

# 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.

**“We see people with myocardial bridges, but nothing is done for them. What is the bridge and what is its significance?”**

— email from an online RCIS review student

**M**yocardial bridges can be quite impressive and quite dramatic. Even the newest of cath lab team members can see that “something isn’t right.” The clinical significance of this phenomena is questionable, however, and we’ll explain why.

The normal coronary artery is an epicardial artery. It normally sits on top of the myocardium. Small branches come off of the artery and penetrate into the myocardium creating the microvasculature, which we have talked about in the past few articles.

“Bridging” is an anomaly in which part of the artery has been encapsulated by the myocardium itself (Figure 1). Instead of laying on top of the myocardium, it is IN the myocardium. This will result in squeezing of the artery during systole (Figure 2). The artery looks normal in diastole when the myocardium is relaxed (Figure 3). The distances of these encapsulations can be anywhere from a few millimeters up to 3 centimeters or more. The longer the bridge, the more dramatic it is on angiography. This can occur in 5–12% of patients and is almost exclusively seen in the left anterior descending (LAD) artery.<sup>1</sup>

Blood flow limitations severe enough to cause ischemia (at rest) or myocardial infarction are unlikely. To realize this concept, we need to revisit coronary blood flow physiology. In the LAD and circumflex, blood flow through the artery to the myocardium occurs in diastole. We think of blood moving around during systole, so this can seem perplexing

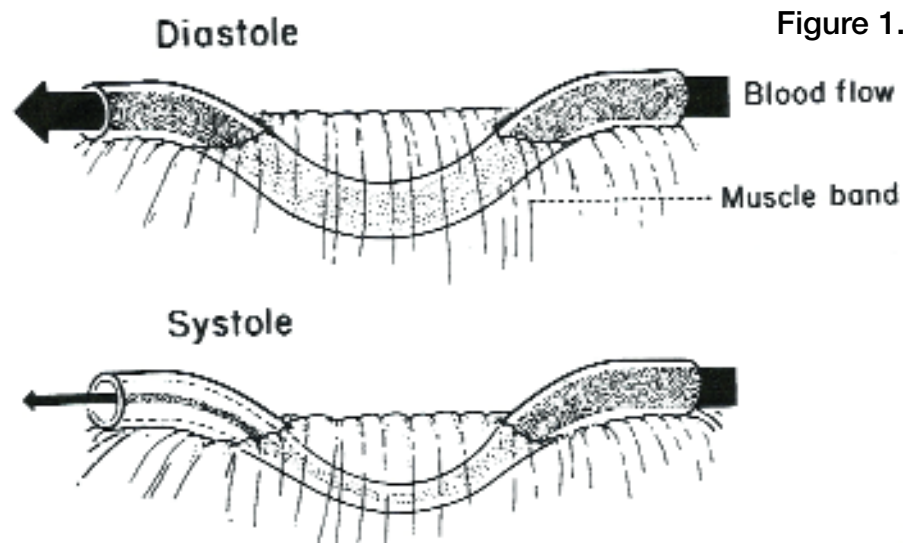
at times. Let’s clarify further.

In previous articles, we talked about how the microvasculature that is in the myocardium can become ‘plugged,’ preventing flow. During systole, this same lack of blood flow can occur, but this time it is reduced because the vessels are being squeezed by the heart in systole.

If you clench your fist, you cannot run water through it. However, during diastole (or “open fist”), the myocardium has relaxed and the blood can flow freely through the vessels and the microvasculature. It should be noted that this concept does not apply to the right coronary artery (RCA). The myocardium covered by the RCA has less muscle mass and does not affect the flow as much.

Some severe bridging can be associated with angina, stress test-induced ischemia and infarction. Most of these occur in cases with the myocardium in a state of hypertrophy. These large hearts are at the end of their compensatory mechanisms, so when there is a state of increased contractility, in exercise for example, the worsening compression of the vessel may produce symptoms. However, these reports appear to be extremely rare.

The female patient from Figures 2 and 3 presented to the cath lab with atypical chest pain.



From Gorlin R. Dynamic Vascular Factors in the genesis of myocardial ischemia. *J Am Coll Cardiol* 1983 Mar;1(3):897-906.

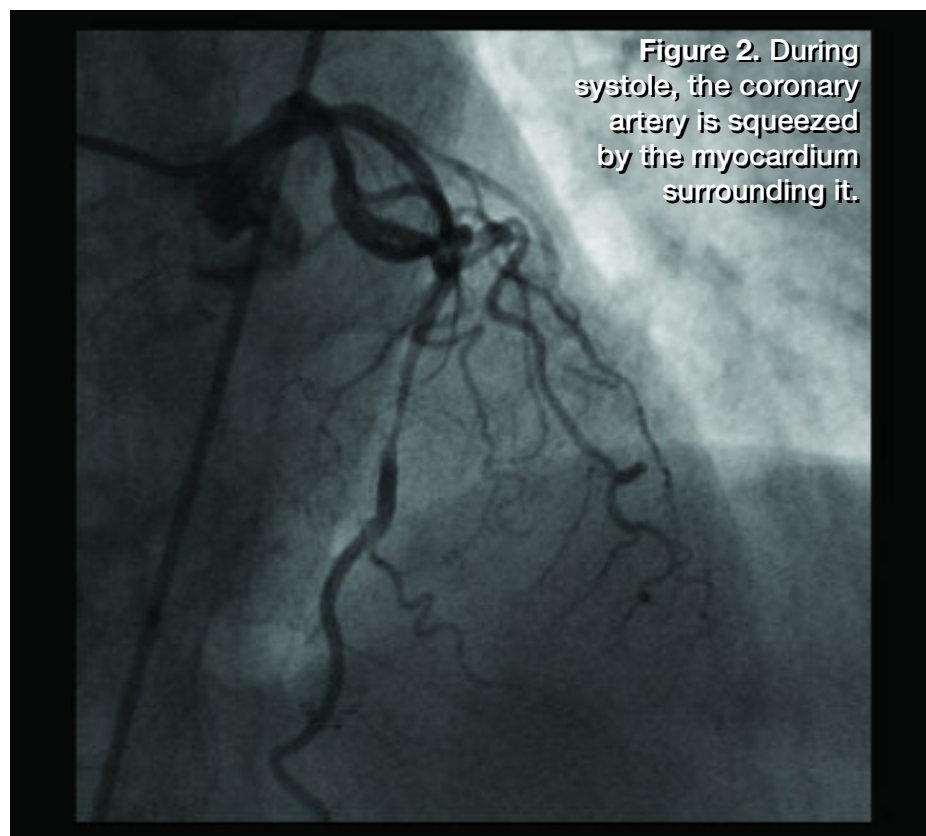


Figure 2. During systole, the coronary artery is squeezed by the myocardium surrounding it.

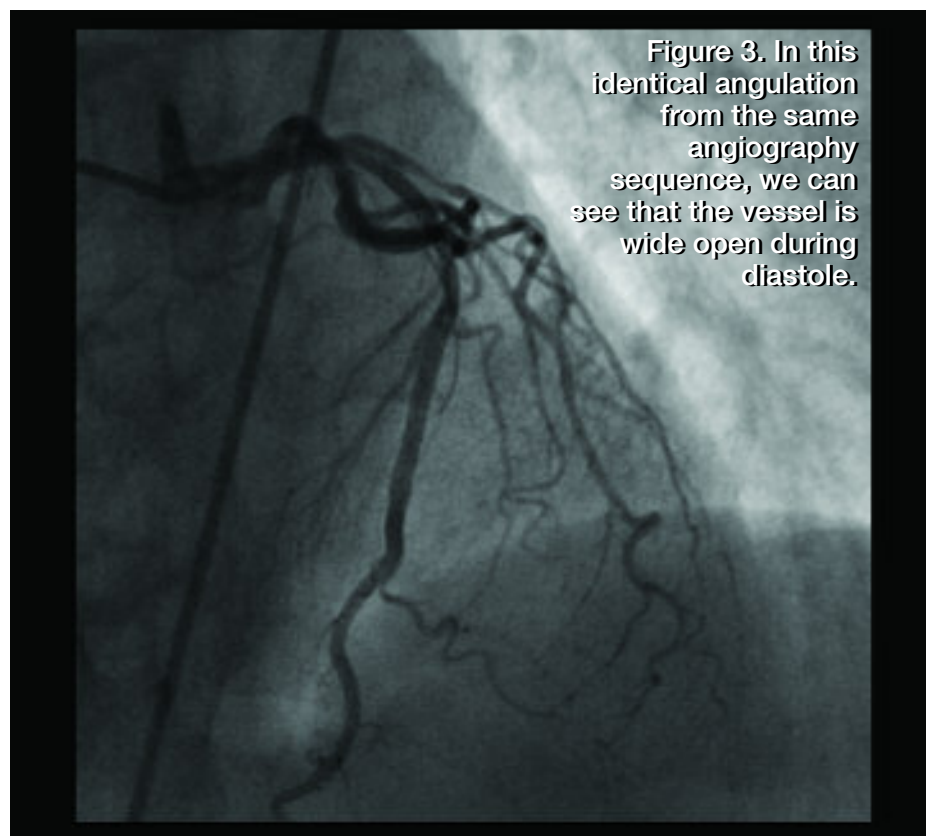
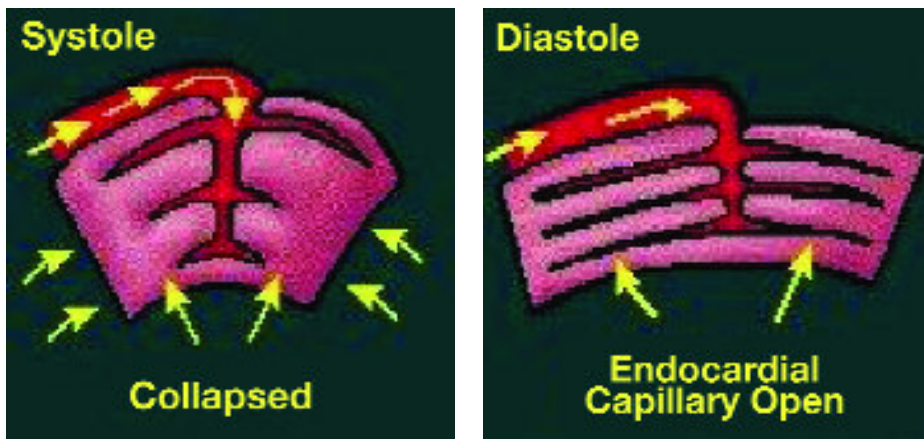
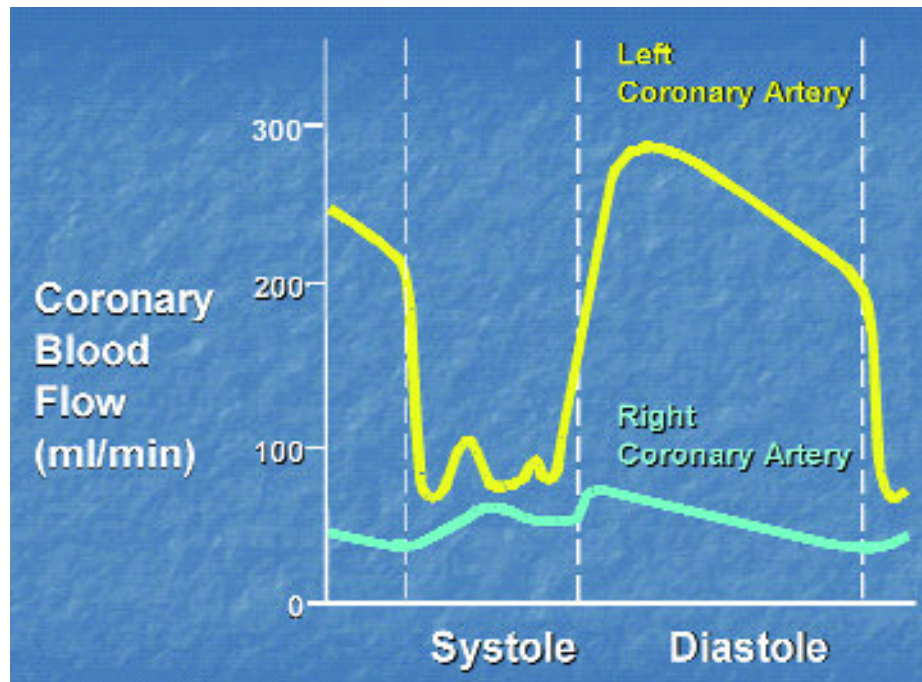


Figure 3. In this identical angulation from the same angiography sequence, we can see that the vessel is wide open during diastole.



Courtesy of Wes Todd, Cardiac Self Assessment, at [www.westodd.com](http://www.westodd.com)



This illustration shows the differences in flow between the LAD and RCA during systole and diastole.



Dr. Avinash Bapat, center, reviews an angiogram with Ana Thompson, RT and Sylvia Felan, RT.

“This patient did not need treatment as there was likely no relation between the chest pain and the bridging,” stated Dr. Avinash Bapat. “The patient will be treated with NTG and beta blockers to keep the heart rate and cardiac workload down. There should not be any complications and the patient should be able to continue her lifestyle.”

The difficulty in establishing an appropriate treatment for patients with a bridge arises from the fact that they are not lesions and therefore do not respond to balloon angioplasty. Stents are a difficult choice because of possibility of fracture and a worse situation than what was initially present. Imagine a stent placed from “healthy-to-healthy” tissue on each side of the

bridge. At the point of vessel compression, the stent would bend just a little bit. Imagine a paperclip that you bend back and forth 60 times a minute. Eventually, it would break. The same thing could happen with the stent.

Once in a while, we see studies or trials that indicate a surgical resection of the vessel to free it from the myocardium can be used. It appears that this is reserved for the most serious of symptomatic bridging patients. For now, medical management, or no treatment at all, is the appropriate course for non-symptomatic patients. ■

*Next month, we will address a question about respiratory effect*

*on intracardiac pressures. Please keep emailing your questions to [tginapp@rcisreview.com](mailto:tginapp@rcisreview.com).*

*Note: to see the angiograms from this article in “real time,” please visit:*

*[www.rcisreview.com/AskTheInstructorSeptember2007.htm](http://www.rcisreview.com/AskTheInstructorSeptember2007.htm).*

#### Reference

1. Braunwald E, Zipes DP, Libby P, Bonow R, eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 7th ed. Philadelphia, Pennsylvania; 2004.