



# Ventricular Septal Defect

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## Abstract

“Ventricular septal defect (VSD) is one of the most common congenital heart lesions.” The most common type of clinically significant VSD is a membranous VSD. Small VSD can be detected by the presence of holosystolic high-pitched systolic murmur. The management of ventricular septal defects (VSDs) is dependent on the size of the defect and degree of shunting. Patients with moderate to large defects who are diagnosed as infants must be continuously monitored in their first weeks of life. Patients who look to be in a good health are scheduled for a follow-up with a pediatric cardiologist 3–4 weeks after the delivery. Monitors growth and change in the cardiac examination can provide to a patient in primary care. The choice of treatment approach is based on the clinical findings and size of the defect. For asymptomatic patients who typically have a small defect, we suggest no intervention. For symptomatic patients, medical therapy for heart failure is generally warranted. Symptoms typically occur in patients with moderate to large defects. For patients who are not adequately managed by medical therapy and/or have evidence of elevated pulmonary artery pressure (PAP) or valvular involvement, we suggest surgical closure of the VS. Medical therapy is focused on reducing the symptoms and complications of heart failure.

## Keywords

Bicuspid aortic valve · Heart failure · Pulmonary hypertension · Interventricular septum  
Immunoprophylaxis · Ventricular septal defect  
Subaortic stenosis

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## Introduction

“One of the most common congenital heart defects is a ventricular septal defect (VSD) (second only to bicuspid aortic valve)” [1].

## Pathophysiology

VSD in the fetus has little effect on the hemodynamic state, resistance equal through the chamber of the heart. Extrauterine like the PVR drop and close of ductus arteriosus lead to change in resistance through the chamber, direction of flow depends mainly on difference of resistance of each circulation, high systemic resistance, and low pulmonary resistance, typically left to right shunt. Small VSD if does not close by own will cause a mild increase in resistance and redirection shunt in reverse of large VSD, depending mainly on the pressure difference between the right ventricle and left ventricle [2–5].

Left to right shunt due to large VSD lead to equal pressure on each side of the ventricle. This effect increases pulmonary blood flow leading to congestion and edema. Pulmonary hypertension due to pulmonary vascular alteration and remodeling. To maintain normal systemic blood flow in patients with considerable left to right shunting left ventricle output must be increased. Increased alpha-adrenergic, increased circulating catecholamine concentrations, and increased angiotensin II and vasopressin concentrations exacerbate cardiac failure [6–10].

## Clinical Features

### Presentation

VSDs of moderate to large size can be discovered in the womb early which can happen on their own or in combina-

tion with other heart problems. Depending on the size and location of isolated VSDs, some will close during pregnancy [11]. Small to moderate VSDs can be missed by fetal echocardiography due to the physiology of equal ventricular pressure in the fetus. The discovery of an in utero VSD or other structural cardiac abnormalities should prompt discussion about possible chromosomal problems and the need for additional testing [12–14].

Patients with VSD present mostly during the neonatal period. Depending on the magnitude of the lesion, the clinical presentation can range from an isolated murmur discovered by chance at a health monitoring visit to acute cardiac failure [15]. Small, restricted VSDs in infants are frequently asymptomatic. Infants with moderate to large VSDs, on the other hand, frequently show indications of heart failure. Small VSD diagnosis by accident with murmur without clinical symptoms can be detected in the first day of life of the neonate. Symptoms appear in moderate and large VSD infants within first weeks of life, with signs and symptoms of heart failure resemble by poor feeding, tachypnea, failure to thrive, hepatomegaly, cardiomegaly, and pulmonary rales [16]. Thrill can palpate in the third and fourth left sternal border. Blowing holosystolic murmur is classically seen in the patient with VSD heard on the left sternal border, moderate and large VSD heard louder than small, rumbling sounds may be heard if the VSD is big enough to cause mitral valve defect. Diastole rumbling sound usually indicates left to right shunt, heard at the apex. A decrescendo murmur in the left sternal border indicates aortic regurgitation and degree splitting of S2 indicates the size of defect [17–20].

## Diagnosis

VSD diagnosis by Echocardiogram, to make the diagnosis, locate the defect, and estimate the size of the shunt, two-dimensional Doppler echocardiography is usually sufficient. Echo is used to confirm the diagnosis that is made typically by holosystolic murmur, locate the defect and measure the size. Chest radiograph if obtained is not that useful and varies depending on the size of the defect also may show signs of heart failure if developed in a baby. ECG is typically normal in most of the cases but also shows left ventricle hypertrophy. Now a days cardiac catheterization is rarely used for diagnosis [21, 22].

## Differential Diagnosis

Usually, VSD can be distinguished from non-cardiac causes of respiratory distress by the presence of systolic murmur on physical exam and diagnostic test. Other anomalies like acyanotic congenital heart disease also can distinguish by ECHO [21, 22].

## Management

### Overview

The extent of the defect and degree of shunting are determined by the clinical examination and echocardiography. Patient with small VSD does not require surgical intervention, these patients are usually asymptomatic, with a good chance of spontaneous closure or a reduction in the extent of the defect over time. Medical therapy patients with heart failure symptoms must receive medical treatment [23]. Medical care may be sufficient to meet the needs of people with moderate defects. Surgical correction is frequently required for people with more severe symptoms, and medication care is used to alleviate symptoms in the meanwhile. Patients with a risk for long-term sequelae and failed medical therapy are recommended to the closure of the defect surgically [24].

### Neonates

Ongoing neonatal surveillance is critical for determining which infants will remain asymptomatic and require no intervention versus those who will develop heart failure and require intervention.

### Small VSD

Small VSD asymptomatic also good chance of spontaneous closure. Patients should have a follow-up assessment by a pediatric cardiologist at 3–4 weeks of age to detect any indications or symptoms of increased left ventricular volume overload. A follow-up evaluation with the cardiologist is scheduled for those patients who remain asymptomatic at roughly 6 months of age. Primary care provides routine care in between visits with the cardiologist. If the patient develops symptoms (e.g., poor weight gain, tachypnea), he or she should be referred to a specialist for a cardiac evaluation as soon as possible. If the murmur is no longer present at the 6-month visit, a repeat echocardiogram is not required unless clinical concerns occur. If the murmur is still present at the 12-month cardiology visit and the patient is asymptomatic and clinically stable, there is no need for additional treatment. Patients with membranous defects usually have an echocardiographic follow-up at 3 years of age. If a patient with a muscular defect stays asymptomatic, no echocardiogram is required. In any symptomatic patient, medical therapy is started. However, because heart failure is not commonly associated with small VSDs, the emergence of new symptoms, especially late in the course of the disease, should prompt a reevaluation of the original diagnosis and a search for other sources of the symptoms [25, 26].

## Moderate to Large VSD

Pulmonary vascular resistance (PVR) diminishes, and infants with moderate to large VSDs frequently become symptomatic within the first few months of life. During the first weeks of life, the primary care provider should keep an eye on the baby for signs of heart failure. Because of the projected decrease in PVR resulting in increased left ventricular (LV) flow. If symptoms arise, medical treatment is recommended, as described in the sections below. Infants who remain asymptomatic should be followed up on and monitored on a regular basis [11, 27].

Asymptomatic patients—all infants with moderate to large VSDs should have regular follow-up during their first year of life, even if symptoms are absent. It is critical to look for signs of pulmonary hypertension in these people. Echocardiography is conducted to determine pulmonary artery pressure if the murmur is diminished but the pulmonic component of the second heart sound (S2) is increased in strength [15]. Symptomatic patient may avoid surgical intervention through medical ways and management, including nutritional support and pharmacological treatment. Nutritional support may help infants with moderate to large VSDs gain weight. Because of the higher metabolic demand, these infants may require a caloric intake of more than 150 kcal/kg per day [28]. Feeding fatigue is common in infants with heart failure, and their intake may be reduced. Providing more frequent feedings is one way to enhance daily calorie intake; however, parents may find this difficult. These infants typically take a long time to eat, and the time required to ensure optimal intake might be significant. To increase caloric intake, nasogastric feedings may be required. Bolus or continuous feeds, which can be administered at any time of day or night, are examples of this. When these procedures are used, it usually means that the VSD will need to be closed. Fluid restriction is often ineffective in the therapy of infants with VSD-related heart failure because it leads to insufficient calorie intake. Although fluid restriction is commonly used to treat adults and older children with heart failure, it is ineffective in infants who are completely reliant on liquids. As stated in the following section, diuretic therapy rather than fluid restriction should be utilized to minimize and avoid volume excess. Supplemental iron should be given to infants with iron deficiency anemia to boost their hematocrit and oxygen-carrying capacity. Medical care for heart failure differs based on the severity of the symptoms of heart failure [29]. The mainstay of treatment is diuretics. Angiotensin-converting enzyme inhibitors have been utilized in the past, but their efficacy appears to be low therefore they are no longer used consistently. Intravenous (IV) inotropic drugs may be administered as a temporary strategy in extreme situations. We rarely prescribe oral digoxin because of the risk of side effects [30–32].

## Closure Interventions

The severity of heart failure, vascular disease (PHVD) progression, pulmonary hypertension or other complications, the likelihood of defect reduction or spontaneous closure, the morbidity and mortality of the procedure in young infants in the center where surgery to be performed, and the likelihood of successful surgical closure are all factors to consider [33–35]. Because infants with Down syndrome are more likely to have PHVD, early surgical intervention in children with moderate to large defects may be necessary. In most cases, primary patch surgical closure is the recommended method. Transcatheter closure is often reserved for patients with defects that are difficult to close operatively (e.g., remote apical muscular defect, multiple muscular defects [“Swiss cheese” septum]) or who are unable to undergo cardiopulmonary bypass for a variety of reasons. Indications if symptoms are persistent with the option of medical management, pulmonary hypertension is established, and reversible shunt (left to right). VSD closure is generally not recommended if pulmonary vascular resistance (PVR) is greater than 12 Wood units (WU). Closure of the defect may result in decreased cardiac output and increased peri-operative mortality in these patients with severe pulmonary hypertension, as previously mentioned [35]. Method of choice for most children with VSD required surgical intervention is direct patch closer under cardiopulmonary bypass. Transcatheter closure several series have documented successful transcatheter closure for muscular, peri-membranous, and residual VSDs following surgical repair. Though the technique has gained favor in some countries, transcatheter closure of VSD remains technically demanding and has a greater complication rate than surgery [36–40].

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