



Chapter 15 – Seizures

NOTE: CONTENT CONTAINED IN THIS DOCUMENT IS TAKEN FROM ROSEN'S EMERGENCY MEDICINE 9th Ed.

Italicized text is quoted directly from Rosen's.

Key Concepts:

- 1. The differentiation between seizures and other causes of altered mentation or abnormal motor activity is not always straightforward and may require synthesizing the history, physical examination, laboratory results, and imaging data.*
- 2. Beginning in the out-of-hospital setting, patients with possible seizure activity should be protected from injury and assessed for hypoglycemia.*
- 3. Status epilepticus is defined as seizures lasting more than 5 minutes or repeat seizures while still postictal.*
- 4. Primary abortive therapy for seizures in the ED setting includes lorazepam; if diazepam is used in status epilepticus, it should be immediately followed by a loading dose of phenytoin, fosphenytoin, or valproic acid.*
- 5. Neuroimaging is recommended for patients with seizures who have head trauma, persistently abnormal mental status, focal neurologic abnormality, or HIV infection.*
- 6. Nonconvulsive status epilepticus should be considered in patients with a prolonged postictal state or otherwise unexplained coma.*
- 7. Patients with a first-time seizure who have no known structural brain pathology, normal serum glucose and sodium levels, and normal neurologic examination can be discharged from the ED with appropriate outpatient follow-up.*

Core Questions:

1. Define seizure and provide an explanation of the classification systems for seizure
2. Define recruitment as it relates to seizure development and progression
3. Differentiate between seizure and syncope
4. Differentiate between neurogenic and psychogenic seizures
5. List 5 diagnoses that can mimic seizures (see Box 15.2)
6. Define status epilepticus and differentiate between convulsive and non-convulsive status epilepticus
7. List 10 causes of status epilepticus in adults (see Box 15.1 and 15.3)
8. Outline management of status epilepticus.
9. List indications for head CT for first seizure.

Wisecracks:



1. List 5 properties of ictal events
2. What medications are needed to treat seizing patients with the following:
 - a. Isoniazid Toxicity
 - b. TCA Toxicity
 - c. Eclampsia
 - d. Hypoglycemia
 - e. Hyponatremia
3. Name 3 key metabolic abnormalities that can cause seizures
4. Name 3 common seizure provokers that can worsen pre-existing seizure disorders
5. What percentage of patients with convulsive status epilepticus will develop non-convulsive status epilepticus?

Rosen's in Perspective

We are back at it again with another episode of CRACKCast. Today, we will be covering the topic of seizures. This content is exceedingly important, as approximately 10% of adults will experience at least one seizure. Additionally, 3% of all persons will be diagnosed with epilepsy, thus further reinforcing the importance of knowing this content. Today, we will go about diving into the nitty gritty of seizure disorders, giving you the knowledge to differentiate between neurogenic seizures, psychogenic seizures, and seizure mimics. Additionally, we will give you the tools to treat your next convulsive status epilepticus patient and a tight list of diagnoses to consider if your conventional treatments are failing. As always, we will give you some tidbits and factoids to make sure you are ready for your next Neurology pimp session. So, buckle up, grab a coffee and enjoy the ride!

Core Questions:

[1] Define seizure and provide an explanation of the classification systems for seizure

While this may seem like a simple question to start off the episode, it is important that we review the basics to build the correct foundation upon which to build. So, let's take a quick jaunt back to medical school lecture halls for the next minute or so.

Seizure is defined by Rosen's 9th Edition as an episode of abnormal neuronal excitation. While this definition is easy to remember, it is a tad simplistic. Seizures are more accurately characterised as periods of neuronal hyperexcitation and hyper-synchronicity. Remembering both the increased activity and increased synchronicity allows you to both explain the initial presentation and progression of seizure activity.



Seizures can be characterized as falling into one of the categories:

1. Primary versus Secondary
 - a. Primary seizures are by definition unprovoked and not linked to some inciting event
 - b. Secondary seizures occur as the result of some underlying pathophysiologic process such as toxic ingestion, trauma, metabolic disturbances, structural lesions etc...
2. Generalized versus Focal
 - a. Generalized seizures are typified by abnormal neuronal activity in BOTH hemispheres of the brain, resulting in a loss of consciousness or change in level of alertness
 - i. Generalized seizures can be further categorized as being either:
 1. Tonic-clonic
 2. Absence-type
 3. Atonic
 4. Myoclonic
 - b. Focal seizures occur in one hemisphere, thus allowing the patient to maintain consciousness
 - i. It is important to note that these seizures can become secondarily generalized
3. Convulsive versus Non-convulsive
 - a. Convulsive seizures are typified by uncontrolled rhythmic movements that can affect any part of the body
 - b. Non-convulsive seizures do not involve rhythmic movements of the body, but instead include:
 - i. Automatism (e.g., lip smacking)
 - ii. Confusion
 - iii. Altered mental status
 - iv. Abnormal behaviour
 - v. Coma

[2] Define recruitment as it relates to seizure development and progression

Recruitment is defined as the activation of neurons by adjacent increased electrical activity of other neurons. Neurons are activated by action potentials following contiguous pathways or deep integrated circuits that cross the midline. When the impulses track into the deep circuits of the subcortex (thus affecting the ascending reticular activating system) or cross the midline, the patient experiences an alteration in consciousness. The process of recruitment explains seizure auras (i.e., alterations in sensation, autonomic deregulations, aphasia, déjà vu, lip smacking, repeated swallowing, picking at clothing secondary to abnormal neuronal activity) and why focal seizures can become secondarily generalized.



[3] Differentiate between seizure and syncope

Seizure versus syncope - the eternal ER debate. As you probably know already, often in clinical medicine, differentiating between the patient who has experienced a syncopal episode versus one who has experienced a seizure is difficult. This process is further muddled by the fact that many people who experience a syncopal event can have brief convulsive or rhythmic motor activity that can make bystanders believe the patient had a seizure.

There are some features, however, that can help you differentiate between these two disease states. We have created a table below that will help you on your next shift to steward management of your next patient with syncope v seizure.

Features of Seizure	Features of Syncope
Presence of post-ictal symptoms (confusion, altered mental status)	Rapid recovery post-event (seconds) without persistent confusion or altered mental status
Longer duration of ictal symptoms (e.g., convulsions, jerking)	Short, seconds-long convulsions or myoclonic activity
Loss of bowel or bladder control	No loss of bowel or bladder control
Tongue biting or lateral tongue lacerations	No tongue biting or lateral tongue lacerations

[4] Differentiate between neurogenic and psychogenic seizures

As with the patient with syncope versus seizure, the patient with neurogenic versus psychogenic seizure presents a unique diagnostic challenge to clinicians in the ED. Often times, differentiating neurogenic from psychogenic seizures when the patient is mid-episode is impossible. Additionally, anywhere from 5-50% of all patients with psychogenic non-epileptic seizures ALSO HAVE EPILEPSY, further complicating treatment, investigation, and disposition of these patients.

There are features classically described in the literature that can help you differentiate between psychogenic and neurogenic seizures, and we have listed them in the table below. Just remember, it is not always as clear cut as we would like it to be.

Features of Psychogenic Non-Epileptic Seizures:

- Often last longer than neurogenic seizures
- Brief or no post-ictal period post-event



- Patients can often recall events during generalized psychogenic seizure events, thus making the diagnosis of neurogenic generalized seizures impossible
- Often include forward-thrusting pelvic movements
- Often have head turning from side to side during event
- Often include gaze deviation away from the examiner during the event
- Patients often avoid noxious stimuli during the event
- No typical metabolic acidosis after event

[5] List 5 diagnoses that can mimic seizures (see Box 15.2)

As you could tell from our discussion above, obviously there are some things that can mimic seizure activity . Given the consequences of being diagnosed with a seizure disorder (e.g, driving restrictions, occupational restrictions, etc...), being able to differentiate between those persons who truly have had a seizure and those who have not is incredibly important. Below, we have listed the top differential diagnostic considerations for seizure activity:

See Box 15.2 in Rosen's 9th Edition for a more comprehensive table explaining the differential considerations for seizure or seizure-like events.

Seizure Mimics:

Cardiac

1. Vasodepressive (vagal) syncope
2. Orthostatic syncope
3. Cardiogenic syncope

Neurologic

1. Stroke, transient ischemic attack
2. Atypical migraine
3. Movement disorders
4. Mass lesions

Toxicologic

1. Intoxication, inebriation
2. Oversedation, over-analgesia
3. Extrapyrmidal symptoms

Metabolic

1. Hypo-, hyperglycemia
2. Thyrotoxicosis
3. Delirium tremens



Infectious

1. CNS infections
2. Tetanus

Psychiatric

1. Pseudoseizure
2. Panic attacks
3. Cataplexy

[6] Define status epilepticus and differentiate between convulsive and non-convulsive status epilepticus

As per Rosen's 9th Edition: "*Status epilepticus is characterized by seizures lasting more than 5 minutes or recurrent seizures, without return to baseline mental status.*"

Remember, this is a change in definition that is significant. Previously, status epilepticus was either a seizure lasting greater than 30 minutes or two seizures with no resolution of post-ictal symptoms in between.

Also, remember that an individual can have both convulsive (i.e., typical generalized seizure activity present) and non-convulsive status epilepticus (NCSE) (i.e., typical generalized seizure activity absent). Often, many patients (approximately 15% of patients with convulsive status) will devolve into non-convulsive status. This can fool the assessor into thinking their therapies for the patient's seizures have been successful, when in reality, the patient is still experiencing the overwhelming and detrimental neuronal hyperactivity associated with this disease state. So, always keep NCSE in mind when dealing with your next post-seizure patient.

[7] List 10 causes of status epilepticus in adults (see Box 15.1 and Box 15.3)

Obviously, knowing how to treat status epilepticus is a must, but it is easy to get bogged down in the procedural nature of treating status epilepticus and forget that there are a multitude of reasons why your patient has been seizing intractably. Knowing this differential and when to consider alternative diagnoses is essential. In short, Owen and I think of the patient who is failing traditional status epilepticus treatments much like the patient with recurrent ventricular fibrillation that is unresponsive to typical ACLS interventions. We consider this differential when things are not going our way in the resus bay. And, much like the H's and T's, this list can help bring your patient back from the brink.



See both Box 15.2 and 15.3 in Rosen's 9th Edition for more a more comprehensive differential for the patient with status epilepticus.

Causes of Status Epilepticus in Adults

Metabolic Disturbances

1. Hepatic encephalopathy
2. Hypocalcemia
3. Hypoglycemia or hyperglycemia
4. Hyponatremia
5. Uremia

Infectious Processes

1. Central nervous system abscess
2. Encephalitis
3. Meningitis

Withdrawal Syndromes

1. Alcohol
2. Antiepileptic drugs
3. Baclofen
4. Barbiturates
5. Benzodiazepines

Central Nervous System Lesions

1. Acute hydrocephalus
2. Anoxic or hypoxic insult
3. Arteriovenous malformations
4. Brain metastases
5. Cerebrovascular accident
6. Eclampsia
7. Head trauma: acute and remote
8. Intracerebral hemorrhage
9. Neoplasm
10. Posterior reversible leukoencephalopathy

Intoxication

1. Bupropion
2. Camphor
3. Clozapine
4. Cyclosporine
5. Flumazenil



6. Fluoroquinolones
7. Imipenem
8. Isoniazid
9. Lead
10. Lidocaine
11. Lithium
12. MDMA
13. Metronidazole
14. Synthetic cannabinoids
15. Theophylline
16. Tricyclic antidepressants

[8] Outline management of status epilepticus.

As you may have expected, Rosen's 9th Edition has a tight little algorithm to help you manage your next patient with status epilepticus. While there are some finer points that it misses, it is generally a pretty solid decision tree that will help you on your next shift. To summarize, the table tells us to do the following:

- In the prehospital setting:
 - Assess airway, breathing, and circulation
 - Attach pulse oximeter
 - Perform EKG
 - Perform fingerstick glucose and treat if it is low
 - Place patient in lateral decubitus position to prevent aspiration
 - Give one of the following medications:
 - Midazolam 10 mg IV/IM/IN - FIRST LINE IF NO IV
 - Lorazepam 2 mg up to max of 10 mg IV - FIRST LINE IF IV
 - Diazepam 5 mg up to max of 20 mg PR - do not use re: poor absorption
- In the ED:
 - If the seizure has stopped, coordinate disposition plan with neurologist, consider NCSE in patients who have not returned to baseline neurologic functioning
 - If the seizure has not stopped
 - Give one of the following second-line therapies:
 - Phenytoin 20 mg/kg IV at max rate 50 mg/min
 - Fosphenytoin 20 PE/kg IM or IV at max rate of 150 mg/min
 - Valproic Acid 20-40 mg/kg at 3-6 mg/kg/min
 - Keppra 1000-3000 mg of 15 minutes
 - If the seizure still has not stopped with second line therapies:
 - **Intubation** and EEG recommended
 - Treat with one of the following third-line medications:
 - Pentobarbital 5 mg/kg IV at 1-5 mg/kg/hr, then 0.5-3.0 mg/kg/hr infusion as needed



- Phenobarbital 20 mg/kg IV at 50-75 mg/min
- Midazolam 0.2mg/kg IV, then 0.1-0.4 mg/kg/hr
- Propofol 2 mg/kg IV at 2-5 mg/kg/hr, then 5-10 mg/kg/hr as needed

See Figure 15.2 in Rosen’s 9th Edition for a more comprehensive description of their status epilepticus management strategy.

Just as a quick reference, we have included a box that describes your benzodiazepine doses to give when first tackling status.

See Table 15.1 in Rosen’s 9th Edition for a more comprehensive table outlining benzodiazepine doses in status epilepticus.

Medication	Adult Dose	Pediatric Dose	Comments
Diazepam	5 mg IV, up to max 20 mg or 10-20 mg PR	0.2-0.5 mg/kg IV/ET or 0.5-1.0 mg/kg PR (max 20 mg)	May repeat in 10 minutes; monitor respiratory status
Lorazepam	2 mg IV at 2 mg/min, up to max 10 mg IV	0.05-0.1 mg/kg IV (max 2 mg)	Preferred IV benzo; may repeat in 10 minutes; monitor respiratory status
Midazolam	5 mg, up to a max of 10 mg IV/IM/IN	0.2 mg/kg IV/IM/IN (max 5 mg)	Preferred IM benzodiazepine; may repeat in 10 minutes; monitor respiratory status

[9] List indications for head CT for first seizure.

Rosen’s is full of lists, we all know this. However, what some of you new to the Rosen’s Emergency Medicine game may not know is there are even MORE hidden lists. We have found a hidden list in Rosen’s that is actually quite helpful. The following is a list of indications for CT for first-time seizure. If your patient does not have any of the proceeding indications for emergent CT scan of the head, they can be dispossessed to neurology and potentially receive an outpatient CT/MRI at a later date.

Indications for Emergent CT Scan:



- New focal deficit
- Persistent altered mental status
- Fever
- Recent trauma
- Persistent headache
- History of cancer
- Anticoagulant use
- Suspicion or known history of AIDS
- Age > 40 years
- Presence of partial complex seizure

In reality, what we are looking for in these patients is serious structural lesions that are causing them to seize. CT is good at picking up most structural abnormalities, but an MRI is far better. If you patient has none of these indications to undergo emergent CT, you may be able to save them some radiation and have the neurologist go right to an MRI. You can always consider giving your friendly neighbourhood Neurologist a shout to help you navigate this decision-making process as well, just to reassure you.

At the end of the day, knowing this list is exceedingly useful. So, use it as a mantra and repeat it to yourself the next time you see the first-time seizure patient in your ED.

Wisecracks:

[1] List 5 properties of ictal events

The following is a list taken verbatim from Rosen's 9th Edition. It explains the five cardinal aspects of neurogenic seizures:

1. *Abrupt onset: History should focus on any evidence of an aura.*
2. *Brief duration. Seizures rarely last longer than 90 to 120 seconds, although bystanders may overestimate the duration. Status epilepticus is the important exception.*
3. *Alteration of consciousness. Generalized seizures are manifest by loss of consciousness; focal seizures are often accompanied by an alteration in consciousness.*
4. *Purposeless activity. Automatisms and undirected tonic-clonic movements are common in ictal events. Tonic-clonic movements are rhythmic and generally do not involve head shaking.*
5. *Postictal state. This is an acute confusion state that typically occurs with all seizure types except focal and absence. This interval represents the transition from the ictal state back to the patient's baseline mental status. It can last from minutes to hours, depending on which specific region of the brain triggered the seizure, seizure duration, age, and use of an antiepileptic drug (AED).*



[2] What medications are needed to treat seizing patients with the following:

- a. Isoniazid Toxicity
- b. TCA Toxicity
- c. Eclampsia
- d. Hypoglycemia
- e. Hyponatremia

Recall the treatments for these seizure-provoking disorders from Box 15.3 in Rosen's 9th Edition.

1. **Toxic ingestion**
 - i. Isoniazid [INH] - Pyridoxine
 1. According to UTD - give an equal amount to isoniazid ingested, up to 5g IV
 - ii. Tricyclic antidepressants - benzos, NO DILANTIN
2. **Eclampsia** - MgSO₄
 - i. 4g bolus over 30 min and then 1g per hour (NB - need 20g bag from pharmacy)
3. **Hypoglycemia** - glucose (± octreotide, glucose infusions in your sulfonylurea ODs)
 - i. For adults - ½-1 amp of D50W repeated as needed
 - ii. For pediatrics - use rule of 50's to best steward glucose bolusing (No D50!)
4. **Hyponatremia** - hypertonic saline - 3% NaCl
 - i. 100 cc of 3% NaCl IV over 10 min
 - ii. Consider the rule of 3's - 3cc/kg of 3% NaCl IV over 30 minutes

[3] Name 3 key metabolic abnormalities that can cause seizures

Quick snapper here. The top 6 metabolic abnormalities causing seizures are:

1. Hyponatremia
2. Hypocalcemia
3. Hypoglycemia
4. Hyperglycemia
5. Uremia
6. Hyperammonemia



[4] Name 3 common seizure provokers that can worsen pre-existing seizure disorders

Knowing this list can help you discern what, if anything, is causing your patient's pre-existing seizure disorder to worsen. Rosen's 9th Edition lists several seizure provokers that can worsen a patient's frequency or severity of seizures. These are:

- Sleep deprivation
- Poor medication compliance
- New medication use
 - Think of medications that lower seizure threshold like antipsychotics
- New illness or infections

[5] What percentage of patients with convulsive status epilepticus will develop non-convulsive status epilepticus?

As stated previously, up to 15% of patients who are successfully treated for convulsive status epilepticus remain in non-convulsive status afterwards. As stated above, the management strategies for these patients change, so keep NCSE on the differential after seizures have abated.