

# Ventricular Septal Defect (VSD)

VSds are the most common cause of congenital heart disease. It is more commonly seen in select genetic syndromes, including Apert's syndrome. Fetal alcohol syndrome, TORCH syndrome, and Trisomies 13, 18, and 21. Caused by a persistent opening in the interventricular septum, both the clinical manifestations and the management of VSD vary greatly depending on the size of the defect. Many small defects will close on their own, but surgical repair is the definitive treatment for large defects to avoid progression to congestive heart failure (CHF).



**PLAY PICMONIC** 

### **Pathophysiology**

#### Opening at Intraventricular Septum

Hole in Wall between Ventricles

Failure of the intraventricular septum to close is the most common cause of VSD. Defects in muscular growth and malalignment of the septum are other contributors. In utero, VSDs affect the fetus very little due to preferential flow through the ductus arteriosis. After birth, the ductus closes and pulmonary pressures fall, causing blood to be shunted from the left to the right side of the heart - making VSD a non-cyanotic heart disease. The degree of shunting depends on both the size of the defect and the difference in pressures between the systemic and pulmonary circulation.

#### Most Common Congenital Heart lesion

#1 Foam-finger from Birth

VSD is the most common congenital heart lesion.

### **Symptoms**

#### High-pitched Holosystolic Murmer (over left sternal border)

High-pitched Whistle from the Merman with Halo-squeezed from heart

Shunting of blood across the abnormal opening causes a harsh, holosystolic murmur that is best heard at the lower left sternal border. The smaller the defect, the louder the murmur will be. Smaller defects may also have a palpable thrill, or vibration, over the chest wall. The murmur may still be present in larger defects, however it will be softer and more blowing in quality. Large defects are more likely to have a palpable heave of the apex as the left ventricle compensates with increased contraction.

### **Dyspnea and Respiratory Distress**

Disc-P-lungs shooting Flare-gun in distress

Due to the increased workload on the heart, dyspnea and respiratory distress are often the most pronounced symptom of VSD. Dyspnea is most pronounced when infants are feeding, and may lead to frequent respiratory infections, poor feeding and failure to thrive.

### Loud Pulmonic S2

Stethoscope girl in a (2) Tutu at Loud Pulmonic-valve

Increased flow across the pulmonic valve combined with an increased pulmonary vascular pressure causes the pulmonary component of the second heart sound to be pronounced. With more severe shunts, fluid backup may manifest as crackles and hepatomegaly on exam.



### **Diagnosis**

### **Echocardiogram**

#### **Echoing Cardiogram**

Echocardiogram is the definitive test for diagnosing VSD, and will also show the size of the shunt and any associated defects. Later in the course of the disease, ECG may show left or right ventricular hypertrophy, and CXR may show signs of pulmonary congestion such as increased vascular markings and cardiomegaly.

#### **Treatment**

### Small VSDs Close Spontaneously

## Small VSD Closing spontaneously with Spartan

75% of small VSDs close spontaneously within the first two years of life, and the ones that persist rarely cause clinically significant disease. Patients with small VSDs should be followed by cardiology with regular echocardiograms.

#### Large VSDs Require Surgery

#### Large VSD with Surgeon

Large VSDs require surgical closure to prevent development of significant strain leading to heart failure. Repair is indicated in any infant (> one year) with signs of pulmonary hypertension, symptomatic patients who have failed medical management, and patients with large VSDs that are not decreasing in size over time.

### **Complications**

#### **Endocarditis**

### In-heart-inflammation

The risk of endocarditis in patients with VSD is low, and surgical closure lowers it further. The risk is increased in the immediate postoperative period. Antibiotic prophylaxis is recommended in patients with unrepaired defects, and for 6 months after repair with prosthetic material.

## Eisenmenger's Syndrome

#### lce-man

A rare, but feared complication of VSD is Eisenmenger's syndrome. This is a phenomenon in which the initially left to right shunt of the VSD converts to a right to left shunt, giving the patient cyanotic disease. This occurs secondary to increased pulmonary vascular resistance from constant increased pulmonary blood flow across the shunt.

#### CHF

#### CHF Heart-balloon

One of the most common complications of large VSD is the development of CHF. CHF is treated with diuretics and ACE inhibitors, as well as inotropes to improve contraction. The defect should be closed as soon as possible to prevent continued strain and further damage.