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It All Makes Sense

Why the Valsalva Maneuver breaks SVT and causes syncope

Understand the four phases of the Valsalva effect and how those phases effect a stable supraventricular tachycardia

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The Valsalva effect is generally divided into four separate phases:

- **Phase 1:** Onset of straining and the beginning of an increase in intrathoracic pressure
- **Phase 2:** The persistent straining and maintenance of the increased intrathoracic pressure
- **Phase 3:** Release of breath-holding and glottic pressure with a sudden drop in the intrathoracic pressure
- **Phase 4:** Sudden increase in cardiac output and aortic pressure

EMS crews often discuss the call where a patient is found on the toilet after suffering a syncopal episode while having a bowel movement. Interestingly, the act of defecation often involves involuntary use of the Valsalva during evacuation of the colon.

The person while inhaling, closes their glottis and tightens the chest and abdominal muscles. This increases not only the intra-abdominal pressure to assist with colon evacuation, but it also increases intrathoracic pressure and produces the Valsalva effect.

Because you may use the Valsalva to break a supraventricular tachycardia in your emergency care of a hemodynamically stable patient, or you may be called to manage a patient who fainted while having a bowel movement, it is important to understand the physiology associated with a Valsalva maneuver.

In the prehospital environment, the Valsalva maneuver may be done voluntarily or involuntarily. The patient's physician may instruct them to voluntarily perform a Valsalva in an attempt to decrease the rapid heart rate following sudden onset of supraventricular tachycardia. If effective, this may prevent the patient from accessing the EMS system and allow them to continue about with their activity after the heart rate is back under control.

On the other hand, a hemodynamically stable patient who presents in a regular narrow-complex tachycardia with a ventricular rate greater than 150 beats per minute may be instructed by the EMS crew to perform a Valsalva maneuver in an attempt to decrease the heart rate.

The patient is asked to inhale, hold their breath and bear down while tightening the chest and abdominal muscles as if they were straining while having a bowel movement. The patient is then instructed to suddenly release and breathe out. An increase in vagal – parasympathetic – tone decreases the heart rate, which averts the need to administer adenosine or other medications.

The end result may cause the patient to experience a syncopal episode due to a decrease in cerebral blood flow and cerebral perfusion, especially if they have cardiovascular disease. While the Valsalva effect may occur during bowel movement, it may also be experienced if the breath is held while lifting heavy weights or while straining when moving heavy objects.

PHYSIOLOGY OF THE VALSALVA EFFECT

Let's explore the 4 different phases of the Valsalva effect.

PHASE 1: INCREASING INTRATHORACIC PRESSURE

During **phase 1**, the patient inhales, closes the glottis and bears down by contracting the thoracic and abdominal muscles. This results in a sudden increase in intrathoracic pressure. With the rise in pressure, the vena cava, aorta and cardiac chambers are compressed. Keep in mind that the pressure in the aorta is inverse to the heart rate. As the aorta is compressed, the intra-aortic pressure rises causing the heart rate to decrease from an increase in vagal — parasympathetic — tone on the sino-atrial node.

However, this decrease in heart rate is short-lived. The increase in atrial pressure and compression of the vena cava impede venous return and decrease left ventricular filling volume and preload. The drop in cardiac output decreases the intra-aortic pressure and produces an increase in heart rate.

The stroke volume directly influences the cardiac output.

Cardiac output (CO) = heart rate (HR) × stroke volume (SV)

Typically, an increase in stroke volume will increase the cardiac output; whereas, a decrease in stroke volume will decrease the output. Likewise, an increase in heart rate will increase cardiac output — a decrease in rate will decrease the output. It is important to note that excessively high heart rates do not allow enough time for ventricular filling, which may lead to a decrease in preload, stroke volume and cardiac output.

The straining and increased intrathoracic pressure continues during phase 2. As the venous return is further impeded and the atria filling is reduced, the left ventricular filling volume and preload continues to decrease. This results in a decrease in stroke volume, which leads to a reduction in the cardiac output.

Cardiac output and peripheral vascular resistance influence the blood pressure.

Blood pressure (BP) = cardiac output (CO) × peripheral vascular resistance (PVR)

Either a drop in cardiac output or peripheral vascular resistance will decrease the systolic blood pressure. In an attempt to maintain or raise systolic blood pressure, the body must increase cardiac output (increase HR or SV), increase peripheral vascular resistance (vasoconstriction), or increase both.

The diastolic blood pressure is actually a measure of the peripheral vascular resistance. As the PVR increases, the diastolic BP increases. Likewise, a decrease in PVR will decrease the diastolic BP. The systolic blood pressure is a measure of the cardiac output.

As the cardiac output continues to decrease, the systolic blood pressure and the pressure in the aorta drop, stimulating the baroreceptor reflex. The baroreceptor triggers the sympathetic nervous system in an attempt to raise the systolic blood pressure and aortic pressure through an increase in cardiac output and peripheral vascular resistance. Because the cardiac output (measured by the SBP) is decreasing and the peripheral vascular resistance (measured by the DBP) is increasing, the pulse pressure (difference between the SBP and DBP) narrows.

PHASE 2: DROPPING CARDIAC OUTPUT, STROKE VOLUME AND SYSTOLIC BLOOD PRESSURE

During **phase 2**, the cardiac output, stroke volume and systolic blood pressure are dropping, while the heart rate and peripheral vascular resistance are both increasing in an attempt to raise the aortic pressure. At this point, one may wonder why a Valsalva is actually used as an intervention in SVT or narrow complex tachycardia, since the heart rate goes up during the procedure when you are already dealing with an accelerated rate.

PHASE 3: DECREASING PRESSURE IN THE THORAX

But as the person breathes out during **phase 3**, the pressure in the thorax suddenly decreases, which releases the compression of the heart chambers, vena cava and aorta. The venous return is no longer impeded and suddenly fills the left ventricle increasing the preload.

PHASE 4: INCREASING STROKE VOLUME, CARDIAC OUTPUT AND INTRA-AORTIC PRESSURE

In **phase 4**, the sudden and drastic rise in venous blood volume increases the stroke volume, cardiac output and intra-aortic pressure. The peripheral vascular resistance is maintained, which leads to an even sharper increase in the intra-aortic pressure. As mentioned previously, an increase in intra-aortic pressure results in a decrease in heart rate. The arterial pressure overshoot causes vagal – parasympathetic –stimulation, which influences the sino-atrial node and decreases impulse formation and heart rate.

Basically, a Valsalva initially creates a low intra-aortic pressure which stimulates the sympathetic nervous system. Following the release of the breath, a vagal – parasympathetic – response is triggered to decrease the heart rate. This vagal influence is what may break the SVT and lower the heart rate, or cause a decrease in cerebral perfusion pressure and cause the patient to have a syncopal episode while straining to have a bowel movement.

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About the author

Joseph J. Mistovich, MEd, NRP, is the chairperson of the Department of Health Professions and a Professor at Youngstown State University in Youngstown, Ohio. He has more than 36 of experience as an EMS educator. He received a Master of Education degree from Kent State University, a Bachelor of Science in Applied Science degree in Allied Health and an Associate in Applied Science degree in Emergency Medical Services from Youngstown State University.

He has authored 15 EMS textbooks and more than 50 EMS journal articles. He is a frequent speaker at state and national EMS clinical and education conferences. He is a member of the Board of Directors for the Committee on Accreditation of Educational Programs for the EMS Professions. He has also served on numerous NHTSA committees and NREMT examination projects.

He is the past recipient of the Watson Award, Chairperson Leadership Award, two Distinguished Professorship Awards in Teaching, a Distinguished Professorship Award in Research and Scholarship, and the Distinguished Chairperson Award for Excellence in Scholarship.

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