ogy books, visit our website www.jaypeebrothers.com, for detailed information on neurology books, visit our website www.jaypeebrothers.com, for detailed information on neurology books

Essentials of CLINICAL NEUROLOGY *for* The Psychiatrist



Editor
Ambar Chakravarty

JAYPEE

Contents

CHAPTER 1: **The Evolution of Neuropsychiatry: From Antiquity to Current Time** **1**
Sayan Malakar, Ambar Chakravarty

CHAPTER 2: **Hysteria: Through the Ages** **9**
Ambar Chakravarty

Commentary 1: Somatic Symptom Disorder: A Brief Note ***17***
Ambar Chakravarty

CHAPTER 3: **Functional Neurological Disorders: An Overview—Pathophysiology, Clinical Recognition, and Outlines of Management** **19**
Amit Halder, Ambar Chakravarty

CHAPTER 4: **Systemic and Neurological Red Flags in the Diagnosis of Psychiatric Diseases** **26**
Sarosh M Katrak

Commentary 2: A Truly Neuropsychiatric Problem! ***38***
Ambar Chakravarty

CHAPTER 5: **Cognition Testing at the Bedside** **42**
Amit Halder, Ambar Chakravarty

Commentary 3: A Relook at Acute Onset Amnesia Syndromes ***58***
Ambar Chakravarty

CHAPTER 6: **Pitfalls in Neuropsychological Testing in Adults with Suspected Dementias** **64**
Atanu Biswas

CHAPTER 7: **Cornerstones in the Diagnosis of Epileptic Seizures in Children and Adolescents: Their Pitfalls and Mimics** **70**
Ambar Chakravarty

Appendix 1: Epilepsy Imitators ***79***

Commentary 4: Utility of Sleep in Seizure Diagnosis ***80***
Ambar Chakravarty

Commentary 5: Geriatric Seizure Mimics: A Problem Oriented Approach ***84***
Ambar Chakravarty

CHAPTER 8: **Seizures and Epilepsy Syndromes: What a Psychiatrist should Know?** **87**
Neenu Alexander, Chaturbhuj Rathore

CHAPTER 9: **Neuropsychiatric Aspects of Epilepsy** **109**
Ambar Chakravarty

CHAPTER 10: **Psychogenic Nonepileptic Seizures: Psychobiology, Clinical Aspects, and Management** **116**
LG Viswanathan, Sanjib Sinha

Commentary 6: Psychogenic Nonepileptic Seizure and Psychogenic Movement Disorder: Are they Really Different? *123*
Ambar Chakravarty

Commentary 7: Psychogenic Nonepileptic Seizures: Is it All in the Mind? *125*
Ambar Chakravarty

CHAPTER 11: **Psychiatric Mimics of Epileptic Seizures: Part 1—Episodic Dyscontrol Syndrome: The Concept** **127**
Ambar Chakravarty

CHAPTER 12: **Psychiatric Mimics of Epileptic Seizures: Part 2—Epilepsy, Rage, Tantrums, and Violence** **131**
Ambar Chakravarty

CHAPTER 13: **An Overview of Stroke Medicine for the Psychiatrist** **135**
Neetu Ramrakhiani

Commentary 8: Stroke Mimics and Chameleons *143*
Ambar Chakravarty

CHAPTER 14: **Neuropsychiatric Aspects of Cerebrovascular Disorders** **147**
Shashank Jaiswal, Subhash Kaul

CHAPTER 15: **An Overview of Movement Disorders for the Psychiatrist** **156**
Adreesh Mukherjee

Commentary 9: Psychogenic Gait Disorders *167*
Ambar Chakravarty

Commentary 10: Astasia–Abasia: Neurologic or Functional? *170*
Ambar Chakravarty

Commentary 11: Diagnosis of Vascular Parkinsonism *172*
Ambar Chakravarty

Commentary 12: Alcohol and Movement Disorders *176*
Ambar Chakravarty

CHAPTER 16: **Neuropsychiatric Manifestations of Parkinson's Disease** **178**
Ambar Chakravarty

Commentary 13: Neural Basis of Impulsive Control Disorders *188*
Ambar Chakravarty

Commentary 14: A Note on Wilson's Disease and its Psychiatric Aspects *192*
Ambar Chakravarty

CHAPTER 17: **An Overview of Headache Disorders and their Neuropsychiatric Aspects** **195**
Sanjay Prakash, Varoon Vadodaria

Commentary 15: A Note on Medication Overuse Headache *206*
Ambar Chakravarty

CHAPTER 18: **An Overview of Neuromuscular Diseases for the Psychiatrists** **210**
Satish V Khadilkar, Varsha A Patil

CHAPTER 19: **Chronic Pain and Chronic Fatigue: A Diagnostic and Therapeutic Challenge** **222**
Satish V Khadilkar, Hiral Halani

CHAPTER 20: **Neuropsychiatric Manifestations of Demyelinating Disorders** **234**
Parthvi Ravat, Santosh Sriram Andugulapati, Sangeeta Ravat, Mayur Thakkar

CHAPTER 21: **Neuropsychiatric Aspects of Autoimmune Encephalitis** **243**
Rahul Kulkarni, Rishikesh Deshpande, Shripad Pujari

Commentary 16: Autoimmune Encephalitis: Some Current Thoughts ***253***
Ambar Chakravarty

CHAPTER 22: **Neuropsychiatric Aspects of Central Nervous System Infections** **255**
Madhu Nagappa, Sanjib Sinha

CHAPTER 23: **Essentials of Sleep Medicine for the Psychiatrist** **267**
Joy D Desai

CHAPTER 24: **Clinical and Radiological Pitfalls in the Diagnosis of Dementias** **275**
Gautam Das, Atanu Biswas

Commentary 17: Pseudodementia and Pseudodepression: Two Sides of the Coin ***283***
Ambar Chakravarty

CHAPTER 25: **Young-onset Rapidly Progressive Dementias: Recognition and Red Flags** **287**
Ambar Chakravarty

CHAPTER 26: **Neuroimaging in the Diagnosis of Dementias** **297**
Sukalyan Purakayastha, Soumik Das

Commentary 18: Utility and Limitations of Neuroimaging and Cerebrospinal Fluid Biomarkers in Diagnosis of Dementia Subtypes ***312***
Ambar Chakravarty

CHAPTER 27: **Neurological Aspects of Autism Spectrum Disorder** **314**
Anaita Udwadia Hegde, Omkar P Hajirnis

CHAPTER 28: **Hyperventilation Syndrome, Panic Disorders, and Related Conditions** **323**
Debasish Roy, Ambar Chakravarty

CHAPTER 29: **Psychiatric Emergencies in Critical Care Units: Recognition and Management** **336**
Joshua Battley, Bappaditya Ray, Venkatesh Aiyagari

CHAPTER 30: **Exploring the Neurobiology of Human Sexuality** **348**
Joy D Desai

CHAPTER 31: **Neuropsychological Aspects of Traumatic Brain Injury** **355**
Sandip Chatterjee, Niloy Biswas

Commentary 19: Chronic Traumatic Encephalopathy ***359***
Ambar Chakravarty

CHAPTER 32: **Neurological Side Effects of Psychiatric Medications** **362**
Shripad Pujari, Amitkumar V Pande, Rahul Kulkarni

CHAPTER 33: **Central Neurological Manifestations of Alcoholism and Substance Abuse** **371**
Sweety Tribedi, Ambar Chakravarty

CHAPTER 34: **Visual Illusions and Hallucinations of Central Origin and their Differentials** **386**
Ambar Chakravarty

Commentary 20: A Note on Auditory Hallucinations ***395***
Ambar Chakravarty

Index **399**

CHAPTER 7

Cornerstones in the Diagnosis of Epileptic Seizures in Children and Adolescents: Their Pitfalls and Mimics

Ambar Chakravarty

JAYPEE BROTHERS

INTRODUCTION

The most important factor contributing to making a wrong diagnosis of epilepsy is obtaining an inadequate and inaccurate history. Observers of spells should be queried directly. All too often, a story within the confines of the clinic proves to be very inaccurate. "Dizzy spells" without loss of consciousness (LOC) may be revealed by witnesses as full tonic-clonic seizures. A careful history is an unbiased history, told in the patient's and observer's own words. Trainees, relatives, and patients must learn to avoid use of too technical terms while presenting the history to the treating physician. Young children are often not expected to mention every details of a spell experienced by them and hence parental account of the incidents observed, assumes paramount importance. By the time multiple physicians have asked a patient the very same question, especially a child (e.g., getting a bad odor before an attack), they often get confused and tend to give a positive answer, even if that was not the case. Witness observations of the sequence of events preceding during and after the event/events contribute a lot in making a proper diagnosis.

This has been assessed very scientifically recently in UK by Reuber and his colleagues[1] using the paroxysmal event observer (PEO) questionnaire. The study concluded that witness reported factors alone differentiated better between syncope and epilepsy than patient-reported factors (accuracy: 96% vs. 85%, $p = 0.0004$). This data improved accuracy of differentiation very significantly not only between epilepsy and syncope but also between syncope and psychogenic nonepileptic seizures (PNES).

The combination of a shaky history and an over interpreted electroencephalogram (EEG) is especially pernicious. As a consequence of this combination of errors, patients may be treated inappropriately for years with anticonvulsants and subjected to a variety of unnecessary social restrictions.

Box 1 lists some very useful queries which may be asked to older children or parents of younger children for making a clinical diagnosis of the event experienced by the child. **Box 2** lists some hallmark clinical character of the events experienced and **Box 3** lays out a road map to arrive at the most likely clinical diagnosis of the event in a logical manner (called a deductive approach by DiMario[2]).

Confusion may arise as to the cause of the seizure. There may be instances where whether a neurological deficit had resulted from a seizure or had been the cause of the seizure, become difficult to decide.

BOX 1: Basic queries for elucidation of clinical history.

- Age at onset?
- Is there more than one type of event?
- What happens just prior to the event?
- Does the child get a warning or any change in behavior noted?
- What is the exact sequence of the event?
- Is the pattern stereotyped or the pattern variable?
- What are the child's posture, tone, respiratory pattern, color, and behavior before, during and after the event?
- Is there any relationship of the event to activity, attention, play, temperament, sleep, and diet?
- Any precipitant of event?
- Any relieving factor?
- Can the events be suppressed by the child?
- Can the child be distracted during the event?
- Any concurrent motor activity, sensory phenomenon, or autonomic feature evident during the event?
- What does the child do immediately afterwards?
- Can the child recall the event in detail?
- The duration, frequency, and periodicity of the event?
- Any family history of similar illness?
- Can the child or observer mimic the event?
- Any video recording done?

BOX 2: Hallmark clinical character of event.

- Paralysis
- Collapse/drop attack
- Generalized posturing/jerking/twitching
- Focal posturing/jerking/twitching
- Ocular movement/deviation
- Autonomic phenomenon
- Sensory phenomenon

BOX 3: Road map to logical thinking.

- Accurate statement of the problem
- Aggregation of facts (based on **Boxes 1 and 2**)
- Identification of the nodal point (clinically the most important issue determining the possible causes of the event, e.g., generalized/focal jerking)
- Generate list of possibilities
- Trim list to most probable ones
- Identify the most probable one
- Validate the diagnosis by appropriate investigations

Focal seizures can cause a postictal transient hemiparesis (Todd's paresis), but cerebrovascular disease can also directly cause hemiparesis and may present as a seizure in about 15% of cases. Bilateral carotid occlusive disease can also cause brief LOC. Differentiating idiopathic epilepsy from epilepsy secondary to cerebrovascular disease can be difficult. The latter diagnosis as the primary diagnosis as the primary causative one may be helped by such features like presence of stroke risk factors while the diagnosis of a primary epileptic condition may be suggested by finding a past history of seizure or a positive family history of epilepsy. Similarly, seizures can induce cardiac arrhythmias, as well as result from them.

The inexperienced clinician tends to overemphasize the rare and obscure. This is particularly easy to do in the differential diagnosis of epilepsy. Most staring spells are simple daydreaming. Most explosive outbursts in children are temper tantrums. Most episodes of abrupt falls or drop attacks in the elderly are either tripping or syncopal—vasovagal or cardiogenic. The paradigm of approach gets altered when it is known that the individual concerned suffers from epilepsy. The first condition to be considered if such a person presents with only a brief LOC would always be epilepsy; however, if he had not had prior history of epilepsy, such conditions like vasovagal or cardiac syncopes should be considered and excluded much before a diagnosis of epileptic seizure be considered.

Great difficulties are often encountered in patients who exhibit features suggestive of mixed seizures. This is not very uncommon. A certain percentage of individuals with documented psychogenic seizures, varying from 10 to 37% or more,[1,3,4] may at other times exhibit epileptic seizures and vice versa. Presumably, the epileptic seizures and their aftermath somehow became a "template" for subsequent nonepileptic spells. By absence of EEG changes during a generalized seizure-like episode, video EEG monitoring can show that the episode under observation is nonepileptic in origin, but it can never show that prior episodes were not epileptic seizures. Inference by analogy is imprecise. Even after establishing a diagnosis of nonepileptic attacks, the experienced clinician should continue to look for the possibility of a mixed disorder. He may, if circumstances so demand, withdraw anticonvulsants (only in hospitalized patients) with the understanding that epileptic seizures may emerge and properly characterized.

Improvement of spells with anticonvulsants need not necessarily mean that the patient has epilepsy and nothing other than that. Placebo response may occur, especially in those with psychogenic components. The efficacy of antiepileptic drugs is not limited to seizures alone. Carbamazepine and sodium valproate are useful mood stabilizers. Phenobarbitone and benzodiazepines are effective both as antiepileptics and as well as anxiolytics. Phenytoin can suppress ventricular arrhythmias. When a positive response to an antiepileptic agent is encountered, the clinician should consider what else besides epilepsy might have been treated. Conversely, some patients with presumed epilepsy may worsen with increasing doses of antiepileptic drugs. This generally should call for looking for presence of associated psychogenic seizures in some cases but not in all. It is well known that absence seizures and juvenile myoclonic epilepsy may worsen following treatment with carbamazepine or phenytoin.

Perhaps the greatest source of mistakes in epilepsy diagnosis is the performance of EEGs.[5,6] Very often these are ordered by general practitioners, who for no uncertain reason lack adequate knowledge what they would really be looking for in the report and performed by technicians not really adequately trained (common problems include for the technician to understand the difference between a sleep EEG and a sleep deprived EEG; problems of interpretation relating to routinely using monopolar montages with reference electrode on ear lobules, inability to recognize common artifacts like electrode pops, pulse artefacts, 60 Hz AC artefacts, etc., and how to minimize them during recording) and lastly and most importantly, the final interpretation by a trained (at least by qualification) neurologist, who often had no clue as to the clinical condition for which EEG had been ordered and who, perhaps trying to play safe, interprets anything which "strikes out" as "dysrhythmic" and subsequently puts the

JAYPEE BROTHERS

TABLE 1: Age-wise distribution of common seizure mimics in children.

Neonate	Infancy/Toddler	Children/Adolescents
• Neonatal sleep myoclonus • Jitteriness • Hyperekplexia	• Benign myoclonus/shuddering • Self-gratification • Breath holding spell • Benign paroxysmal vertigo • Gastroesophageal reflux/Sandifer syndrome • Benign tonic upgaze • Benign paroxysmal torticollis • Stereotypies • Parasomnias • Infantile colic	• Syncope • Psychogenic nonepileptic seizures • Tics • Migraine equivalents • Parasomnias

final impression as " seizure disorder". Such a term does not exist in the International League Against Epilepsy (ILAE) nomenclature!

Result

Parents and relatives get panicky; move from doctor to doctor and the poor patient (often a child), gets loaded up with unnecessary antiepileptic drugs almost all of which have potential side effects, especially in relation to cognition and behavior. It is very strongly felt that the reporting neurologist must exhibit some degree of restraint in "wording" the conclusive sentence. One must remember that *it is always better to underinterpret an EEG than overinterpreting them.* If a patient really has got epilepsy, it would reveal its true nature in due course of time.

Table 1 depicts the age-wise distribution of common conditions which are often mistaken as epileptic seizures in children.

PHYSICAL AND NEUROLOGICAL STATUS

Head injuries, shoulder dislocation, and tongue bite involving the lateral aspect of the tongue are strongly suggestive of epileptic seizures.[7] Bilateral extensor plantar responses are common in the postictal stuporous state.

ELECTROENCEPHALOGRAM

The pitfalls in EEG diagnosis of epileptic seizures had already been touched upon. It is an important diagnostic tool as paroxysmal changes are seen in 34% cases after a first seizure and as much as 50–70% if performed within 24 hours.[8,9] A sleep-deprived EEG can reveal additional spike discharges in additional 13–31% cases. EEG has got a low sensitivity and should be used as a test to exclude epilepsy but it is very useful in the classification of different types of seizures and epilepsy syndromes **(Appendix 1)**. Minor EEG findings should not be over interpreted as 2.8% children and 0.4% adults may display minor paroxysmal changes in absence of epilepsy.[10] The request for EEG should come from a specialist as a survey in a district hospital has revealed that as many as 56% of EEG done were unnecessary.[11]

BRAIN IMAGING

The CT scan of the brain is widely available but in absence of focal neurological signs its yield is relatively low (4–6%).[12,13] MRI scan of the brain with appropriate sequences is the investigation of choice, but it should be ordered only by specialists in the field. In a case series of 166 patients with first unprovoked seizures, MRI was positive in 47% cases.[14]

LABORATORY TESTS

Routine blood tests like blood glucose, electrolytes, calcium, and blood counts are of clinical significance in only a small proportion of cases.[15,16] In patients with generalized tonic-clonic seizure (GTCS), serum lactate levels were higher than in patients with syncope and PNES. Serum lactate levels in male patients with GTCS were higher than females. It has been suggested that a cutoff value of 2.43 mol/L may be considered appropriate to distinguish GTCS from nonepileptic events. This cutoff value was less sensitive for female patients. If the serum lactate value is determined within 2 hours from ictus

JAYPEE BROTHERS

then this cutoff value can be useful in differentiating GTCS from other nonepileptic events such as syncope and psychogenic nonepileptic events in male patients.[10] In addition to astute clinical observation, imaging, and EEG, serum lactate can also be a helpful diagnostic tool in assessing patients with impaired consciousness. However, this needs to be tested in larger population of patients before its routine clinical use.

SERUM PROLACTIN IN SEIZURE DIAGNOSIS

Ever since the original report by Trimble in 1978 about the role of prolactin in seizure diagnosis is still remains unsettled. In a series of 200 patients undergoing video EEG monitoring in an epilepsy clinic, serum prolactin was measured within 20 minutes of the seizure or seizure-like event. Abubakr and Wambacq found serum prolactin to be elevated in all 22 patients with GTCS, in 27 out of 32 patients with complex partial seizures but in only 42 out of 146 patients with psychogenic seizures.[11] Sensitivity was 100% for tonic-clonic seizures and 84.6% for complex partial seizures. Serum prolactin was elevated in 84.4% of cases with epileptic events but only in 28.8% of patients with nonepileptic events. The American Academy of Neurology Technology and Assessment Subcommittee reviewed 10 studies and found a sensitivity of 52.6% and specificity of 92.8% for all types of epileptic seizures. The criteria for establishing an elevated prolactin level was unclear as some studies took doubling of base line serum prolactin values whereas others considered an absolute value of 16–45 ng/mL. The absolute value is important as doubling of value from 1 to 2 ng/mL is unlikely to be of any significance. So, ideally the serum prolactin level must double and also reach a minimum value of 15 ng/mL to be of clinical significance. Clinical conditions that interfere with prolactin levels such as pregnancy and lactation and intake of neuroleptic and dopaminergic drugs should be carefully taken into account. Serum prolactin levels really do not actually provide any significant additional information and it is often the case of the glass being half full or half empty. They do not really help in distinguishing psychogenic nonepileptic events from organic ones. The serum prolactin estimation does not help in coming to a diagnosis in an epilepsy monitoring unit as the issue is usually solved with the integration of the clinical and electrophysiologic data. In some settings, the serum prolactin estimation can be of value where epilepsy is strongly considered. A negative prolactin level can be useful in an outpatient setting with suspected tonic-clonic seizures. Therefore, serum prolactin estimation plays only a minor role in the diagnosis of epileptic seizures where an astute clinical observation and history plays a far more important role. All laboratory tests with the exception of video EEG play only a minor supplementary role with false positives and false negatives and do not substitute a clinical observation which is still considered a gold standard in the diagnosis of epilepsy.

EXCLUSION OF OTHER PAROXYSMAL EVENTS MIMICKING EPILEPTIC SEIZURE

Anoxic/Hypoxic Paroxysmal Event (Syncope)

Syncope is almost ten times more common than epilepsy.[12] This is particularly relevant in the elderly, and in patients above 70 years they have a 23% chance of having a syncope in the next 10 years.[13] A standard 12-lead ECG is the initial investigation for the evaluation of syncope. Echocardiography is positive only in patients with a suggestive cardiac history and with abnormalities in their ECG.[14] The tilt table test can only be recommended in patients with an atypical history of LOC according to the guidelines of the European Society of Cardiology.[15] When syncope is accompanied by jerking movements, it may be confused with epilepsy. This was frequently seen in a group of 56 medical students with self-induced syncope.[16] Syncope generally has a prodromal phase of lightheadedness, gradual fading of the visual and auditory surroundings, blurred vision, and generalized weakness. The patient is usually pale with epigastric discomfort with a lot of sweating. There is no cyanosis or a rising feeling in the epigastric region as seen in seizures. There is loss of postural tone with a fall usually without injuries and a brief LOC. There may be a tonic phase and a few clonic jerks but only after a few seconds and never accompanying the fall. There may be some vocalizations and automatisms, but they are usually very brief. If there is prolonged cerebral hypoxia there may be decorticate posturing and opisthotonos. Urinary incontinence may also occur. According to the Lempert's study on intentionally provoked syncope, clonic jerks are multifocal, arrhythmic, and are probably less frequent than those observed in "spontaneous" syncope. Jerking movements range from 12 to 46%[17,18] among blood donors who faint. Recovery is usually rapid and eye contact is very quickly established. But occasionally recovery may be delayed and some degree of postictal confusion may occur which may mimic a seizure. Eyes are usually open during the episode as also occurs during a seizure. However, eyes usually roll upward during a syncope whereas eyes usually turn laterally to either side during a seizure depending on the side of location of the epileptic

JAYPEE BROTHERS

focus. The patient may still feel very weak after recovery, look pale, and have a lot of diaphoresis. A large number of patients have a precipitating factor like emotional stress, prolonged standing in a hot crowded place, carotid sinus stimulation, low salt intake or dehydration or raised intrathoracic pressure like during coughing. The common causes of syncope are listed in **Box 4** and may include benign causes like vasovagal attacks or malignant life-threatening causes like dangerous cardiac arrhythmias. If the syncope occurs a few moments after exercise, it may indicate reflex syncope but an attack during exercise indicates a more sinister cause like cardiomyopathy or a conduction defect. Neurally mediated vasovagal syncope generally occurs in the upright position but venepuncture-induced syncope can occur in supine position as well. Abdominal pain, that may be confused with epigastric aura of temporal lobe seizures, may occur at onset. Auras comprising epigastric, vertiginous, visual, or somatosensory experiences may occur in both vasovagal and cardiogenic syncope.

BOX 4: Classification of syncope.

- *Insufficient pumping action of the heart*:
  - Arrhythmia (atrioventricular block, paroxysmal supraventricular and ventricular tachycardia, and long QT syndrome)
  - Structural cardiac disease (valvular disease, congenital heart disease, obstructive cardiomyopathy, cardiac tamponade, pulmonary embolism, and global myocardial ischemia)
- *Insufficient vascular tone, leading to orthostatic hypotension*:
  - Autonomic failure
  - Primary (multiple system atrophy and pure autonomic failure)
  - Secondary (diabetic and other neuropathies
  - Drugs (antidepressants and beta-blockers)
- *Insufficient circulatory volume*:
  - Hypovolemia (Addison's disease, diuretics, and hemorrhage)
- *Inappropriate neural control over the circulation*:
  - Reflex syncope (vasovagal syncope, carotid sinus syndrome, visceral syncopes, micturition, deglutition, glossopharyngeal, cough, Valsalva, and sneeze)

Transient Ischemic Attack

A transient ischemic attack (TIA) involving the vertebrobasilar territory can involve the ascending reticular activating system and cause transient LOC. Such events are accompanied by other brainstem features such as dysarthria, diplopia, vertigo, ataxia, or hemiparesis and be easy to distinguish. More than 75% of TIAs last longer than 5 minutes and this is longer than either a seizure or a syncope.[19]

Carotid Sinus Syncope

Carotid sinus hypersensitivity is a common cause of unexplained falls in elderly people generally above the age of 60 years. Testing for such hypersensitivity should be part of routine workup of elderly subjects with falls and transient LOC of very brief duration. Such testing should always be done with ECG and blood pressure monitoring in a hospital setting. Some subjects with carotid sinus hypersensitivity may have falls without definite LOC where sinus pressure may occur from head turning or tight collars. Sinus hypersensitivity causes peripheral vasodilatation causing hypotension as well as slowing of heart rate. Both these features need to be looked for during a carotid massage test **(Fig. 1)**. However, such tests carry the risks prolonged asystole, transient or permanent neurological deficits, stroke, and sudden death; hence, the justification of the aforementioned warning.

Long QT Syndrome

This form of cardiogenic syncope needs special mentioning as it may be associated with convulsive syncopes that cause unexplained sudden death in a young person and may closely mimic a GTCS. The condition is often familial with autosomal dominant inheritance resulting from a mutation affecting sodium or potassium channels. Syncope results from ventricular tachyarrhythmia, generally torsades de pointes triggered by fear, fright, emotionally charged exercises, and sleep. ECG is diagnostic.

Brugada syndrome is characterized by three types of ST segment anomalies and is another type of genetic ion channel disorder caused usually by SCN5A mutation. This

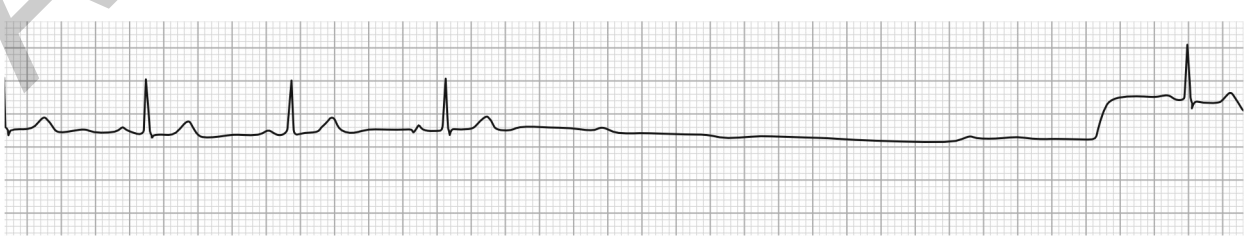

FIG. 1: Sinus arrest induced by carotid massage.

JAYPEE BROTHERS

may imitate an epileptic attack and may cause sudden death.[20]

Such conditions highlight the need for a proper ECG recording in every subject presenting with a history suggestive of a convulsion for the first time. A recent report highlights the need for searching for occult cardiac arrhythmias (asystole/complete heart block) in some subjects presenting with intractable "epileptic seizures" with implantable loop recorder (ILR) on a long-term basis.[21]

Anoxic Epileptic Seizures

At times, not very commonly though, true epileptic seizures can be triggered by nonepileptic syncope, and hence the name. This is not synonymous with reflex anoxic seizure, which is a syncope. Neurally mediated syncope may trigger a prolonged clonic seizure with myoclonic jerks and Valsalva maneuver-induced syncope can trigger a vibratory tonic epileptic seizure in children. On the other hand, epileptic seizures may manifest with syncope like attacks in Panayiotopoulos syndrome (*epileptic seizure imitating syncope*).

PSYCHOGENIC PAROXYSMAL EVENT

Psychogenic pseudosyncope and dissociative seizures remain diagnostic challenges to general practitioners and specialists. They constitute about 30% of cases in syncope clinics and about 25% cases who are referred for refractory epilepsy in tertiary epilepsy centers.[22-24] These events start by external or internal triggers like unpleasant people or surroundings and flashback of traumatic internal events. The prodromal phase is characterized by symptoms such as tachycardia, epigastric discomfort, and difficulty in breathing with tachypnea. Such patients may have neurological symptoms such as tremor, weakness, headache, or sensory changes. They may also present with pure psychogenic features such as visual and auditory hallucinations, intense fright, depersonalization, dream-like states, or feeling of impending death. The patient may stare blankly, have bizarre movements, and may fall down very slowly without any injury. Unresponsiveness may be very prolonged and last for >10 minutes. Some patients may have pseudostatus epileptics. Movements are asynchronous and demonstrative and usually out of phase with intermittent vocalizations. Pelvic thrusting, side-to-side head movements, and forcible eye closure are commonly seen. *La grande courbure* of Charcot or intense opisthotonus like body arching is not commonly seen nowadays. There may be quick return of consciousness after the ictus and patient may only be partially amnestic about it. Total amnesia is rare.[25] The "eye sign" is important. Convulsive movements with closed eyes are highly suggestive of PNES. Eyes are generally open during GTCS and syncope and if closed before, would open during the event The resistance offered to passive opening of eyes is also an important feature to note in psychogenic cases.

These events have been named differently but the accepted terminology is PNES as decided by epilepsy experts. The American Psychiatry Association names them as conversion disorders [the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-4)—somatoform section].[25] The most suitable term could be dissociative convulsion. The International Classification of Diseases, Tenth Revision (ICD-10) states that the common theme shared by dissociative disorders is a partial or complete loss of the normal integration between memories of the past, awareness of identity, and immediate sensation and control of body movements (World Healing Organization 1992).[26] Dissociative convulsions may begin in early life and usually begin in a specific situation caused by external (place, time, and witness) or internal triggers (flash backs and emotions). Bowman and Markand's[27] study of 45 cases revealed that dissociative convulsions begin in the young adulthood (20–30 years of age) and are mostly seen in women (78%). Past history of a traumatic event is mentioned by 84% of patients (sexual abuse 67%, physical abuse 67%, and other trauma 73%). Psychiatric comorbidity is common. These include somatoform disorders (89%), dissociative disorders (91%), affective disorders (64%), personality disorders (62%), posttraumatic stress disorders (49%), and other anxiety disorders (47%).[27]

Suggestive features of PNES are given in **Box 5**.

PAROXYSMAL EVENTS OCCURRING DURING SLEEP

Electric shock-like movements, called hypnic/hypnagogic jerks/myoclonus, can occur at the onset of sleep. Body rocking, head banging, or rolling can occur at the sleep–wake transition.[28,29] In adults above 50 years, rapid eye movement (REM) sleep behavior disorders are seen. These conditions often resemble frontal lobe seizures and are difficult to differentiate. In the pediatric age group, sleep walking, sleep talking, nocturnal terrors, and different types of non-rapid eye movement (NREM) parasomnias can be seen. Some motor phenomenon of different types of nocturnal seizures can mimic these various parasomnias. This could be due to cortical release of subcortical motor

JAYPEE BROTHERS

BOX 5: Psychogenic nonepileptic seizures (PNES)—suggestive features.

- Atypical auras
- Preictal behavior changes
- Gradual onset
- "Pseudo sleep" at onset
- Eye closure during unresponsiveness
- Eye fluttering
- Discontinuous seizure activity
- Gradual cessation
- Absence of postictal state
- High seizure frequency
- Excessive variation in seizure manifestations
- Funny vocalization
- Resistance to eye opening
- Prolonged duration
- Occurrence during clinic visits
- Suggestibility
- Precipitation of typical attack by suggestion
- Attacks only in the presence of others
- Vocalizations consisting of gagging, retching, gasping, screaming, crying, or moaning
- Emotional display during events
- Emotional triggers
- Retained consciousness and recollection of events with bilateral jerking activity

learned patterns during such episodes.[30] Night terrors happen in the first 3 hours of sleep, when the child suddenly wakes up from sleep, screams, and is terrified. This may last up to 10 minutes. Sleepwalking is found in children of 5–10 years of age and in the first half of sleep. The child walks about in a trance like state with eyes often open and may wander out of the room but usually returns to bed. Several other clinical conditions may resemble epileptic events and will be discussed later on in the appropriate context. Distinctive features of sleep parasomnias and nocturnal frontal lobe epilepsy are depicted in **Table 2**.

CONCLUDING REMARKS

The present chapter would hopefully serve to reinforce three principles: (1) The primacy of the clinical history in the determination of epilepsy diagnosis; (2) the utility of the EEG is at its maximum when the prior probability can be shifted from the extremes (by a good history); and (3) many of the pitfalls can only be avoided by secure knowledge of normal maturational patterns and variation in childhood EEGs. Some of the common errors in pediatric EEG reporting include misinterpreting movement artifacts, high amplitude delta slowing during hyperventilation, and normal sleep markers including vertex waves, spindles, K-complexes, and hypnagogic

TABLE 2: Sleep parasomnias and nocturnal frontal lobe epilepsy.

Feature	Non-REM parasomnias (somnambulism; night terrors)	Nocturnal frontal lobe epilepsy
Onset age	<10 years	*Variable*: Generally childhood or adolescence
Positive family history	60–90%	<40%
Number of attacks/day	1–2	>3
Attacks/month	<1–4	20–40
Clinical course	Disappear by adolescence	Frequency generally stable
Duration of disorder	Around 7 years	Around 20 years
Duration of episodes	Seconds to 30 minutes	Seconds to 2–3 minutes
Semiology	Variable complexity	Very stereotyped on VEEG
Triggering factors	Sleep deprivation; fever; stress	Usually none
Associated conditions	Obstructive sleep apnea	Usually none
Ictal EEG	Slow waves; no epileptiform features	Usually normal; often obscured by muscle artifacts; epileptiform features in <10%
Timing of sleep	First half of night—generally after first 30 minutes	Any time
PSG stage	Stage III–IV	Generally stage II; may be later

(EEG: electroencephalogram; PSG: polysomnography; REM: rapid eye movement; VEEG: video electroencephalogram)

JAYPEE BROTHERS

hypersynchronous discharges as epileptiform discharges. In children, benign variants such as wicket waves, benign epileptiform transients of sleep (BETS), and rhythmic midtemporal theta bursts of drowsiness (RMTD) can all mimic epileptiform discharges to an inexperienced reader. The diagnosis of epilepsy is still based on careful elicitation of history from patient (if not too young) and relatives and often repeated analyses of seizure semiology, either physically or videographically (home videos/mobile phones). This still would constitute the closest to a gold standard for diagnosis, although currently often combined with video EEG monitoring. Video EEG, of course, has its own limitations such as its inability to record seizures arising from inaccessible areas like supplementary motor seizures, recording only one form of seizure discharge in children with polymorphic seizure semiologies, and occasional precipitation of generalized seizures (even status) during recording (with a drug withdrawal protocol). Detailed history elicitation by an experienced clinician is more sensitive than EEG or almost any other investigation in the evaluation of epilepsy. Finally, the use of EEG should be framed within a consideration of the costs and consequences of a falsely positive or falsely negative result.

LEARNING POINTS

- The art of listening has to be mastered in differentiating seizures from close mimics. This is time consuming and can be acquired with practice.
- If a dissociative disorder is suspected then suggestible questions like depersonalization and derealization can be helpful. Sense of panic and dissociation are common in the prodromal phase but not often volunteered by patients.[31]
- Patients with a first seizure should ideally be seen by an epileptologist with experience in that field. This gives a diagnostic yield of about 96%.[33] Patient should not be treated in case of diagnostic uncertainty; it is better to adopt a policy of "wait and see".
- Video EEG is very useful in diagnosis particularly in dissociative seizures and a 48-hour recording may clinch the diagnosis in the majority of cases.[32] There indeed are pitfalls with this technique as well. In difficulty to diagnose cases, a combined tilt table test and carotid massaging done under EEG control may be helpful.[33,34] Provocation of a seizure by an experienced clinician under protected environment with proper counseling is accepted ethically.

REFERENCES

1. King MA, Newton MR, Jackson GD, Fitt GJ, Mitchell LA, Silvapulle MJ, et al. Epileptology of the first-seizure presentation: clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. Lancet. 1998;352:1007-11.
2. Schreiner A, Pohlmann-Eden B. Value of the early electroencephalogram after a first unprovoked seizure. Clin Electroencephalogr. 2003;34:140-6.
3. Walczak TS, Jayakar P. Interictal EEG. In: Engel J Jr, Pedley TA (Eds). Epilepsy: A Comprehensive Textbook. Philadelphia: Lippincott-Raven; 1997. pp. 831-48.
4. Smith D, Bartolo R, Pickles RM, Tedman BM. Requests for electroencephalography in a district general hospital: retrospective and prospective audit. BMJ. 2001;322:954-7.
5. Ramirez-Lassepas M, Cipolle RJ, Morillo LR, Gumnit RJ. Value of computed tomographic scan in the evaluation of adult patients after their first seizure. Ann Neurol. 1984;15:536-43.
6. Breen DP, Dunn MJG, Davenport RJ, Gray AJ. Epidemiology, clinical characteristics, and management of adults referred to a teaching hospital first seizure clinic. Postgrad Med. 2005;81:715-8.
7. Pohlmann-Eden B, Schreiner A. Epileptology of the first seizure presentation. Lancet. 1998;352:1855-6.
8. Turnbull TL, Vanden Hoek TL, Howes DS, Eisner RF. Utility of laboratory studies in the emergency department patient with a new-onset seizure. Ann Emerg Med. 1990;19:373-7.
9. Dunn MJ, Breen DP, Davenport RJ, Gray AL. Early management of adults with an uncomplicated first generalised seizure. Emerg Med J. 2005;22:237-42.
10. Doğan EA, Ünal A, Ünal A, Erdoğan Ç. Clinical utility of serum lactate levels for differential diagnosis of generalized tonic–clonic seizures from psychogenic non-epileptic seizure and syncope. Epilepsy Behav. 2017;75:13-7.
11. Abubakr A, Wambacq I. Diagnostic value of serum prolactin levels in PNES in the epilepsy monitoring unit. Neurol Clin Pract. 2016;6(2):116-9.
12. Soteriades ES, Evans JC, Larson MG, Chen MH, Chen L, Benjamin J, et al. Incidence and prognosis of syncope. N Engl J Med. 2002;347:878-85.
13. Sararin FP, Louis-Simonet M, Carballo D, Slama S, Rajeswaran A, Metzger JT, et al. Prospective evaluation of patients with syncope: a population based study. Am J Med. 2001;111:177-84.
14. Sarasin FP, Junod AF, Carballo D, Slama S, Unger PF, Louis-Simonet M. Role of echocardiography in the evaluation of syncope: a prospective study. Heart. 2002;88:363-7.
15. Brignole M, Alboni P, Benditt DG, Bergfeldt L, Blanc JJ, Thomsen PE, et al. Task Force on Syncope, European Society of Cardiology. Guidelines on management (diagnosis and treatment) of syncope–update 2004. Executive Summary. Eur Heart J. 2004;25:2054-72.
16. Lempert T, Bauer M, Schmidt D. Syncope: a videometric analysis of 56 episodes of transient cerebral hypoxia. Ann Neurol. 1994;36:233-7.
17. Lin JT, Ziegler DK, Lai CW, Bayer W. Convulsive syncope in blood donors. Ann Neurol. 1982;11:525-8.
18. Newman BH, Graves S. A study of 178 consecutive vasovagal syncopal reactions from perspective of safety. Transfusion. 1982;41:1475-9.

JAYPEE BROTHERS

19. Warlow CP, Dennis M, van Gijn J, Hankey G, Bamford J, Sandercock P, et al. Stroke: A Practical Guide to Management, 2nd edition. Malden: Wiley–Blackwell; 2000. pp. 28-105.
20. Kanjwal K, Karabin B, Kanjwal Y, Grubb BP. Differentiation of convulsive syncope from epilepsy with an implantable loop recorder. Int J Med Sci. 2009;6(6):296-300.
21. Mattson RH. Value of intensive monitoring. In: Wada HA, Penry JK (Eds). Advances in epileptology. New York: Raven Press; 1980. pp. 43-51.
22. Gates JR, Ramani V, Whalen S, Loewenson R. Ictal characteristics of psychogenic seizures. Arch Neurol. 1985;42:1183-7.
23. Benbadis SR, O'Neill E, Tatum WO, Heriaud L. Outcome of prolonged video-EEG monitoring at a typical referral epilepsy center. Epilepsia. 2004;45:1150-3.
24. Bowman ES. Why conversion seizures should be classified as a dissociative disorder. Psychiatr Clin N Am. 2006;29:185-211.
25. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th edition. Washington, DC: American Psychiatric Association; 1994. p. 477.
26. World Health Organization. The ICD-10 classification of mental and behavioural disorders. Clinical description and diagnostic guidelines. Geneva, Switzerland: World Health Organization; 1992;151:737-9.
27. Bowman ES, Markand ON. Psychodynamics and psychiatric diagnoses of pseudoseizure subjects. Am J Psychiatry. 1996;153:57-63.
28. American Academy of Sleep Medicine. The International Classification of Sleep Disorders (revised). Chicago, IL: American Sleep Disorders Association; 2001.
29. Tassinari CA, Rubboli G, Gardella E, Cantalupo G, Calandra-Buonaura G, Vedovello M, et al. Central pattern generators for a common semiology in fronto-limbic seizures and parasomnias. A neuroethologic approach. Neurol Sci. 2005;26:225-32.
30. Stone J, Carson A, Sharpe M. Functional symptoms and signs in neurology: assessment and diagnosis. J Neurol Neurosurg Psychiatry. 2005;76:2-12.
31. Deacon C, Wiebe S, Blume WT, McLachlan RS, Young GB, Matijevic S. Seizure identification by clinical description in temporal lobe epilepsy: how accurate are we? Neurology. 2003;61:1686-9.
32. Lobello K, Morgenlander JC, Radtke RA, Bushnell CD. Video-EEG monitoring in the evaluation of paroxysmal behaviour events: duration, effectiveness and limitations. Epilepsy Behav. 2006;8:261-6.
33. Zaidi A, Clough P, Cooper P, Scheepers B, Fitzpatrick AP. Misdiagnosis of epilepsy: many seizure-like attacks have a cardiovascular cause. J Am Coll Cardiol. 2000;36:181-4.
34. Grubb BP, Gerard G, Roush K, Temesy-Armos P, Elliott L, Hahn H, et al. Differentiation of convulsive syncope and epilepsy with head-up tilt testing. Ann Intern Med. 1991;15:871-6.

JAYPEE BROTHERS

APPENDIX 1

Epilepsy Imitators

There are a range of conditions associated with recurrent paroxysmal events that may imitate and be misdiagnosed as epilepsies. It is important that these disorders are considered in the evaluation of paroxysmal events as misdiagnosis rates in epilepsy are high throughout the world. History remains the key to a correct diagnosis with video recordings very helpful. There are some conditions in which epileptic and nonepileptic events can coexist. The following is a list of conditions whose clinical manifestations may closely mimic those of epileptic seizures and these conditions may be designated as epilepsy imitators by the ILAE in 2017.

- *Syncope and anoxic seizures*:
  - Vasovagal syncope
  - Reflex anoxic seizures
  - Breath-holding attacks
  - Hyperventilation syncope
  - Compulsive Valsalva
  - Neurological syncope
  - Imposed upper airways obstruction
  - Orthostatic intolerance
  - Long QT and cardiac syncope
  - Hypercyanotic spells
- *Behavioral, psychological, and psychiatric disorders*:
  - Daydreaming/inattention
  - Self-gratification
  - Eidetic imagery
  - Tantrums and rage reactions
  - Out of body experiences
  - Panic attacks
  - Dissociative states
  - Nonepileptic seizures
  - Hallucinations in psychiatric disorders
  - Fabricated/factitious illness
- *Sleep-related conditions*:
  - Sleep-related rhythmic movement disorders
  - Hypnagogic jerks
  - Parasomnias
  - REM sleep disorders
  - Benign neonatal sleep myoclonus
  - Periodic leg movements
  - Narcolepsy–cataplexy
- *Paroxysmal movement disorders*:
  - Tics
  - Stereotypies
  - Paroxysmal kinesigenic dyskinesia
  - Paroxysmal nonkinesigenic dyskinesia
  - Paroxysmal exercise-induced dyskinesia
  - Benign paroxysmal tonic upgaze
  - Episodic ataxias
  - Alternating hemiplegia
  - Hyperekplexia
  - Opsoclonus-myoclonus syndrome
- *Migraine-associated disorders*:
  - Migraine with visual aura
  - Familial hemiplegic migraine
  - Benign paroxysmal torticollis
  - Benign paroxysmal vertigo
  - Cyclical vomiting
- *Miscellaneous events*:
  - Benign myoclonus of infancy and shuddering attacks
  - Jitteriness
  - Sandifer syndrome
  - Nonepileptic head drops
  - Spasmus nutans
  - Raised intracranial pressure
  - Paroxysmal extreme pain disorder
  - Spinal myoclonus

JAYPEE BROTHERS

For detailed information on neurology books, visit our website www.jaypeebrothers.com, for detailed information on neurology books, visit our website www.jaypeebrothers.com, for detaile

Essentials of CLINICAL NEUROLOGY *for* The Psychiatrist

The present volume is perhaps the only comprehensive text published from Asia in English language to discuss the essentials of clinical neurology primarily meant for both trainee and trained psychiatrists. Through 34 chapters and 18 commentaries, the neuropsychiatric manifestations of nearly all commonly encountered neurological disorders have been discussed in reasonable detail highlighting important diagnostic and therapeutic issues. Basic clinical neurological aspects of conditions such as headache, epilepsy, strokes, and movement disorders have been discussed specially for trainee psychiatrists to keep them abreast with current developments in these fields. Neurological and systemic medical illnesses may initially present with psychiatric symptoms and thus may cause delay in implementing appropriate therapy. This major pitfall or red flag in clinical psychiatric practice have been stressed upon in more than one chapter in the book. Dementias lie at the borderland between neurology and psychiatry. Diagnostic aspects of dementias, their various subtypes, and specially the rapidly progressive ones including the ones which are treatable have been discussed in detail in more than one chapters. Diagnosis of functional neurological disorders is a major challenge for both the neurologists and psychiatrists. The positive and negative features of such disorders have also been highlighted in some chapters and commentaries. The historical notes on the development of the subspeciality of neuropsychiatry and the changing concepts on the nature of the illness commonly designated as hysteria would be of interest to read for both neurologists and psychiatrists.

Ambar Chakravarty MD FRCPC FRCP FRCPE FICP FIAN is an Honorary Professor and Emeritus Consultant in the Department of Neurology at the Vivekananda Institute of Medical Sciences, Kolkata, West Bengal, India. A recipient of several awards at both national and international levels and Lifetime Achievement Awards, Dr Chakravarty is a member of the Royal College of Physicians of London and Edinburg, Royal College of Physicians and Surgeons of Canada, Indian Academy of Neurology, and Indian College of Physicians. He has published extensively in national and international peer-reviewed journals on varied neurological topics ranging from headaches, epilepsy, movement disorders, ataxias, neuro-ophthalmology, neurological histories to neuroscience of art and creativity. He had been the Founder Editor of the Journal of Association of Neuroscientists of Eastern India (1996–2000) and an Editor-in-Chief of Frontiers of Neurology (2012–2016). He has published 14 monographs on neurological topics and has delivered around 20 orations so far. He is a Past President of the Indian Academy of Neurology (2005–2006).

Printed in India

Available at all medical bookstores or buy online at www.jaypeebrothers.com



JAYPEE BROTHERS
Medical Publishers (P) Ltd.
EMCA House, 23/23-B, Ansari Road,
Daryaganj, New Delhi - 110 002, INDIA
www.jaypeebrothers.com

Join us on facebook.com/JaypeeMedicalPublishers

Shelving Recommendation
NEUROLOGY

ISBN 978-93-5696-550-8

9 789356 965508