Discussion to: Infarct exclusion repair of postmyocardial infarction ventricular septal rupture with a hybrid patch and septal occluder device compared with patch only

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Presenter: Dr Adam Williams

Dr Joseph Sabik, MD (Cleveland, Ohio). [applause] Our invited discussant is Tirone David, American Association for Thoracic Surgery past president.

Dr Tirone David (Toronto, Ontario, Canada). Thank you, Dr Williams. Perhaps send me the manuscript. I enjoy reading it. But more importantly, what I enjoy is that Duke University surgeons have adopted this infarct exclusion technique, which I continue to believe that likely is the best approach to treat this disastrous disease because if you don’t do something about it, most of them died. And I assume—I am also pleased to see that you take the patient to the operating room as soon as the diagnosis is made. I think in delaying operation is looking for more problems than it’s worth. If you think they require mechanical support circulation, repair the hole first, and then put in support like extracorporeal membrane oxygenation (ECMO) or something else. But that’s the end of my comments that are positive. I don’t quite understand the rationale. Why intraoperatively put a septal occluder device 40 material where the pericardial alone can do the same thing? And I’m puzzled, their ventricular function, right ventricular function, is better in those 10 patients who had the device. I would expect the opposite. Rigid structure in the septum would be detrimental to ventricular function, mostly septal recovery and healing. But that’s such a show we have to accept the data, which is an interesting point. I’ve never done that, but I imagine the operation becomes a bit more complicated to sew a patch to exclude infarct with a septal occluder device 40 material away, particularly in posterior ventricular septal defect, where most of the suturing is done along the mitral annulus in the base of the septum. Would you care to comment about the difficulty in putting a patch down into the base of the heart?

Dr Adam Williams (Durham, NC). Well, thank you for those comments, Dr David, it’s a real honor to have you as a discussant. So yeah, I agree with you. The right ventricular function improvement is hard to explain. I mean, as you know, the 3 determinants of right ventricle function are coronary perfusion, septal function, and afterload pulmonary artery pressure. So maybe putting in this septal occluder device somehow restores some structure to the septum and improves right ventricle function. With regards to your comment about why we put it in, I think this makes it an easier repair because when we put the device in there, at least when I do it, I include when I put the patch in, the stitches go through myocardium, and then I go through the edge of the septal occluder device, so it helps anchor it in there. Because as I’m sure you know better than I do, a lot of those bites are in marginal sometimes in kind of marginal endocardial or myocardial tissue tears through pretty easily, so it’s nice to have this structure in there to include in your bites for the patch repair.

Dr David. So, the septum is dead. That’s why it ruptured. Have you ever seen dislodgement of the device a week or 10 days later when the myocardial is replaced by clot and fibrosis?

Dr Williams. Yeah, so we have not seen any of these dislodged. They’re included in the securing of the patch to the myocardium, so it would be very difficult for it to dislodge and go anywhere because we really incorporate our bites in the myocardium to include the septum.

Dr David. But you don’t sew the patch to the device either, only the muscle, isn’t it?

Dr Williams. The patch goes on to the septal occluder device—so I usually will take a bite of the muscle and
then through the edge because the septal occluder device has a waste. And then there’s a 10-mm oversizing on each of those discs. So, the actual disc is lying on healthy myocardium. So, we incorporate myocardium and the septal occluder device in the patch closure.

Dr David. Well, that’s all very interesting and I’ll try next time. Unfortunately, we don’t do very many anymore. When I was a young surgeon, I’m not exaggerating, I was doing this operation at least once or twice a month. Now I see it once or twice a year. So, it’s very difficult to become an expert in an operation if you see it twice a year. But thank you very much for your contribution.

Dr Williams. Thank you. [applause]

Unidentified Speaker 2. I agree with Dr Tirone David, and I’m humbled to follow him. And I congratulate you on a great presentation. It’s becoming rarer, but around COVID time, in 2020 and 2021-22, we started seeing more of those again. I don’t know what the experience of everybody else. But presumably, because patients were scared to go to hospitals. And many of them instead of undergoing percutaneous coronary intervention on an emergency basis experienced myocardial infarction or ventricular septal defect. Putting this in place close to the mitral apparatus, although it might make sense that you’re providing space and scaffold to show, mechanistically the mechanics of it is not compelling for me at least. My question is this: Many of them come in with shock. The ones who scare me are the shock with end-organ failure. Because they get transferred from another hospital, they have renal shutdown, the bilirubin is up. You said you put balloon pumps, but many of those patients balloon pumps are not enough. Can you comment on those patients specifically that you hesitate to take to the OR right away because renal shutdown, bad liver, yet if you don’t operate on them, outcome is a 100% mortality.

Dr Williams. That’s a challenging problem when they show up in shock with end-organ failure. We use the balloon pump as just a temporizing measure to get them to the operating room. It isn’t a definitive treatment. And we don’t usually do these in the middle of the night. So, when they come in, if they come in late in the day, we’ll put the balloon pump in and then do them as a first case the next morning. In the patch septal clot patient cohort, we didn’t have any that we put them on ECMO and then took them to the operating room. There was 1 in the patch-only group where we put them on ECMO and then took them to the operating room. And that patient ultimately died. But there were, in both cohorts, 2 patients who had ECMO support, perioperatively.

Unidentified Speaker 2. So, were there patients that were just too sick to take to the operating room?

Dr Williams. Nonoperative weren’t included in this study. We only included in the study patients with postmyocardial infarction ventricular septal defects who underwent operative repair with either patch alone or patch/septal occluder device.

Unidentified Speaker 2. Do you have that number, do you have that denominator?

Dr Williams. No, I don’t have that denominator.

Unidentified Speaker 2. Okay, just a thought. When those patients present to us, we usually put in an Impella device (Abiomed) and wait for the end-organ dysfunction to reverse. And then we take them to the operating room. It used to be a traditional teaching that you wait 4 to 6 weeks for the tissue to strengthen. But in our experience, 2 weeks is long enough. And we take them to the operating room with the Impella in place. The tissues, surprisingly, are better at 2 weeks. And we don’t have to use that device. But thanks for an excellent presentation.

Dr Williams. Thanks.

Unidentified Speaker 3. Yeah, Sandozo from Syracuse. The way I should put these people on ECMO for a few days. And the interesting phenomenon we notice is that initially the ventricular septal defect is say 1 cm. After a few days, it becomes 3 cm. So, my concern is with this kind of occlusion device that your size at may be, say, 1 cm and then becomes 3 cm. So, after a few days they may not do anything. So, my question for you is, what’s your recurrence? What sort of residual leak after a few days? And what’s the reason for the people who died? Because they have failed the repair again, or just multiorgan system failure?

Dr Williams. So, in both cohorts, we had only trivial residual shunts, and there were 2 of them in each cohort who had a trivial residual shunt. So, it really didn’t make any difference in terms of the residual shunt. Now, with these septal occluder devices, since they’re expandable, if that infarct hole enlarges, it’s expandable. These nitinol septal occluder device will expand and could potentially occupy more of that hole as the infarct progresses and the hole becomes larger.

Unidentified Speaker 3. Thank you.

Dr Sabik. Thank you for the great discussion. [applause]

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