

Ask the Clinical Instructor

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

Questions are answered by:
Todd Ginapp, EMT-P, RCIS, FSICP

Todd is the Cardiology Manager for Memorial Hermann Southeast in Houston, Texas. He also teaches an online RCIS Review course for Spokane Community College, in Spokane, Washington, and regularly presents with RCIS Review Courses.



While monitoring during cases, we notice that the compensatory and non-compensatory pauses create different pressure levels. Why is this?

—RCIS Review Course Participant

Yes, they often do. We'll try to explain why, briefly.

A detailed explanation of the ECG identification of what is non-compensatory versus compensatory can be complex and is beyond the scope of this article. We would like to know whether the "cause of the pause" is atrial or ventricular. Simply put, premature atrial contractions (PACs) often do not display compensatory pauses. The presence of ectopic P waves, usually absent of a full compensatory pause (R-R interval containing the premature contraction is <2 times the R-R interval of basic rhythm — see Figure 1) and a relatively narrow QRS complex morphology help differentiate PACs from premature ventricular contractions (PVCs). If the sinus impulse is able to conduct despite the PVCs, then the PVC is termed "interpolated" and no compensatory pause occurs. Remember, this is a brief explanation of a complex conduction system scenario. Exceptions can, and do, apply.

In the heart, those complexes that cause a compensatory pause are those whose impulse does not reach the normal (sinus) pacemaker. Lack of the compensatory pause is generally attributed to the impulse from the premature complex conducting retrograde towards the sinus node and resetting it. Although this is by no means a fixed rule, PACs (and premature junctional contractions, or PJC) tend to reset the sinus, while PVCs tend not to reset the sinus. Thus, if a

premature complex is followed by a compensatory pause, you should suspect a PVC.

For the remainder of this article, let's focus on the PVC and associated compensatory pause, because it has a more dramatic effect on the hemodynamics we see on the monitor.

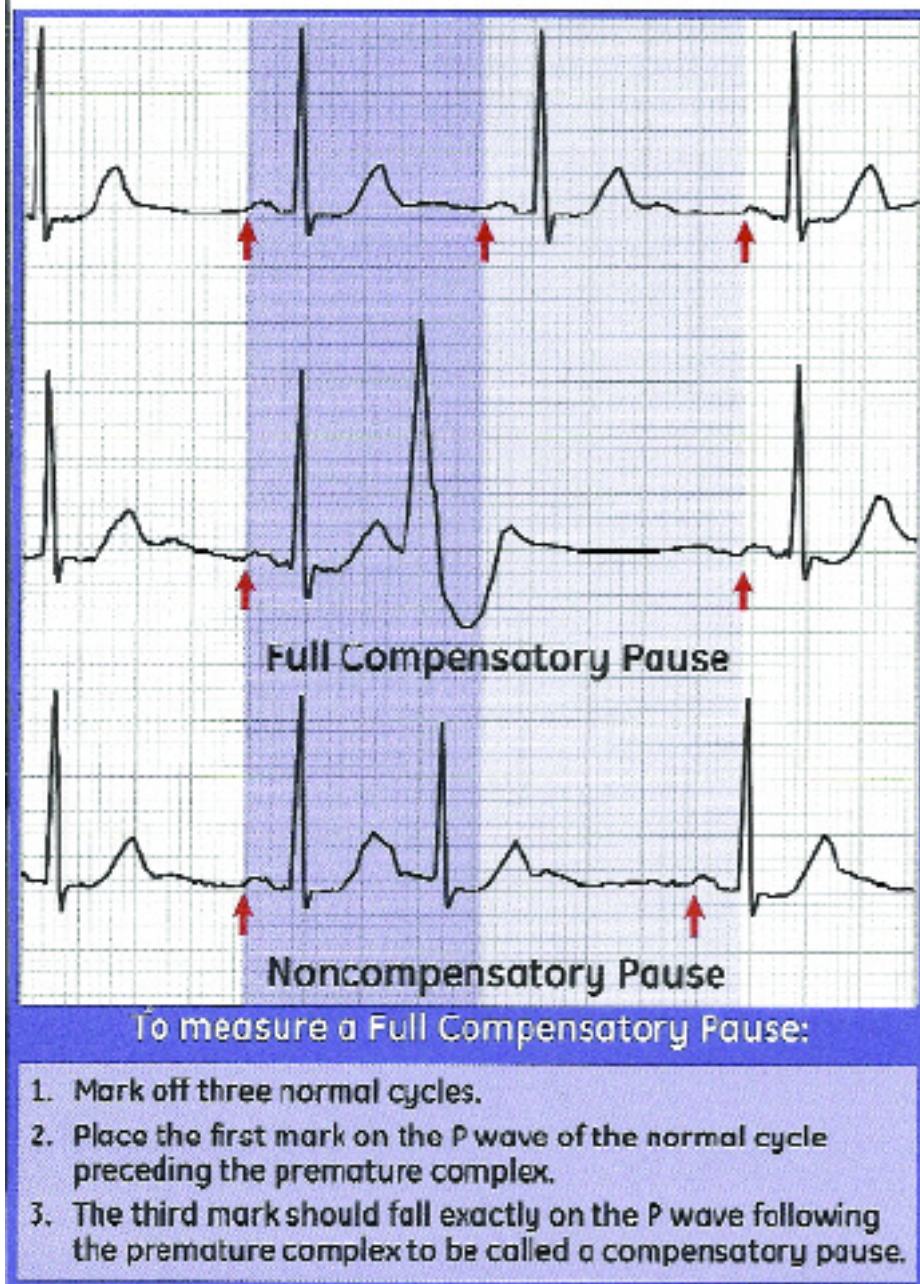
During a PVC and the associated compensatory pause, we see a demonstration of the Frank-Starling Principle. In this rule, the assumption is that the heart will pump out all the blood returned to it (preload). During the compensatory pause, there is an increased diastolic filling period as a result of the pause.

With the heart expanding and filling up more than normal, the next beat (assuming it is a sinus beat) will have more blood to pump, creating a higher pressure than preceding sinus beats. If you have ever had a PVC, you would notice this as the "hard thump" or palpitation that doesn't "feel right."

In Figure 4, we see that there is a slight compensatory pause (refer to Figure 1) and that the standard pressure or the normal beats is approximately 120mmHg. The post-PVC beat is approximately 140mmHg. This demonstrates the increase in blood that the heart accumulates during the extended diastole during/after the PVC, and how the healthy heart attempts to eject all the blood it has received. It also requires a more forceful contraction, which we see as an increase in the pressure.

While we are thinking about "healthy hearts," this is a great place to interject something that we might see during an "unhealthy heart" condition called hypertrophic obstructive cardiomyopathy (HOCM).

Figure 1. Full Compensatory Pause vs. Noncompensatory Pause



If you review Figure 1, you will see that these PACs do not create a compensatory pause.



With this strip, it is difficult to tell whether a "true" compensatory pause is created. A PJC? (Refer to Figure 1) As seen at www.gehealthcare.com

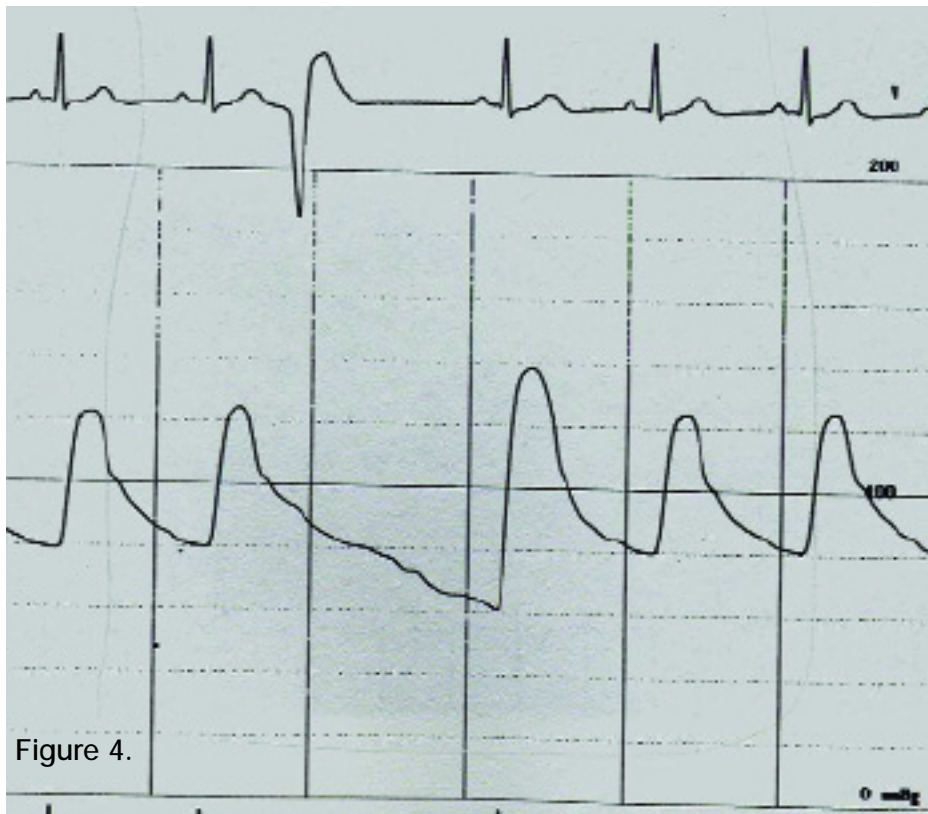


Figure 4.

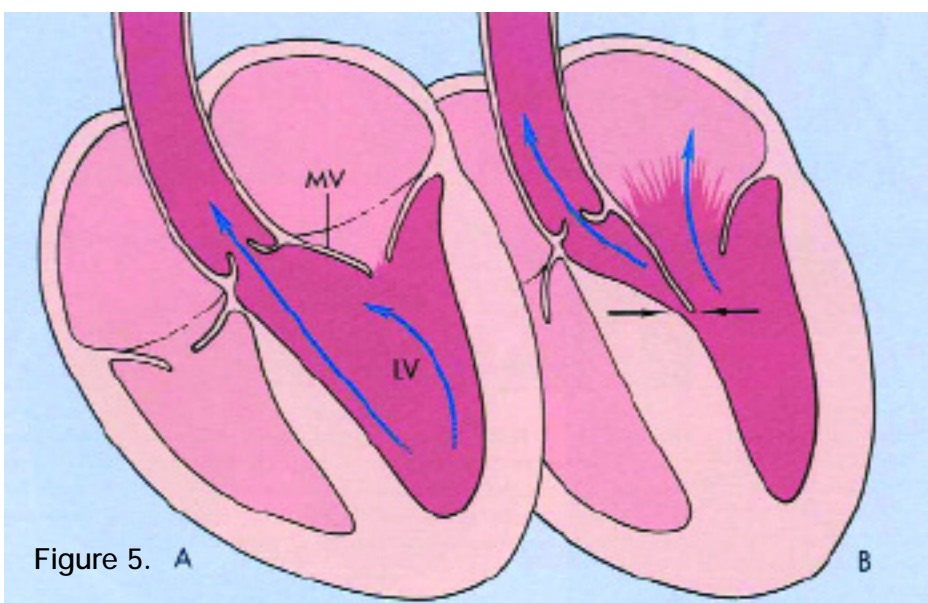


Figure 5. A B

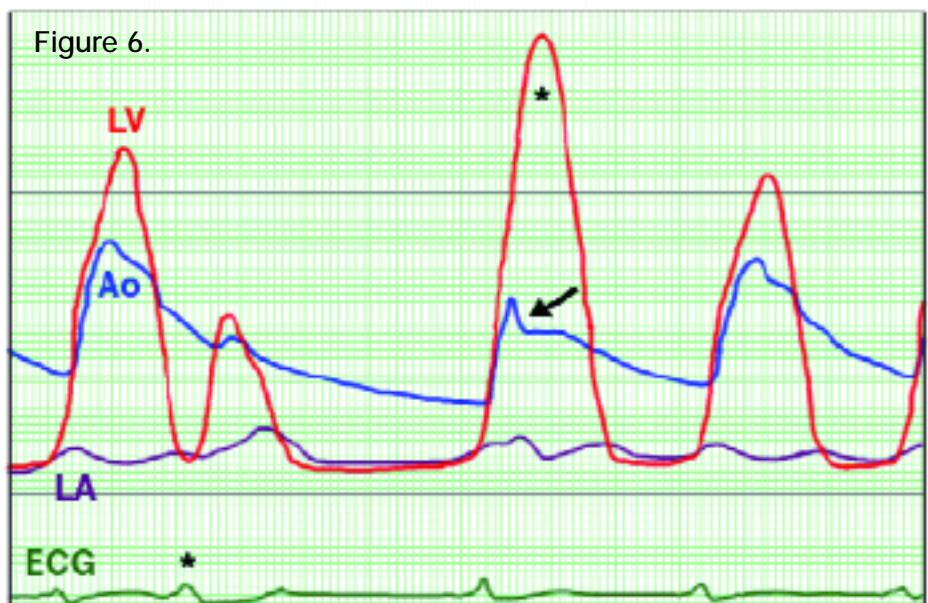


Figure 6.

"The Brockenbrough response is due to the marked increase in contractility and decrease in afterload that happens after a pause following a PVC (*), resulting in an increase in the gradient and a decrease in ascending aortic pressure. In this patient, the beat after a premature contraction is shown (* on LV line) with an increase in gradient across the outflow tract. There is a spike and dome pattern on the ascending aortic pressure trace (arrow) with a small pulse pressure."

Source: Braunwald's Heart Disease, 6th edition.

In the normal heart (Figure 5, image "A") during systole, the flow of blood moves freely from the left ventricle through the aortic valve to the body. On the right (Figure 5, image "B"), in a diseased heart with HOCM, the pathway is obstructed by an enlarged septum (indicative of chronic failure) and sometimes the mitral valve. This creates less flow "out" during systole because of the obstruction, as well as an intracavitary gradient that could be visible on pullback (a topic which could be a whole section by itself).

The **Brockenbrough sign** is observed in HOCM patients with outflow tract obstructions and a weakened heart. The weakened heart can not totally rely on the Frank-Starling principle, as the myofibrils have been nearly stretched to their limit and there is an obstruction caused by the bulging septum. The result is that the left ventricular pressure increases and the ascending aortic

pressure decreases, with an increase in the intraventricular gradient.

In Figure 4, we saw the increase in aortic pressure after the PVC. Figure 6 shows the 'abnormal' response of the aortic pressure, LOWER after the PVC, which is indicative of an obstructed/failing heart.

While the Brockenbrough sign is most dramatically demonstrated using dual catheters or a dual-chamber recording catheter (for example, Vascular Solutions' Langston Dual-Lumen pigtail catheter) it can be seen on routine physical examination as a decrease of the pulse pressure in the post-PVC beat in individuals with HOCM.

Understanding these principles can bring you a step closer to the basics of hemodynamics of the healthy, and unhealthy, heart. ■

Ask your question at tginapp@houston.rr.com

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