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VENTRICULAR SEPTAL DEFECT: A COMPREHENSIVE LITERATURE REVIEW OF PATHOPHYSIOLOGY, DIAGNOSIS, AND TREATMENT

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ABSTRACT

Background: Ventricular septal defect (VSD) is the most prevalent congenital heart defect, characterized by an abnormal communication between the ventricles, leading to a left-to-right shunt. Its clinical spectrum ranges from asymptomatic small defects to large shunts causing heart failure, pulmonary hypertension, or Eisenmenger syndrome. Early diagnosis and appropriate management are critical to prevent long-term complications.
Methods: This literature review synthesizes current evidence on the classification, pathophysiology, diagnostic modalities, treatment options, and long-term outcomes of VSD. A comprehensive analysis of peer-reviewed articles and clinical guidelines was conducted to present up-to-date insights into the multidisciplinary approach to VSD care.
Results: Anatomical subtypes of VSD vary in clinical significance, risk of complications, and management strategies. Advances in imaging, particularly echocardiography and cardiac MRI, have improved diagnostic precision. While small defects often resolve spontaneously and are managed conservatively, large or symptomatic defects may require surgical or transcatheter closure. Surgical repair remains the standard for most cases, but selected muscular and perimembranous defects can be treated percutaneously. Long-term follow-up is essential due to risks of arrhythmias, conduction block, residual shunts, or valve dysfunction. Emerging technologies—including biodegradable devices, minimally invasive surgery, and experimental gene therapy—may reshape future management paradigms.
Conclusions: VSD management requires individualized, anatomy-guided decision-making supported by multidisciplinary expertise. Technological progress has expanded therapeutic options, yet lifelong surveillance remains necessary to mitigate late complications and optimize outcomes.

KEYWORDS

Heart Septal Defects, Ventricular, Pulmonary Hypertension, Echocardiography, Cardiac Catheterization, Transcatheter Closure

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1. Introduction

Ventricular septal defect (VSD) is the most frequently diagnosed congenital cardiac malformation, characterized by an abnormal communication between the right and left ventricles, resulting in a left-to-right shunt under normal hemodynamic conditions *[1]*. Its clinical significance lies not only in its prevalence but also in its potential to cause congestive heart failure, pulmonary hypertension, and growth retardation if left untreated *[2]*.

The reported incidence of VSD varies with the sensitivity of diagnostic methods. With the advent of high-resolution echocardiography, VSDs are identified in up to 4 per 1,000 live births, although many small muscular defects close spontaneously during infancy and go undetected into adulthood *[3]*. While most VSDs are diagnosed during infancy, some remain silent until adolescence or adulthood, where they may be discovered incidentally or through evaluation of a murmur, stroke, or endocarditis *[4]*.

Understanding the anatomy, pathophysiology, and long-term implications of VSD is essential for guiding patient management across the lifespan. The purpose of this review is to provide a comprehensive synthesis of the pathophysiology, diagnostic strategies, and evidence-based treatment options for VSD, based on current literature and clinical guidelines *[5]*.

2. Classification and Etiology

Ventricular septal defects are classified according to their anatomical location within the interventricular septum. The most common types include perimembranous, muscular, inlet, and outlet (supracristal) defects. Perimembranous VSDs, located near the membranous septum and adjacent to the tricuspid and aortic valves, account for approximately 70–80% of all cases *[6]*. Muscular defects may occur anywhere along the muscular septum and are further subdivided into apical, central, and marginal types *[7]*. Inlet VSDs are associated with atrioventricular septal defects, while outlet VSDs—more common in East Asian populations—carry a higher risk of aortic valve prolapse and regurgitation *[8]*.

Etiologically, most VSDs are congenital, resulting from incomplete closure of the interventricular septum during embryogenesis. The defect often arises from disruption in the fusion of the muscular and membranous septum around the 4th to 8th week of gestation *[9]*. Several genetic syndromes, such as trisomy 21, 13, 18, and DiGeorge syndrome, are associated with increased incidence of VSDs, indicating a strong genetic component in pathogenesis *[10]*.

In contrast, acquired VSDs, though rare, can occur secondary to myocardial infarction (post-infarction VSD), blunt chest trauma, or iatrogenic injury during interventional or surgical cardiac procedures *[11]*. Post-infarction VSD is a life-threatening complication, often presenting with acute hemodynamic instability and requiring urgent surgical repair.

Environmental factors, including maternal diabetes, alcohol exposure, teratogens, and rubella infection, have also been implicated in disrupting normal septal development during embryogenesis *[12]*.

A precise understanding of the anatomical subtype and underlying etiology of VSD is vital for prognostication and determining the most appropriate treatment strategy, as different types carry distinct risks for complications such as arrhythmias, aortic valve disease, or pulmonary hypertension.

3. Pathophysiology

The primary physiological disturbance in ventricular septal defect (VSD) is the formation of a left-to-right shunt between the ventricles, driven by the higher pressure in the left ventricle during systole. This causes oxygenated blood to re-enter the pulmonary circulation, leading to increased pulmonary blood flow and volume overload of the pulmonary vasculature and left-sided heart chambers *[13]*.

Over time, this volume overload results in left atrial and left ventricular dilation, as well as elevated pulmonary venous return, predisposing patients to pulmonary congestion and congestive heart failure, particularly in infancy. The degree of overload correlates with defect size and pulmonary vascular resistance *[14]*.

In moderate to large VSDs, the persistent elevation of pulmonary blood flow stimulates remodeling of the pulmonary vasculature. This involves medial hypertrophy, intimal proliferation, and eventually obliterative changes in small pulmonary arteries, increasing pulmonary vascular resistance *[15]*. If left uncorrected, these changes may lead to irreversible pulmonary arterial hypertension and ultimately to Eisenmenger syndrome, a life-threatening condition characterized by shunt reversal, cyanosis, and right heart failure *[16]*.

The natural history of VSDs varies depending on anatomical location and size. Small muscular defects often close spontaneously during infancy due to the growth of surrounding myocardium or endocardial proliferation. This spontaneous closure may be complete or partial, leaving a restrictive residual shunt that requires continued surveillance *[17]*.

Perimembranous VSDs frequently develop aneurysmal tissue over time, often involving the septal leaflet of the tricuspid valve. This tissue may partially occlude the defect, contributing to either spontaneous closure or creation of a "tunnel-like" shunt with altered hemodynamics *[18]*.

Importantly, the atrioventricular conduction system lies adjacent to perimembranous VSDs. Progressive aneurysmal transformation or surgical manipulation in this area increases the risk of conduction abnormalities, including bundle branch block or even complete heart block, particularly following surgical closure *[19]*.

In severe cases, long-standing left-to-right shunting leads to right ventricular hypertrophy and eventual right ventricular failure, especially when pulmonary pressures approach or exceed systemic levels. The clinical manifestation of Eisenmenger syndrome includes clubbing, polycythemia, hemoptysis, and significantly reduced life expectancy *[20]*.

Thus, the pathophysiology of VSD is a dynamic interplay between hemodynamics, vascular remodeling, and anatomical evolution, which must be closely monitored to guide timing and modality of intervention.

4. Clinical Presentation

The clinical presentation of ventricular septal defect (VSD) is influenced by the size, location, and hemodynamic impact of the defect, as well as the patient's age at diagnosis *[21]*. In infants with large or nonrestrictive VSDs, symptoms typically emerge within the first weeks of life and include tachypnea, failure to thrive, feeding difficulties, and recurrent respiratory infections due to pulmonary overcirculation and left-sided volume overload *[22]*.

These infants may present with diaphoresis during feeding, hepatomegaly, and signs of congestive heart failure. On auscultation, a harsh, pansystolic murmur is commonly heard at the left lower sternal border and may be accompanied by a palpable thrill. The murmur is typically loudest in small restrictive defects with high-velocity shunting *[23]*.

Small VSDs, especially muscular types, are often asymptomatic and discovered incidentally during routine pediatric examination. Many of these defects close spontaneously during early childhood and do not require intervention but warrant echocardiographic surveillance *[24]*.

Despite being clinically silent, small VSDs are not without risk. Infective endocarditis has been reported in small or residual defects, particularly in those with persistent high-velocity jets, making prophylaxis an important consideration in selected patients *[12]*.

In older children and adolescents, persistent or partially closed VSDs may result in exercise intolerance, fatigue, or subtle signs of volume overload. Outlet-type defects are especially prone to causing aortic valve prolapse, which can progress to aortic regurgitation due to distortion of the valve cusp *[11]*.

Arrhythmias, including right bundle branch block, AV block, or supraventricular tachycardia, may be observed in patients with post-surgical scars or membranous aneurysmal transformation, particularly when the conduction system is affected *[25]*.

In adults with long-standing uncorrected VSDs, especially those with large defects, progressive pulmonary vascular disease may result in Eisenmenger syndrome. Clinical signs include cyanosis, clubbing, polycythemia, and symptoms of right heart failure, typically presenting in the third or fourth decade of life *[26]*.

Adults with previously undiagnosed VSDs may first present with stroke from paradoxical embolism, new-onset arrhythmias, or an incidental murmur noted during evaluation for unrelated complaints. These cases underscore the importance of lifelong cardiology follow-up, even for patients with previously minor or repaired defects *[27]*.

5. Diagnosis

The diagnosis of ventricular septal defect (VSD) begins with clinical auscultation. A harsh holosystolic murmur, most pronounced at the left lower sternal border, is a characteristic finding in most cases and is often accompanied by a systolic thrill, especially in small restrictive defects *[28]*. The murmur's intensity is inversely related to the size of the defect, as smaller VSDs generate higher-velocity flow that produces louder sounds *[29]*.

Transthoracic echocardiography (TTE) is the primary diagnostic modality, allowing direct visualization of the VSD, assessment of its size, location, and shunt direction. It also enables evaluation of cardiac chamber enlargement and estimation of pulmonary artery pressures through color and spectral Doppler imaging *[22]*.

In cases where acoustic windows are limited, particularly in neonates, postoperative patients, or individuals with complex anatomy, transesophageal echocardiography (TEE) offers superior spatial resolution, especially for identifying perimembranous or outlet-type defects, and is routinely used during intraoperative or catheter-based evaluations *[30]*.

Cardiac magnetic resonance imaging (MRI) plays a growing role in the noninvasive assessment of shunt fraction (Qp/Qs), ventricular volumes, and right heart pressures, particularly in adolescents and adults, or when echocardiographic views are suboptimal *[31]*.

Cardiac catheterization is no longer routinely performed for diagnosis in children but remains indispensable for evaluating pulmonary vascular resistance (PVR) and shunt magnitude in patients with suspected pulmonary hypertension or uncertain operability. It provides the most accurate hemodynamic data for surgical decision-making *[32]*.

Angiography, performed during catheterization or device closure, offers detailed visualization of muscular, apical, or multiple fenestrated VSDs, and is essential for selecting and deploying occlusion devices in transcatheter procedures *[33]*.

The differential diagnosis of a holosystolic murmur includes atrioventricular septal defects, tricuspid regurgitation, and ruptured sinus of Valsalva aneurysms. Distinguishing between these conditions requires high-resolution imaging of the septal and valvular structures using color Doppler echocardiography *[34]*.

Together, a structured diagnostic approach that integrates clinical signs, echocardiography, cardiac MRI, and catheterization when indicated, provides the anatomical and hemodynamic clarity necessary for effective treatment planning.

6. Treatment

Management strategies for ventricular septal defect (VSD) are determined by the size of the defect, its hemodynamic significance, the presence of clinical symptoms, and the risk of complications such as pulmonary hypertension or endocarditis *[28]*.

6.1 Conservative Management

Small, hemodynamically insignificant VSDs, particularly muscular types, are often managed conservatively, as many of them undergo spontaneous closure within the first years of life *[31]*. These patients are monitored through regular clinical assessments and echocardiography, focusing on ventricular size, shunt flow, and valvular function *[22]*.

Medical therapy is used in symptomatic infants with large VSDs who are not immediate surgical candidates. Treatment includes diuretics (e.g., furosemide) to reduce pulmonary congestion, ACE inhibitors to decrease afterload, and beta-blockers in selected cases to reduce myocardial oxygen demand *[35]*.

Nutritional support and fluid restriction may also be necessary, especially in failure-to-thrive infants. With optimized medical management, some patients may stabilize enough to delay or avoid surgical intervention, particularly if the pulmonary vascular resistance remains favorable *[36]*.

6.2 Interventional and Surgical Treatment

Surgical closure remains the gold standard for large or symptomatic VSDs, especially those associated with left heart volume overload, failure of medical therapy, or evidence of pulmonary hypertension *[29]*. Surgery typically involves patch closure via median sternotomy and cardiopulmonary bypass. The surgical risk is low in isolated VSD repair, particularly in high-volume centers *[24]*.

However, complications such as complete heart block, residual shunts, and valvular damage remain concerns, especially in defects adjacent to the conduction system or valves *[30]*.

Transcatheter device closure has emerged as a viable alternative for selected patients, particularly those with muscular or select perimembranous VSDs, where the anatomy is favorable and proximity to the conduction system is minimal *[33]*. Devices such as the Amplatzer occluder and duct occluders have been used with high closure rates and low complication profiles *[8]*.

However, transcatheter closure carries a risk of atrioventricular block, especially in perimembranous VSDs, due to mechanical pressure on the AV node region. This has led to greater caution and patient selection protocols to mitigate risk *[34]*.

Comparative studies suggest that while surgery offers definitive repair, transcatheter approaches reduce hospital stay, recovery time, and eliminate sternotomy-related morbidity, though long-term outcomes are still under evaluation *[6]*.

Therefore, treatment of VSD should be individualized based on anatomy, age, comorbidities, and institutional expertise. Hybrid techniques and novel devices may further expand treatment options in complex or high-risk patients *[32]*.

7. Prognosis and Long-Term Outcomes

Long-term outcomes after ventricular septal defect (VSD) closure are generally favorable, particularly in patients who undergo early and uncomplicated repair *[37]*. In children who receive surgical or transcatheter closure during infancy, survival rates approach those of the general population, and most individuals lead active, asymptomatic lives *[23]*.

However, residual shunts are not uncommon, especially in patients with multiple or muscular defects, and may persist despite technically successful closure *[38]*. These may be hemodynamically insignificant but still require lifelong monitoring due to the risk of infective endocarditis or progressive valve dysfunction *[9]*.

One of the most serious post-surgical complications is atrioventricular (AV) block, which can occur due to proximity of the patch or device to the conduction tissue—particularly in perimembranous VSDs. In some series, permanent pacemaker implantation was required in a subset of patients following surgical repair *[11]*.

Arrhythmias and ventricular dysfunction may also develop in later years, often related to surgical scarring, residual defects, or progressive pulmonary vascular disease. Patients with prior open-heart surgery are at elevated risk of atrial fibrillation and ventricular arrhythmias, necessitating periodic rhythm monitoring *[26]*.

Even patients with previously small, untreated VSDs are not entirely free from long-term risk. In adults, such defects have been associated with stroke due to paradoxical embolism, infective endocarditis, and progressive aortic regurgitation, particularly in outlet-type defects *[12]*.

Patients with Eisenmenger syndrome, resulting from long-standing uncorrected shunts, have a markedly reduced life expectancy. They may develop right heart failure, hemoptysis, and intractable arrhythmias, and are generally not candidates for defect closure in adulthood *[27]*.

For all patients with a history of VSD—whether repaired or unrepaired—lifelong cardiology follow-up is recommended, with special attention to changes in ventricular size, function, valvular integrity, and arrhythmia surveillance *[20]*.

8. Future Directions

Ongoing advances in the diagnosis and management of ventricular septal defect (VSD) continue to reshape clinical strategies and patient outcomes. One major area of innovation lies in noninvasive imaging, particularly the refinement of three-dimensional echocardiography and fusion imaging technologies, which enhance spatial resolution and procedural planning without radiation exposure *[39]*.

Cardiac MRI protocols are also evolving, allowing for faster acquisition, higher resolution, and automated quantification of shunt flow, ventricular function, and myocardial fibrosis. These advances are expected to improve pre-interventional assessment and post-repair surveillance, particularly in complex or residual VSDs *[7]*.

In the field of intervention, development of next-generation occlusion devices has focused on reducing the risk of conduction disturbances and improving conformability in varied septal anatomies. Innovations in self-expanding, low-profile devices may enable safer closure of perimembranous defects and expand indications for transcatheter therapy *[17]*.

Progress in biomaterials has led to research into resorbable scaffolds, hydrogel-based patches, and biodegradable occluders that could reduce long-term foreign body reactions and facilitate tissue integration. Some of these materials are currently in preclinical testing and may improve long-term safety profiles for pediatric patients *[10]*.

Although still in early phases, molecular and gene therapies targeting cardiac septal development are under exploration. These strategies aim to modulate early cardiac morphogenesis, offering theoretical potential for in utero correction or postnatal prevention of shunt progression in genetically predisposed individuals *[18]*.

Minimally invasive surgical techniques, including robot-assisted and thoracoscopic VSD closure, have demonstrated reduced morbidity, shorter hospital stays, and excellent cosmetic outcomes in select populations. As surgical instrumentation evolves, these approaches may become increasingly adopted, especially in older children and adolescents *[19]*.

As these technologies mature, collaborative research efforts, clinical registries, and randomized studies will be essential to evaluate their safety, effectiveness, and long-term impact on patient care.

9. Conclusions

Ventricular septal defect (VSD) remains the most common congenital heart malformation, with significant variability in anatomical presentation, natural history, and clinical outcomes *[15]*. Early diagnosis through advanced imaging modalities has markedly improved the ability to stratify risk, monitor progression, and guide timely interventions *[4]*.

Surgical repair continues to serve as the gold standard for large and hemodynamically significant defects, while transcatheter approaches offer promising alternatives for muscular and select perimembranous VSDs. Innovations in device technology and minimally invasive techniques are progressively shifting the paradigm toward less invasive management with comparable efficacy *[14]*.

Despite these advances, patients remain at risk for late complications, including arrhythmias, conduction abnormalities, and valve dysfunction. Lifelong surveillance is therefore imperative, even in those with closed or spontaneously resolved defects, to detect evolving sequelae or emerging hemodynamic burden *[16]*.

Future directions emphasize the integration of biomaterials, gene-based therapies, and personalized care pathways supported by longitudinal data and collaborative registries. A multidisciplinary approach, tailored to anatomical and physiological complexity, will remain central to optimizing outcomes in VSD care *[40]*.

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