

# Technique for the Repair of Truncus Arteriosus to Maintain Pulmonary Artery Architecture

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Due to improved techniques, the timing for the surgical correction of truncus arteriosus (TA) has moved to the neonatal period. Although this has reduced the mortality, patients frequently develop branch pulmonary artery stenosis resulting from excision of the pulmonary arteries from the truncal root and advancing the pulmonary bifurcation anteriorly and laterally. Tension created at the distal anastomosis of the right ventricle to pulmonary artery conduit can result in stenosis, which often requires balloon dilation or surgical reconstruction. Further, distal anastomotic stenosis results in early right ventricular to pulmonary artery conduit failure. Therefore, maintenance of the pulmonary artery architecture is critical and is the basis of our approach for repair of TA.

The ideal patient for our approach has the diagnosis of TA type I; however, this technique can be utilized in some patients with TA type II provided the branch pulmonary arteries exit the truncal root from the left posterior lateral position. The critical tenet for this repair is the maintenance of the pulmonary arteries in situ, thereby not removing them from the truncal root. As a result, the proximal left pulmonary artery serves as the pulmonary bifurcation. This technique is not applicable in those patients with TA type III, or some patients with TA type II, where excision of the pulmonary arteries from the truncal root is required.

In general, preoperative cardiac workup consists of history, physical examination, and transthoracic echocardiography. Additionally, all extracardiac anomalies are identified.

In our experience, cardiac catheterization is infrequently required.

After sternotomy, the thymus is removed, and the pericardium is opened. Our preference is bicaval venous and distal ascending aortic cannulation in all patients. In those patients with TA and interrupted aortic arch, a 3.5-mm Gor-Tex graft is sewn to the innominate artery and is used for arterial cannulation. Vessel loops are used to occlude the right and left pulmonary arteries just before initiating cardiopulmonary bypass. A left ventricular vent is placed preferentially in the right superior pulmonary vein but can be placed in the left atrial appendage. The heart is then cross-clamped and arrested. Visualization of the truncal root is obtained using a hockey-stick incision beginning low in the truncus and extending onto the left pulmonary artery. This incision also allows for excellent exposure of the truncal valve should it necessitate repair. The truncal root is then septated with a 0.4-mm polytetrafluoroethylene (PTFE) patch and 6-0 Prolene in a running fashion. Care should be taken when constructing the PTFE patch used to septate the truncal root. Too large of a patch can bulge into the pulmonary artery bifurcation and obstruct pulmonary artery blood flow.

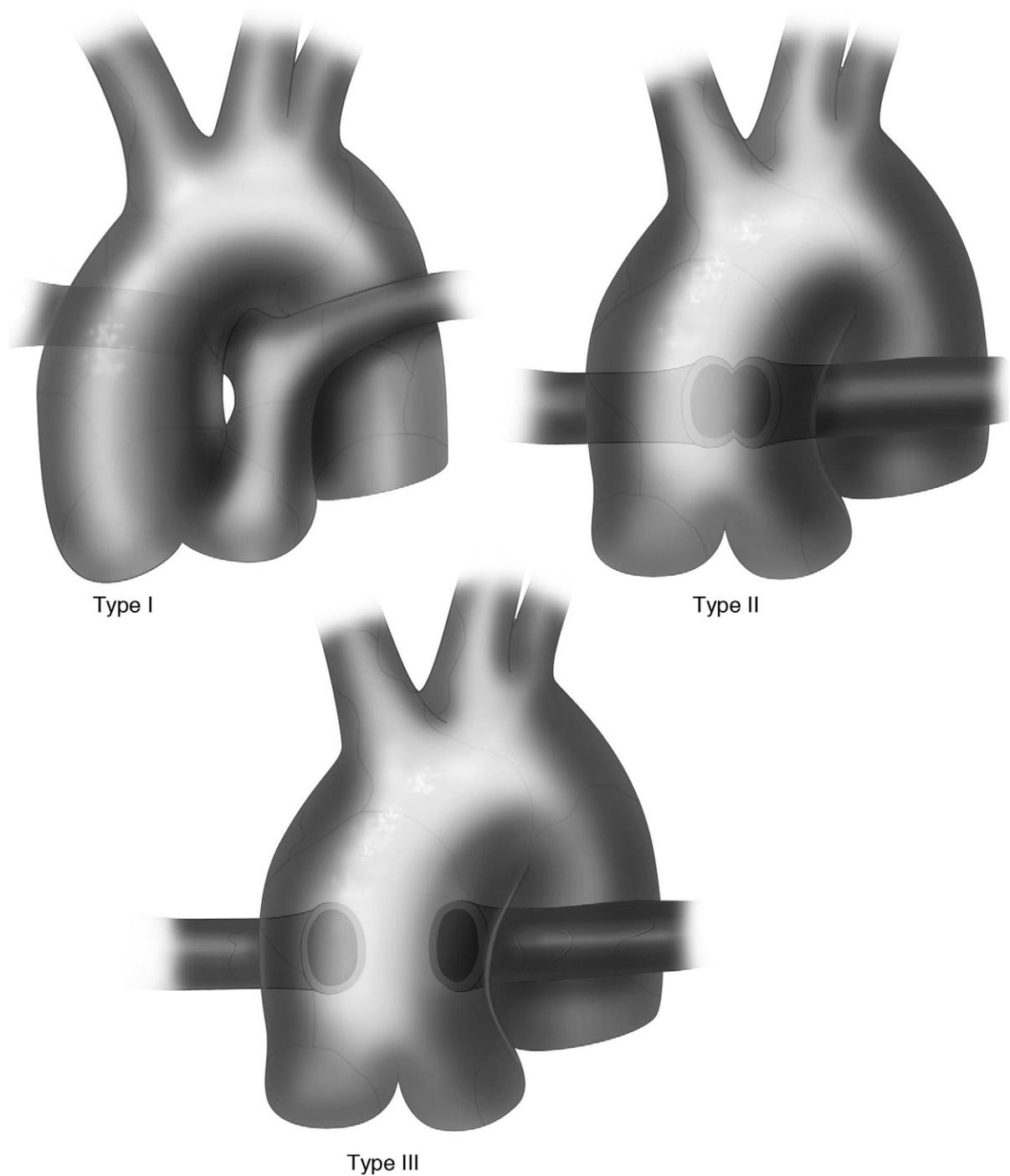
Once the truncal root has been septated, a right ventriculotomy is accomplished and the ventricular septal defect is closed in a standard fashion. An appropriate sized aortic homograft is then thawed and used to establish right ventricle to pulmonary artery continuity. The anterior leaflet of the mitral valve provides a unique hood for the anastomosis to the right ventricle. The homograft is tailored to remove the excess aortic tissue and the distal anastomosis is performed. Routinely, the sternum is easily closed at the conclusion of the procedure.

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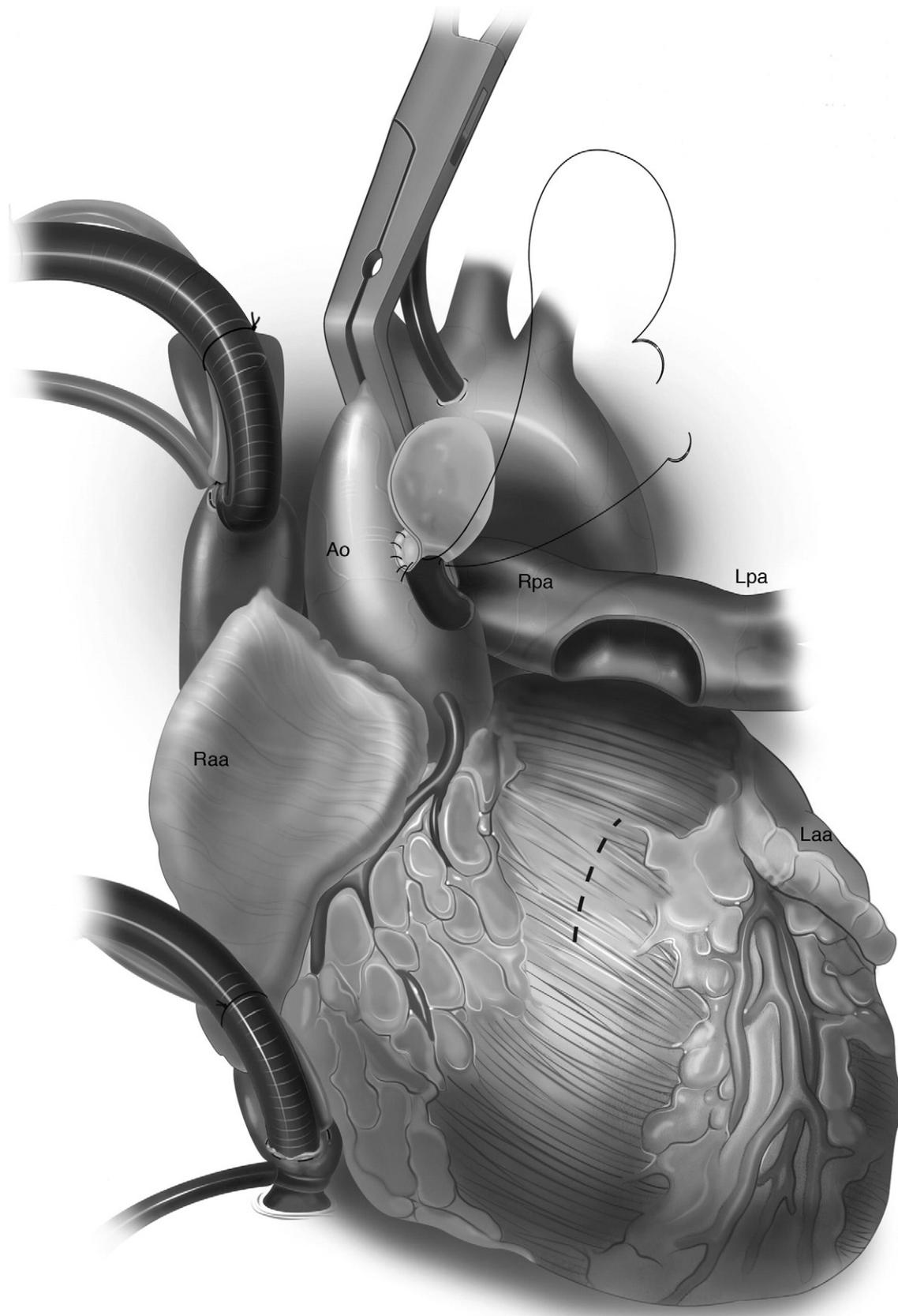
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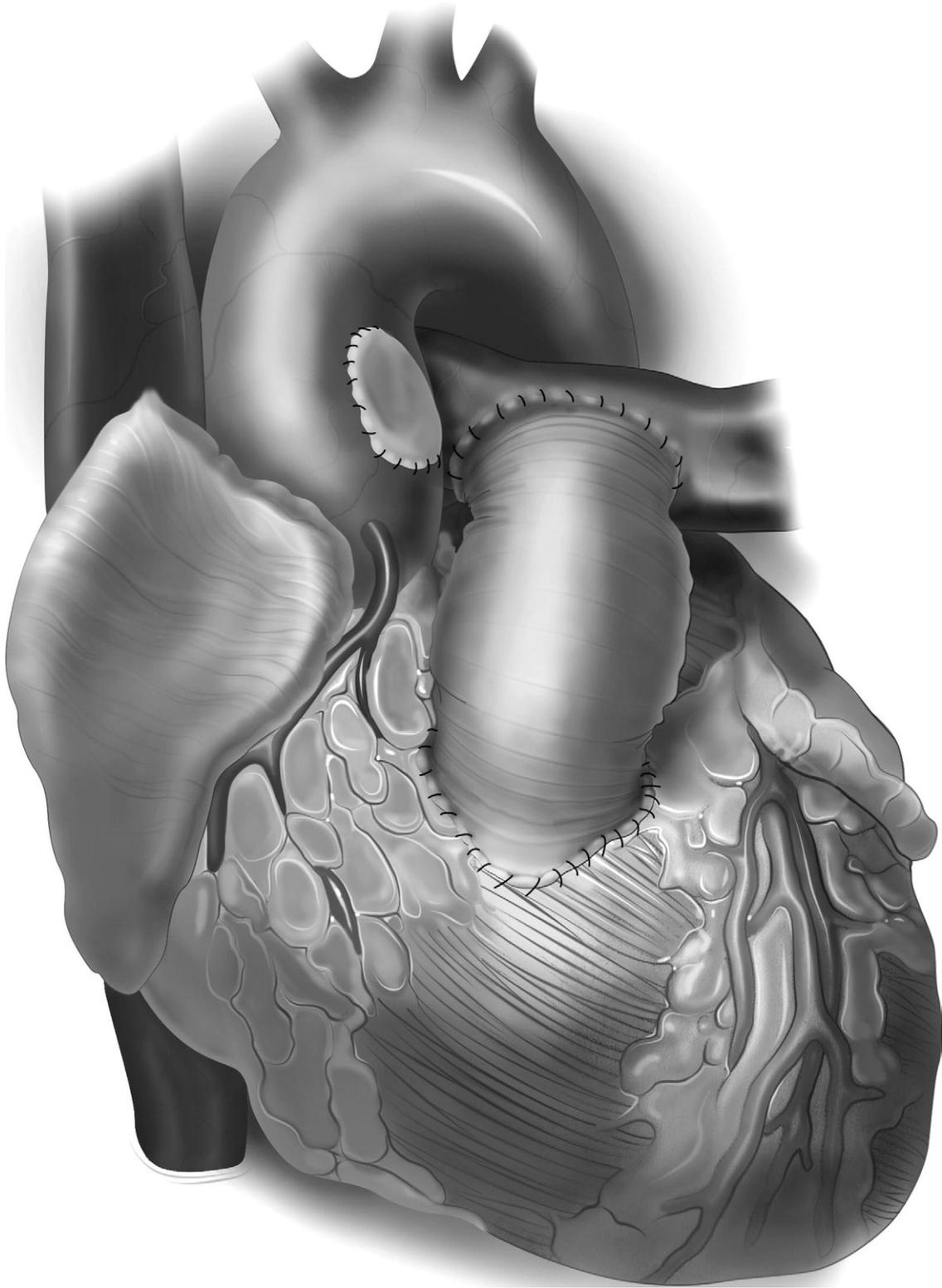
## Operative Technique



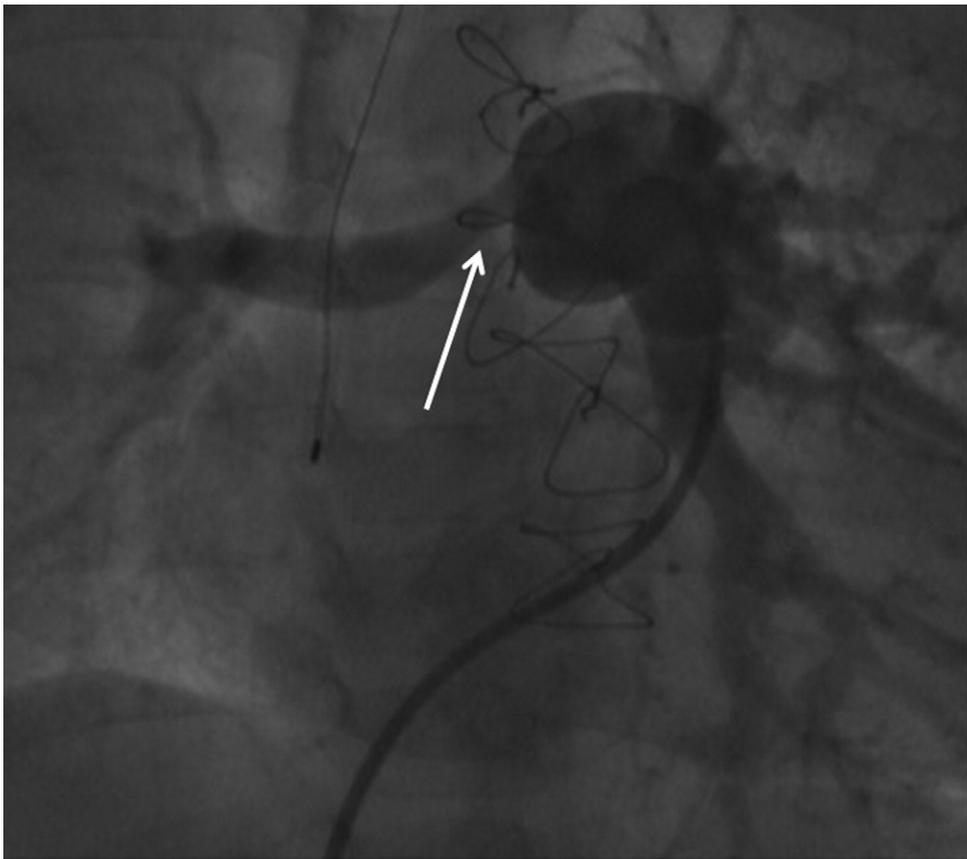
**Figure 1** Truncus arteriosus, type I, II, and III. In TA type I, there is a common anterior-posterior window between the main pulmonary artery and the ascending aorta. In type II, the branch pulmonary arteries arise from the posterior portion of the aorta, and in type III, the branch pulmonary arteries arise from the lateral aspects of the ascending aorta.



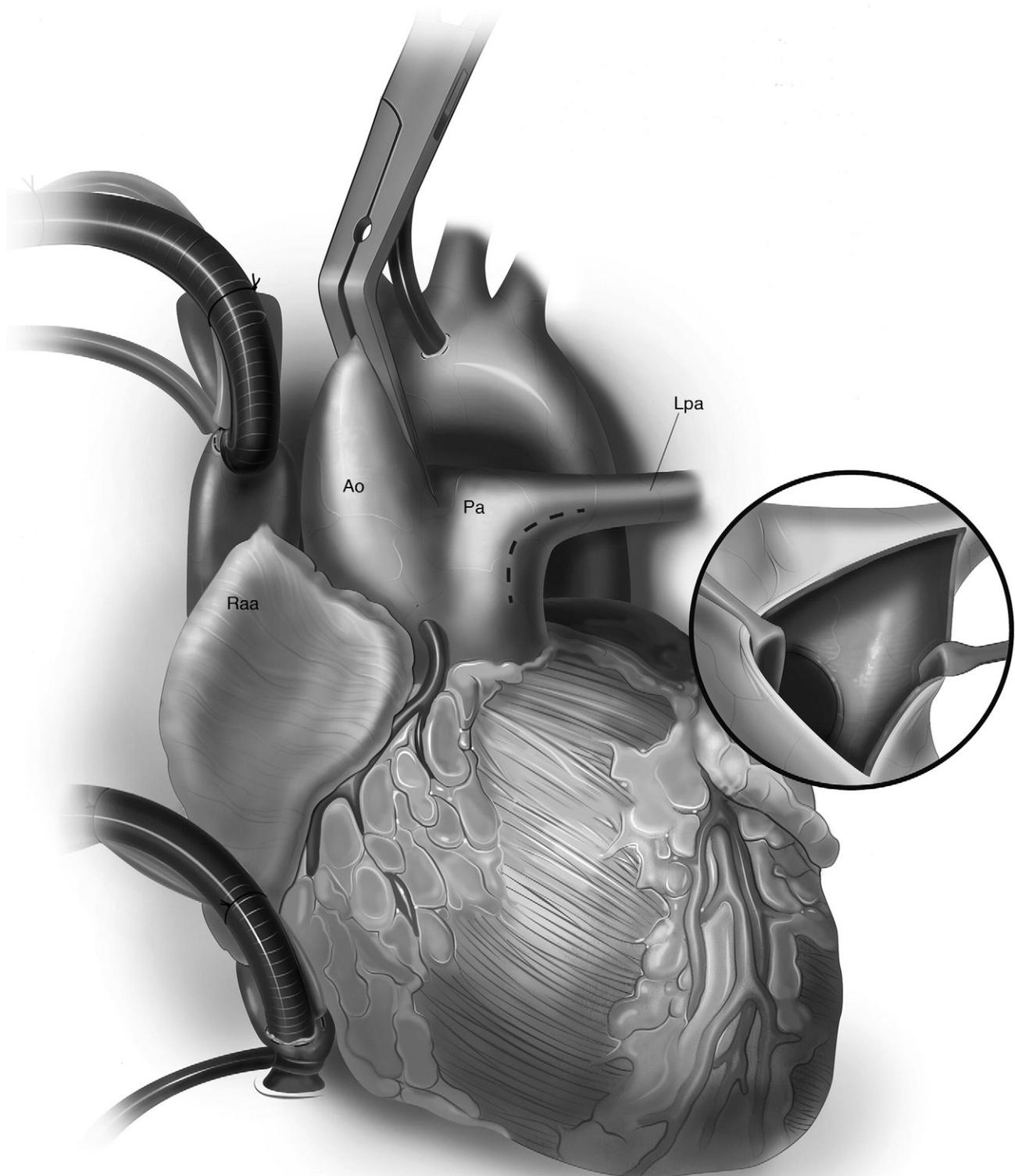
**Figure 2** Historic TA repair. The heart has been arrested; the pulmonary arteries have been excised from the truncal root and moved anterior and laterally, establishing a pulmonary artery bifurcation adjacent to the ascending aorta. The truncal root has been repaired using a patch. The dotted line represents the location of the right ventriculotomy, which is used to expose the ventricular septal defect. Ao = aorta; Laa = left atrial appendage; Lpa = left pulmonary artery; Raa = right atrial appendage; Rpa = right pulmonary artery.



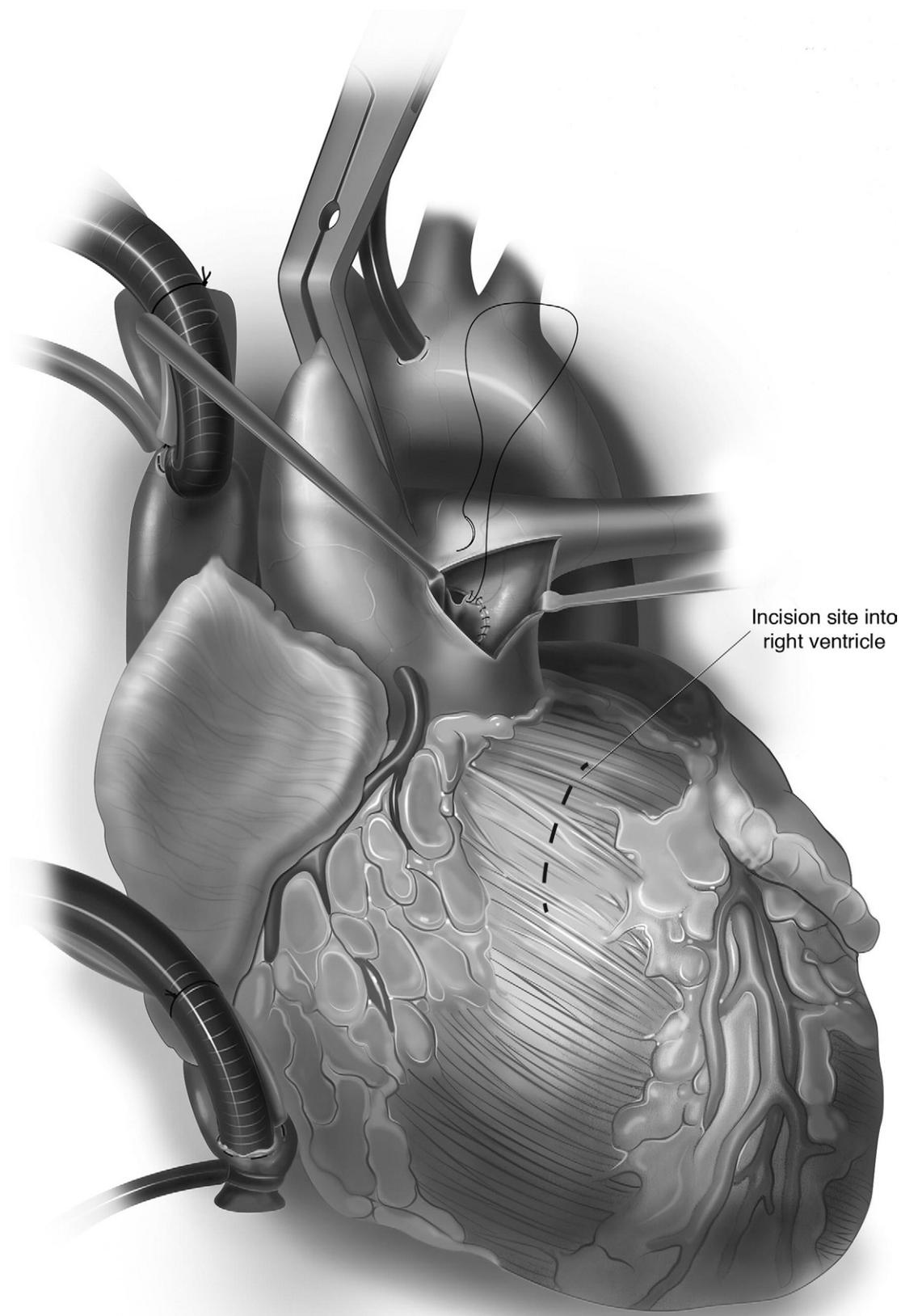
**Figure 3** Historic TA repair with a right ventricular to pulmonary artery valved conduit.



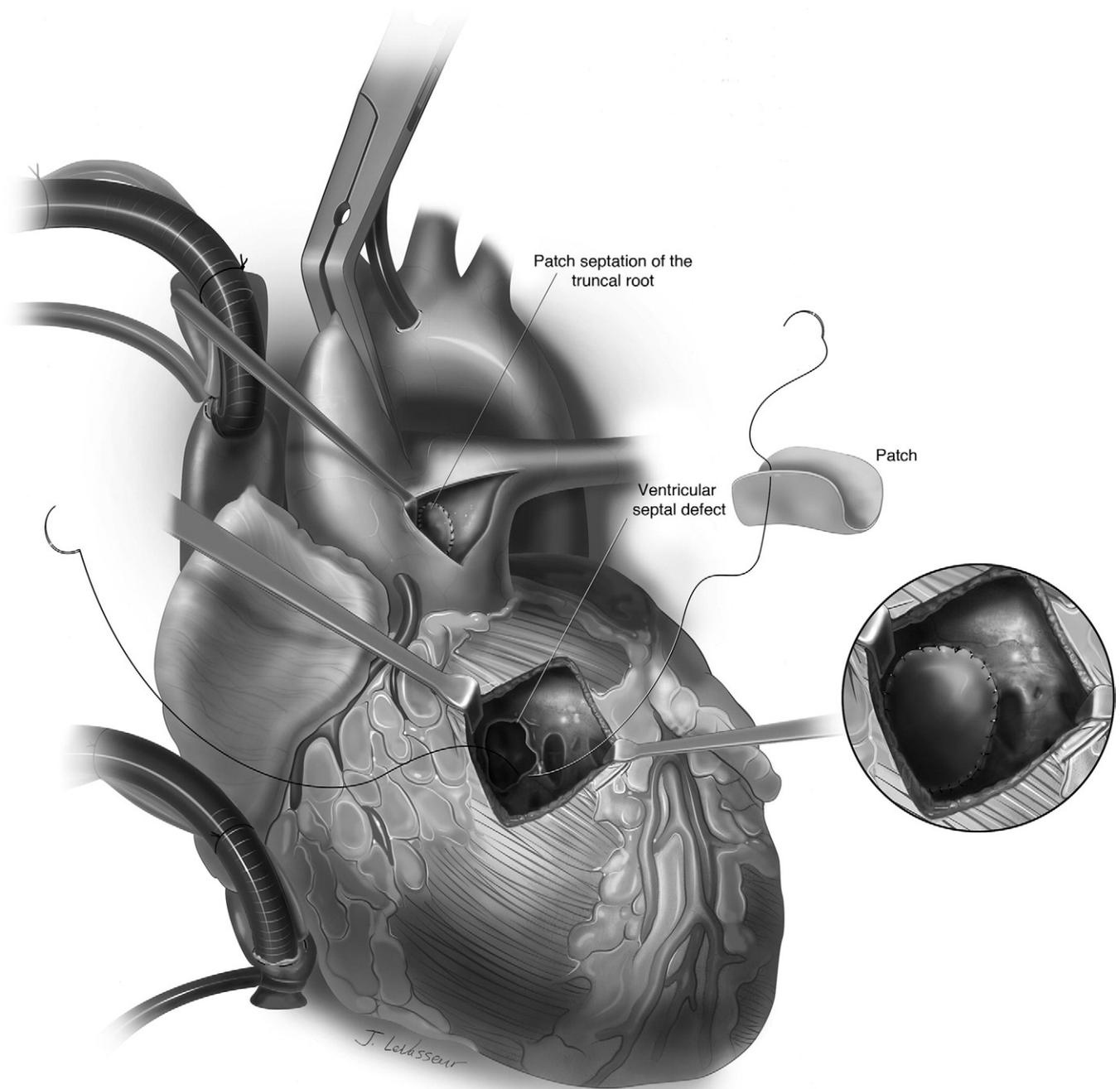
**Figure 4** Pulmonary artery angiogram 6 years after repair from a patient treated with the standard approach mobilizing the pulmonary arteries and moving them laterally. The angiogram demonstrates right pulmonary artery stenosis (arrow) just lateral to the pulmonary artery bifurcation.



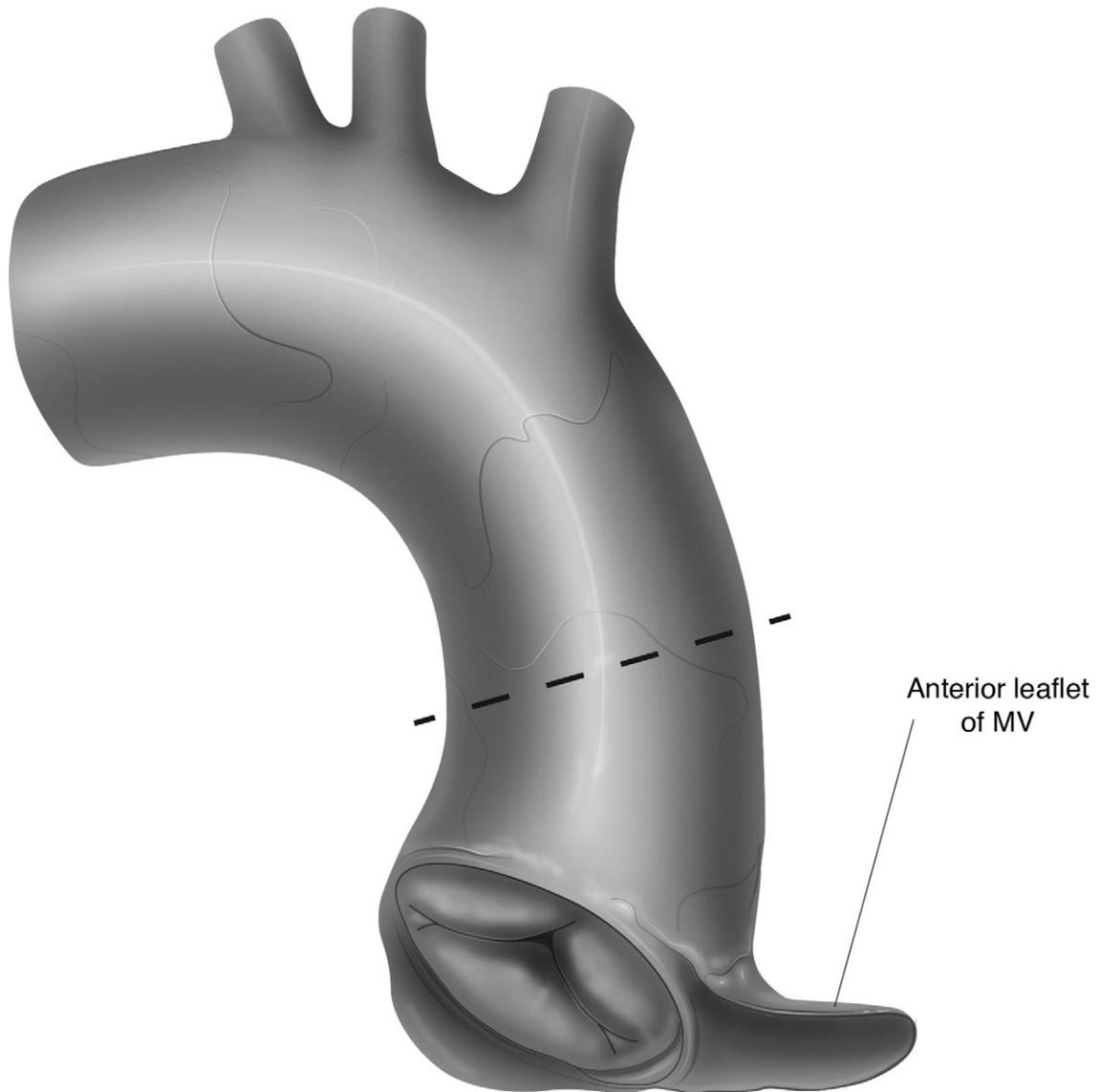
**Figure 5** Our approach with TA type I. The heart has been cross-clamped. A hockey-stick incision has been made in the truncus extending onto the left main pulmonary artery. Retraction using 2 ragnels demonstrates the right pulmonary artery orifice superiorly and the common anterior-posterior window inferiorly. Looking inferiorly within the incision allows optimal access and visualization of the truncal valve in case of repair. Ao = aorta; Lpa = left pulmonary artery; Pa = pulmonary artery; Raa = right atrial appendage.



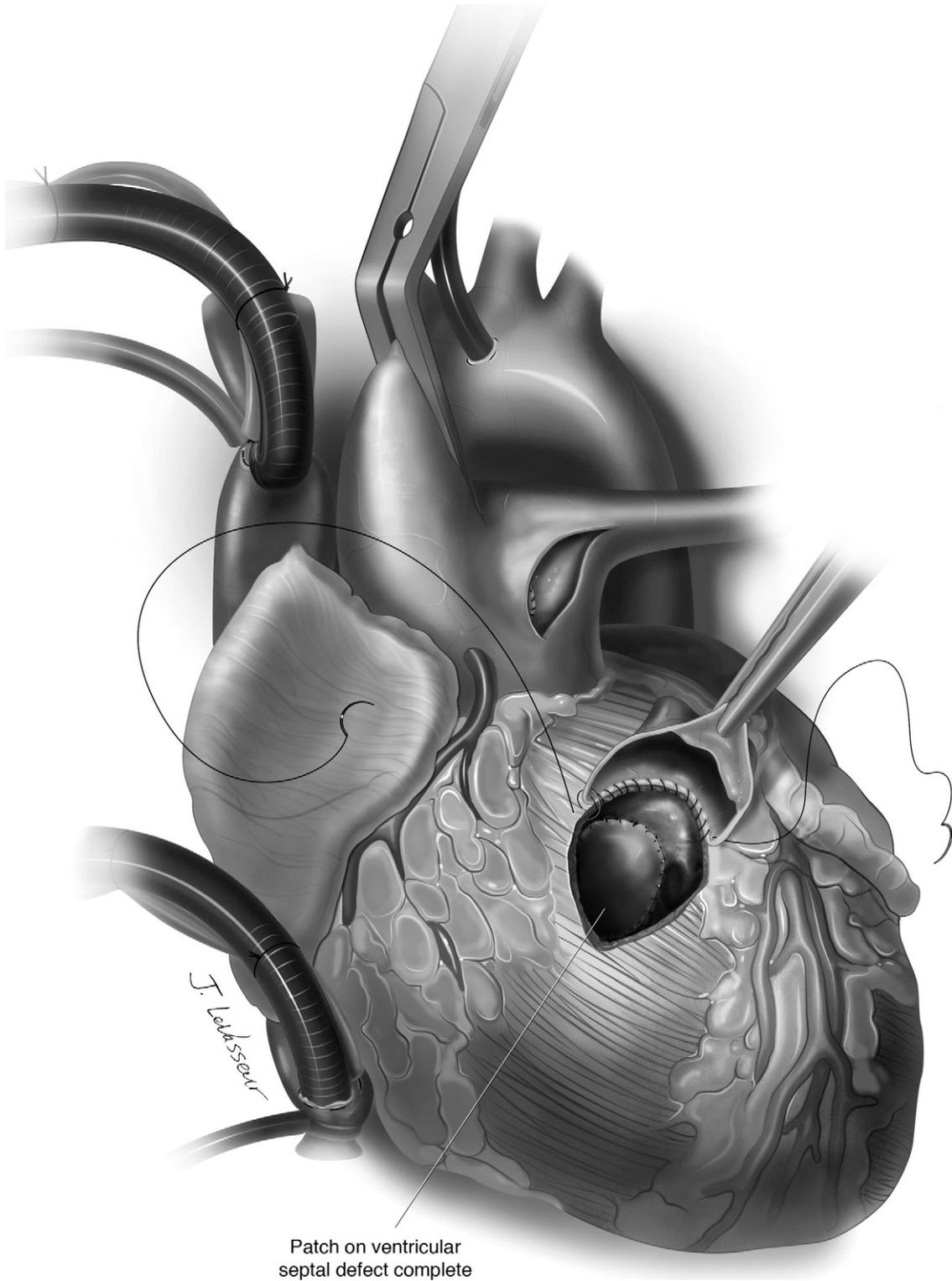
**Figure 6** A 0.4-mm PTFE patch is used to close the defect within the trunk using a running 6-0 Prolene suture. Care must be taken not to injure or incorporate the right pulmonary artery orifice within the suture line or patch. Once the PTFE patch has septated the truncal root, the original truncal arteriotomy functions as the new pulmonary artery bifurcation. The dotted line represents the location for the right ventriculotomy, which will be used to repair the ventricular septal defect and establish right ventricle to pulmonary artery continuity.



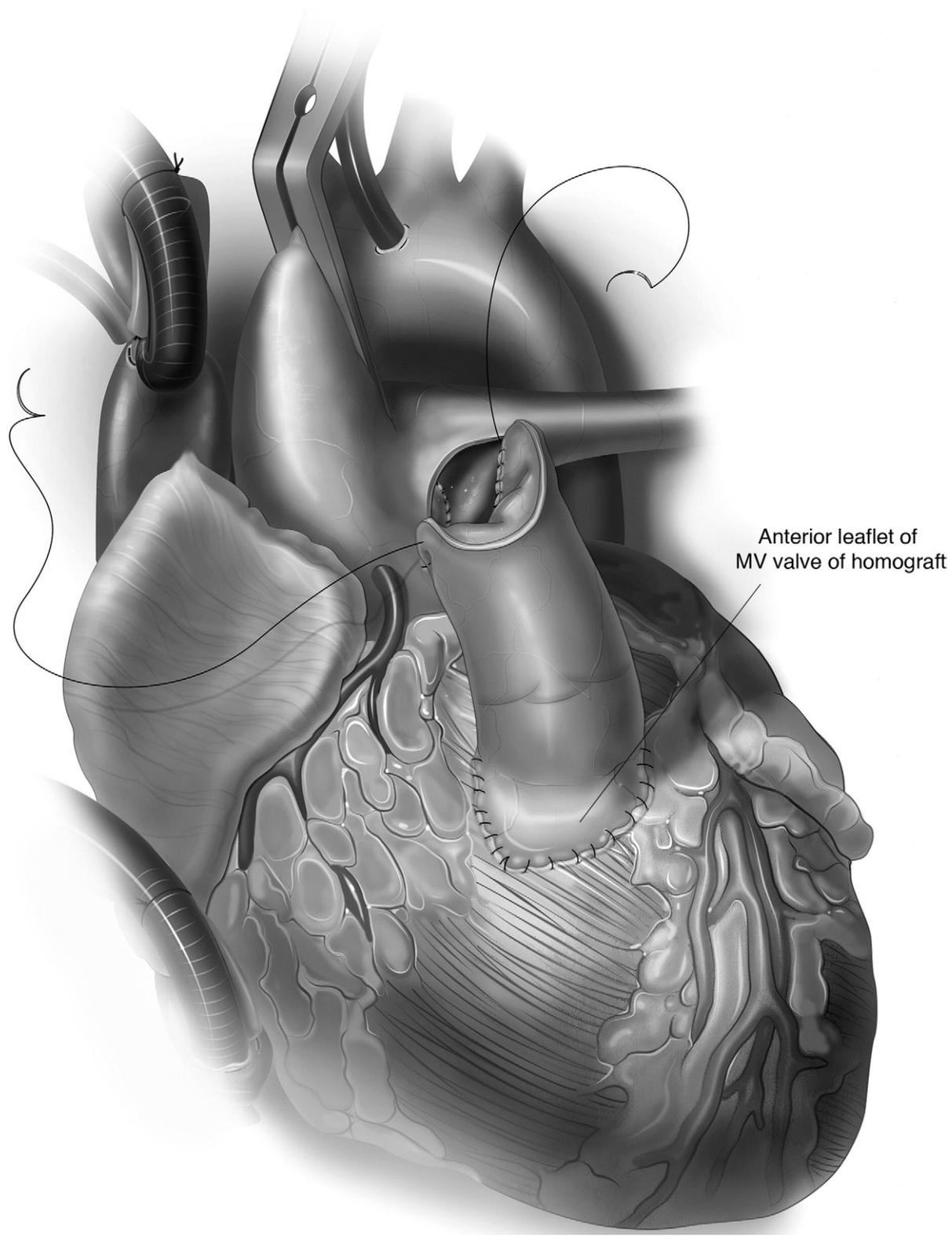
**Figure 7** A right ventriculotomy is made and the ventricular septal defect is exposed. The ventricular septal defect is exposed again using 2 ragnel retractors. A 0.4-mm PTFE patch is brought into the field, and using a running 6-0 Prolene suture, the defect is repaired.



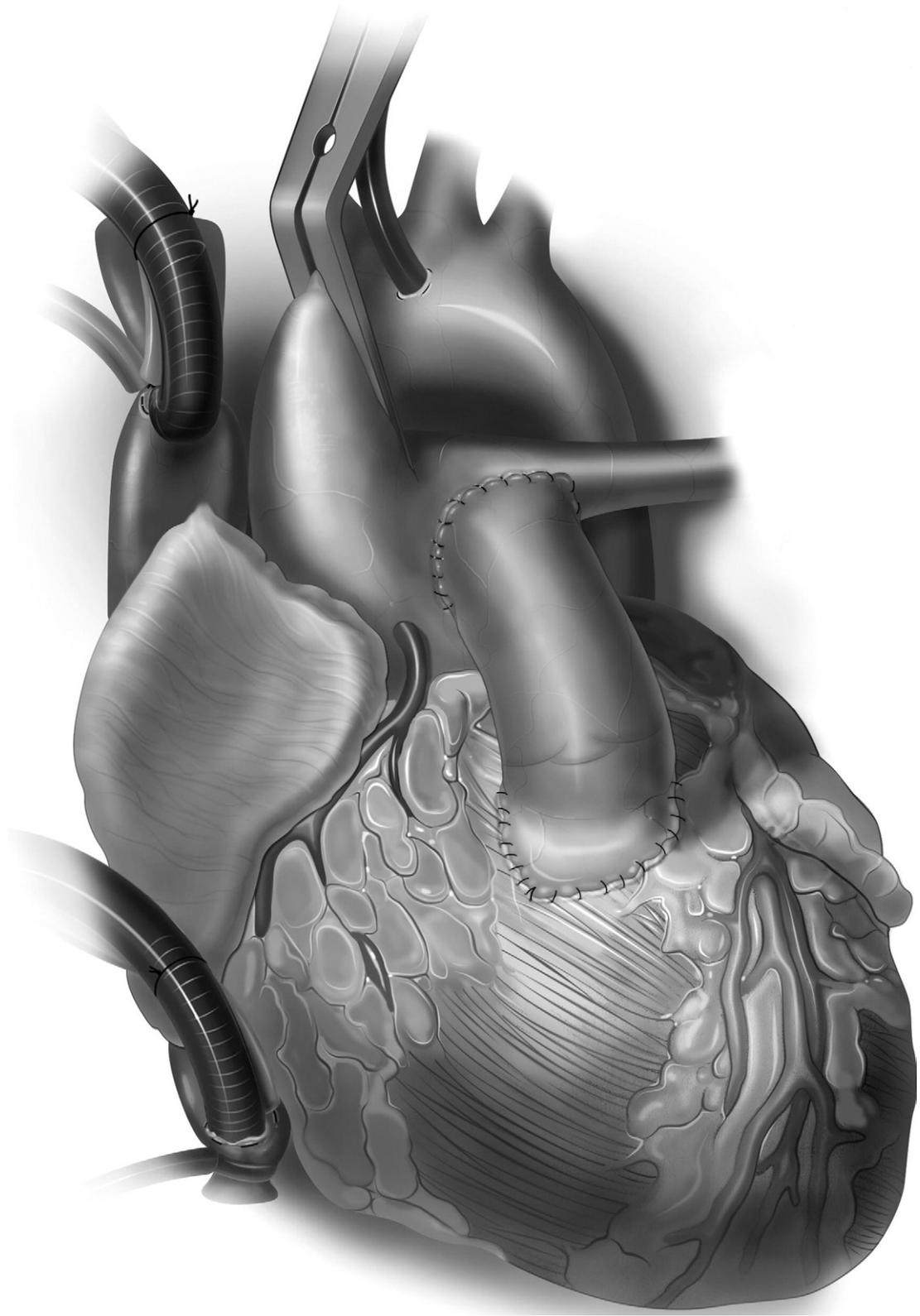
**Figure 8** The aortic homograft is thawed and prepared in the usual fashion. The dotted line indicates the approximate position of the distal anastomosis. MV = mitral valve.



**Figure 9** The proximal end of the conduit is sewn into place using a 6-0 Prolene suture. The anterior leaflet of the mitral valve is used as a hood for the proximal anastomosis.



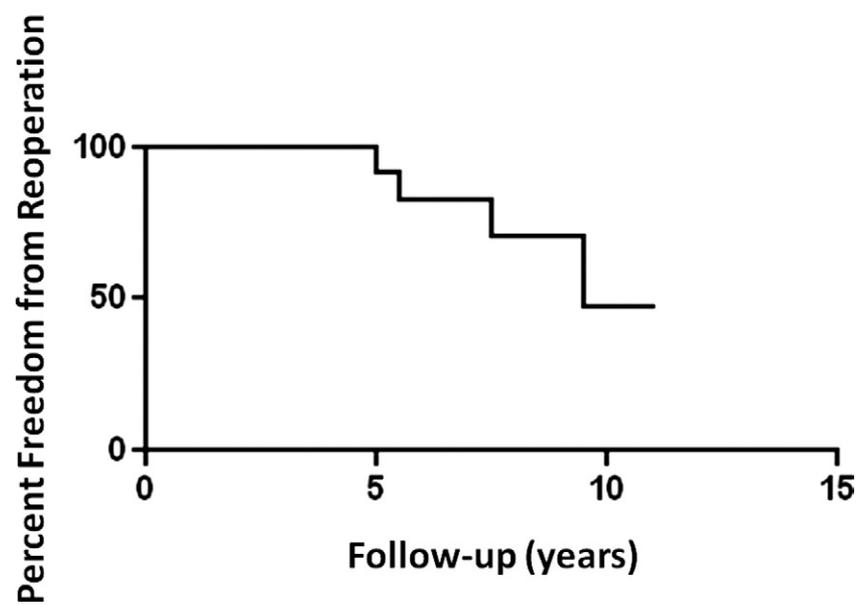
**Figure 10** The proximal anastomosis has been completed and the homograft is then tailored to limit the amount of graft tissue. The distal end of the conduit is sewn into place with a running 6-0 Prolene suture. MV = mitral valve.



**Figure 11** The complete repair after establishing right ventricle to pulmonary artery continuity.



**Figure 12** Pulmonary artery angiogram of a patient who underwent our modified repair to maintain the pulmonary artery architecture, which demonstrates widely patent pulmonary arteries.



**Figure 13** Freedom from reoperation curve for the 22 patients who received our modified repair for TA.

**Table 1** Description and Follow-Up of Conduit Stenosis After Truncus Repair

Patient	Type	Age at Repair (d)	Homograft Size (mm)	Follow-Up (y)	Peak Gradient (mm Hg)
1	I	14	11	9.4	50
2	II	42	11	7.5	60
3	I	80	12	8	35
4	I	30	13	8.5	35
5	I	10	13	5.4	30
6	I	13	9	4	50
7	I	15	11	.3	20
8	I	7	13	5	55
9	I	14	12	0.75	15
10	I	57	13	6.5	20
11	II	12	11	9.5	68
12	I	90	12	11	50
13	I	18	11	5.5	62
14	I	37	13	6.5	40
15	I	42	12	10.5	50
16	I	28	12	2	20
17	I	9	9	2.3	56
18	I	34	14	2.3	64
19	I	3	9	2.3	30
20	I	40	12	1	33
21	I	30	12	1.5	30
22	I	21	11	0.5	50

## Conclusions

Postoperatively, patients are hyperventilated, hyperoxygenated, and sedated for the first 12 to 24 hours after the operation. Patients are typically extubated within 48 hours and discharged from the hospital within 14 days after repair.

The benefit of preserving the pulmonary artery architecture is a concept described for nearly 20 years.<sup>1</sup> However, unlike previous reports, our repair maintains the branch pulmonary arteries within their native position and provides a competent valve in the pulmonary position.<sup>1,2</sup> Maintenance of the branch pulmonary arteries in situ prevents distal anastomotic tension, as well as the development of branch pulmonary artery stenosis. Although it has been reported that the use of an aortic homograft in the pulmonary position is inferior to a pulmonary homograft,<sup>3</sup> this data are primarily based on patients with tetralogy of Fallot and other right ventricular outflow tract obstructions. We feel that the aortic homograft is uniquely suited for our repair of patients with TA. During implantation, the homograft is tailored to the right ventricular outflow tract to minimize the amount of graft and thus reduce the amount of future graft calcification.

The anterior leaflet of the mitral valve provides a unique hood, thereby limiting the need of an additional patch.

Over the past 14 years we have exclusively used this repair for all patients with TA type I and for those patients with TA type II, whose anatomy is amenable to this repair. A total of 22 patients have undergone this repair with no surgical or hospital mortality. Table 1 lists the characteristics of the patients at the time of implant and follow-up. Freedom from reoperation for conduit failure was 47% at 9.5 years (Fig. 13). This repair for TA demonstrates superior intermediate term results, with improved conduit longevity, as a result of maintaining the branch pulmonary architecture.

## References

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2. Alfieris GM, Gangemi JJ, Schiralli MP, et al: Modified repair of truncus arteriosus to maintain pulmonary artery architecture. *Ann Thorac Surg* 90:1038-1039, 2010
3. Niemantsverdriet MB, Ottenkamp J, Gauvreau K, et al: Determinants of right ventricular outflow tract conduit longevity: a multinational analysis. *Congenital Heart Dis* 3:176-184, 2008