

Introduction

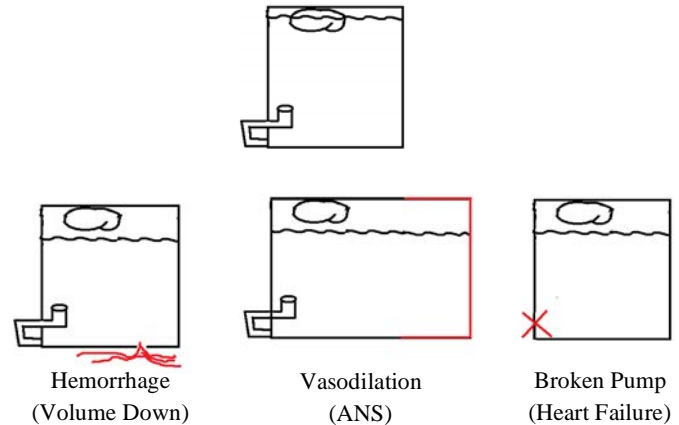
Syncope's a symptom defined as a **transient loss of consciousness** due to global cerebral hypo perfusion. The heart is a pump; it pushes blood into the vasculature, the "tank." The brain is at the top of the tank. Gravity works against the heart by pulling the blood towards the ground. Thus, it's necessary to have a strong pump, a normal sized tank, and enough blood to fill the tank to get the blood to the brain. If blood can't get up to the brain, we pass out, i.e. **syncope**. It all comes down to **blood pressure** - a product of multiple factors (equation to right). The etiologies of syncope are vast; each affects one of these elements directly. But they all can be narrowed down to: a broken pump, too big a tank, and not enough fluid. It's important to realize how the history and physical relates to the potential etiology and the tests that need to be done to confirm suspicions.

Neurocardiogenic (Vasovagal)

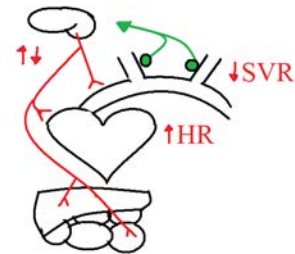
The vagus nerve goes everywhere: visceral organs, blood vessels, and the brain. It's both afferent and efferent. Its signal to the blood vessels causes them to dilate, reducing systemic vascular resistance. The signal given to the heart is bradycardia. If the **Vagus nerve activates** more than it should (for whatever reason), it can cause **bradycardia** (cardio-inhibitory) or **hypotension** (vasodepressor). In both cases blood pressure falls, blood to the brain falls, and the person passes out. Lots of things can cause the Vagus to fire: visceral stimulation, such as **cough / defecation / micturition**, an **overactive carotid sinus** as in **turning the head** or **shaving**, and, because the vagus nerve comes from the brain, **psychotropic** causes such as the sight of blood. Vasovagal is both **situational** and **reproducible**. Do a **tilt-table** test to confirm suspicions.

Orthostatic Hypotension

Normally, when going from sitting to standing the blood follows gravity and pools in the legs. The person does not feel it but there's a drop in blood pressure. It's sensed by the same baroreceptors that could go overactive in vasovagal. These carotids send a signal that causes an almost immediate compensatory vasoconstriction and increased heart rate (which is why we don't pass out every time we stand). But this reflex can fail if there's insufficient sympathetic tone or volume. If the **autonomic nervous system is broken** (as in the **elderly** or a **diabetic**) or there's something fighting against the sympathetic tone (such as **sepsis**) there can be **no reflex sympathetics** to compensate, causing a person to pass out. In other words - SVR is insufficient. However, if there's **insufficient preload** to begin with, standing up exacerbates the condition; CO is insufficient. A decreased preload is seen in people with **hypovolemia** (diuretics, diarrhea, dehydration and hemorrhage). In both cases **vital signs** are highly suggestive of the disease. A **decreased systolic BP of 20**, a **decrease diastolic BP of 10**, an **increase in HR of 10**, or symptoms when moving from a laying position to a standing one give away the diagnosis. This person is said to be "orthostatic." Give back the volume with **IVF** if volume's down, or give **pressors**.



$$BP = \frac{CO}{HR} \times SVR$$



- *Overactive Vagus → HoTN, Bradycardia*
- *Situational Syncope*
  - o *Visceral → Cough, Defecation, Micturition*
  - o *Turning Head/ Shaving*
  - o *Site of blood → Psychogenic*
- *Tilt Table*
- *Failure of Reflex Sympathetics*
  - o *Elderly/DM → Broken ANS*
  - o *Sepsis → Inflammatory Cytokines*
  - o *Anaphylaxis → Same as sepsis*
  - o *Addison's Disease*
- *Hypovolemia*
  - o *Hemorrhage*
  - o *Dehydration*
  - o *Diuretics*
- *Postural Hypotension*
  - o *Laying → Standing*
  - o *SysBP ↓ 20, DiaBP ↓ 10, HR ↑ 10*
  - o *Rehydrate or Add Constrictors*

Mechanical Cardiac Disease

This is a rare cause of syncope. If there's a giant **obstruction to outflow** from the heart (saddle embolus, aortic stenosis, HOCM, LA Myxoma) the cardiac output suffers. Because the patient is living, syncope occurs with an increase in cardiac demand, i.e. **sudden onset with exertion**. There might be an audible murmur, but these diseases are structural so get an **Echo**. Cardiac output suffers because there's an obstruction to outflow. For a more thorough discussion of this phenomena please see hypertrophic cardiomyopathy in the cardiomyopathy section.



- *Structural Lesion*
  - o *PE, AS, HOCM, LA Myxoma*
- *Post Exertional Syncope*
- *ECHO*
- *Treatment Etiology dependent*

Arrhythmia

Arrhythmias are typically a disorder of **automaticity**. If the heart goes **too fast**, there's not enough time to fill (↓ **preload**). If the heart goes **too slow**, heart rate suffers + with it BP. Syncope will occur **suddenly**, without warning. An **ECG** will show the arrhythmia IF symptoms are occurring at the time of ECG, but it usually requires a **24-hr Halter** monitor to catch symptoms occurring with the arrhythmia. This will require **antiarrhythmics** or an **AICD** to flip them into a normal rhythm.

- *Sudden onset syncope, without prodrome*
- *Rapid change in CO*
  - o *Too fast = ↓ Preload*
  - o *Too slow = ↓ HR*
- *ECG → ECHO*
- *Antiarrhythmics or Defibrillator*

Neuro

Some things LOOK like a syncopal episode but actually aren't. If you see someone "pass out," consider these diseases. This section is even more brief than usual; only one neuro cause is actually syncope. Decreased blood flow to the posterior circulation - **vertebrobasilar insufficiency** – may result in the patient passing out. Diagnose it with a **CT Angiogram** by looking at the vertebrobasilar arteries.

If the patient is **post-ictal** after "passing out" they may have had a **seizure**. Diagnose with an EEG. If the patient has a **focal neurologic deficit** they may have had a **stroke**. Diagnose with a **CT** or an **MRI**. If the patient simply **falls asleep** and **wakes refreshed** consider **narcolepsy**; treat with **amphetamines** and regularly scheduled naps.

Put it in practice - handling syncope: "Woman 3-2-1 PE"

	<i>History</i>	<i>Physical</i>	<i>Diagnosis</i>
<b>VV</b> (Vaso Vagal) - Visceral Organs (micturition, defecation, cough) - Carotid Stimulation (turning head, shaving) - Psychogenic (site of blood)	Situational, often Reproducible, with a positive prodrome	Vagal stimulation produces asystole or a ↓ SYS BP of 50 mmHg	Tilt Table
<b>O</b> rthostatics - Volume Down - Autonomic Nervous Dysfunction	Orthostatic hypotension	Defined as ↓SYSBP by 20 ↓DIA BP by 10 ↑HR by 20 Sxs of orthostasis	Volume and Reassess, chase causes of hypotension if refractory to fluid
<b>M</b> echanical Cardiac	Exertional syncope	Murmur	Echo
<b>A</b> rrhythmia	Sudden Onset, unprovoked,	None	24-hour Holter
<b>N</b> euro (vertebrobasilar insufficiency)	Sudden Onset, unprovoked, very rare	Focal Neurologic Deficit	CTA
<b>P</b> ulmonary Embolism	PE	PE	Wells Criteria, CT scan
<b>E</b> lectrolytes (bG, Tsh)	None	None	BMO