Reading

Professional Development: Syncope

Syncope

"The only difference between syncope and sudden death is that in one you wake up"
G.L. Engel

The Dinner Mystery

It was a special day for Mary. Her and John had been married for 40 years and were in an upscale restaurant with family and friends celebrating. Spirits were high as the guests reminisced about times gone by. While most of the guests were finishing their main course, a second bottle of wine arrived. Nobody was aware that things were about to go horribly wrong. The night would indeed be one to remember but for all the wrong reasons. Mary was the first to sense that something was awry; she felt slightly dizzy. She tried to take a few deep breaths to shake the feeling. Suddenly she felt a hot, prickly sensation on her skin. Her vision was becoming narrow and tunnel like.

"Are you all right?" John had during their 25 years together, developed an intuitive ability to sense when his wife was not feeling well. To Mary, his voice seemed distant, she didn't answer.

Suddenly it became obvious to even the most casual observer that Mary was in trouble, she was very pale, she was trying to speak but nothing came out. In rapid order things went from bad to worse.

As the horrified dinner guests watched on, Mary's eyes "rolled back" in her head, her arms seemed to jerk slightly, and there was a loud forceful snoring sound that got the attention of other patrons in the restaurant. For what seemed like forever but was realistically about a minute, Mary was completely unresponsive and slumped awkwardly in her seat in the dimly lit restaurant. Guests and family alike feared the worst.

"Oh my god, she is choking!" someone at another table yelled.

"Is she having a stroke?" Mary's younger sister asked to no one in particular.

"Quickly, call an Ambulance!" Mary's son yelled to a nearby waiter

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"8644 call".

You look at the clock on the radio in the dash, 17:15, this will probably be the last call of the shift.

"8644 at King and Main" you respond.

"8644 I have a call for you. It's a code four at 9877 Queen Av, the Aquawit restaurant. It's for a 67year old female, unresponsive"

"10-4, 10-8"

As you begin making your way through the sluggish afternoon traffic, carefully avoiding cell-phone users and cab drivers, you get a further update;

"8644 the patient is now conscious, breathing OK. Pale and sweaty"

"Sounds like a syncopal episode." your partner says while logging the information on the shift log.

With that you begin to ponder the mysteries of syncope. What is the actual mechanism that causes the loss of consciousness? What is the underlying problem? What are the possible serious causes versus the benign? How should the assessment and treatment proceed to ensure that nothing important is missed?

Welcome to a journey into the pathology of syncope. We will explore the various types and causes of syncope. We will find ways that we can look at the external clues, clinical findings, that will give us an internal view of what is really happening deep within the machinery of life.

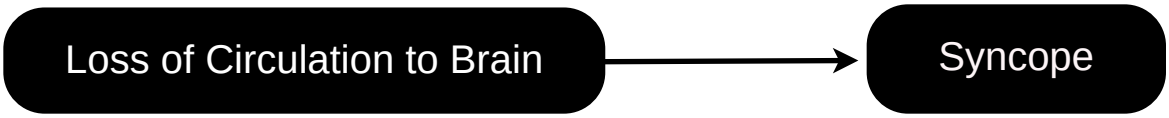
Like searching the scene of a crime, we will discover clues that will allow us to establish what happened before we arrived. Like our well known friends on CSI, let's take our flashlights and start our investigation.

What Is Syncope?

Syncope is derived from the Greek word *synkoptein*, which means "to cut short". In the BLS Patient Care Standards, it is defined as; "a transient complete loss of consciousness due to cerebral ischemia, followed by recovery to previous mental status" (MOH 1999).

Often the term "pre-syncopal" is used to describe someone who feels or appears as if they are going to pass-out. Let's begin by looking at what actually causes the loss of consciousness, and then we can begin to unravel the various events that may lead up to that point.

You may recall that the reticular activating system (RAS), located in the brain stem is responsible for keeping us awake. So it stands to reason that a loss of oxygen to the RAS will cause a loss of consciousness. In syncope, the loss of oxygen is due to a loss of blood flow (perfusion) to the RAS and/or the rest of the brain. Yes, believe it or not it's that simple! Lets review;



The Underlying Problem

Now for the interesting part; what causes the loss of blood flow to the brain? This is where the various *types* of syncope come in. Again, tracing the underlaying cause backwards will let us categorize the causes in a relatively simple fashion.

Basically there are three factors that determine blood circulation to the brain (as well as anywhere else in the body); blood volume, vessel diameter, and heart function (rate and contractility).

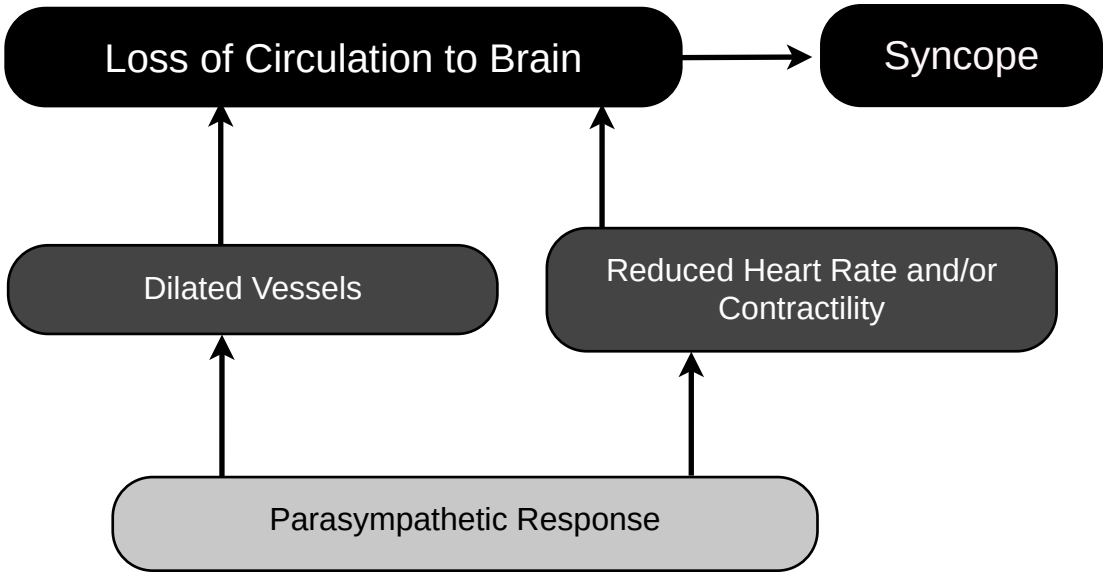
Blood volume, on its own, rarely causes isolated syncope. If someone looses enough blood to impair circulation to their brainstem, they are in a late stage of shock and will not really be considered a 'syncope patient'. Often a slight drop in volume can *trigger* a syncopal episode but it is not because there is not enough fluid in the body, it is due to the reflex that the drop in volume triggers. We will discuss that in more detail later. Most commonly, dilation of blood vessels and/or a drop in heart rate will be the direct cause of the lack of blood flow to the brain that causes syncope.

Recall that blood vessel diameter is closely regulated minute by minute by two opposing forces; the sympathetic and the parasympathetic nervous systems.

The sympathetic nervous system will constrict veins to increase the amount of blood coming into the heart as well as constrict arteries.

The parasympathetic nervous system will dilate veins to decrease the blood returning to the heart as well as dilate arteries.

Heart function is similarly controlled by the sympathetic (increases heart-rate/strength), and the parasympathetic (decreases heart-rate/strength) nervous systems.



By adding to our simple diagram we can really start to get down to the nitty gritty. Anything that causes a strong parasympathetic response or anything that has a direct effect on the heart rate and/ or contractility can lead to a syncopal episode. Almost too simple, isn't it?

Types of Syncope

Vasovagal Syncope

Lets begin by discussing the most common type of syncope (it is also the most confusing). ***Vasovagal*** syncope, also called ***neuro-cardiogenic*** syncope.

The direct cause of a vasovagal syncope is a really strong parasympathetic response that causes massive venodilation and a slowing of the heart rate, or even a period of asystole (Jardine, Ikram & Crozier 1996). Mary, in the opening story, suffered a vasovagal syncope and we will use her to help us understand exactly what lead to her strong parasympathetic response.

As Mary was eating and drinking, her parasympathetic nervous system was activated (not enough to cause her to pass out though, that happens later). The parasympathetic nervous system plays a major role in digestion, it makes sure that the digestive tract is getting lots of blood by dilating vessels. The parasympathetic nervous system also has an inhibitory effect on the sympathetic nervous system, which will inhibit any sympathetic induced vasoconstriction. In addition, alcohol has a direct vasodilatory effect which further compounds the enlargement of the vessels. Keep in mind, this is the bodies normal response to eating. I am sure we have all enjoyed the 'warm glow' when enjoying a good meal. In Mary's case, however, something interesting happened; due to her dilating vessels, her ventricles suddenly got only partially filled. Imagine for a moment, that you are walking up the stairs without paying attention, you think there is another step but there isn't, your foot suddenly slams down on the top floor with a bone jarring force (if I'm the only one who have ever done this I apologize for the poor choice of analogy).

The ventricle goes through a similar experience. As the sympathetic nervous system begins to compensate for the dilated vessels, the heart attempts to beat stronger by triggering a sympathetic response. The heart consequently contracts forcefully against a partially empty ventricle (under-filled from the vasodilation). The result is a misinterpretation by stretch receptors in the left ventricle (c-fibers) that the ventricle is way too full (the very opposite of what is really happening)! It sends those faulty signals to the brain. The brain stem responds quickly to prevent damage to what it now believes is an overfilled ventricle. A powerful protective reflex mechanism (Bezold-Jarish reflex) is stimulated. The reflex triggers a strong parasympathetic response leading to profound bradycardia or asystole as well as massive venodilation, all in an effort to reduce the pressure within the ventricle (that was really only part full to begin with) (Kikushima et al 1999; Moya et al. 2001).

This faulty parasympathetic reflex is what caused Mary to faint on her anniversary. Anything that cause a drop in pre-load, volume of blood returning to the heart, can trigger a Bezold-Jarish reflex:

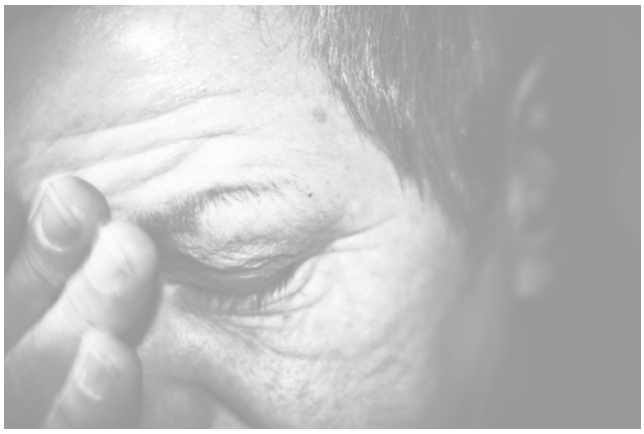
- prolonged standing or sitting
- nitroglycerine administration
- dehydration
- warm environment
- donating blood (or other blood loss)

The Bezold-Jarish Reflex

Drop in Pre-Load from:
- Eating
- Standing / sitting
- Ntg. administration
- Dehydration
- Warm environment
- Blood loss

Compensatory Sympathetic Response

Forceful contraction



Miss-interpreted as overfilled ventricle

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strong protective parasympathetic reflex

- **Massive venodilation**
- **Heart rate slowing (or asystole)**
- **Decreased Contractility**

In addition to the above described rather complex mechanism, a strong emotion can directly trigger a strong parasympathetic response (Miller & Kruse 2005). The exact emotion that may trigger the reflex varies from person to person. For example, the sight of blood, the smell of decaying flesh or receiving upsetting news, to name a few. Keep this in mind when giving death notifications and sit the person down before delivering the news! Also, consider the risk of syncope when someone has suffered a graphic looking injury or seems a bit uneasy when you pull out the IV supplies. Be aware that emotion triggered vasovagal syncope, is *not* the same as *situational syncope*. Situational syncope is covered next.

Situational Syncope

Direct stimulation of the vagus nerve can lead to a 'crossing of wires' whereby a stimulus from the vagus nerve can cause a reflex vasodilation and heart slowing (Miller & Kruse 2005).

One type of situational syncope is caused by GI tract irritation. The GI tract is heavily innervated by the vagus nerve (from top to bottom).

GI upset, cramping or irritation of the esophagus can trigger a powerful vagal response and lead to syncope.

The bladder is also innervated by the vagus nerve and the stimulus from emptying the bladder can trigger syncope (micturition syncope) (Miller & Kruse 2005). If you haven't already attended to a patient who passed out in the middle of the night while having a pee, you will soon.

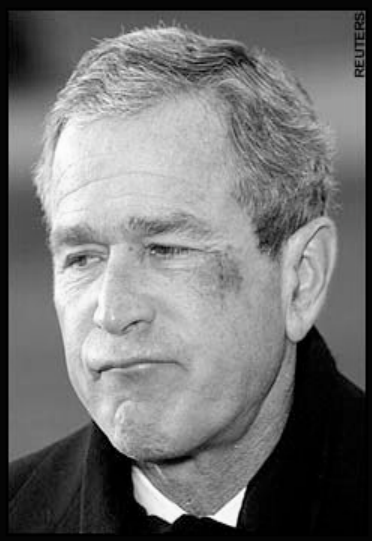
Other triggers include anything that puts pressure on the baroreceptors located in the carotid arteries. Some people have overly sensitive baroreceptors and pressure on the neck from shaving, tight collars or even turning the head can cause them to faint (Miller & Kruse 2005).

In summary, some causes of situational syncope;

- peeing
- pooing
- nausea / vomiting
- GI cramping / diarrhea
- coughing
- shaving
- tight collars
- head turning

The Pretzel Incident

On January 14th 2002 George W. Bush suffered a syncopal episode while watching a football game in the Whitehouse. A pretzel the president was eating got lodged in his throat causing a situational syncope. The only witnesses to the incident were the president's two dogs Barney and Spot. The president spontaneously regained consciousness and only suffered minor scrapes and bruises on his face during the incident. Bill Clinton's former press secretary later joked that President Clinton would never have been able to emerge from an empty room with scrapes and bruises, and convince people that it was simply caused by a pretzel.



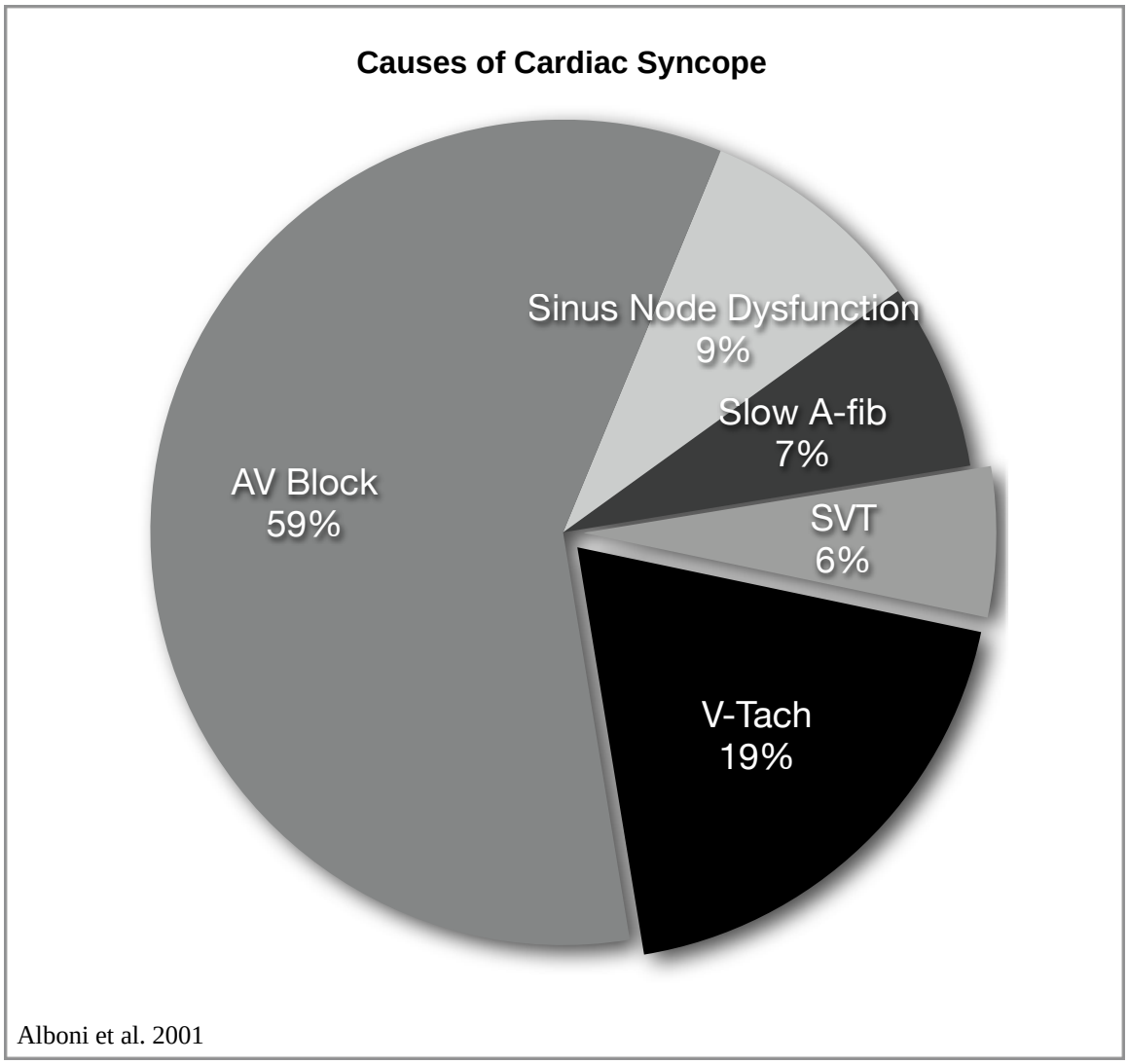
http://archives.cnn.com/2002/ALLPOLITICS/01/14/bush.fainting
http://www.ippsr.msu.edu/Documents/MPLPSummer03Newsletter.pdf

Cardiac Syncope

The underlying cause of cardiac syncope is rather straight forward; the heart goes too fast or too slow to provide adequate cardiac output. Cardiac syncope make up 10-30% of all syncopal episodes. The most common causes are various bradycardias with 3rd degree A-V block being the most common culprit and 2nd degree type II being the second most common cause (Alboni et al. 2001; Kadri et al. 2006).

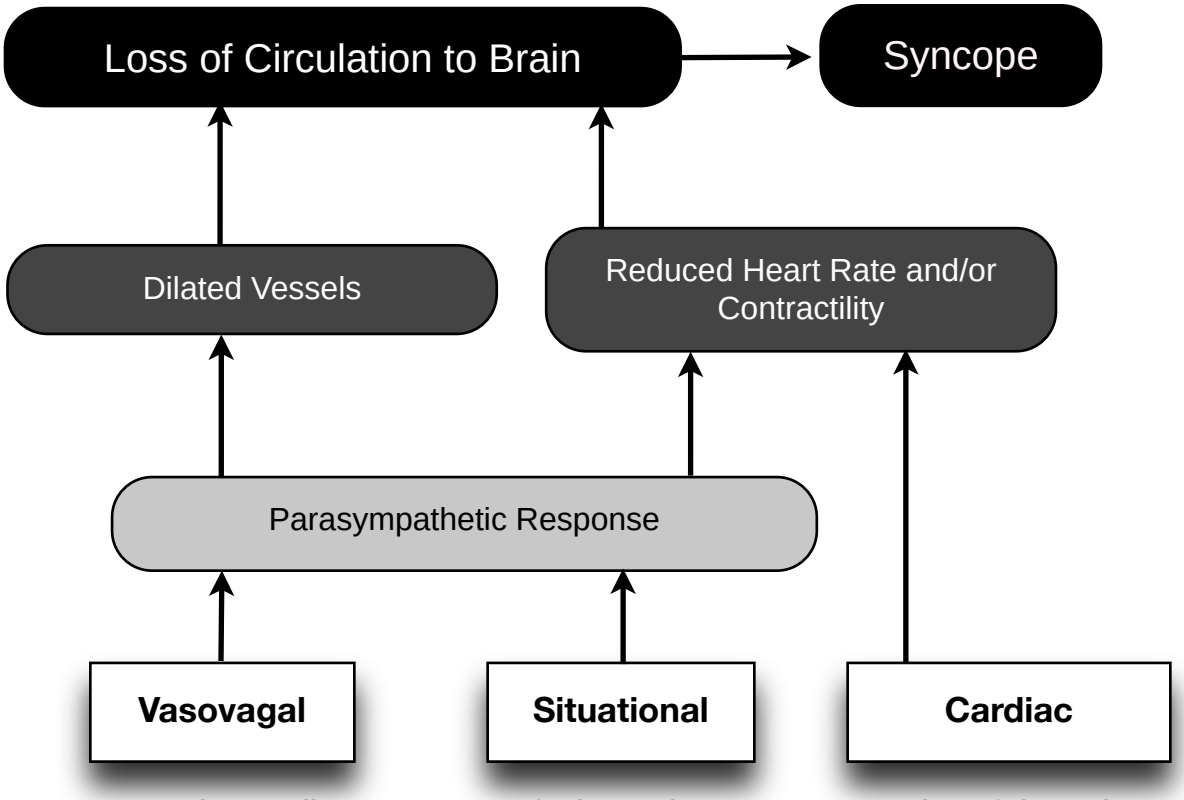
Out of the tachycardias, SVT is the most common rhythm to cause syncope, followed by V-tach (Kadri et al. 2006).

It is important to determine if a syncope patient suffered a cardiac syncope, as it has twice the mortality compared to other causes (Miller & Kruse 2005)! We will discuss how to tell the different types on syncope apart in the next section.



Alboni et al. 2001

Overview of Causes



Now we can take our diagram one step further and get an easy overview of the various underlying causes of syncope.

Other Causes

The above types of syncope makes up the bulk of incidents. There are other causes, but rather than getting into great detail on issues that we are unlikely to encounter, we will focus our efforts on what is common. Here is a list of some of some other causes of syncope:

- pulmonary embolism
- aortic stenosis
- TIA (implies massive circulatory compromise or directly effected RAS, so not super likely)
- subclavian steal
- hypoglycemia

Assessment

Asking the Right Questions

Now that we have a solid understanding of what causes various types of syncope, we can incorporate that knowledge into the hands-on assessment and treatment of our patients. The assessment portion will allow us to develop an educated guess (working assessment) on what type of syncope the patient suffered. Does it matter to us? Yes, a cardiac syncope carries a much higher risk to the patient than vasovagal or situational syncope. Knowing that our patient suffered a cardiac syncope should increase our vigilance and the sense of urgency. There is also a great sense of satisfaction in having a true understanding on what is happening to our patient.

A detailed, systematic history is what will truly help us understand what type of syncope the patient experienced. When obtaining the incident history, it is important to determine exactly how the patient felt and looked prior to the syncope, and how long those symptoms lasted for. The nature of the actual syncope also must be determined in some detail. Ask any bystander who witnessed the episode exactly what the patient looked like during the syncope and how long it lasted. Finally, ask how the patient feels now, after the syncope.

Be careful not to *lead* the patient and bystanders to answer the questions to fit what you expect to hear. For example asking;
"How did you feel just before you fainted?"
is a much better question than;
"So, no fluttering sensation or chest pain before you fainted, right?"
Having said that, some patients require more direct questioning due to confusion or poor communication skills, in those cases the above question can be narrowed a bit;
"How did your chest feel just before you fainted?"
This will allow the patient to describe the symptoms in his/her own words.

The patient's past medical history is also an important indicator of what type of syncope they are most at risk for.

Before the Syncope

Ok, so we have arrived on scene of Mary's fainting spell. We have managed to delicately maneuver the stretcher around the tables in the dimly lit dining room. We find Mary sitting at her table, vomiting into her cotton napkin. She looks terrible. She is pale, sweaty and embarrassed. Now for the important part, what type of syncope did Mary have? As mentioned before, *what* the patient was doing before fainting is one important clue that should be determined. If the patient was peeing, pooping, shaving or coughing, it is likely that he/she suffered a situational syncope. It is very unlikely that Mary was doing any peeing, pooping or shaving before she passed out. She may have been coughing or gagging on food that went down the wrong way which also might cause a situational syncope, but that would be easy to establish from bystander accounts. So, while your partner hooks up the oxygen, monitor and gets some vital signs, lets start asking Mary and her family exactly what happened.

Vasovagal syncope has a fairly distinct pre-syncopal period. Environments that promote vasovagal syncope include warm, crowded environments, and situations that include prolonged standing or sitting (Vaddadi et al. 2007). Also, any exposure to emotional stimuli such as needles, graphic images or upsetting news can trigger vasovagal syncope and should be inquired about (Vaddadi et al. 2007). In the moments before a vasovagal syncope the patient will usually describe feeling very warm and lightheaded. There may also be darkening or blurring of vision,

Obama's Fainting Ladies

During the US presidential campaign the media picked up on a very common, recurring theme during Barack Obama's speeches. During the speeches Barack would stop to point out that someone in the audience had fainted. He would call for EMT's and often toss a water bottle to the collapsed person. The similarity between the incidences made some wonder if it was part of the 'show'. Knowing what we now know about syncope, is it likely that someone might faint while standing in the warm, crowded environment of a campaign rally, emotionally overwhelmed? You be the judge.

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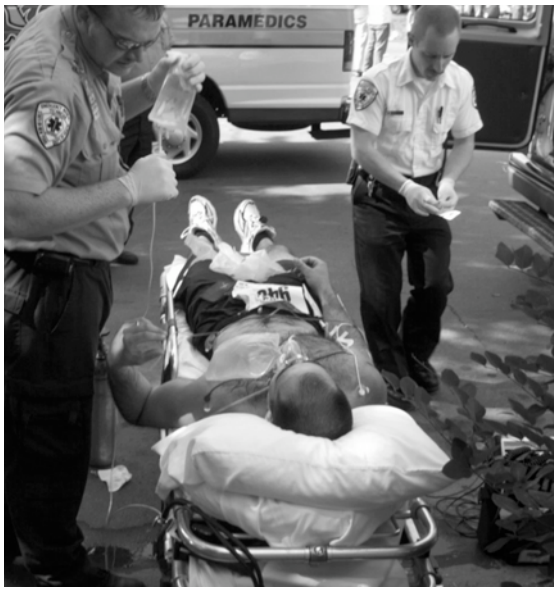
nausea, shortness of breath or repeated yawning (Rollinson 2005; Sheldon et al. 2002; Graham & Kenny 2001; Miller & Kruse 2005). The symptoms are likely to last greater than 10 seconds and may last several minutes (Alboni et al. 2001). Bystanders usually report that the patient looked very pale, sweaty and seemingly distant and lethargic. They may also confirm that the patient was yawning repeatedly (the yawning is actually a sign of the parasympathetic response). All these signs and symptoms are due to the initial parasympathetic, then sympathetic response. Once the Bezold-Jarish reflex triggers the final, faulty parasympathetic response, the patient loses consciousness.

In cardiac syncope, on the other hand, there is usually no warning, or a very short period of palpitations prior to fainting (Kadri et al. 2002). It makes sense if we consider the mechanism that lead to cardiac syncope. As soon as the heart enters into the arrhythmia that causes the syncope, cerebral perfusion drops and the patient faints suddenly and with little or no warning.

Also, syncope that occurs during exertion



is likely to be of cardiac origin (Miller & Kruse 2005). However, if the syncope occurs after exercise it is likely a vasovagal mechanism (Miller & Kruse 2005). This again highlights the importance of gathering a clear history of events. For example, an athlete that drops during exercise is much more worrisome than an athlete who passes out after exercising.

Another aspect of the incident history that can be established during this time is how well the patient can recall the moments before the syncope. If the patient is unable to recall the events just prior to fainting, it is likely that the syncope was actually a seizure (Kadri et al. 2006). Other details of the period before the collapse that suggest a seizure are mood changes, trembling, hallucinations or confusion prior to the event (Sheldon et al. 2002).

Drink to This

Studies have shown that drinking water prevents syncope. Not that we can (or should) tell our patients to drink water if they are feeling dizzy. However, if you know someone who is prone to syncope and they have to be in a situation that may trigger one, then you can suggest that they drink a glass of water prior to the event.

(Lu et al. 2003)

During the Syncope

The description of the syncope itself clearly must come from the bystanders. In addition to the duration of the loss of consciousness, ask the bystanders exactly what they saw. Again, avoid suggesting things that may lead the answers.

Vasovagal syncope usually causes a standing patient to kneel over. While in the sitting position, the person will simply lose postural tone and 'go limp'(Miller & Kruse 2005). During the syncope the skin color might be flushed or turn very pale or cyanotic (Miller & Kruse 2005) and will usually be covered in sweat (Sapin 2004). The duration of the syncope is usually seconds to a few minutes (Sapin 2004).

One common, and confusing, factor is the limb jerking that may occur during a vasovagal syncope. Literature reports that as many as 66% of syncope patients will have mild seizure activity during their fainting spell (Vaddadi et al. 2007). The misdiagnosis of syncope as seizure is quite common. As many as one in five patients being treated for seizures, may in fact have suffered syncope (Vaddadi et al. 2007)! So how can we differentiate between seizure and syncope? Essentially, a syncope patient will loose postural tone whereas a seizure patient will become rigid. Head turning during the event (as if the patient was trying to look over their own shoulder) and tongue biting are signs more specific to seizures than the limb jerking alone (Sheldon et al. 2002).

After the Syncope

The period that follows the syncope is also riddled with clues as to the cause of the 'black out'. This period is also the time that we usually get to see the patient so we can get some really reliable information about this phase of the episode. After the strong parasympathetic response has caused the patient to lose consciousness, a strong sympathetic response is triggered in order to correct the mistake. The signs of this sympathetic response is what we see after the syncope, the patient is often pale, sweaty, nauseated, weak and dizzy (Sapin 2004; Miller & Kruse 2005; Alboni et al. 2001). This period usually resolves within thirty minutes to an hour. If the recovery is very fast, and the patient feels normal immediately after the event it is more likely to be a cardiac cause. This quick recovery to normal is misleading as our 'gut feeling' about the patient who recovers fast and looks fine by the time we get there is

probably better, as compared to someone who has a relatively benign vasovagal episode but looks really crappy afterwards.

Urinary incontinence can be expected after approximately 10% of syncopal episodes but is more indicative of a seizure (Alboni et al. 2001; Miller & Kruse 2005). In addition, a syncope patient will usually have full awareness of their surroundings following the syncope whereas a seizure patient will usually be very confused for some time.

Past Medical History

Once we have established a clear picture of the events before, during and after the seizure we can turn our attention to past medical history and medications.

Any past medical history of heart problems is concerning Not surprisingly, a history of heart disease increases the chance of having cardiac syncope (Miller & Kruse 2005; Alboni et al. 2001). On the other hand, if the patient does not have heart disease and did not have palpitations prior to the syncope, a cardiac cause is very unlikely (Alboni et al. 2001). Since cardiac syncope is much more dangerous than other causes, take note of any cardiac history in the patient's past.

Finally, many drugs can predispose a person to syncope. Cardiovascular, neurologic, Parkinson's, Alzhemier's and antidepressant medications can all lead to syncope and should be noted, especially any new drugs or changes in dosages of current drug regimes (Miller & Kruse 2005).

	Vasovagal	Situational	Cardiac	Seizure
Environment	•Prolonged standing / sitting •Crowded / warm environment •Emotional trigger •Graphic images •After exertion	•Pooping •Peeing •Shaving •Tight collar •Turning head •Coughing •GI upset	•Can occur anywhere •Suspect if *during* exercise	•Stressful environment •Strobe lights
Before	•Feeling warm •Lightheaded •Nausea •Blurred vision •Yawning	•Sudden	•Sudden •Short period of palpitations / chest pain	•Confusion •Trembling •Mental status changes •Patient unable to recall
During	•Kneeling forward •Seizure activity •Slow / no pulse •Pale / flushed / cyanotic	•Same as vasovagal	•Drop suddenly •Pale / cyanotic	•Head turning •Urinary incontinence
After	•Pale •Nauseated •Dizzy •Weak / tired	•Same as vasovagal	•Feels normal •Chest pain •Palpitations	•Confused / postictal •Tongue trauma
Past Medical History	•History of fainting spells	•Same as vasovagal	•Cardiac history	•Seizure history

Treatment

Oxygen

Treatment for the syncope patient is relatively straight forward. According to the BLS Patient Care Standards, *'treatment should consist of establishing a patent airway and; assisting ventilation or administration of high concentration oxygen, if required based on assessment'* (MOH 2007). So, what does that mean? Another section of the manual lists, specifically, what assessment findings should prompt high concentration oxygen;

- Altered mental status
- Poor skin colour
- Respiratory distress
- Abnormal heart rate or blood pressure
- Abdominal pain
- Chest pain
- The incident history indicates a potentially serious underlying condition

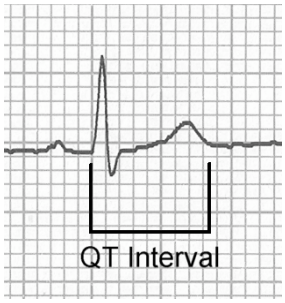
So, wait a minute...we just learned that if the patient looks really good and feels fine right after their syncope then its likely to be a cardiac cause, which is serious, so would warrant high concentration oxygen. But if they are pale, sweaty and nauseous then they fit the 'poor skin colour' category and should also get high concentration oxygen. Then we might as well give all post syncope patients high concentration oxygen..? Exactly!

Monitor

Cardiac monitoring is not really a treatment but it is part of what we do to the patient along with oxygen, so we can definitely add it to our to-do list. Look for any abnormalities with the rhythm. If you are using the LP12 turn on the QRS beeper to get a beat by beat indication of the rhythm. The beeps will allow you to quickly pick up on irregularities even when you are not looking at the monitor.

12/15 Lead

Obtaining a 12/15 lead is also important. Obviously, look for S-T elevation that could indicate that the syncope was caused by an AMI. Also, check the Q-T interval. The Q-T interval is measured from the beginning of the QRS complex to the end of the T wave. A Q-T interval longer than 500 ms is considered abnormal and could lead to arrhythmia. 500 ms amounts to 12.5 small boxes, or 2.5 large boxes on the ECG paper. At a quick glance, it will just look like the T-wave is far away from the rest of the complex. Be aware that factors such as heart rate, age and sex also alters the Q-T interval. The full story on Q-T intervals is beyond the scope of this package, just keep it in the back of your mind and if it looks abnormal point it out to the receiving staff.



Position

Patient positioning should be guided by the patients blood pressure and comfort. If the patient is hypotensive, they should be kept supine or left lateral position. If the blood pressure is adequate, you can leave it up to the patient. The BLS Patient Care Standards also suggest to *'...keep patient movement to a minimum'* (MOH 2007). So....well, don't jostle the patient too much!

Bolus

How about a bolus? If the blood pressure is low when we assess the patient, should we give a bolus, or wait and see if the vasodilation and heart rate will correct itself?

One study investigated the effect of a saline bolus on adolescent patients susceptible to syncope. The researchers induced syncopal episodes with a tilt table. Essentially the subjects were strapped to a table and tilted from supine to standing, thus inducing a drop in preload and subsequent Bezold-Jarish reflex and syncope. Initially, the researchers successfully induced syncope in all subjects.

However, after a one liter saline bolus none of the subjects fainted when tilted on the tilt table. Given that data it certainly seems like a saline bolus is a reasonable idea if the patient meets the medical directive (Burklow et al. 1999).

Summary

Syncope is a very common EMS call. Often the cause is relatively benign but in some cases a potentially life threatening condition lurks under the surface. Hopefully this self-study package has provided some added insight into syncope and its causes.

With good, thorough history gathering and informed use of additional assessment tools we can hopefully be better able to determine the underlying cause of the syncope and prepare for complications.

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