Operative Techniques in Thoracic and Cardiovascular Surgery

Repair of Postinfarct Ventricular Septal Defect: Anterior Apical Ventricular Septal Defect

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A post–myocardial infarction (MI) ventricular septal defect (VSD) is a rare but frequently fatal complication, occurring in less than 1% of patients suffering MI in the era of early reperfusion therapy.[1] In patients with this complication receiving medical therapy alone, mortality rates exceed 90%, whereas in patients undergoing surgical repair, mortality ranges between 19% and 60%. Cooley performed the first open repair of a VSD in 1957. Daggett et al[2] developed an infarctectomy and patch technique in 1977, which was the standard approach used by many surgeons. The prognosis for these patients remained poor, with mortality rates of approximately 50%. David and Armstrong[3] described an endocardial patch technique with infarct exclusion in 1995, with documented reductions in operative mortality to 19% in the Toronto series. Despite advances in the perioperative management of these patients, VSD repair still has the highest operative mortality among operations reported to the Society of Thoracic Surgeons adult cardiac surgery database.[4]

The diagnosis of a post-MI VSD should be suspected by the history of a recent MI along with a new pansystolic murmur. These patients often have significant hemodynamic compromise. Some patients may have small defects that limit hemodynamic impairment, and may lead to a delayed diagnosis. Other diagnoses that should be considered in the setting of recent MI and new cardiac murmur include ruptured papillary muscle with mitral regurgitation or ischemic mitral regurgitation. A left-to-right shunt is usually present, and the diagnosis can be confirmed with either transthoracic or transesophageal echocardiography. Ventriculography performed during coronary angiography can also confirm the diagnosis.

Anterior defects typically occur in the anterior septum in patients who have left anterior descending artery occlusion. Areas of left ventricular myocardium beyond the infarction can be hyperdynamic owing to unloading of volume into the lower-pressure right ventricle (RV). However, the acute volume overload presented to the RV can lead to severe right ventricular dysfunction and congestive heart failure. In a minority of patients, VSDs are detected late after the original MI because of investigations for an incidentally discovered pansystolic murmur. In these patients, the defects are usually small and rarely cause hemodynamic compromise.

Preoperative Considerations

Without surgical intervention, the prognosis for patients with post-MI VSD is grave. Mortality ranges from 60%-70% in the first 2 weeks, with approximately 90% mortality at 3 months.[4] However, patients who undergo surgical repair in the setting of profound shock also have high operative mortality. Therefore, it is important for the surgeon to evaluate the patient carefully and optimize the cardiopulmonary status before proceeding to the operating room.

Many patients with post-MI VSD will require preoperative cardiovascular support with an intra-aortic balloon pump. The intra-aortic balloon pump improves hemodynamics, augments coronary perfusion, and may permit safer coronary angiography. Vasodilators and inotropes should be administered as necessary to preserve organ perfusion. Inhaled nitric oxide or other pulmonary vasodilators are important adjuncts in this setting to decrease pulmonary resistance. However, a reduction in pulmonary vascular resistance may increase shunting, so this must be considered before administering these agents. Preoperative coronary angiography must be performed to define coronary anatomy and the need for concomitant revascularization.[5] Extracorporeal membrane oxygenation has been used with success to stabilize patients and correct metabolic abnormalities before surgery.

The timing of surgery is critical. Although high risk, patients with continued hemodynamic decline should undergo early surgical intervention, often within 24 hours of diagnosis, to minimize the progressive organ dysfunction. Though the data are retrospective, a Society of Thoracic Surgeons (STS) report showed that patients with a longer time interval between MI and surgical repair experience improved outcomes. Therefore, patients who demonstrate progressive hemodynamic improvement can await surgical repair until the clinical condition has been optimized, typically a period of 1-7 days.

When concomitant coronary revascularization is planned, saphenous vein harvest should occur simultaneously with the sternal incision. To avoid prolonged aortic cross-clamp times, only severely stenotic coronary lesions should be bypassed. Revascularization of the occluded artery

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responsible for the VSD is of questionable benefit, as this territory generally represents nonviable myocardium. We favor performing the anastomoses before VSD repair, permitting infusion of cardioplegia through the conduit. Because the infarcted myocardium is friable, manipulation and retraction of the heart during coronary revascularization should be done carefully to prevent dislodging necrotic intracardiac muscle.

Operative Considerations

There are many different techniques that have been used successfully to repair post infarction VSD. Most techniques break down into patch techniques, either single or double, or infarct exclusion techniques. Primary repair techniques and percutaneous or hybrid techniques using closure devices are used less frequently.[6]

It is often difficult to determine exactly where the actual communication in the infarcted, hemorrhagic septum is located, and the quality of the tissue often makes suture placement difficult. Excessive debridement is generally discouraged. Placing sutures a significant distance away from the infarcted septum and generous patches is a good idea. Continuous or interrupted suture techniques may be used. Reinforcing the patches or the ventriculotomy closure with buttresses is sometimes helpful, and avoiding tension on the suture lines is imperative.

Here we demonstrate the 2-patch technique and the exclusion techniques. The quality of the superior septal tissue affects how the sutures are placed to complete the septal repair and how the 2 patches are related. The 2 patches can be completely separate or connected to reconstruct the junction between the septum and the anterior wall.

The exclusion technique is sometimes hard to visualize. A patch of bovine pericardium, dacron, or other material is used to separate the left ventricle from the infarcted septum. Some find it helpful to think of it conceptually as creating a new left ventricle by constructing a new septum parallel to the infarcted septum and leaving the old septum as part of the RV cavity (Figs. 1-14).

Infarct Exclusion Technique

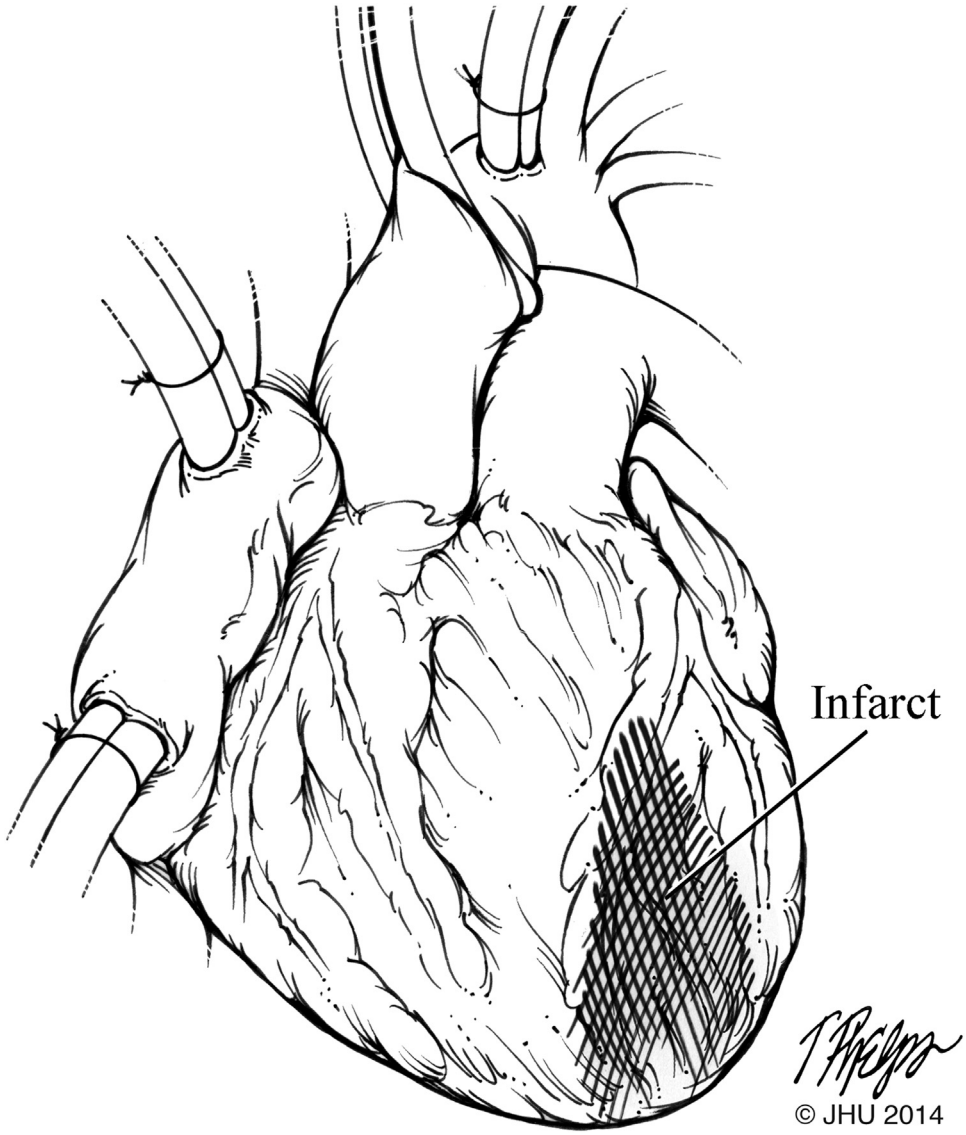

Figure 1 Infarct in the region of the left anterior descending (LAD) coronary artery. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

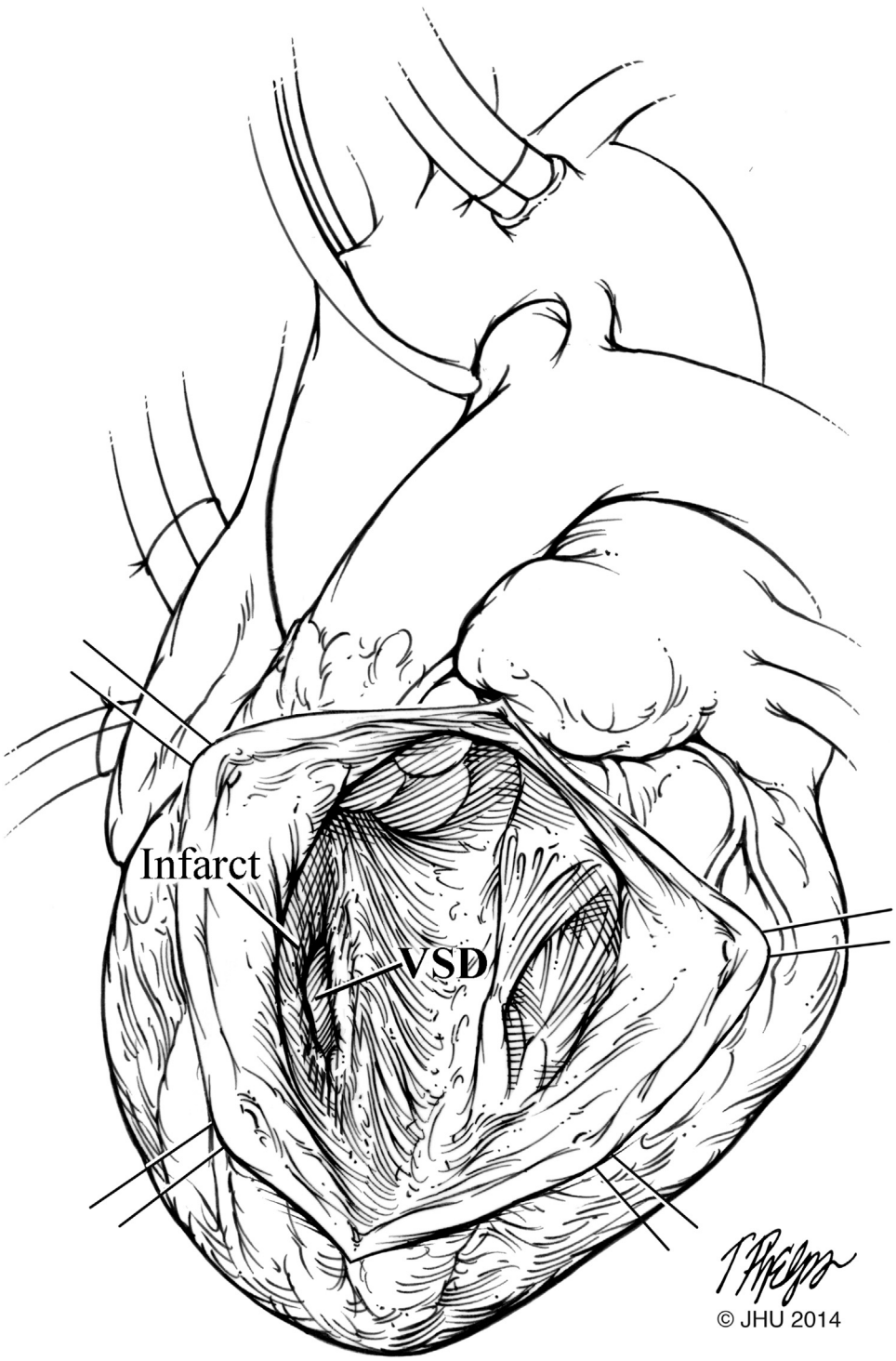

Figure 2 Anterior ventriculotomy through anterior wall infarct parallel to LAD vessel exposing infarcted septum with ventricular septal defect (VSD). LAD = left anterior descending. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

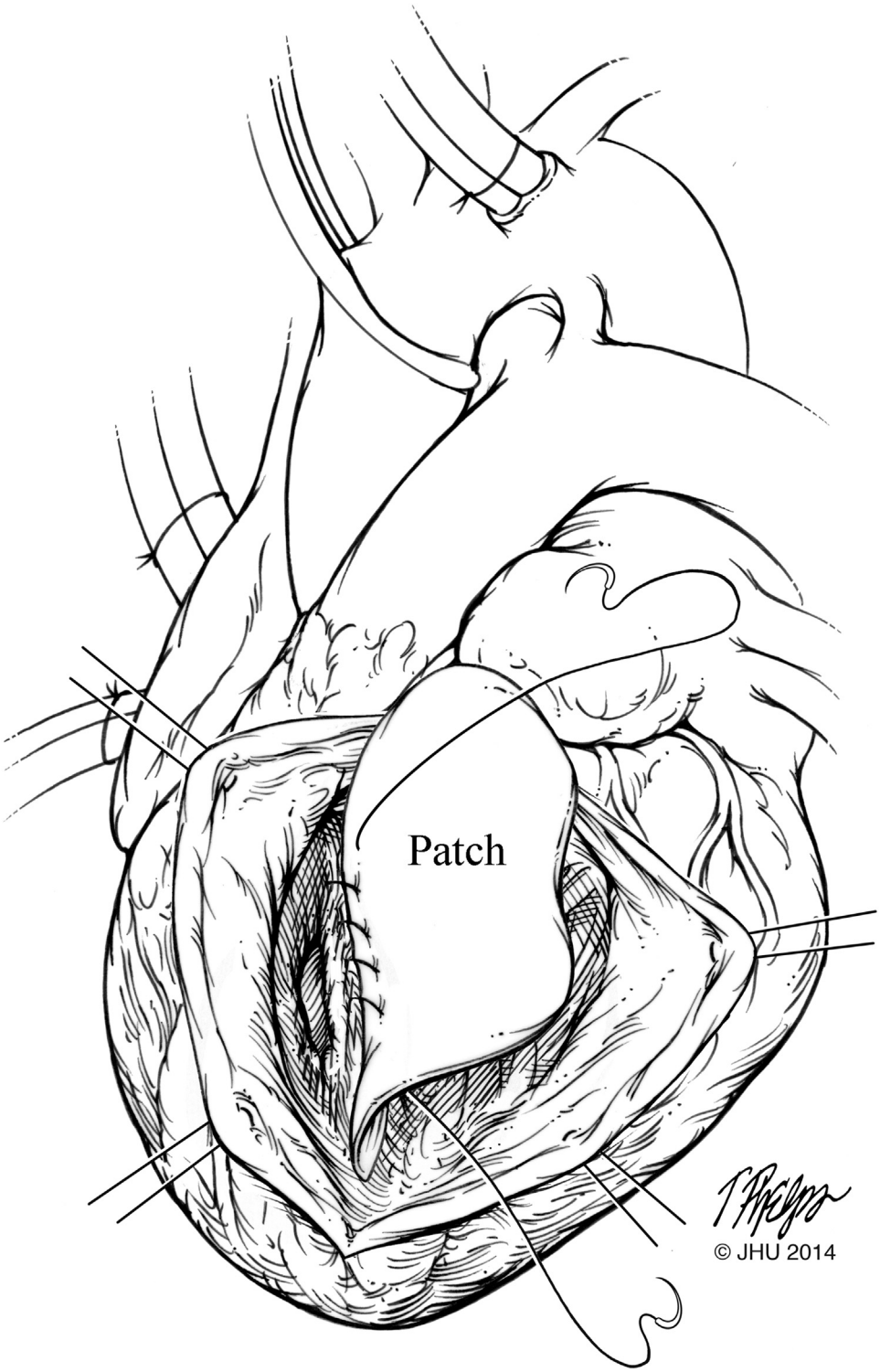

Figure 3 Patch begun in inferior border of septum. The patch will be used to create a "neoseptum" between the LV and the old septum where the VSD lies. Deep bites are taken through healthy tissue. Interrupted pledgeted sutures may be used primarily or to reinforce a continuous suture line. Drawings by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

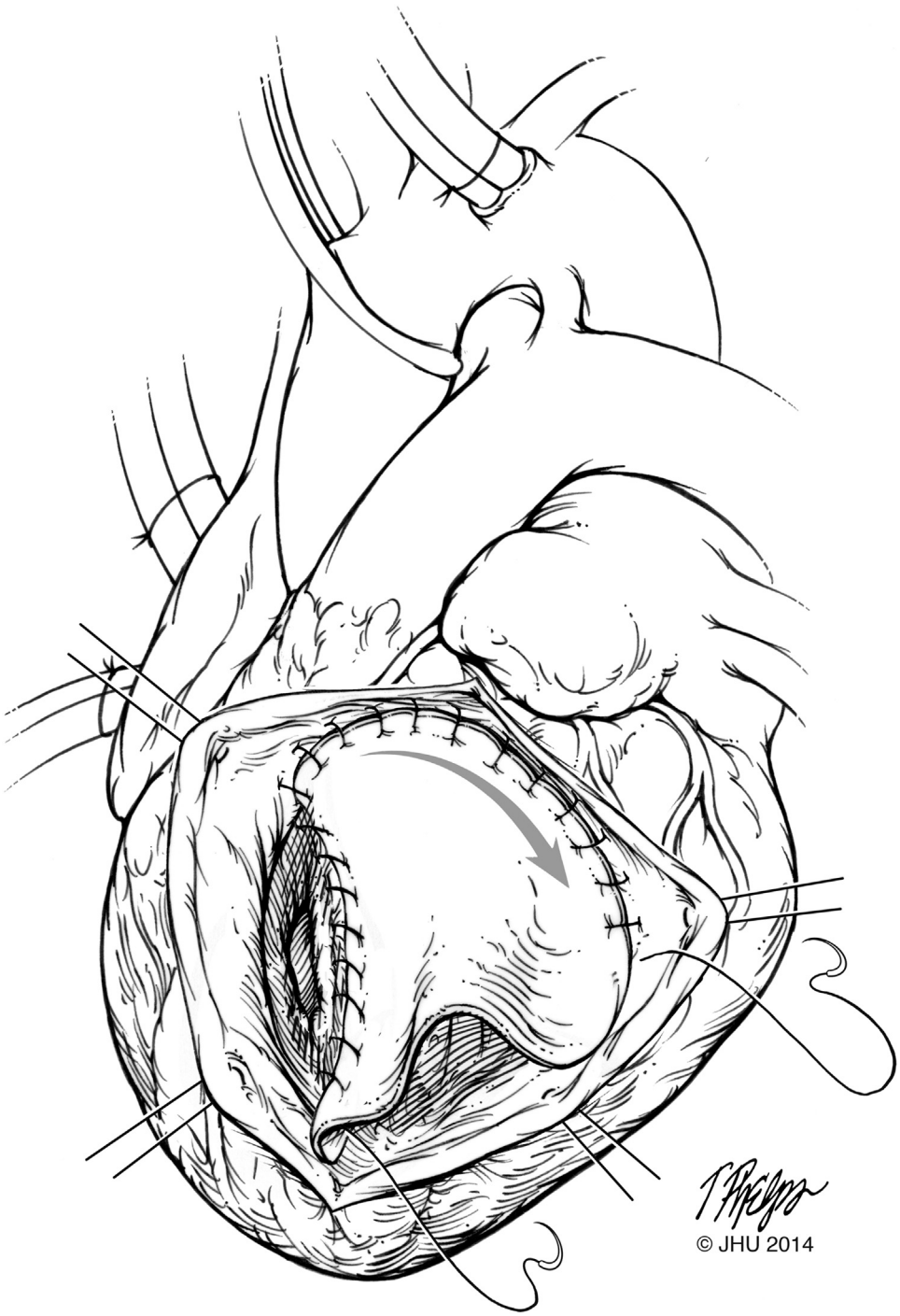

Figure 4 Suture line continued cephalad creating "neoseptum." Sutures must be passed through healthy tissue. The annulus of the mitral valve can be used to secure patch to healthy tissue. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

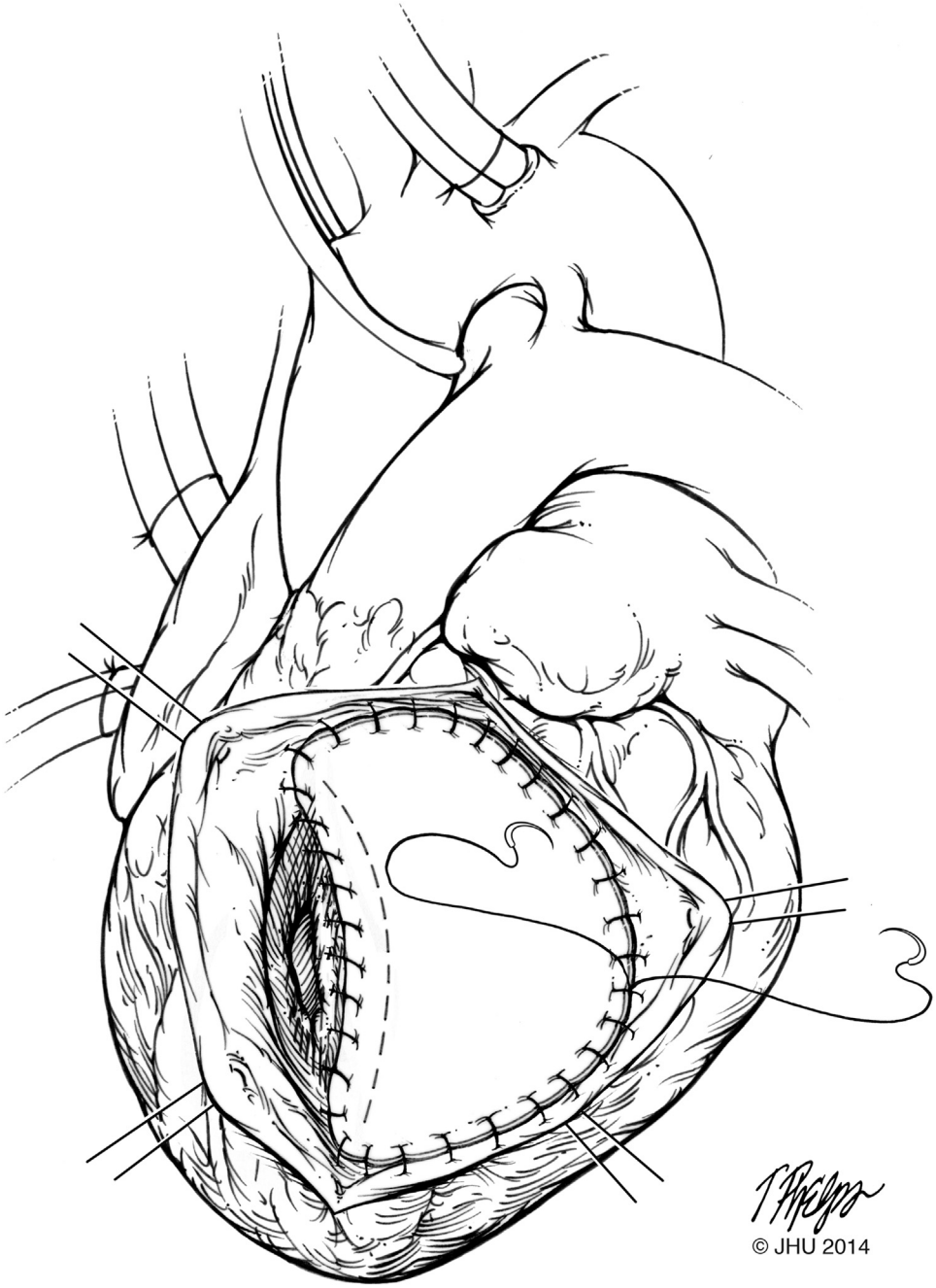

Figure 5 Continuation of suture line inferiorly and to completion. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

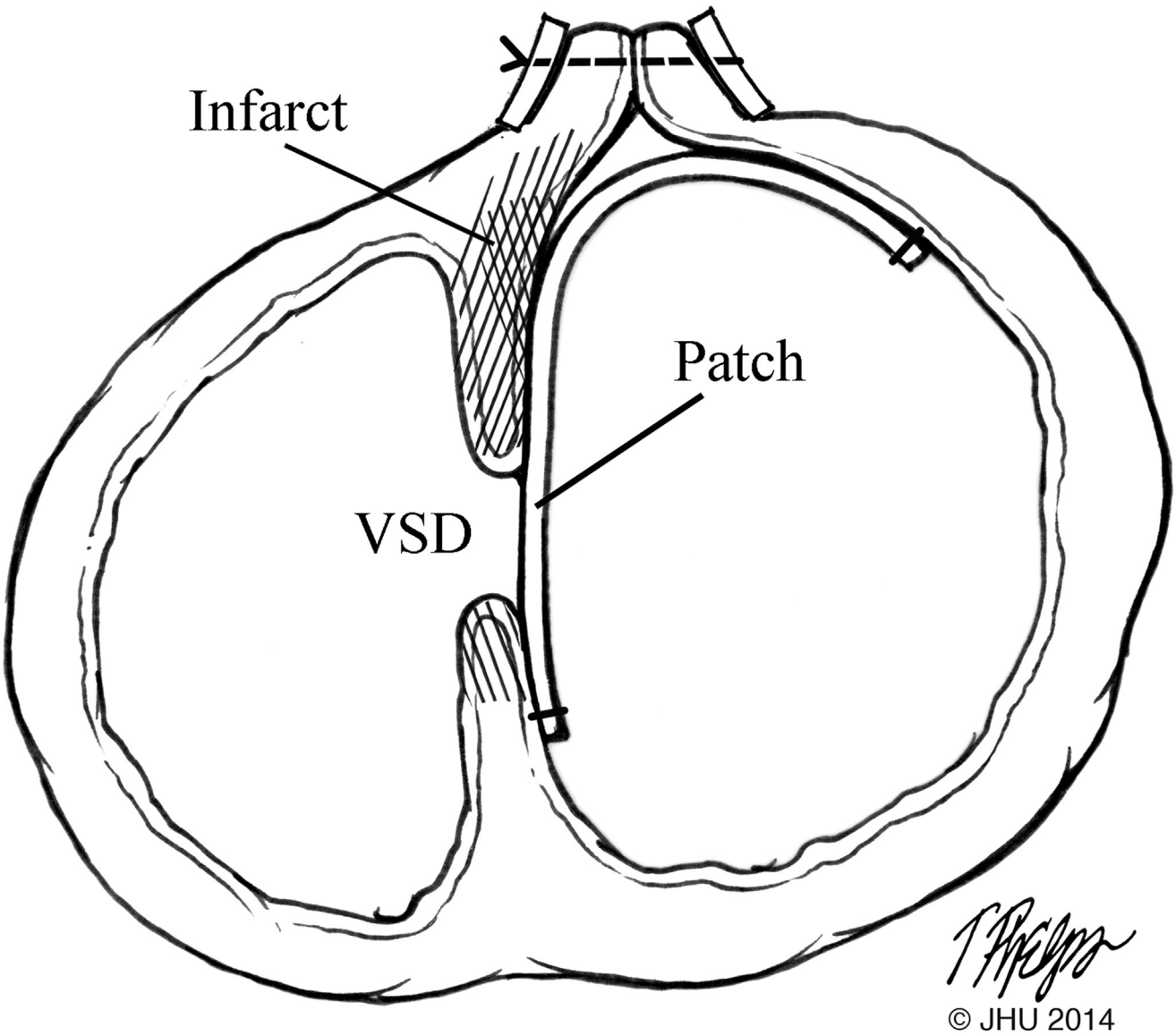

Figure 6 Cross-section of ventricle demonstrating "neoseptum" and separation of old infarcted septum from high pressure LV. VSD = ventricular septal defect. Drawings by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

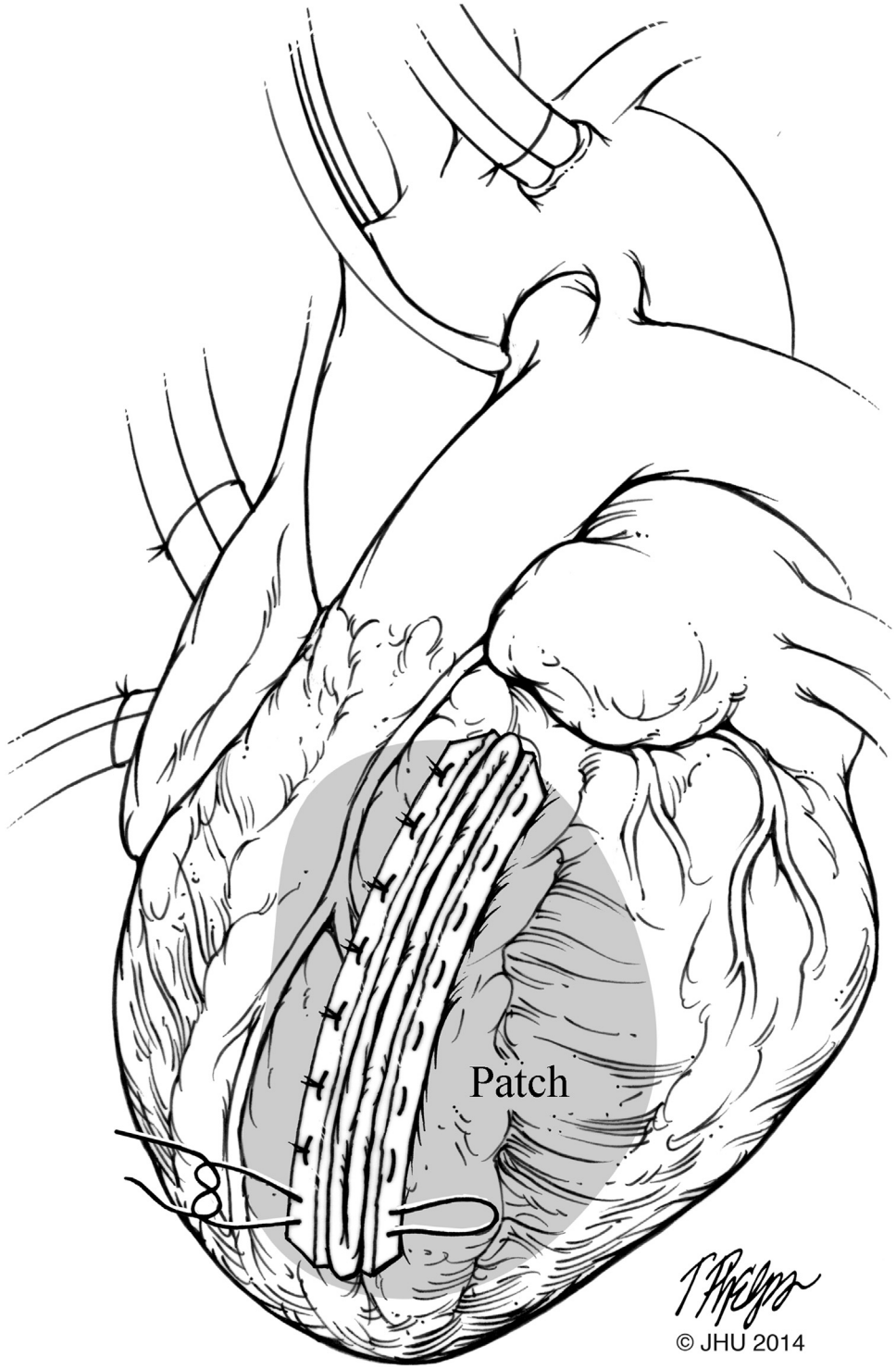

Figure 7 Completion of repair by linear closure reinforced with strips of felt or pericardium. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

Two Patch Technique

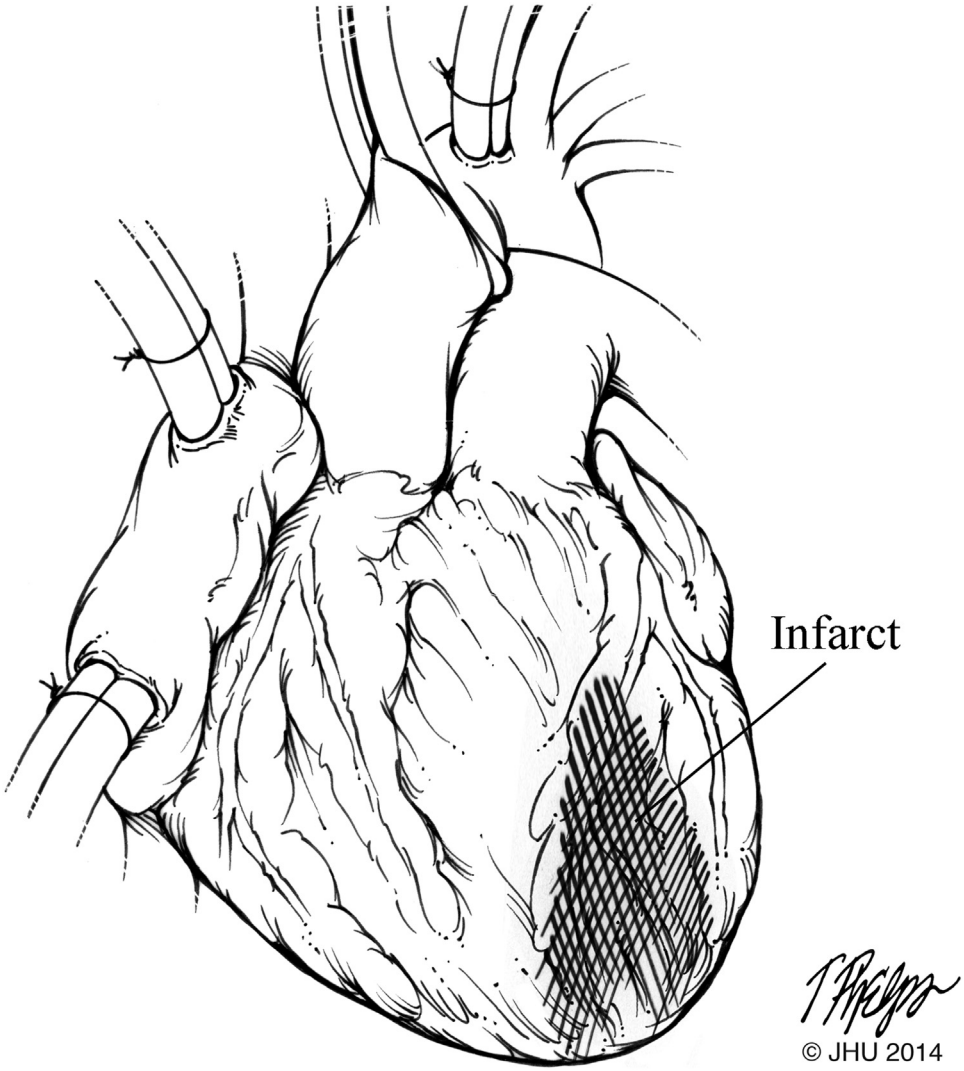

Figure 8 Infarct in the region of the left anterior descending (LAD) coronary artery. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

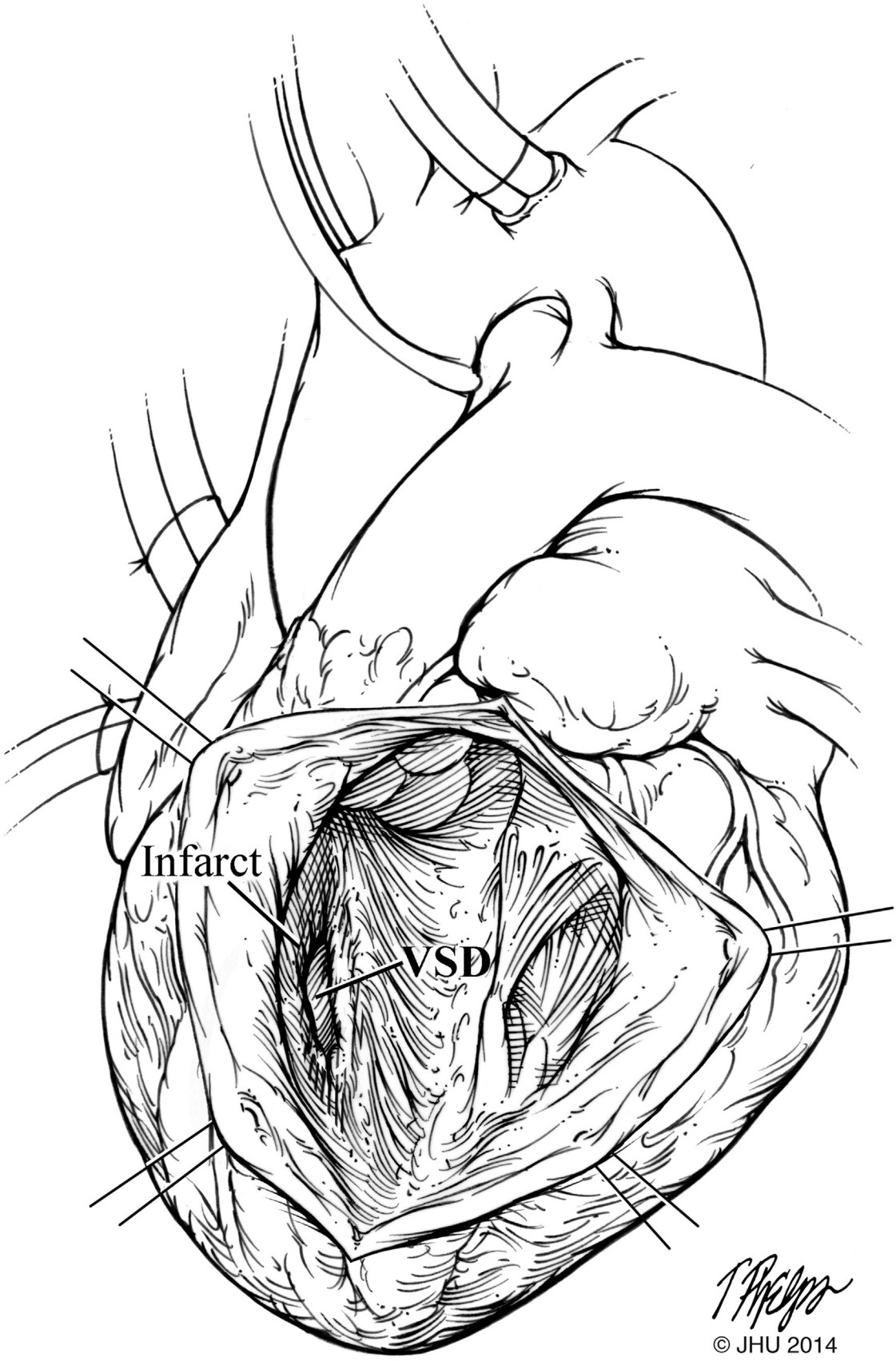

Figure 9 Anterior ventriculotomy through anterior wall infarct parallel to LAD vessel exposing infarcted septum with ventricular septal defect (VSD). Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

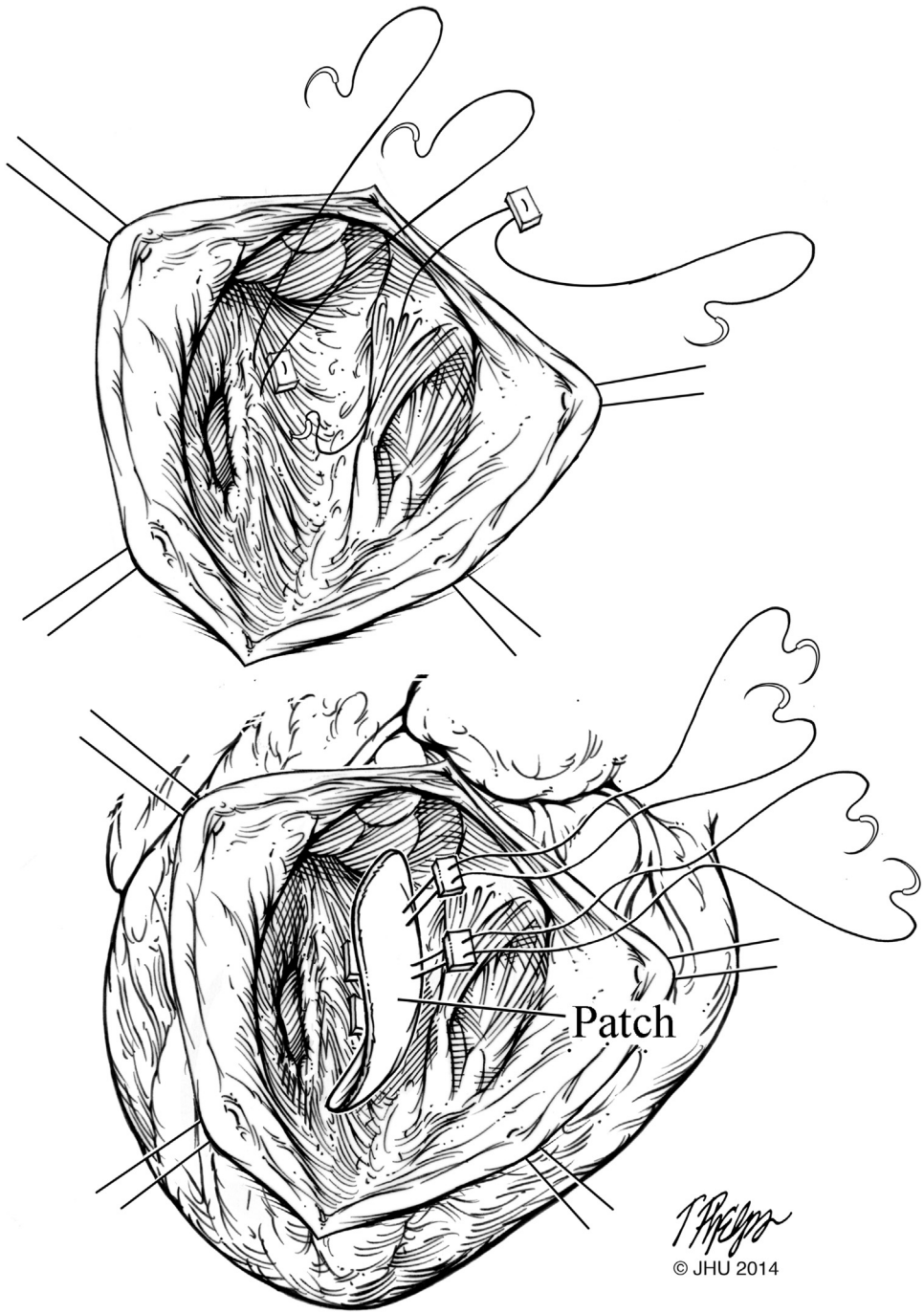

Figure 10 Interrupted pledgeted sutures placed at base of septum through healthy tissue away from infarcted tissue and VSD. Sutures then placed through "first" patch, which is cut to size. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

Figure 11 Completed septal patch repairing VSD. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

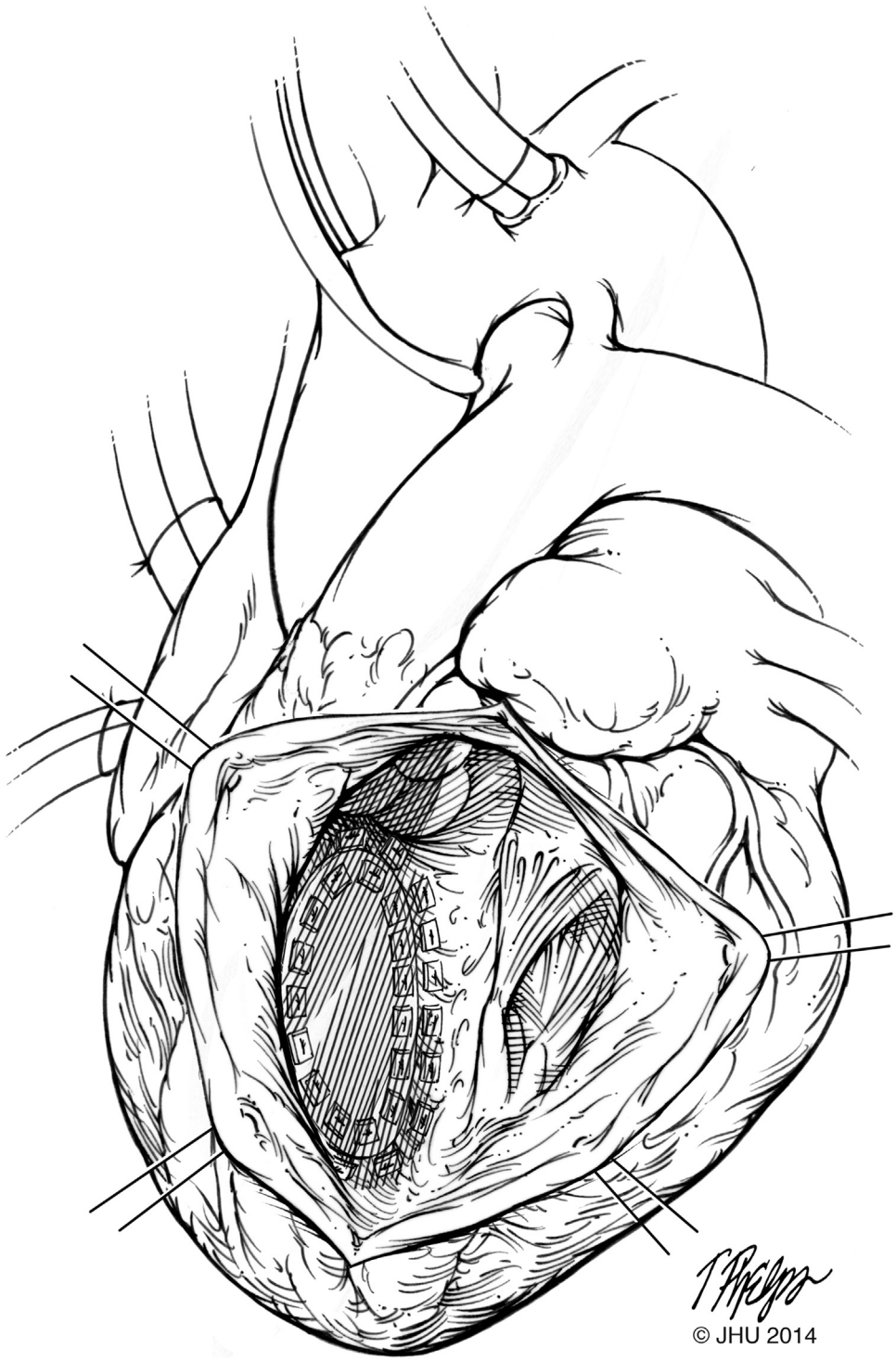

Figure 12 Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

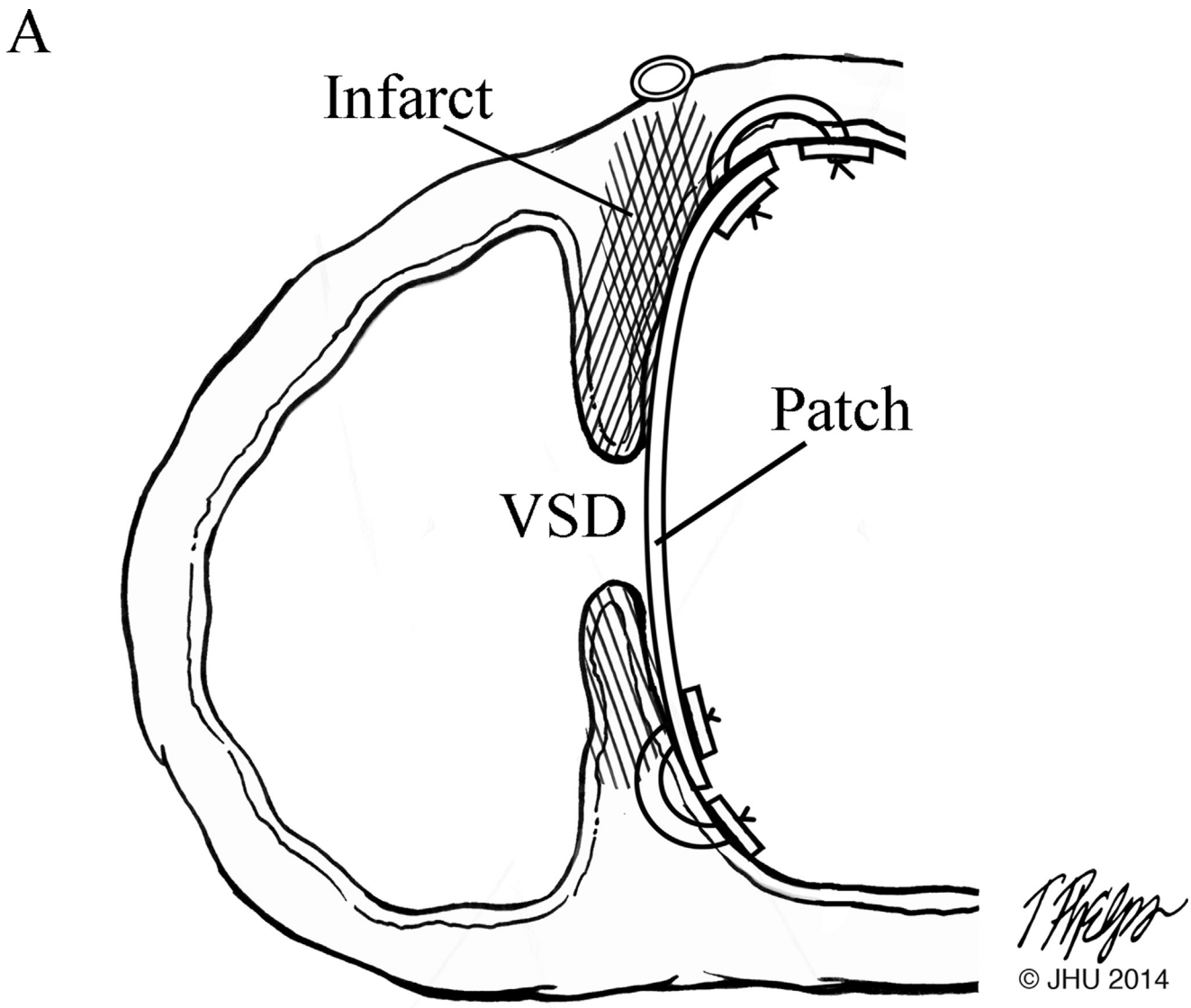

Figure 13 (A) Cross-section of ventricle demonstrating patch repaired with sutures entirely within LV. VSD = ventricular septal defect.

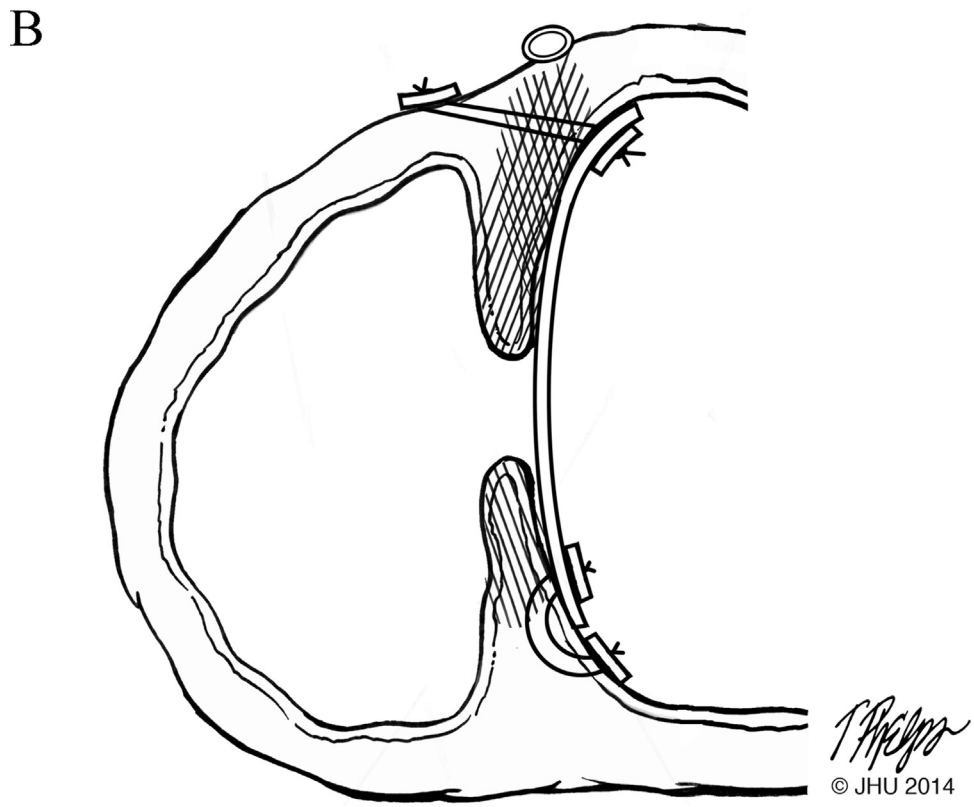

Figure 13 *(Continued)* (B) Cross-section of ventricle demonstrating patch repaired with sutures placed on anterior wall of right ventricle owing to poor quality of superior septal tissue.

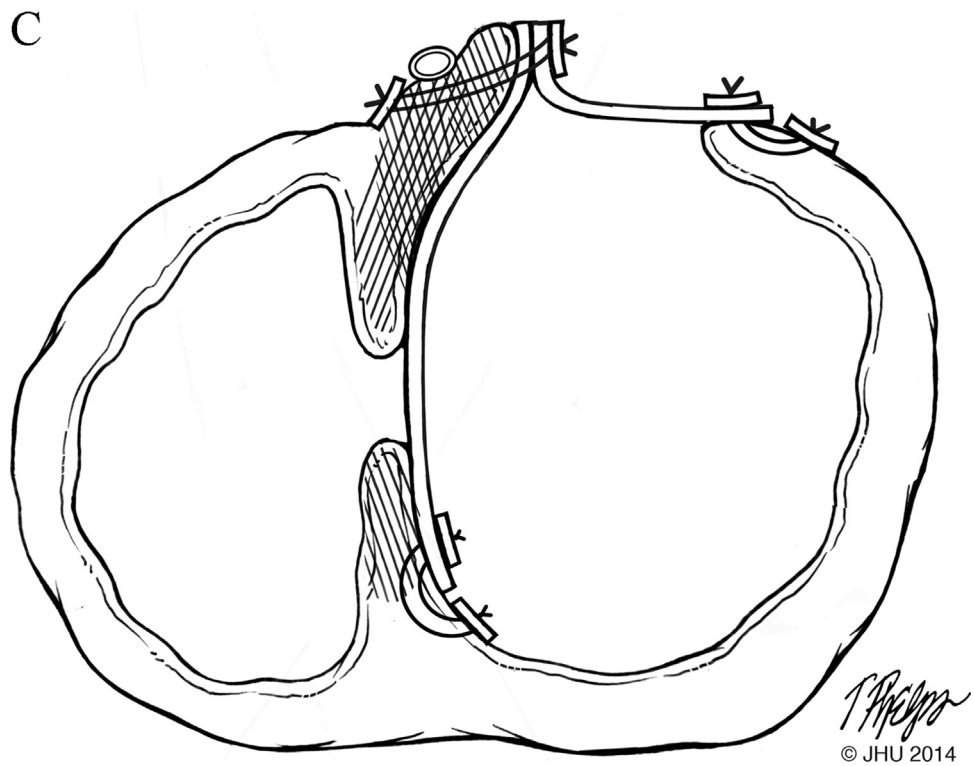

Figure 13 *(Continued)* (C) Cross-section of ventricle demonstrating patch repaired with sutures from anterior surface of RV and through patch to complete repair of septum and anterior LV wall with a patch-to-patch junction of septum and anterior wall. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

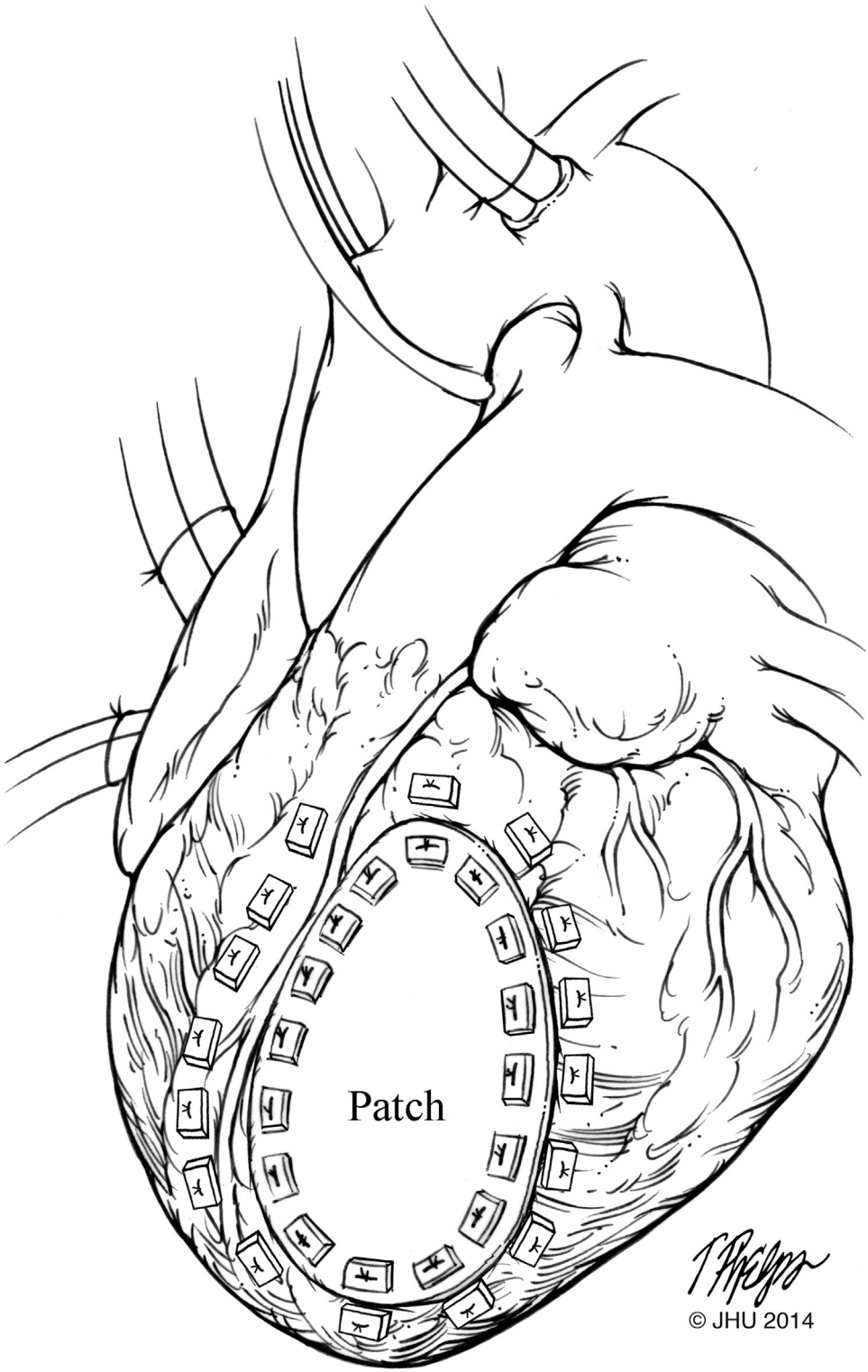

Figure 14 Completed 2-patch repair with use of second patch to reconstruct anterior LV wall. Drawing by Timothy H. Phelps, MS FAMI, Associate Professor, Department of Art as Applied to Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

Postoperative Management

Management in the intensive care unit is similar to any patient with severely compromised ventricular function, including ventilator and fluid management. Early institution of continuous hemodialysis is sometimes beneficial. Arrhythmias are common and striking a balance between antiarrhythmic medications and inotropes is sometimes difficult. The intra-aortic balloon pump and inotropic support often remain necessary for prolonged periods. We customarily wean inhaled nitric oxide before discontinuation of the intra-aortic balloon pump.

Conclusions

Postinfarction VSD is a rare complication of MI, yet these patients have high mortality. Both a national registry study from Europe as well as data from the STS national database documented 40% mortality after postinfarction VSD repair.[4,7] A single-institution series of 32 patients undergoing postinfarction VSD repair reported a 31% operative mortality, but among patients who survived to hospital discharge, the 5-year actuarial survival was 79%.[8] Careful patient selection and timing of repair before the onset of organ dysfunction offers the best possible chance of survival with surgical repair. We routinely employ 1 of the 2 techniques described in this report to treat postinfarction VSD. There are no good data favoring the use of either technique because postinfarction VSD is such a rare condition. The decision on which technique to use is patient dependent and often made in the operating room, based on the extent of the defect as well as the relative condition of the septal tissue surrounding the defect. A recent report from the STS database suggests that the use of percutaneous technology to treat VSDs does not seem to have had an effect on the use of surgical intervention, as annual numbers of procedures have remained relatively constant.[4] The keys to a successful operation include the following: (1) optimal timing of surgery with adequate preoperative resuscitation, (2) close collaboration with anesthesia and perfusion colleagues intraoperatively to maintain adequate organ perfusion, (3) accurate and rapid assessment of the extent of the defect size and devitalized tissue in the septum and anterior wall, and (4) determination of the optimal surgical technique and an efficient intraoperative strategy to achieve a satisfactory and durable repair.

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