

Ask the Clinical Instructor

A Q&A column for those new to the cath lab

Questions are answered by:
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“Once in a while, when we are removing a sheath, the patient becomes sweaty, the pulse rate goes down and the BP goes down. The doctors say that this is a vasovagal response, but I thought it was the beginning of shock because the BP went down. I’m confused.”

— CVT student from Wisconsin.

A vasovagal reaction is nothing more than a fainting episode. It can sometimes be seen referred to as neurally mediated syncope. When you hear about someone who is squeamish of blood and then sees blood, what happens to them? They sometimes fall to the floor during a ‘faint.’ You might say that physical collapse during a vasovagal reaction is the body’s protective mechanism. So why is this an issue in the lab? What position are

our patients usually in? When they are already supine, we have taken away that normal protective mechanism, so sometimes we have to do a few other things.

You can more easily understand what is happening during a “vagal” episode once you understand a little physiology.

One section of our body’s every day control is the autonomic nervous system (ANS). These are reactions/functions that we generally do not think

about controlling. We can control them temporarily (*i.e.*, holding our breath), but eventually the body wins out and takes over. For purposes of this article, we will focus on the ANS responses involving smooth muscle, the heart and some glandular tissue.

The ANS splits into two branches relative to this topic. It branches into the sympathetic nervous system (SNS) and the parasympathetic nervous system (PSN). The SNS is also known as the “fight-or-flight” mechanism. When someone jumps out at you from behind a corner and you get an adrenaline rush where your hair stands on end, a little jumpy and sometimes a little short of breath, that is your SNS kicking in. This can also be thought of as the gas pedal for your body.



On the other side of the spectrum, the PNS controls our vegetative functions, such as our digestive system, tears and sweat, and, as we will see, our heart rate and BP. We truly cannot control these functions, but we rely on them to work without conscious control. Most of the PNS involves the vagal nerve. When it is stimulated in some manner, the PNS response is enhanced; hence the term “vagal” or “vasovagal” response. We can also think of this as the brake pedal for the body. A side note is that the vagus

nerve actually gives off branches, called cardiac nerves.¹ This helps establish the link between the PNS and the heart rate.



In our daily lives, these two systems are intended to balance each other. There is enough acceleration and braking at the right times to keep everything on the straight and narrow. When the two systems are in balance, all is well and we feel fine. In our scenario of someone jumping out at us creating an adrenaline rush (or the gas pedal has been pushed) because of SNS stimulation, we hope that, at some point, our PNS will kick in, or ‘put on the brakes,’ to bring us back to a state of normalcy. We really couldn’t live for long in a constant state of adrenaline surge. The brakes of our body prevent those problems from occurring.

In a vasovagal reaction, there is simply too much brake pedal. It has either overridden the gas pedal, or the gas pedal is simply not functioning. Something has stimulated the vagus nerve, causing the body to think that it needs to slow down. The predominance of the PNS over the SNS causes the individual to “faint,” because cardiac output suddenly drops due to bradycardia and hypotension.

What can cause a vasovagal reaction? We see some of these reasons all the time on television and in movies.

The sight of blood, bad news or something shocking can precipitate the response. The trigger can vary from person to person.



In the cath lab, most vasovagal responses are seen during the painful processes of our work. The local lidocaine injections or sheath insertions can certainly cause a vasovagal response, either through pain or stimulation of nerves. Manual compression after a sheath removal can also cause a reaction. For this reason, EVERYONE needs to be paying attention and watching the monitor during the first few minutes of a sheath removal. (That's why having atropine immediately available is a standard during sheath removals.)

There are also other causes that I will address in a future article, such as internal carotid artery ballooning, baroreceptor stimulation, bearing down (as in valsalva or defecation), etc. These deserve more attention than space here will allow.

When there is a stimulation of the PNS, what really happens? What happens during the “too much brake pedal” phase is a little different than other syndromes we see. The typical patient will usually have some early warning (prodromal) symptoms:

- Weakness, nausea, diaphoresis, light-headedness, yawning, sense of impending darkness (*i.e.*, the tunnel is closing in). Most experienced cath lab professionals will

– In some elderly patients, a vagal reaction may occur without bradycardia and appear as unexplained hypotension.²

These signs and symptoms can last for at least a few seconds before consciousness is lost (if it is lost), which typically happens when the person is sitting up or standing. When standing sufferers pass out, they fall down (unless this is impeded); when in this position, effective blood flow to the brain is immediately restored, allowing the person to wake up. As we mentioned, our patients who are supine on the table do not have this mechanism available to them.

Understanding the vasovagal sequences, and an appropriate reaction to them, is extremely important. The significance of these episodes can sometimes be taken for granted. “The only difference between syncope and sudden death is that in one you wake up.”³

So what do we do in the cath lab with these patients? Remember,

However, in the cath lab, they are already supine, so other management techniques are required. First, BE CALM. These events are usually self-limiting. Yes, the combination of bradycardia and hypotension is sometimes dramatic, but keep in mind that these are usually easily controlled.

Administration of atropine for bradycardia (0.5mg every 5 minutes as needed up to 3mg) and IV bolus (500cc of normal saline) are the first steps. Yes, you can try to raise the legs to help get blood to the central core, but in reality, you will be able to administer the atropine and fluids faster than you can find items to put under the legs. If you have a venous sheath in place, give as much fluid as you can through the sheath so fluid rapidly enters the central circulation. You may be tempted to reach for the button on the transcutaneous pacemaker, but you should leave it alone. The atropine and the fluids are usually enough to resolve the situation. Even though the heart rate and BP will eventually return to normal, the patient will likely still feel very bad.

Just make sure to provide comfort measures. Explain to the patient that they had a “fainting” spell and that they are OK. If you have ever suffered from a syncopal episode, you probably understand this feeling.

Vasovagal reactions generally do not need further treatment or investigation. Should they repeat often, the physician may later perform tilt table testing and/or electrophysiology testing. Aggressive management is generally not warranted.

If you have any questions about this article, or you or your lab have a question about a particular topic, please send an email to tginapp@rcisreview.com.

Next month, we will address a question on atropine.

References

- 1 Moore KL and Agur AMR. *Essential Clinical Anatomy, Third Ed.* Philadelphia, PA: Lippincott Williams Wilkins; 2007.
- 2 Kern M, ed. *The Cardiac Catheterization Handbook, 4th Ed.* Philadelphia, PA: Mosby, An Affiliate of Elsevier Science, 2003.
- 3 Engel GL. Psychologic stress, vasodepressor syncope, and sudden death. *Ann Intern Med* 1978;89:403-412.

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tell you patient yawning should get you to check things out a little closer.

These can be followed by signs/symptoms:

- Loss of skin color, abrupt bradycardia, diaphoresis, pupillary constriction, and finally, decreased cerebral perfusion resulting in syncope. Sometimes people will also just feel “crappy.”

when the brake pedal is pushed, the normal response is to lower the heart rate and blood pressure, which results in a reduction in cardiac output. If the individual is standing and the cardiac output is suddenly reduced, perfusion to the brain is significantly decreased, causing them to pass out. Once supine, blood flow to the brain resumes and they wake up.