

Ventricular Septal Defect

Ventricular septal defect is a condition of abnormal interventricular communication due to a septal defect, resulting in intracardiac shunting of blood.

ANESTHETIC CONSIDERATIONS:

- **Shunt size and direction**
- **Paradoxical emboli – bubble precautions**
- Atrial tachyarrhythmias
- Associated cardiac defects
- **Pulmonary HTN** due to ongoing volume overload of the RV and pulmonary circulation (Eisenmengers)
- RV failure in pregnancy and with diastolic dysfunction
- Associated congenital anomalies

ANESTHETIC GOALS:

- Maintain preoperative hemodynamics to avoid exacerbation or reversal of shunt
- In presence of L-R shunt:
 - Preload – high-normal to maintain adequate systemic CO in presence of intracardiac shunt
 - Rate/rhythm – NSR
 - PVR – high-normal
 - Contractility
 - SVR – low-normal
- In presence of R-L shunt:
 - Preload – normal
 - Rate – high-normal to improve RV function
 - Rhythm – NSR
 - PVR – low
 - Contractility – high-normal to improve RV function
 - SVR – normal

HISTORY

- Heart failure
 - Exertional dyspnea, fatigue
 - FTT, diaphoresis with feeding in infants
- Arrhythmias – palpitations, presyncope
- Pulmonary HTN

PHYSICAL

- General – poor weight gain, pallor, cyanosis suggests shunt reversal or infundibular pulmonic stenosis
- CVS
 - Cardiomegaly
 - Holosystolic murmur loudest at LLSB – murmur becomes shorter, softer, and lower pitched as RV pressure increases; small muscular VSDs high pitched, occur only in early systole as muscle contraction closes defect
- Resp – increased work of breathing, pulmonary edema
- GI – hepatomegaly

INVESTIGATIONS

- Labs – CBC, lytes, Cr, liver enzymes, coags, ABG as indicated
- EKG – LVH, RVH, prior MI
- CXR – cardiomegaly, prominent PA, increased pulmonary vascular markings
- Echo
 - VSD size and location, RVP and PAP, gradients
 - Doppler – shunting pattern, holosystolic flow + diastolic flow
 - Contractility, orifice size, and relative ventricular pressures affect intensity of turbulence
 - Less flow if poor LV function, pulmonary HT, larger defects
- Cardiac cath
 - Performed if uncertain anatomy or uncertain PVR
 - PVR >6 Wood U/m2 despite pulmonary vasodilators considered unoperable

TABLE 18-1 (LAKE)

Size	Shunt	Gradient	PVR	RVP	RVH	LVH	Murmur
Small	Small L-R	High	N	N	No	Yes	Holosystolic
Medium	Mod to Lg L-R	20 mmHg	Mild ↑	Mild ↑	Mild	Yes	Holosystolic
Large	Lg L-R Small R-L	None	↑	↑	Yes	Yes	Decrescendo
Large with ↑PVR	R-L	None	↑	↑	Yes	No	Minimal or absent

OPTIMIZATION

- **Medical**
 - Manage CHF
 - See CHF document

- Endocarditis prophylaxis
 - Not required in presence of isolated VSD with L-R shunt (unless associated valve defect)
 - Required if cyanotic CHD
 - Required for 6mo post-repair if prosthetic material used
- **Surgical repair**
 - Goal – prevent pulmonary vascular obstructive disease, treat intractable CHF
 - Indications – refractory heart failure, FTT, recurrent pulmonary infections, pulmonary HTN
 - Contraindications – PVR: SVR > 0.7
 - Techniques
 - Percutaneous – transcatheter closure
 - Open – via median sternotomy

ANESTHETIC OPTIONS

- Regional – caution, uncontrolled SVR exacerbates R-L shunt
- GA – careful induction to maintain PVR: SVR ratio

ANESTHETIC SETUP

- **Drugs**
 - Vasopressors, vasodilators (systemic and pulmonary) to manipulate SVR:PVR
 - Inotropes if evidence of RV failure
- **Monitors**
 - Consider need for artline, CVL, TEE

MANAGEMENT OF ANESTHESIA

- Similar principles as ASD (see ASD seminar)

DISPOSITION & MONITORING

- Monitor for low cardiac output syndrome

COMPLICATIONS

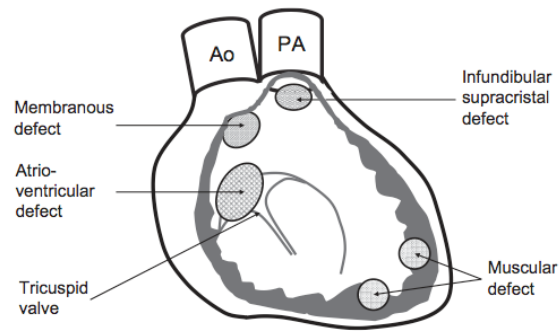
- Air embolism
- RVOT obstruction
- Irreversible pulmonary HT → RV failure
- Eisenmenger's syndrome with shunt reversal → hypoxemia

OBSTETRICS

- Higher incidence of fetal cardiac lesions
- Pregnancy has minimal impact on small non-restrictive VSD
 - L-R shunt may increase due to ↑CO in pregnancy, however this is counterbalanced by ↓SVR
- Large shunts and PAH in pregnancy – risk of arrhythmias, ventricular dysfunction, progression of PAH
- VSD patients who survive to adulthood usually have Eisenmenger syndrome
 - Pregnancy contraindicated

PATHOPHYSIOLOGY

- **Epidemiology**
 - Most common congenital heart defect in children
 - Accounts for 30% of pediatric CHD
- **Etiology**
 - Congenital
 - Acquired
 - MI – 1-2%, 1wk post-MI new holosystolic murmur, ↑risk with ventricular septal aneurysm, presents with sudden worsening CHF, rapid progression to cardiogenic shock
 - Trauma – blunt (contusion →necrosis →perforation), penetrating (laceration)
- **Classification**
 - Type I
 - Subarterial (aka supracrystal, 5%) – in region of subpulmonic infundibulum beneath AV valves; associated AI from AV prolapse
 - Type II
 - Perimembranous (aka infracristal, 75%) – in membranous septum near TV leaflet; associated TV aneurysms, subaortic stenosis
 - Type III
 - Inlet (aka AV canal, 5%) – in posterior septum of atrioventricular canal; associated TV and MV defects
 - Muscular (15%) – within muscular or trabecular septum; may become smaller during late systole; multiple defects (> 3) = swiss cheese septum



- **Associated cardiac defects**
 - Bicuspid aortic valve, aortic coarctation, RVOT obstruction, complex CHD
- **Natural Hx**
 - Diagnosed ~2-6 wks of age by new murmur
 - Coincides with expected ↓ in PVR and associated shunting across defect
 - Spontaneous closure
 - More common than with ASD
 - Common in Type IV defects, may occur in Type I and II, rarely in Type III
 - Common in defects < 5mm, rare if > 6.5mm
 - Size of nonrestrictive VSD may decrease over time and become restrictive
- **Characterization by size**
 - Small
 - PVR: SVR < 0.3 and Qp/Qs < 1.4
 - Moderate
 - PVR: SVR > 0.3 and Qp/Qs 1.4 – 2.2
 - LAE, LV dilation, ↑PA pressures
 - Large unrestrictive
 - PVR: SVR > 0.3 and Qp/Qs > 2.2
- **Characterization by pathophysiology**
 - Restrictive
 - LV pressure > RV pressure
 - L-R shunt; magnitude of shunt determined by pressure gradient across VSD
 - ↑er L-R gradient, however limited flow through shunt; ↑PA diameter, N RVSP, N PVR, LVH
 - Minimal additional cardiac or pulmonary workload
 - Nonrestrictive
 - RVSP = LVSP
 - Shunt direction *and* magnitude determined by PVR:SVR ratio
 - L-R shunt – ↑pulmonary blood flow, ↑pulmonary HT, pulmonary edema, ↑myocardial work, LVH, RVH, ↑risk pulmonary infections
 - R-L shunt – hypoxemia
- **Pathophysiologic consequences**
 - Shunting
 - L-R shunt
 - LV output must to maintain systemic blood flow at normal levels; eventual high output heart failure
 - CHF
 - When PVR ↓s in infancy → overcirculation → CHF
 - Pulmonary HTN

REFERENCES

- Cote (p. 343-44, 479-80), Chestnut, Coexisting (p. 45-46)
- Pediatric Cardiac Anesthesia (Lake), Chpt 18
- Anesthetic implications of grown-up congenital heart disease. Lovell. BJA 2004;93:129-39
- ACC/AHA 2008 guidelines for management of CHD. Circulation 2008;118:e749-52