

# Ask the Clinical Instructor

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

**Questions are answered by:  
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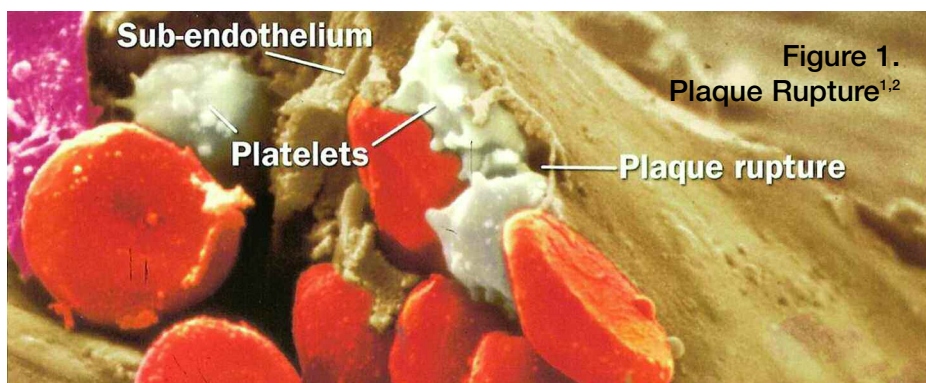
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**During angioplasties, the doctors with whom I work are often asking for different medications to be administered through the coronary catheter. Just when I think I have it figured out, the next doctor uses yet a different medication. What is the reason?**

**—Email received at  
tginapp@RCISreview.com**

you have encountered the scenario where a stent was placed in a relatively well-flowing artery, only to have the next contrast injection result in contrast “hanging” (no reflow) in the artery. If the contrast isn’t moving, neither is the blood flow. This occurs as a result of impairment of the microvasculature within the heart vessel circulation, most commonly



**Figure 1.  
Plaque Rupture<sup>1,2</sup>**

This is one of the most frustrating parts of the job for the technologist or RN. Just when you think you know what might be going on, something changes! However, in this case, things haven’t really changed on you. Here’s why. The physicians are using these medications to help prevent or actually treat slow — or no — reflow (which we will refer to as “no reflow” in this article). There are numerous medications physicians can use and as with catheters and wires, each physician has their own idea of what works best, based upon their education and training, studies and trials, or personal experiences. Be comforted that even though the doctors are using different medications, they are all trying to accomplish the exact same thing.

If you have been participating in angioplasties for any period of time,

within the distribution area of the infarct. It can be caused by endothelial swelling, inflammation, edema, tissue compression or foreign body obstruction. Here we will focus on the most common reason, which is foreign body obstruction from plaque and clot.

All acute myocardial infarctions (AMIs) are caused by some degree of blood clot (Figure 1). STEMIs (ST elevation MIs) are caused by a plaque rupture with resultant clotting at the site of the injury. During this clotting sequence, clot can spontaneously move “downstream,” if not at the time of injury, then almost certainly during the passage of the coronary wire, balloon or stent.

During the post-balloon period, clot and plaque can move downstream as well. When the stent is deployed, a “cheese-grater” action

can occur on the remaining plaque at the lesion site. The plaque and/or clot can fragment, and parts of it flow downstream. When the microvasculature is blocked, there can be reduced flow to the myocardium, which can result in localized hypoxia to a certain portion of myocardium (based upon the extent of the obstructions). Of course, this is not desired, as it can either worsen the existing infarct area or even create an additional infarct area. Studies have shown that patients with no-reflow phenomenon have a poor clinical prognosis.<sup>3</sup> The impaired perfusion is associated with adverse left ventricular remodeling, heart failure and reduced survival.<sup>4</sup>

Basic anatomy reminds us that arteries turn into arterioles, and arterioles turn into capillaries. At the capillary level, we call it the “microvasculature.” As we can see



in Figure 2, the capillary bed is intended to have a small passageway for red blood cells (RBCs) to pass through, one at a time, to promote efficient exchange of oxygen with the capillary wall.

If we know that the capillaries are designed for single RBCs to pass through (as we saw in Figure 2), what happens if a foreign body larger than the RBC makes it this far? Of course, it will become lodged and block the blood flow. If a clot or plaque has broken into many pieces, it will now shower debris downstream and block many of the capillary pathways.

By administering the appropriate medication(s), either in advance of ballooning/stenting, or after ballooning/stenting, the goal is to dilate the microvasculature. Smaller pieces that are blocking the flow could then “pass through” to the venous side (Figure 3). Another thought is that if there is some debris blocking the flow, then maximizing the flow capability of the unblocked portions of the microvasculature will be beneficial to the patient.

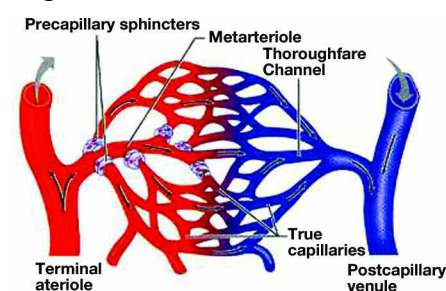
It’s important to note how the ECG relates to reperfusion during angioplasty. The rapid reduction in ST-segment elevation after reperfusion therapy indicates early, full and prompt restoration of myocardial tissue perfusion.<sup>5</sup> Failure to see a reduction of ST elevations or a return of ST elevation after balloon/stenting is quite indicative of a no-reflow state. The physician will administer medications to dilate the “plugged” microvasculature, as well as to help reduce the clot burden that may be present.

Reducing the clotting ability of the body through administration of medications (heparin, enoxaparin sodium [Lovenox, sanofi-aventis, Bridgewater, NJ] or bivalirudin [Angiomax, The Medicines Company, Parsippany, NJ], for example) will help with early clot reduction. Administration of anti-platelet therapy (aspirin, clopidogrel [Plavix, Bristol-Myers Squibb/Sanofi Pharmaceuticals Partnership]) and glycoprotein (GP) IIb/IIIa inhibitors (such as abciximab [ReoPro, Eli Lilly & Co., Indianapolis, IN] or enoxaparin) will help with the resolution of clot burden over the first few hours after a procedure. IIb/IIIa inhibitors are also good at increasing microvasculature flow that has been obstructed by clot. It should be remembered that it can take some time, during which there will be reduced flow to the affected myocardium.

Clot removal can also occur through the use of a thrombectomy device (AngioJet [Possis Medical, Inc., Minneapolis, MN], Export Catheter [Medtronic, Inc., Santa Rosa, CA], Pronto Catheter [Vascular Solutions, Minneapolis, MN] and Rescue Catheter [Boston Scientific, Natick, MA] are all examples of this type of device). This is generally done at the beginning of a case, but after a wire is placed in the artery. At that time, some clot inevitably will be pushed downstream.

Dilating the microvasculature can be accomplished with numerous

Figure 3.



medications, including calcium channel blockers, nitrates and potent vasodilators, with some of the more common ones listed in Table 1. The specific pharmacological actions of each are beyond the scope of this article. All are an attempt to achieve the same result, which is vasodilation of the microvasculature and restoration of normal coronary flow.

The physician can administer these medications via the coronary catheter or actually through the balloon, placed as far distally as possible. This assures that the maximum administration is delivered distally, instead of to branches that do not have the flow problem. The RN and technologist should communicate diligently about the “table” dose of the medication, as it can be significantly different than the IV dose that the RN may be used to. It is helpful to confirm the desired concentration of the medication with the physician prior to preparation, and write that concentration on a label and place it on the syringe. All are potent medications that can cause problems if too much is given. Contact your department educator for specific education on the pharmacokinetics of these medications. ■

*Next month: A question concerning TIMI flow assessment and how it relates to this topic.*

To see images of examples of slow/no reflow, please visit <http://www.rcisreview.com/AskTheInstructor.htm>. Images will load upon your arrival.

Table 1.  
The more common  
pharmacological agents  
for no reflow situations

Verapamil (Calan)  
Nicardipine (Cardene)  
Adenosine (Adenocard)  
Nitroglycerine (Tridil)  
Nitroprusside (Nipride)

Ask your question, or any question you and your co-workers have had in your cath lab, to [tginapp@rcisreview.com](mailto:tginapp@rcisreview.com)

#### References

1. Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes (2). *N Engl J Med*. 1992;326:310-318.
2. Photos courtesy of Boehringer Ingelheim International GmbH, by Lennart Nilsson, Braunwald E, et al. 2002. <http://www.acc.org/clinical-guidelines/unstable/unstable.pdf>
3. Ito H, Maruyama A, Iwakura K et al. Clinical Implications of the ‘no reflow’ phenomenon. A predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction. *Circulation* 1996;93:223-228.
4. Braunwald E, Rezkalla SH, Kloner RA. No-reflow phenomenon. *Circulation* 2002;105:2332.
5. Weherens X, Doevendans PA, Ophuis TJ, et al. A comparison of electrocardiographic changes during reperfusion of acute myocardial infarction *Am Heart J* 2000;139:430-436.

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