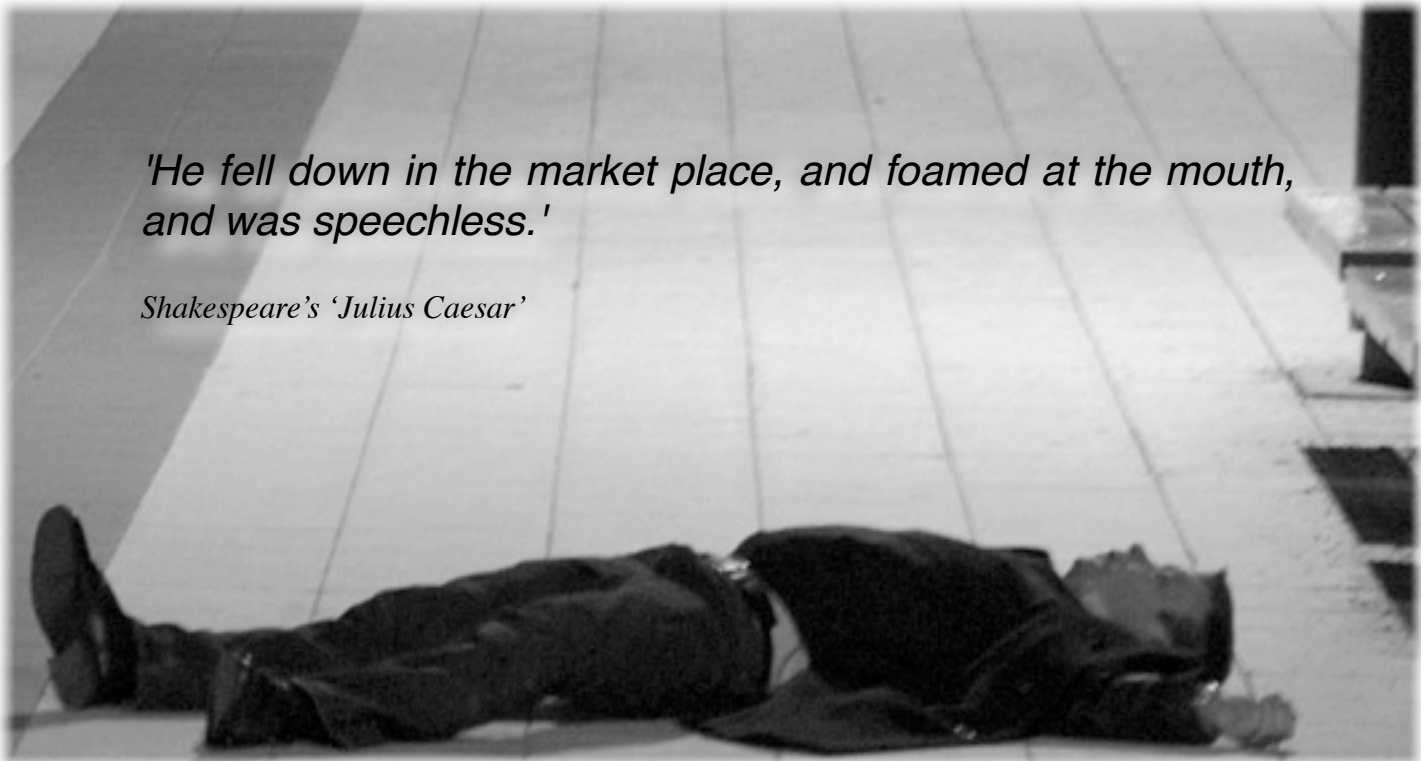


Reading

CEPCP

Professional Development: Seizures



*'He fell down in the market place, and foamed at the mouth,
and was speechless.'*

Shakespeare's 'Julius Caesar'

The Ravages of a Seizure

Tim had been in his seizure for almost twelve minutes by the time the EMS crew arrived. During those minutes his blood had turned into a dangerous toxic river, starved of oxygen and teeming with acids. The acids were coming from two sources: the cells who had been forced to turn to anaerobic metabolism because of the lack of oxygen, and the carbon dioxide that was being retained from Tim's failure to breathe during his seizure. It was a cruel, ironic wrinkle in human physiology, that during a seizure, when demands couldn't be higher due to the forceful contractions of muscles, oxygen supply was abruptly cut off by apnea.

On this particularly sunny afternoon it was causing a direct threat to Tim's life; the acids irritated the heart, threatening to send it into a disorganized, non perfusing rhythm. Already it was beating dangerously fast and becoming irritable and occasionally the ventricles would trigger independently, causing premature ventricular contractions. To make matters worse, the contents of ruptured skeletal muscle cells were also traveling through the blood stream. These large, awkward proteins (myoglobins) threatened to get caught in the delicate, fine capillary system embedded

within Tim's kidneys which would cause them to shut down. The Paramedics were all too aware of these imminent dangers and started taking immediate steps to interrupt this dangerous downward spiral.

The other people in the mall had been startled when they heard Tim's initial shout. It sounded like he was purposefully shouting out in pain or fear. In reality what happened was a seizure-induced contraction of the diaphragm and vocal cords, forcing the air out of a partially closed vocal cords. The events that followed are similar to those captured eloquently in the song, 'Seizure' by the death metal band, 'Malevolent Creation';

*Convulse, twisted, falling to the ground
Saliva flowing free, tasting, choking*

to the reaction of onlookers

*Cursed by this disease
Those around you panic
Onlookers start to freeze.*

Luckily one onlooker didn't freeze but instead grabbed her cell phone and dialed 9-1-1.

Introduction

It is only in the last two hundred years or so that seizures have been recognized as a disorder of the brain. Before this time the general consensus was that supernatural powers caused someone to have a seizure. There are even biblical accounts of what might now be interpreted as seizure activity;

Mark 9; 16-18

¹⁶He asked them, "What are you arguing about with them?" ¹⁷Someone from the crowd answered him, "Teacher, I brought you my

son; he has a spirit that makes him unable to speak; ¹⁸and whenever it seizes him, it dashes him down; and he foams and grinds his teeth and becomes rigid; and I asked your disciples to cast it out, but they could not do so."

We have developed a better understanding of seizures since those days, but much remains unknown. Seizure disorders remain a confusing diagnostic challenge as they often coexists with psychiatric diseases as well as alcohol and drug abuse. It can be very challenging as a Paramedic to sort out the 'real' seizures from the 'fake'; the epileptics, from the withdrawal seizures; or even seizures from

syncope. Adding to this challenge is the fact that by the time EMS arrives on the scene, the seizure has usually stopped, leaving only the after-effects and the bystander accounts of the episode as clues to what happened.

This review will hopefully add to your knowledge of seizures and help you ask some rather specific questions from bystanders to help you make the correct working assessment and be able to relay crucial information to the receiving staff in order to help them ultimately make the correct diagnosis. We will also review what we can do if a patient is seizing while in our care, whether we are working in a PCP or an ACP capacity.

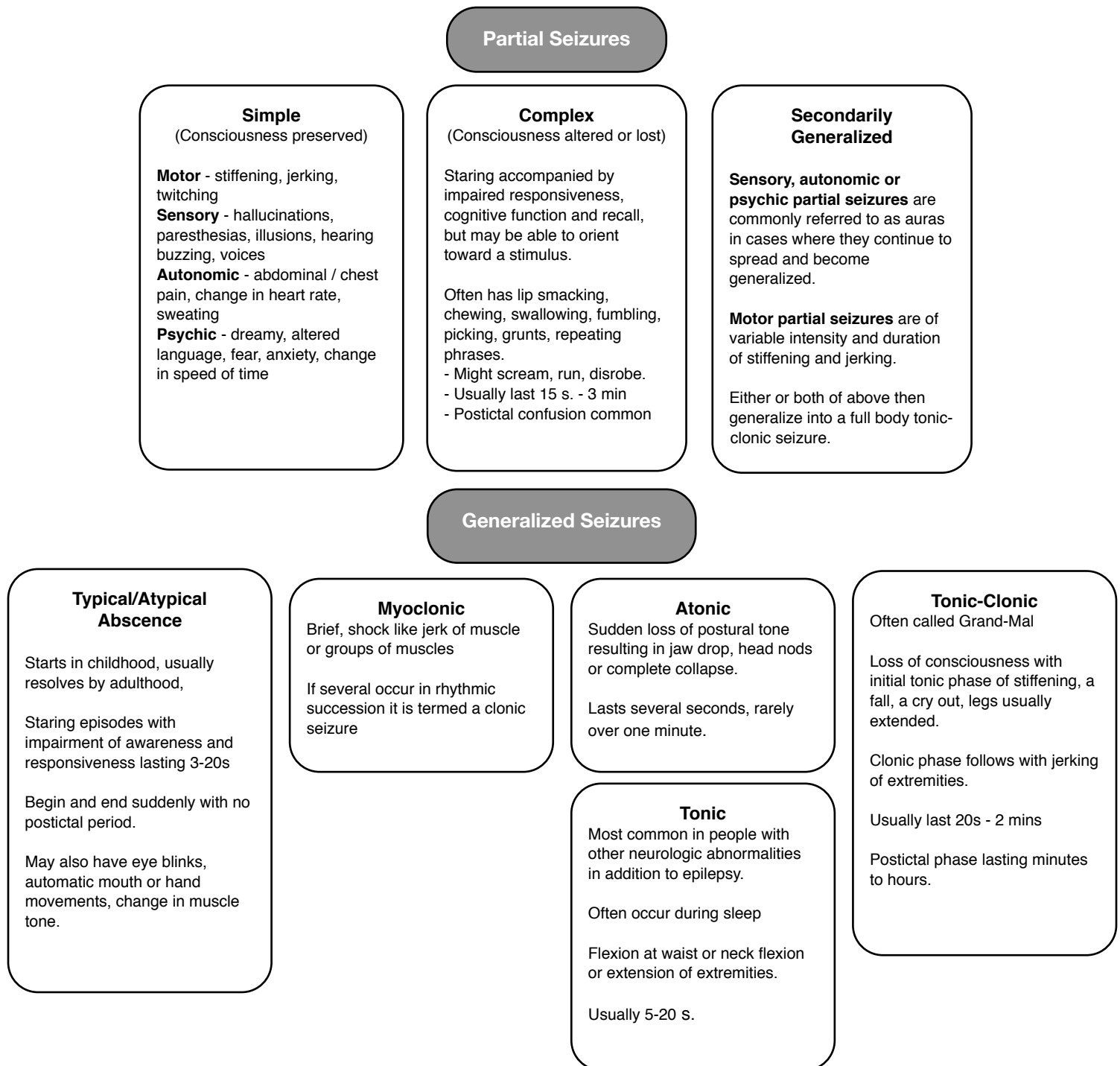
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Classifications

Seizures can be divided into two main categories, partial or generalized, depending on if the seizure is occurring in a specific part of the brain or throughout. There are also several subcategories in the generalized and

partial categories. Here is a summary and brief descriptions of these categories. We will not get too caught up in these various classifications as it is of limited use for us as paramedics. Working instead with the principle that; 'common things are common', we will instead focus on the types of seizures we commonly come across on the road.



Grand-Mal Seizures

The full body, grand-mal, tonic-clonic seizure, is the type of seizure Paramedics are most likely to encounter. It can have a variety of causes such as; alcohol abuse / withdrawal, hypoxia, febrile illness, head injury and many more. It is important for Paramedics to have a clear understanding of what a grand-mal seizure typically looks like so that when a patient has a seizure, or a bystander is describing a seizure, the Paramedic can clue in to discrepancies that may indicate that the event is, or was not, a seizure. So, let's slowly walk through a typical grand-mal seizure.



The **tonic phase** is where the patient usually loses consciousness and the entire body stiffens. If the patient is in a standing position during the tonic phase, they will fall to the floor. A cry is often heard as air is forced out of partially contracted vocal cords from the contracting diaphragm, and commonly the patient is apneic during this phase. The tonic phase usually lasts 5-20 seconds.



The **clonic phase** immediately follows, where the initial tonic phase gives way to clonic convulsive movements. During the clonic stage there are alternating stiffening and loosening of muscles, causing violent jerking of the extremities. Each spasm is accompanied by pupillary contraction and dilation. Voiding usually occurs at the end of the clonic phase. The patient continues to be apneic during this stage which normally lasts about 30 seconds.

After the seizure stops, the **postictal** phase follows, during which the patient may be confused, combative or unresponsive. The patient may also exhibit strange behaviour during this phase and pick at things or undress. There is usually strong evidence of an autonomic response, exhibiting as tachycardia, diaphoresis and paleness.

Febrile seizures

Febrile seizures are seizures that occur in an infant or young child (6 months to 5 years) in conjunction with a fever, or recent fever and without a history of afebrile seizures (Warden et al. 2003).

There are two classifications of febrile seizures depending on how severe they are. A **simple febrile seizure** lasts less than 15 minutes, only occurs once in a 24 hour period and has a generalized onset (both arms, legs etc.) A **complex febrile seizure** lasts greater than 15 minutes and/or recurs in a 24 hour period and/or has focal features (Warden et al. 2003). Differentiating between these two types of febrile seizures has some important implications. If, for example, a patient has a single, simple febrile seizure; the risk of developing epilepsy is only slightly higher than the general population (Warden et al. 2003). However, if the patient has a complex febrile seizure the risk of developing epilepsy is 30-50 times greater than the general population (Warden et al. 2003). More importantly, the occurrence of a complex febrile seizure is strongly linked to meningitis. One study found that nearly 1 in 5

children who had a complex febrile seizure had meningitis (Chin et al. 2005).

Detecting meningitis as a possible cause of a febrile seizure is clearly an important task. Besides the presence of a complex febrile seizure, other assessment findings to look for are petechial rash (small, flat, red non blanching spots), nuchal rigidity (stiff, sore neck) or persistent lethargy or coma after the seizure (Fetveit 2007).

The good news is that a febrile seizure is usually a relatively benign event that will not require any invasive interventions. Having said that, it is also important not to get lulled into a false sense of security and shrug these seizures off as, 'no big deal.' Always consider other causes, such as trauma, poisonings, meningitis, hypoglycemia, etc. Do a thorough assessment, keep the monitor on and don't be shy with the oxygen.

A common question is whether the patient should be cooled down or not. There is no evidence that cooling measures are useful to decrease the chance of a recurrence of seizure activity (Warden et al. 2003). Also, there is no evidence that treating the fever

- Febrile seizures occur in 2% to 5% of all children between the ages of 6 months and 5 years.
- These seizures might appear frightening to observers but are generally harmless.
- Simple febrile seizures often occur in the first 24 hours of the febrile illness and only occur once. If the seizure recurs, your child should be reevaluated.
- A febrile seizure might be manifested by body stiffening; twitching of the face, arms and legs, or both; eye rolling; jerking of the arms and legs; staring; or loss of consciousness. Febrile seizures generally last <1 minute but can last up to 15 minutes.
- Your child might appear not to be breathing, and the skin color might become darker. If so, call 911 or emergency personnel and lay the child on the floor on his or her back and DO NOT place your fingers in the child's mouth.
- Febrile seizures do not cause brain damage or paralysis.
- A child who has febrile seizures has only a slightly increased risk of having a seizure disorder compared with that of a child who has never had a febrile seizure.
- Febrile seizures tend to run in families.
- Febrile seizures can recur with subsequent febrile illnesses. Medicines are generally not given to prevent simple febrile seizures.
- Use of medicines such as acetaminophen or ibuprofen for fevers have not been shown to prevent febrile seizures.



Figure 1

Warden et al. 2003

with medications decreases the chances of the child having a seizure (Warden et al. 2003).

Regardless, we as pre-hospital providers would not administer such medications, but it might help in comforting a parent who feels they did not treat the fever aggressively enough.

Often the main task will be to calm upset parents. Watching a child have a febrile seizure is very upsetting. Often the parents think the child is choking and that is indeed how the call to 9-1-1 is often received. Calming the parents while still insisting on bringing the patient to the hospital are your main goals. Figure 1 lists some useful facts that can be used to educate upset parents

Fake Seizures?

Before we get too carried away spotting 'fake' seizures, we need to clarify what that means. There is a category of seizures called 'Psychogenic Non-Epileptic Seizures' (PNES). PNES are **not** the same as fake seizures, but fake seizures fall into the PNES category.

PNES by definition are, '*...episodes of altered movement, sensation, or experience*

similar to those due to epilepsy but caused by a psychogenic process and not associated with abnormal electrical discharges in the brain.' (Kumar 2004). It is important to note that just because PNES are psychogenic they are not necessarily 'faked'. Most often, the episodes are very real to the patient and are thus not a deliberate attempt to trick caregivers (Panayiotopoulos 2005).

However, it is clearly valuable to be able to determine when an episode is/was a PNES so that inappropriate treatment is not administered, either in the pre-hospital setting or later in the follow up care. Again, do not be mistaken, PNES patients do need treatment, just not anti-epileptic treatment.

So what are the distinguishing features that can help us tell when a seizure is of the psychogenic kind? As usual there is not one 100% full proof way of telling, but the list below outlines some features that indicate PNES.

Features of PNES

- Eyes closed during seizure (Syed et al. 2008; Korff et al. 2005; Chung et al. 2006)
- No oral lacerations (Oliva et al. 2008)
- No incontinence (Oliva et al. 2008)
- Weeping during seizure (Walczak & Bogolioubov 1996)
- Precipitated by stress (Tojek et al. 2000)
- Irregular movements, switching from one extremity to the other, side to side head movement, pelvic thrusting, abrupt cessation (Kotagal 2002)
- Usually occurs when awake, in the presence of others (Tojek et al. 2000).



Alcohol Related Seizures

Seizures are more prevalent in patients who abuse alcohol than in the general population. Most of the seizures related to alcohol abuse are caused by alcohol withdrawal. When a habitual drinker stops drinking there is an activation of the sympathetic nervous system; the heart rate and blood pressure increases and the body temperature increases causing sweats, chills and goosebumps. The term, 'quitting cold turkey' is a reference to the appearance of the skin of a person in withdrawal, with the goose bumps and cold sweats appearing like the skin of a plucked turkey.

Along with the sympathetic response, the withdrawal causes brain hyperexcitability that can trigger a seizure in an individual who is seizure prone. Additionally, the hyperexcitability can, over time, lead to seizures even in individuals without any such predisposition, a phenomena called the 'kindling

effect' (Rathlev et al. 2000). Essentially it is as if the brain is a glowing ember. Every time the body goes through withdrawal, the ember is blown on, causing it to glow more intensely. With enough blowing the flames flare to life and the patient suffers a full blown seizure.

Because a person without a predisposition to seizures require several withdrawal episodes before a seizure manifests, those types of seizures are unlikely in patients who have abused alcohol less than 10 years (Rathlev et al.). Usually withdrawal seizures are

thus generally seen in patients in their middle age or older.

Be careful not to assume withdrawal seizure in all alcoholics who suffer a seizure though. One study found that half of patients who presented to the ER with 'alcohol related seizures' were not suffering withdrawal seizures ,but had a variety of other causes for their seizures (Rathlev et al. 2002). Head traumas, CVAs, toxicity and intracranial lesions were identified as causes in some of those patients (Rathlev et al. 2002).

A typical withdrawal seizure is tonic-clonic with a short postictal period and will occur within 3 days of stopping drinking (Hillbom 1980). If the incident history doesn't fit the typical picture, don't assume withdrawal and dig deep for other causes.

It is important that patients who have suffered a withdrawal seizure are monitored carefully and transported to the ER. There is a definite risk that those patients will have another seizure within the following six hours (Rathlev et al. 2000). Treating the patient with midazolam or diazepam may help

prevent a recurrence seizure. Consider patching for orders if your patient is exhibiting strong withdrawal signs and symptoms to prevent a seizure, or if the patient has had a withdrawal seizure in order to prevent a recurrence.



Seizures and Injuries

Injuries resulting from seizures are relatively common and can result from a variety of causes. First, if a person is in a standing position when the seizure occurs they usually fall down without any way of breaking their fall. Clearly such a fall can lead to a variety of injuries. Then, once the seizure occurs, the forces from opposing muscle groups violently contracting can lead to fractures.

Thoracic and/or lumbar compression fractures are a well recognized complication of seizures as well as femur fractures and hip or shoulder dislocations (Kalaci et al. 2008; Vanheer et al. 2008; Mehlhorn et al. 2007). For some patients, the discovery of a seizure disorder is made when they wake up with back pain from a vertebral fracture, caused by an overnight seizure that they didn't even realize they had (Aboukasm & Smith 1997)! To complicate matters, many anti-seizure medications cause lowered bone density, putting those patients at additional risk (Kulak et al. 2004).

Head injuries have also been reported following seizures, most of them minor lacerations. However, skull fractures and intracranial bleeding have occurred as well

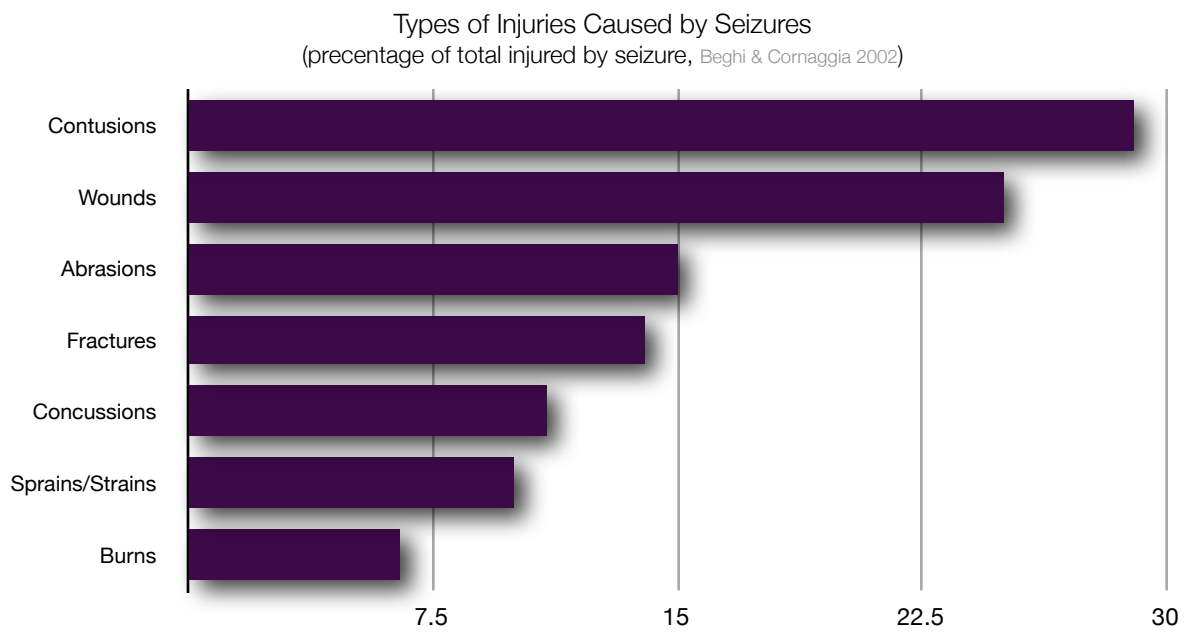
(Russell-Jones & Shorvon 1989).

The take home message for paramedics is to be very vigilant and perform a thorough hands-on assessment of post-seizure patients in order to rule out any occult injuries. If the patient is complaining of neck or back pain or has point tenderness, then take spinal precautions.

More overt injuries also occur due to seizures. More than one out of ten patients with epilepsy report having suffered a seizure while bathing or swimming and studies have shown that children with epilepsy are over seven times more likely to suffer drowning or near drowning than children without epilepsy (Wirrell 2006).



C-1 Fracture secondary to epileptic seizure. (Torreggiani 2001)



Things That go Crunch in the Night...

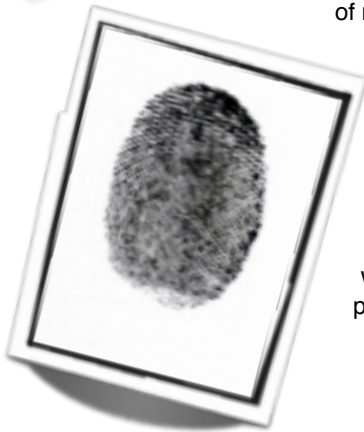


A seemingly routine case of death-during-the-night caught forensics investigators by surprise in Dublin, Ireland in 1999. A 77 year old man had gone to bed in the evening, as usual. Family members would later say that he seemed totally normal that evening.

The next morning they found the gentleman dead, lying peacefully in his bed, having passed away during the night. Investigators were shocked and concerned to find that the cause of death pointed to severe trauma.

The deceased's pelvis was broken in two places, both hips where dislocated, there was over one liter of blood in the pelvis and there was evidence of multiple pulmonary embolisms from fat and bone marrow. The devastating injuries were a mystery, especially since family members denied having heard any unusual noises through the night.

So how can an elderly man go to bed healthy and the next morning show evidence of such violent trauma without anyone having heard any kind of commotion? Through a detailed investigation of the patient's past medical records it was determined that he had a distant history of seizures. Consequently the injuries were ultimately attributed to a violent seizure that happened while the patient was asleep.



Status Epilepticus

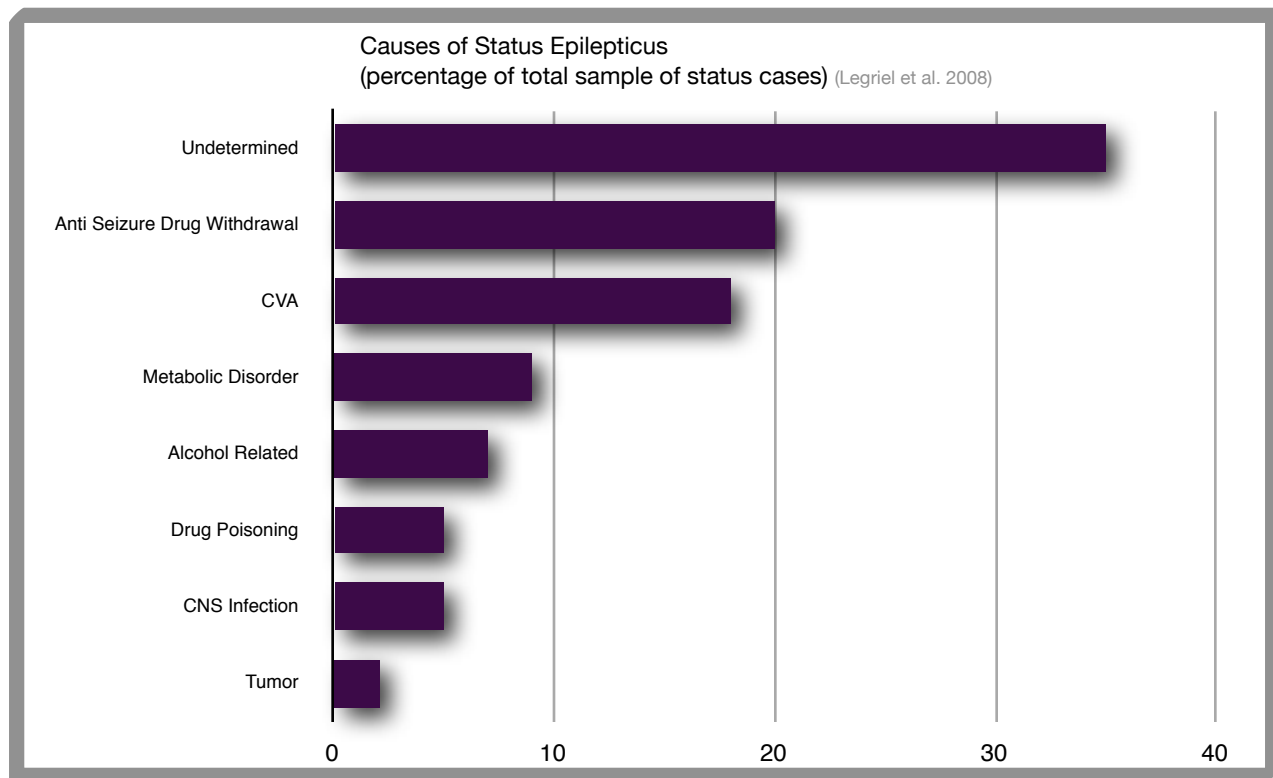
Status epilepticus is defined as a seizure that lasts longer than 30 minutes, or two or more seizures without full recovery in between (Chin et al. 2008). The seizure can be of any kind, although the status grand-mal seizure is the most dangerous and easiest to recognize. As the introduction to this reading alluded to, status epilepticus is a life threatening condition unless it can be stopped. One in five patients who seize for longer than thirty minutes will not survive (Chen & Wasterlain 2006). Not only do the seizures literally kill brain cells but other organs fail when exposed to the severe acidosis and hypoxia present during a prolonged seizure (Legriel et al. 2008).

The best chance of stopping a seizure is within the first 5-10 min's of the onset. The longer it continues the harder it becomes to stop. Animal studies have shown that after

15-30 minutes a seizure becomes resistant to first line drugs, and anesthesia may become necessary to save the patient's life (Chen & Wasterlain 2006). An important take-home message lies within the knowledge that the longer the seizure lasts the harder it will be to stop, and that pre-hospital pharmacology might be rendered useless; focus on transport! During a prolonged seizure the motor activity tends to become less violent with time (Chen & Wasterlain 2006). The decreased motor activity does not necessarily mean that the patient is improving though, and the implications of the patient having a prolonged seizure remain.

Considering status epilepticus is the most common neurological emergency in childhood and is an area where pre-hospital care has proven to make a big difference in outcomes (Chin et al. 2008), it is imperative that paramedics remain well trained in the management of these challenging patients. So what are the

underlying causes of status epilepticus? Here is a chart showing the various causes;

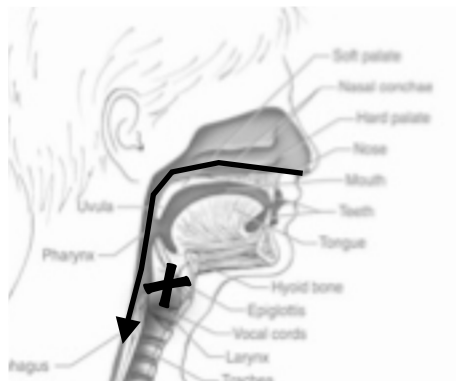


Airway Management

Managing the airway in a patient who is in an active seizure might be very challenging. Their mouth will be in trismus (clenched shut). Also, there is often oral trauma with bleeding adding to the challenge. In a prolonged seizure the patient may need to be ventilated unless they

are spontaneously breathing. Keep in mind, if the patient's diaphragm, intercostal muscles and/or vocal cords are seizing it might be very difficult to ventilate their lungs. Forcing air into such a patient is likely to cause gastric distention with potentially disastrous consequences.

In short, there may not be any great options. Consider a nasopharyngeal airway for patients who are older than five years old. If required, attempt some gentle ventilations preferably with some cricoid pressure applied to avoid gastric distention. Suction can be performed through the nasal airway with a french catheter. Never get tempted to force anything into the airway.



Why Oxygen for the Postictal Patient?

A topic of discussion that comes up fairly often is whether high concentration oxygen is really required for a postictal patient whose oxygen saturation is adequate. The BLS Patient Care Standards clearly indicate it should be given, but why? What benefit can oxygen have on a neurological issue that has resolved by the time paramedics arrive? If the oxygen saturation is 100% we can't really bump it up any higher, Can we?

In addition to flooding the plasma with fresh oxygen to restore oxygen supplies it basically comes down to being prepared for another seizure. Most tonic-clonic seizures render the patient apneic. Luckily there is about two liters of air in the lungs at the end of a normal expiration. All the oxygen in that two liters can be used up before the 'tank is empty'. So, how much actual oxygen is in there? Well, we know atmospheric air has 21% oxygen in it (we will round it to 20% to make the math easier). Simply stated the amount of oxygen available is 20% of the two liters.

$$20\% \text{ of } 2,000 \text{ ml} = 400 \text{ ml}$$

There we have it. If the patient stops breathing, due to a seizure for example, there is 400 ml of oxygen available before the patient runs out. How long will that last?

Physiology texts estimate 3-4 ml/kg/min of oxygen consumption for normal resting metabolism in adults. A seizure would increase the demands quite a bit but we will stick with 4 ml/kg/min for now.

$$4 \text{ ml} \times 70 \text{ kg} = 280 \text{ ml/min.}$$

So we can quickly see that the 400 ml of oxygen in the lungs would run out in less than two minutes!

Fortunately, we have a way of topping up the tank before the seizure. Lets say that we

achieve an amazing seal with the non-rebreather mask and actually get 100% oxygen delivered to our postictal patient. After about 5 breaths the 100% oxygen in the tidal volume has 'refreshed' the air in the lungs and the 2 liters of air in the lungs is now comprised of 100% oxygen. So, when our patient stops breathing and starts seizing, we now have;

$$100\% \text{ of } 2,000 \text{ ml} = 2,000 \text{ ml} \text{ of oxygen available before running out.}$$

Using our 280 ml/min consumption rate, we now have a little over 7 minutes of oxygen available. I like those numbers much better!

$$\text{NRB} = 7 \text{ min's}$$

$$\text{Room air} = < 2 \text{ min's}$$



Pharmacology of Seizure Management



Pre-hospital management of seizures have been proven to be safe, effective and beneficial to patients (Chin et al. 2008). Thanks to continued research and improvements to our EMS systems, we now have a safe and non-invasive method of delivering a cutting edge benzodiazepine to patients in active seizures.

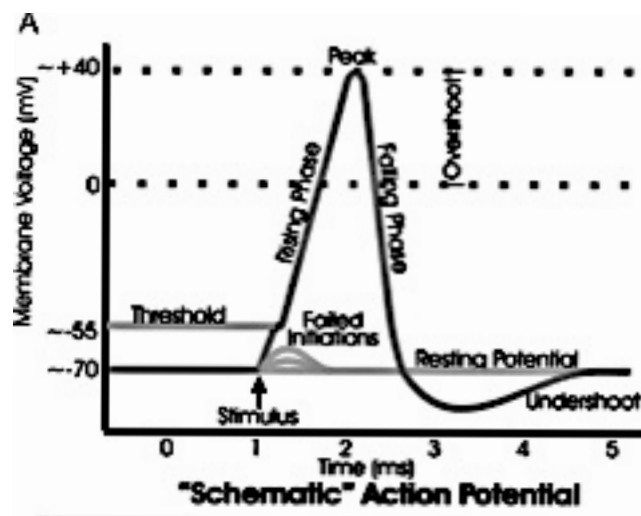
Diazepam and midazolam are both benzodiazepines that work by enhancing chloride's transportation into the cells. Because chloride is a negatively charged ion, it lowers the voltage within the cell making the resting potential 'more negative'. The action potential depicted shows a resting potential at -70 mV: a few 'failed initiations' occurred as the stimulus didn't push the resting potential past the threshold

potential. If the initiation pushes the voltage past the threshold, a depolarization occurs. It is easy to see that lowering the resting potential by stuffing the cell with negatively charged ions will lower the chance that a given initiation will push it past the threshold.

Since midazolam is shorter acting, has a faster onset and can be given intranasally, intramuscularly, as well as intravenously it has largely replaced diazepam as the drug of choice to treat seizure patients. In studies comparing diazepam to midazolam, midazolam is as effective as diazepam in controlling seizures and has a similar rate of side effects (Rainbow et al. 2002; Warden & Frederick 2006; Appleton et al. 2009). Regardless of which drug is chosen the seizure will stop within five minutes in about half the patients who are treated

(Rainbow et al. 2002; Appleton et al. 2009).

However, an alarmingly high rate of patients will require airway interventions after treatment. In one study of 107 patients treated with either midazolam or diazepam, more than twenty percent became apneic after drug administration and most of those patients required intubation (Rainbow et al. 2002). The risk of respiratory depression after midazolam administration has prompted a 'black box warning' by the FDA prompting providers to ensure that airway devices and a BVM is readily available whenever midazolam is administered. The warning also prompts providers to ensure continuous cardiac and pulse oximetry monitoring throughout the recovery period (Roche 2000).



For up-to-date doses, refer to your current medical directives. As of this printing the dose is 0.1 mg/Kg to a maximum of 5 mg IV. If you are administering the midazolam via the IN or IM route the dose is doubled to 0.2 mg/Kg to a maximum of 10 mg.

The midazolam comes provided in 5 mg/ml in a 2 ml vial. When being delivered IV, the drug needs to be mixed with saline to make a 1 mg/ml solution. The safest thing to do is draw up 4 ml normal saline in a 10 cc syringe, then draw up 1 ml of midazolam. That will provide 5 mg in 5 ml which is the maximum dose. As long as the patient weighs over 50 kg, that will also be the correct dose.

The IM dose requires a bit more math as it can't be diluted. There is a shortcut though for those 4 am calls. Simply take the patient's weight, double it, then double it again, move the decimal and voila...you have the volume you need to give.

Let's try it; a patient weighs 20 kg, double it and we have 40, double it again and we have 80, move the decimal two steps left and we end up with 0.8 ml...go ahead and check it if you want! Of course if the patient weighs over 50 kg just give the full vial (2 ml). Let's double check; $50 \text{ kg} \times 2 = 100$, $100 \times 2 = 200$, decimal moved twice...2.0 ml! To figure out the IN dose just add 0.12 ml to the IM volume and you are all set.

The various routes of Midazolam administration have their own unique benefits and drawbacks.



<p>The intravenous (IV) route has a very fast onset (offset by the time required to initiate IV access), but also carries the biggest risk of respiratory depression and other side effects (Roche 2000).</p> <p>There is also the inherent difficulty in establishing an IV on a patient who is seizing to consider.</p> <p>When midazolam is given IV, it is important to dilute the drug to a 1 mg/ml concentration and administer the drug slowly over 2-3 minutes as this will help prevent respiratory depression (Roche 2000).</p>	<p>The intranasal (IN) route is a little bit slower than the IV route, but unless an IV is already established when the patient starts to seize, the time required to establish an IV will outweigh the increased time of onset (Mittal et al. 2006).</p> <p>In order to ensure that the drug is atomized over a large surface area, divide the dose between both nares.</p> <p>Some pre-hospital providers feel that the intranasal route inhibits airway management and question how much drug is really absorbed if the patient has a stuffy nose or ends up reflexively snorting the drug back out. When researchers studied the switch from diazepam to midazolam IN or IM in New South Wales, Australia they soon found that paramedics preferred the IM route because of its ease of use and effectiveness (Rainbow et al. 2002).</p>	<p>When administering midazolam via the IM route, guidelines simply state to deliver the drug deep into a large muscle. The vastus lateralis is the best option using a 1.5" needle for anyone larger than a toddler.</p>



Back to the Mall

Lets bring our minds back to the mall and Tim, seizing for over twelve minutes now. As we approach him, we see that he is still in a seizure. Based on the response time alone, we know that he has been seizing for a dangerously long time. The cyanosis serves as a visual alarm bell, reminding us that he is dangerously hypoxic. We quickly recall that he ran out of oxygen within the first couple of minutes of his seizure and his cells have now turned to inefficient, dirty, anaerobic metabolism. The blood trickling from the side of his mouth tells us that he is most likely seizing 'for real' and that managing his airway might be a challenge. Your partner calmly but efficiently supports the patient's head and starts evaluating what can be done with the airway, and ventilation.

As an ACP, (if you are a PCP you can still play this mind game, it will be very valuable for an ACP partner to be able to double check the dose with you) you start thinking about the most efficient way to pharmacologically stop Tim's seizure. You know this is a limited time offer, if you waste time, Tim might not respond to the drug at all. You decide IM midazolam is the way to go. You know it will take a bit longer to work as compared to the IV route but you also know that establishing an IV will easily eat

up that time. You open your drug bag, grab a 3 cc syringe, fit it with a blunt needle and draw up the full 2 ml of midazolam (Tim clearly weighs over 50 kg). Then you exchange the blunt needle for a 1.5" long needle. A quick cut at the bottom of Tim's pants and then a firm rip exposes the vastus lateralis in his thigh. A quick swab and then effortlessly the needle penetrates deep into the muscle. You withdraw the plunger on the syringe and to your relief, it won't move, no blood flows back into the syringe. You deliver the drug slowly, deep into the muscle mass where it will be absorbed and eventually make its way to Tim's brain and hopefully move the resting membrane potential further from the threshold potential, thus causing more 'failed initiations' (see figure p. 13).

In the meantime, your partner has been busy too. She immediately noticed that Tim was completely apneic and knew she needed to act quickly. She lubed up a #30 nasal airway, which easily passed through the right nare. Next, she hooked up a BVM to oxygen and tried to ventilate. She noticed there were times in Tim's contraction / relaxation cycles where she could squeeze the BVM with little resistance. She fought the urge to force air in as she knew it would take the path of least resistance and go down the esophagus. As soon as the fire department arrived she instructed a fire fighter to firmly press down on the cricoid

cartilage, essentially occluding the esophagus thus preventing air from going down that way, as well as gastric contents from coming up. Soon Tim's colour was improving.

With all the priority items out of the way we can now take a deep breath and think about what to do next. The monitor is attached, a blood sugar is obtained and vital signs are measured. Our thoughts turn to what might be causing Tim's seizure. There is a mnemonic that provides a mental check list of causes, VITAMINS;

Vascular - intracranial/subarachnoid hemorrhage, infarct, AV malformation

Infectious - meningitis, encephalitis, abscess

Traumatic - head injury, could be old

Autoimmune - lupus, CNS, vasculitis, MS

Metabolic/Toxic - substrate alterations, ETOH, drugs

Idiopathic/Iatrogenic - hereditary, medications

Neoplastic - tumors

Structural / **pS**ychiatric - congenital defects

However, a medic alert provides the answer as it lists 'epilepsy' as a medical condition. It makes sense as you recall that anti-seizure-drug withdrawal is the second leading cause of status epilepticus. To your relief, the seizure subsides as you are packaging Tim on the scoop stretcher. His respiratory effort improves so much that the BVM can be replaced with a non-rebreather. You continue to watch the breathing with a healthy level of paranoia though, knowing that the midazolam could suppress it again. In less than fifteen minutes you depart the scene, another life saved...literally!

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