wake to stage 1 transition and have no EEG correlate associated with the jerks [53].

4.5. Transient ischemic attacks

Transient ischemic attacks (TIAs) rarely present a diagnostic challenge, because symptoms of TIAs are typically negative, and symptoms of seizures are typically positive. In addition, focal symptoms in TIA are strokelike, that is, maximal at onset, whereas focal seizure symptoms tend to "march" or evolve over seconds. Although rarely seizures can manifest with ictal negative symptoms (e.g., aphasia, negative myoclonus), they usually also include positive symptoms, which make the diagnosis easier. Similarly, limb-shaking TIAs occur but are very rare [54]. The confusion between TIAs and seizures may be more likely when the seizure is unwitnessed and the patient appears with a focal deficit (e.g., Todd paralysis or aphasia), especially because both will improve over time (minutes). Usually, age and associated symptoms help differentiate the two. Contrary to a common misconception, TIAs exceptionally (if ever) cause a LOC.

4.6. Migraines

Complicated migraines and migraine auras can cause positive focal symptoms in all five senses and, as such, may mimic focal (simple partial) seizures or epileptic auras. In addition, both migraine and seizure focal symptoms "march." The key differentiating factor, as usual, is the time course: migraine symptoms tend to evolve in minutes, whereas seizure symptoms evolve in seconds. Usually, associated symptoms (migrainous headache or more obvious seizure symptoms) make the diagnosis easy. Basilar migraine can also cause LOC.

4.7. Transient global amnesia

Transient global amnesia consists of dramatic episodes of anterograde amnesia. Patients are alert and otherwise cognitively intact but cannot form new memories, and they ask repetitive questions about their environment. This lasts several hours and then resolves. The cause is not known, but transient global amnesia is not thought to represent a TIA or a seizure and usually does not recur [55].

5. Conditions and issues specific to young children: Misdiagnosis of epilepsy in children

The differential diagnosis of seizures is broader in children than in adults [56,57], with many nonepileptic but nonpsychogenic conditions to be considered. Physiological and organic events predominate in infants and young children, and psychiatric disorders become more common in later childhood and adolescence. About

50% have psychological disorders (90% being PNEAs), but other psychiatric diagnoses are also found (e.g., episodic dyscontrol with rage attacks, behavioral outbursts, panic/anxiety disorder, and factitious disorder by proxy). PNEAs occur in older children and adolescents and have the same characteristics as in adults. However, the gender difference of female predominance is not seen until adolescence [52], so that PNEAs are as common in preadolescent boys as in girls. The other 50% have nonpsychogenic conditions, the most common of which is nonepileptic inattention with staring spells [58]. Other diagnoses include stereotyped mannerisms, hypnic jerks, parasomnias, tics, gastroesophageal reflux with posturing or laryngospasm, arousals, shuddering attacks, and apneas [56,57,59,60].

Nonepileptic staring spells represent a common challenge. Children are occasionally inattentive, and the families report brief episodes of staring and unresponsiveness with no motor manifestations. Several features can help distinguish absence seizures from benign nonepileptic staring spells in otherwise normal children [58]. Three features suggest nonepileptic events: (1) the events do not interrupt play; (2) the events were first noticed by a professional such as a schoolteacher, speech therapist, occupational therapist, or physician (rather than by a parent); and (3) the child, while staring, is responsive to touch or interruptible by other external stimuli. Other features that have been found to suggest nonepileptic or behavioral rather than epileptic staring include lower age and lower frequency [61]. By contrast, factors that suggest an epileptic etiology include twitches of the extremities, urinary incontinence, and upward eye movement. Such benign nonepileptic staring spells are particularly likely to be noticed and reported by overvigilant parents in a child who has or has had clear seizures.

Tics can superficially resemble simple partial seizures with motor symptoms, but several features distinguish them [62]. Tics are not episodic and tend to occur throughout the day, although they can fluctuate. They are sporadic rather than repetitive, stereotyped (the same movement repeats itself without evolving, and the same muscle group is involved), and disappear in sleep. They are preceded by an urge to move that is temporarily suppressible and followed by a sense of relief. Tics are particularly common between ages 5 and 10.

Shuddering attacks are benign paroxysmal spells of older infants and young children, and can mimic several seizure types, including tonic, absence, and myoclonic. These episodes are usually benign, have no association with increased morbidity or mortality, and tend to remit spontaneously. The condition is seen in older infants and young children. Parents describe the paroxysmal episodes as a sudden flexion of the neck and trunk and adduction of the arms. A shiverlike movement of the trunk ("like a chill") occurs, and the body may stiffen. Consciousness does not seem to be altered. The episode usually lasts 5 to 15 seconds. Unlike epileptic seizures, shuddering attacks do not occur during sleep [63,64].

Breath-holding spells (cyanotic infantile syncope) affect children aged 6 months to 5 years. Typically, a clear trigger is present, with the child being upset and crying. At the end of expiration, the child is unable to relax and inhale and becomes apneic and cyanotic. The child may appear angry and upset about this uncomfortable feeling, loses consciousness, may have urinary incontinence, and becomes stiff or even opisthotonic. The EEG during the event typically shows high-amplitude slowing followed by suppression, as is seen in syncope of any cause. When the child relaxes and breathes again, consciousness is gradually regained. These cyanotic breath-holding spells could be easily confused with epileptic events, but they are not primarily epileptic phenomena. They can evolve into epileptic seizures and even status epilepticus, but the initial event is not epileptic. Anemia should be ruled out and may require treatment. Cyanotic breath-holding spells are to be distinguished from pallid infantile syncope, which is associated with brief cardiac asystole and overlap with seizures [65].

Gastroesophageal reflux associated with laryngospasm in infants may cause events that look like seizures, with limb posturing, eye deviation, and even opisthotonus (Sandifer syndrome). These events tend to occur in sleep and in the postprandial period, and recurrent vomiting is typically associated with them [66]. The diagnosis can be made with an esophageal pH probe, and treating the reflux usually resolves the problem.

Benign myoclonus of infancy [67] must be differentiated from infantile spasms. This requires video/EEG monitoring as the events appear clinically similar. This benign phenomenon resolves spontaneously within a year without neurologic sequelae.

Mannerisms are common in young children, in particular those with a mental handicap. Mannerisms can look odd and unnatural and occasionally mimic motor seizures. Similarly, self-stimulating behaviors, including masturbation, can be erroneously interpreted as seizures.

Spasmus nutans is a benign triad of head nodding, head tilt, and pendular nystagmus, which typically occurs between 4 and 12 months of age [68].

6. Benign nonspecific symptoms misinterpreted as seizures

This phenomenon has no name and is not written about because it does not fit under psychogenic seizures or other organic conditions described above. It is best described as "overvigilance" and is commonly seen at epilepsy centers. It basically consists of the overinterpretation of benign or nonspecific symptoms as seizures. Unexplained symptoms are common in everyday life and include transient dizziness, limb numbness, head sensations, and various mild and brief involuntary movements. The misinterpretation of these symptoms as seizures is more likely to occur in anxious patients (or caregivers) with hypochondriacal tendencies. It is also more common in patients who also have or have had seizures or who have other organic conditions. Another setting is the intensive care unit, where many patients who are very ill can have nonspecific abnormal movements such as shivers, twitches, and tremors that are of concern to caregivers or intensive care unit personnel, but are neither epileptic nor psychogenic. Video/EEG recordings will usually clarify the situation, but because many of the mild nonspecific symptoms mimic simple partial seizures or auras rather than more severe seizures, the mere presence of a normal ictal EEG does not *in itself* exclude seizures. However, the video, that is, the characteristics of the movements, usually does, as they are nonclonic, nontonic, and not myoclonic [69]. At times the distinction can be difficult, and when in doubt it is preferable to be conservative rather than label the episodes as seizures.

6.1. The role of misread (overread) EEGs in the misdiagnosis of epilepsy

Many patients (about a third) who have been misdiagnosed as having epilepsy have had previous EEGs interpreted as epileptiform that contributed to the misdiagnosis of epilepsy [3,70,71]. In fact, sometimes patients are diagnosed with epilepsy and treated based solely on an EEG, despite that fact that they have no symptoms or that their symptoms are not at all suggestive of seizures. This has led many experts to point out that EEG can in fact be bad for you. There are many well-described normal variants that can be misread as epileptiform, but in reality the vast majority of overread patterns are simple fluctuations of sharply contoured background rhythms or fragmented alpha activity [70–73]. The reasons for the overinterpretation of EEGs are complex, and have been discussed elsewhere [71,72], but the fact that the diagnosis of seizures should be clinical cannot be overemphasized. In chil-

dren, benign centrotemporal (rolandic) spikes on EEG are a common "red herring'" because they occur frequently in asymptomatic children. Most errors in diagnosis are made because the EEG is overread as abnormal and is interpreted outside of the clinical context [71,72].

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