

# INTERVENTIONAL SURGICAL CLOSURE OF VENTRICULAR SEPTAL DEFECT

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### **Abstract**

Transcatheter closure of a ventricular septal defect (VSD) is a type of heart procedure. It closes a hole between the left and right ventricles of the heart. It does not make a cut (incision) in the chest wall. The heart has four chambers: two upper (atria) and two lower (ventricles). Blood that is high in oxygen flows from the left atrium to the left ventricle and out to the body, where the vital organs use the oxygen. Blood with less oxygen flows from the right atrium to the right ventricle and out to the lungs. There, it picks up more oxygen. Normally, a wall is present between the left and right atria and between the left and right ventricles. A child with a VSD has a hole in the wall between the left and right ventricles. The hole causes blood to flow abnormally from the left ventricle into the right ventricle. As a result, too much blood can go to the lungs. This causes the heart and lungs to work harder.

**Keywords**: VSD, transesophageal echo (TEE), aneurysma, ventricular angiogram.

## Introduction

For patients with hemodynamically significant ventricular septal defects (VSD), surgical repair is considered the preferred therapeutic option with data supporting its safety and effectiveness. Perimembranous and muscular VSDs have been targets for percutaneous device closure, and this is yet another area of interventional cardiology that has evolved substantially with the revision of delivery systems and devices. The experience and outcomes have been substantially different for these two types of VSDs with divergent current strategies as a result. These experiences, outcomes, and strategies are described in this section.

Given the recognized success of surgical repair, appropriate patient selection for percutaneous VSD closure is critically important. Contraindications for VSD device closure include: irreversible pulmonary vascular disease, contraindication to antiplatelet therapy, inadequate rim ( <4 mm ) between the defect and cardiac valves and aortic valve cusp prolapse. AV canal type (inlet) and conal septal (doubly committed, supracristal) type VSDs are not considered appropriate anatomy for device closure. Based on anatomy, most perimembranous VSDs are not appropriate anatomic candidates due to the inadequate rims. The anatomic location of the defect risks damage to the aortic valve, tricuspid valve, and the conduction system. However, some defects with aneurysmal septal tissue may provide a safe anatomical target in which to place a device. For patients with a perimembranous VSD, this specific anatomy is generally considered the only potential candidate

**165** | Page





for device closure when surgical repair is available, due to the high risk of heart block and other adverse events.

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There are various techniques and approaches for VSD device closure. For all cases, venous and arterial access points are obtained based on patient anatomy and any vessel occlusions. A full hemodynamic assessment is performed with particular attention to the Qp:Qs, pulmonary artery pressure, and pulmonary vascular resistance. A left ventricular angiogram is routinely performed in a long axial oblique angulation, profiling the VSD, in order to measure and provide fluoroscopic reference for catheter and device positioning. The device delivery and deployment are performed under echocardiographic guidance, most commonly with transesophageal echo (TEE). The most widely described and used technique is the antegrade approach using an arterial-venous wire loop or rail. The VSD is crossed with a wire from the left ventricle (LV) to the right ventricle (RV). The wire is advanced to the SVC or the PA where the end of the wire is then snared and externalized through the venous access point (most commonly femoral or internal jugular vein). An appropriate device is selected and the delivery sheath is then advanced from the venous side, over the arterial-venous wire loop, and into the LV. The wire is removed and the device is deployed in a similar fashion to ASD devices. The LV disc is advanced either in the LV cavity or ascending aorta, being careful to avoid opening the device near the aortic valve and risk damaging the aortic valve leaflets. The device is then pulled into the VSD and the RV disc is unsheathed on the RV side of the defect. Appropriate position is confirmed by TEE prior to the release of the device.

A retrograde approach can also be employed and may have advantages over the antegrade approach. As smaller delivery systems have been developed, delivering the device from the arterial side may eliminate the need for an arterial-venous wire loop and may decrease overall procedure time. The delivery of the device mirrors the antegrade approach. The RV disc is deployed, the device is then pulled back into the defect and the LV disc is unsheathed and positioned on the LV side of the defect. An additional approach that can be considered is a perventricular approach, in particular for infants with muscular VSDs that cannot be closed surgically.

As mentioned above, the experience and outcomes for device closure of perimembranous VSDs and muscular VSDs have been significantly different and must be distinguished. For perimembranous VSDs, the Amplatzer Membranous VSD Occluder device demonstrated promised in initial phase I trials in the USA with high rates of complete closure and acceptable rates of adverse events. There was a relatively high frequency of complications in this cohort with adverse events reported in 29% of cases and there were two patients who developed complete heart block requiring pacemaker implantation. A lower weight (<10 kg) was associated with a higher incidence of adverse events. However, as case numbers and follow-up time accumulated, higher rates of complete heart block were reported with acute onset without identifiable risk factors and variable timeline post-procedure. One center reported an incidence of complete heart block in 22% of cases performed. As a consequence, the Amplatzer Membranous VSD Occluder did not obtain FDA approval, and this experience motivated new approaches and further device development. One modification, as mentioned above, is the selection of defects with aneurysmal septal tissue. A single-center case series of 15 patients with this anatomy reported a modified technique for perimembranous VSD closure with placement of the device within the aneurysmal tissue of the ventricular septum. Using this technique, this group reported complete closure in 54% of cases,

but there were no cases of heart block. Newer, softer devices may also result in reduced incidence of heart block. A recent meta-analysis of percutaneous perimembranous VSD device closure studies reported a pooled incidence of residual shunt in 16% of cases and a pooled estimate of complete heart block in 1% of cases. A second meta-analysis comparing percutaneous device closure and surgical closure of perimembranous defects demonstrated no difference in residual shunt, valve regurgitation, or heart block between the two approaches. Therefore, innovative device design and technical approach continue to evolve for patients with perimembranous VSD, but surgical repair remains the first line approach for the majority of patients due to the recognized incidence of permanent heart block. Additional devices are approved outside of the USA for VSD closure (for both perimembranous and muscular defects) and multiple further device trials are ongoing internationally. For patients with muscular VSDs, there is one FDA approved device in the USA – the Amplatzer

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Muscular VSD Occluder, which obtained approval in 2007. Of note, multiple other devices have been used off-label and described for VSD closure (muscular and perimembranous) including the Amplatzer Ductal Occluder, Amplatzer Septal Occluder, and Amplatzer Vascular Plug. Data from the initial device trial in the USA demonstrated a high rate of procedural success (85%) and a high rate of complete defect closure (92%) at 12-month follow-up, resulting in FDA approval. Major adverse events occurred in 10% of cases including two procedure-related deaths. Adverse events associated with smaller patient size (< 5 kg). Arrhythmia or conduction abnormalities were reported in 20% of the procedures but no patients had sustained heart block in follow-up (three patients had a persistent right bundle branch block). Mid-term results after device closure of muscular VSDs is encouraging with no reported cases of late heart block, increased atrioventricular valve insufficiency, or ventricular dysfunction in a multi-center cohort. Given the increased risk seen with infants (<5 kg), a perventricular hybrid approach should be considered. In conclusion, transcatheter closure of a VSD uses a flexible tube called a catheter. This tube contains a small device used to close the hole. The healthcare provider threads the tube through a blood vessel in the groin and into the heart, next to the wall between the ventricles. The provider then releases the small device and uses it to plug up the hole in the wall. Then the tube is removed from the body.

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