

APPROACH TO SEIZURES

A SEIZURE IS DEFINED AS ABNORMAL NEUROLOGIC FUNCTIONING CAUSED BY ABNORMALLY EXCESSIVE ACTIVATION OF NEURONS

EPILEPSY IS RECURRENT UNPROVOKED SEIZURES

EPIDEMIOLOGY AND CLASSIFICATION:

- ~1% of ED visits, half had alcohol or low-anticonvulsant levels implicated as cause
- CLASSIFICATION:
 - Primary vs secondary
 - Generalised vs focal (partial)
 - Generalised → abnormal activity in both hemispheres
 - Focal (partial) → one hemisphere
 - Simple → consciousness maintained
 - Complex → consciousness lost
 - Can become secondarily generalised
 - STATUS EPILEPTICUS:
 - At least 30 minutes of persistent seizures or recurrent seizures without inter-ictal recovery to full consciousness
 - Some advocate shortening this period
 - SECONDARY SEIZURES → raft of causes:
 - Intoxication/poisoning
 - Encephalopathy
 - Organ failure
 - CNS infection
 - CNS tumour
 - Pregnancy (eclampsia)
 - Supra/subtherapeutic anticonvulsant levels

Table 15-1

Classification of Seizures in a General Adult Population

SEIZURE TYPE	PERCENTAGE
Generalized	
Tonic-clonic	35
Absence	1
Myoclonic	<1
Others	2–3
Partial	
Simple partial	3
Complex partial	11
Secondarily generalized	27
Mixed partial	12
Unclassified	9

- Distribution in kids is different → relatively high incidence of febrile seizures:
 - Occur in 2-5% of kids age 6 months to 5 years with 20-30% having recurrence

- First time seizure in infants younger than 6 months may indicate underlying pathology warranting further assessment

PATHOPHYSIOLOGY:

- Seizures occur when abnormal increased electrical activity in initiating neuron activates adjacent neurons and propagate
- Cellular pathophysiology is not well understood
- Clinical seizure activity typically, but not always reflects the initiating focus
- When ictal discharge extends below the cortex to the reticular activating → consciousness is altered
 - In generalised seizures, the focus is **OFTEN DEEP AND MIDLINE** → hence prompt LOC

DIAGNOSTIC APPROACH:

Table 15-2 Differential Considerations for the Diagnosis of Seizure*

DISORDER	CLASSIFICATION	ICTAL-LIKE MANIFESTATIONS
Syncope	Vasodepressive vs dysrhythmogenic (including long QT syndrome) vs orthostatic Preictal or postictal twitching	"Fit vs. faint"
Hyperventilation syndrome		Mood disturbances Posturing of extremities
Prolonged breath-holding	More typical in children	Tonic-clonic movements Loss of urinary continence
Toxic and metabolic disorders	Alcohol abuse/withdrawal Hypoglycemia Phencyclidine Tetanus Strychnine and camphor Extrapyramidal reactions	Delirium tremens, blackout Abnormal behavior Buccolingual spasms Myotonic spasms Myotonic spasms
Nonictal CNS events	Transient ischemic attacks Transient global amnesia Hemiparetic migraine Carotid sinus hypersensitivity Narcolepsy	Posturing, deviation of eyes Drop attacks, "fit vs. faint" Similar to postictal state, absence status Todd's paralysis Drop attacks, "fit vs. faint" Drop attacks, "fit vs. faint"
Movement disorders	Hemiballismus, tics	Convulsions
Psychiatric disorders	Fugue state Panic attacks	Similar to postictal state, absence status Twitching, altered mental state
Functional disorders	Pseudoseizure	May closely resemble ictal activity; patients may have both true seizures and pseudoseizures

*Electroencephalography provides the definitive diagnosis in unclear cases.
CNS, central nervous system.

- An incorrect diagnosis is both expensive and has implications for the patient in terms of loss of driving privileges → hence the main task in ED is to determine whether the patient is having a "true" seizure → ictal activity can only be irrefutably by EEG
- Primary differential is **SYNCOPE**
 - Sudden LOC with abnormal movements can be ictal or syncopal → one study showed myoclonic activity in 90% of syncope patients together with:
 - Frequent head turns, upward gaze, oral automatisms and righting movement → makes discrimination difficult
 - Generally → ictal righting movements are more forceful and prolonged than the "twitches" seen with syncope. **POST-ICTAL** state does not occur with syncope
- **SUGGESTIONS OF ICTAL DIAGNOSIS:**
 - Retrograde amnesia
 - Loss continence
 - Tongue biting

- Metabolic acidosis that has resolved by time of repeat analysis (if blood drawn at time of seizure)

RAPID ASSESSMENT AND STABILISATION:

- Early access, BSL → hypoglycaemia is the most metabolic cause of seizure (but prolonged seizure activity may CAUSE hypoglycaemia) → IV dextrose
- Airway protection is paramount → prepare for RSI if seizure not aborted with anticonvulsants
- Benzodiazepines are first line (lorazepam 0.1mg/kg, diazepam 0.2mg/kg, midazolam 0.1mg/kg):
 - Diazepam can be given rectally, ETT, IO
 - Lorazepam has longest half life, hence leads to less recurrence of seizures in status
- Second line → phenytoin 20mg/kg (fosphenytoin is pro-drug that can be given IM and more quickly IV, but is more expensive), phenobarb 20mg/kg, valproate 10-15mg/kg
- Third line → general anaesthetic (isoflurane, propofol) → intubation mandated. Include neuromuscular blocker to reduce hyperthermia/metabolic burden
- SPECIFIC SCENARIOS:
 - ISONIAZID TOXICITY → pyridoxine
 - ECLAMPSIA → magnesium 6g over 15mins then 2g/hour → ~ 10% will have second seizure
 - HYPONATRAEMIA (water intoxication) → hypertonic saline

PIVOTAL FINDINGS:

- Once seizure activity is controlled and airway secured → turn attention to obtaining more information
- HISTORY:
 - WAS THIS TRULY A SEIZURE? → difficult, but ictal events have six properties:
 1. Abrupt onset (most generalised seizures do NOT HAVE AN AURA)
 2. Brief duration (overestimation by observers, but ~120seconds normally)
 3. Altered mental status
 4. Purposeless activity → automatisms, tonic-clonic activity
 5. Unprovoked (especially with regard to emotional activity, exception obviously withdrawal and febrile convulsions in kids)
 6. Post-ictal state
 - a. Also incontinence and tongue biting commonly occur
 - DOES THIS PATIENT HAVE A HISTORY OF SEIZURES?
 - If yes → thorough history/exam and drug levels should suffice
 - Intercurrent illness, drug/alcohol use, recent change in medications, compliance
 - Supratherapeutic doses of carbamazepine/phenytoin can cause seizures
 - If no → consider medical, traumatic, toxicologic or neurologic causes of seizures

- **PHYSICAL EXAMINATION:**
 - Physical manifestations of convulsive ictal activity include:
 - HT
 - Tachycardia
 - Tachypnoea
 - From sympathetic stimulation
 - Resolve with cessation of seizure activity
 - With more prolonged convulsions → skeletal muscle damage, lactic acidosis and rhabdomyolysis (rarely)
 - Autonomic discharge may result in urinary or faecal incontinence and vomiting → aspiration risk
 - Look for potential sequelae of seizure activity:
 - Posterior shoulder dislocations
 - Head trauma
 - Tongue biting
 - Complete neurologic exam:
 - TODD'S PARESIS → persistent focal deficit after a seizure → indicates focal origin but can also indicate a stroke

ANCILLARY TESTING:

- **LAB TESTS:**
 - BSL
 - Anticonvulsant levels as appropriate
 - Electrolyte (calcium and sodium) and LFT
 - LP if suggestion of meningitis
- **IMAGING:**
 - If patient is fully recovered, does not have headache and has a normal neurological examination after single brief seizure → no need for CT in ED → can occur later at discretion of treating physician
 - **INDICATIONS:**

Table 15-4

Indications for Emergent Head CT for New-Onset Seizure Patients

Acute intracranial process is suspected
 History of acute head trauma
 History of malignancy
 Immunocompromise
 Fever
 Persistent headache
 History of anticoagulation
 New focal neurologic examination
 Age older than 40 years
 Focal onset before generalization
 Persistently altered mental status

- **EEG:**

- Not available in ED
- Consider in diagnosis of non-convulsive status or to monitor the intubated and paralysed patient

MANAGEMENT:

- Search for reversible insults → HYPOGLYCAEMIA, HYPOXIA, ISONIAZID TOXICITY
- Abortive therapy as previous
- The decision to institute anticonvulsant therapy is NOT made by us

DISPOSITION:

- Patients may be discharged home if they have a normal neurological exam, no comorbidities, no known structural brain disease and do not require anticonvulsants in ED