



## Ventricular Septal Defect

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### Abstract

Ventricular septal defect(VSD) is most prevalent congenital heart defect, which is generally distinguished by an atypical opening in the inter ventricular septum. This abnormality leads to the permission of blood to shunt from left ventricle to the right ventricle, which further leads to the over circulation in pulmonary, results in volume overload, and also increase the risk for potential failure of the heart, in condition if its left untreated. VSD severity totally based on the defect's size and the pulmonary vascular resistance. This following article for VSD scrutinises the pathophysiology, it's classifications, clinical manifestations, the diagnostic evaluation approaches and the strategic management. Advancements in surgical procedures and catheter-dependent interventions has lead in outstanding improvement of outcomes for the affected individuals from the same. In advance diagnosis and pertinent management always play a vital role in the reduction of morbidity and the mortality corresponding to VSD.

**Keywords:** Ventricular septal defect, congenital heart disease, cardiac surgery, paediatric cardiology, echocardiography, heart failure

### Introduction

Ventricular septal defect (VSD) is most prevalent congenital heart disease(CHD) which gets diagnosed, approximately 2-6 per 1,000 live births it is occurring. Also nearly 20-30% it accounts of all congenital heart defects, and this data representing the occurring frequency in the environment. This particular condition is a result of incomplete inter ventricular septum closure in the course of fetal development, further leading to left-to-right shunting of the blood. In situation of small defect in closure, it may gets close eventually or spontaneously whereas, the larger defects results in remarkable hemodynamic compromise and calls attention for the intervention medically or surgically.

## 2. Embryology and Pathophysiology

### 2.1 Embryological Basis

The development of the inter ventricular septum during gestation period takes place between the 4 and 7 weeks, consisting muscular portion which raised from ventricular myocardium with a membranous portion which derived from the cushions of endocardium. The fusion of these takes place for the successful closure and a healthy development of heart functioning but fusion failure leads to defect which opens the communication channel among left ventricle and right ventricle.

### 2.2 Hemodynamic Consequences

Cardiac functioning deflects due to the impact of VSD and it depends upon the size of the defect occurs and pulmonary vascular resistance:

1. Small VSDs(<3mm): It is often asymptomatic and gets closed spontaneously.

2. Moderate VSDs(3-6mm): It can lead to a remarkable left-to-right shunt, further leads to volume overload.
3. Large VSDs(>6mm): It is having potential to render outcome of increased pulmonary blood flow, which leads to pulmonary hypertension, heart failure and Eisenmenger syndrome if it's left untreated.

### 3. Classification of Ventricular septal defect

VSDs classification relies on the location and its size

1. Perimembranous VSD (70-80%)- Located near the tricuspid valve in the membranous septum.
2. Muscular VSD (5-20%)- Found in multiple numbers in the muscular portion of the septum.
3. Inlet VSD (5-10%)- It is located near the atrioventricular valves.
4. Outlet VSD (5-7%)- It is more in Asian population, and found near the pulmonary and aortic valves.

#### 3.2 Based on Size

1. Small (or restrictive) VSD: Shows high resistance to the flow and minimal hemodynamic effect.
2. Moderate VSD: Significant shunting is reflected.
3. Large (Non-restrictive) VSD: Left to right shunting, Eisenmenger syndrome.

### 4. Clinical Manifestation

1. Small VSD: Asymptomatic or murmur may present.
2. Moderate to Large VSD: Failure to thrive, tachypnea, dyspnea, frequent respiratory infections, signs of heart failure, Eisenmenger syndrome.

### 5. Diagnostic approaches

#### 5.1 Clinical Examination

1. Murmur
2. Thrill: It is palpable in larger defects.
3. Signs of heart failure: Hepatomegaly, pulmonary congestion and tachycardia.

#### 5.2 Imaging Studies/Radiological Studies

1. Echocardiography
2. Chest X-ray
3. Cardiac MRI
4. Electrocardiogram (ECG)
5. Cardiac Catheterization

### 6. Management studies

#### 6.1 Medical management

1. Diuretics: To reduce pulmonary congestion.
2. ACE Inhibitors: To reduce afterload.
3. Nutritional support: For infants having poor weight gain, high-calorie feeding is preferred.
4. Pulmonary vasodilators: They are used in pulmonary hypertension case.

#### 6.2 Surgical management

1. Open heart surgery
2. Transcatheter closure

#### 6.3 Nursing management

1. Assess cardiac function support
2. Monitor nutritional support and assess adequate weight gain
3. Allow adequate rest and activity
4. Prevent infection
5. Provide parental education and psychological support
6. Assist in pre and post operative care

#### 6.3 Prognosis and Long-term follow-up

1. Surgical closure have an excellent rate te of success (>95%).
2. Patients with VSD requires lifelong monitoring and follow-up.
3. In small VSDs, spontaneous closure occurs in 30-50% only.
4. Early interventions prevent complications.

### Conclusion:

With variable clinical presentation the ventricular septal defect (VSD) remains a general congenital heart disease and early diagnosis and adequate interventions considerably improves the outcome in the patient. The need of open-heart surgery reduces somewhat with the advancement in interventions such as imaging techniques and catheter-based interventions.

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