Atrial Septal Defect

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

ANESTHETIC CONSIDERATIONS:

- Shunt size and direction
- Paradoxic emboli – bubble precautions
- Atrial tachyarrhythmias
- Pulmonary HTN
- RV failure
- Associated valvular defects
- Associated congenital syndromes

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

- Most patients asymptomatic until adulthood; symptoms once Qp/Qs >3
- Children may present with CHF, frequent respiratory infections, FTT, fatigue
- Heart failure – dyspnea, fatigue, exercise intolerance
- Arrhythmias – palpitations, presyncope
- Pulmonary HTN (in <5% of patients)

PHYSICAL

- CVS
  - Elevated JVP
  - Cyanosis (shunt reversal)
  - Systolic murmur LUSB (↑flow across PV), wide and fixed split S2
  - Atrial fibrillation/flutter
  - Pulmonary HTN
- RESP
  - Pulmonary edema
  - Wheeze (dilated PA's compressing bronchi)
- GI
  - Hepatomegaly
- CNS
  - Neurologic deficits (CVA from PAE)

INVESTIGATIONS

- Labs – CBC, lytes, Cr, liver enzymes, coags, ABG as indicated
- EKG – RAE, incomplete RBBB with RV dilation, RVH, AFib
- CXR – RA, RV and PA enlargement
- Echo – TEE (gold standard) for ASD size, location, shunt direction and severity; color Doppler, bubble study
- Cardiac cath – usu not required for diagnosis; 10% increase in O2 saturation between SVC/IVC and RA, and RV:PA gradient may by 15-30mmHg across a normal pulmonary valve

OPTIMIZATION

- **Medical**
  - Endocarditis prophylaxis
    - Not required in presence of isolated ASD with L-R shunt (unless associated valve defect)
    - Required if cyanotic CHD
    - Required for 6mo post-repair if prosthetic material used
- **Surgical**
  - Early closure desirable (prior to 6-8 mo) to ↓risk of developing irreversible pulmonary vascular disease
    - Closure beneficial even in patients over 60 y.o.
  - Indicated when Qp/Qs ≥1.5, RV volume overload
    - Percutaneous – transcatheter device closure of PFO or secundum ASD
    - Open – via sternotomy; suture closure or patch repair via right atriotomy
  - If need to postpone corrective surgery in infancy – PA banding to ↓pulmonary blood flow until definitive closure possible

ANESTHETIC OPTIONS

- **Neuraxial anesthesia**
  - Epidural preferred over spinal – rapid ↓SVR can cause L-R shunt to become R-L shunt with hypoxemia
- **General anesthesia**
  - 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**
  - SpO2 to detect hypoxemia as a precipitant or a result of shunt reversal (R-L shunt)
  - Consider need for artline, CVL, TEE
  - Ensure all fluid tubing is bubble-free

MANAGEMENT OF ANESTHESIA

- **Induction**
  - Inhalational induction accelerated in L-R shunt (↑pulmonary blood flow ↑s uptake) and slowed in R-L shunt (less blood flow through lungs = less uptake); theoretical, may not be clinically significant
  - Intravenous induction slowed in L-R shunt (↑pulmonary blood flow dilutes drugs) and accelerated in R-L shunt (drug bypasses pulmonary circulation); theoretical, may not be clinically significant
    - Etomidate useful for hemodynamic stability
    - Ketamine useful to maintain SVR and contractility with minimal effect on PVR
  - Rapid intubation to avoid hypoxemia and hypercarbia which can cause shunt reversal (R-L shunt)
    - Adequate depth of anesthesia to avoid ↑SVR and worsening L-R shunt
- **Maintenance**
  - Balanced anesthesia technique
  - In presence of L-R shunt
    - Caution with factors that ↑L-R shunt (prolonged ↑SVR (drugs), ↓PVR (high FiO2, hypocarbia))
    - May need to ↓L-R shunt (↓SVR (volatiles, histamine release), ↑PVR (positive pressure ventilation))
  - In presence of R-L shunt
    - Caution with factors that ↑R-L shunt (↓SVR (volatiles, most iv induction agents, histamine release), ↑PVR (positive pressure ventilation))
    - May need to ↓R-L shunt (↑SVR (drugs), ↓PVR (high FiO2, relative hypocarbia, optimal PEEP))
    - ETCO2 *underestimates* PaCO2
  - Avoid air emboli - ensure no bubbles in iv tubing, avoid N2O
- **Emergence**
  - Adequate analgesia to prevent catecholamine-induced SVR
  - Avoid hypoxemia and maintain normocarbia

DISPOSITION & MONITORING

- Monitor for arrhythmias
- Risk of low cardiac output syndrome if preop CHF
- Careful hemodynamic monitoring and ICU if pulmonary HTN and RV failure

COMPLICATIONS

- Paradoxical embolism
- Arrhythmias
- Pulmonary HT
- Hypoxemia (shunt reversal)
- RV failure

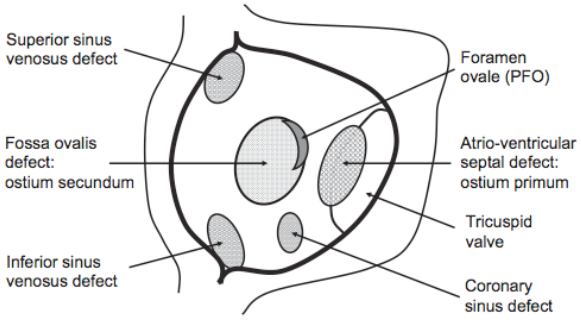
OBSTETRICAL

- Modest L-R intracardiac shunt from ASD well-tolerated in pregnancy
  - Shunt may ↑due to ↑CO however this is counterbalanced by ↓SVR
- Increased blood volume of pregnancy in setting of large ASD → progression of heart failure, pulmonary HTN
  - Pregnancy contraindicated in Eisenmenger syndrome
- ASD transmission to fetus in 8-10% of cases
- Serial echocardiograms during pregnancy to identify reversal of shunt flow
- Hemorrhagic shock can transiently reverse shunt flow
- Early epidural indicated
  - Pain with L+D ↑s catecholamines and SVR → worsen L-R shunt → risk of pulmonary HT and RV failure
- Epidural preferred over spinal for operative delivery
  - More gradual onset sympathectomy avoids rapid SVR which could cause L-R shunt to become R-L shunt with hypoxemia
  - Administer supplemental O2 and monitor SpO2 to prevent and detect shunt reversal

PATHOPHYSIOLOGY OF ASD

- **Classification**
  - PFO (probe-patent 27%) – defect in foramen ovale; associated migraines
    - Unlike an ASD, PFO's do *not* permit L-R shunt but will permit R-L shunt if RAP > LAP (eg: sneezing)

- Primum ASD (15%) – inferior septum; component of endocardial cushion defect, associated cleft MV and MR
- Secundum ASD (75%) – mid-septum in region of fossa ovalis; associated MVP
- Sinus venosus ASD (10%) – upper septum near opening of SVC; associated with anomalous pulmonary venous return
- Coronary sinus ASD (rare) – communication between LA and coronary sinus with blood flow from LA to RA via coronary sinus; associated complex CHD
- Common atrium – complete absence of atrial septum; associated asplenia



- **Epidemiology**
  - ASD accounts for 7-10% of all pediatric CHD and 30% of adult CHD
    - More common in females
    - 2nd most common CHD in adults
- **Natural Hx**
  - Small defects (< 3mm) – small shunt, minimal hemodynamic consequences; majority close spontaneously
  - Medium-sized defects (3-8mm) – 80% close spontaneously
  - Large defects (> 8mm) – large shunt, significant hemodynamic consequences; usu require surgical closure
- **Pathophysiology**
  - Minimal R-L shunting when Qp/Qs <1.5
  - RV dilation when Qp/Qs > 1.8 with ↑risk RV dysfunction and pulmonary HTN
  - Symptoms when Qp/Qs > 3
- **Shunting**
  - Direction and magnitude of shunting depends on difference b/w RAP and LAP, as determined by:
    - Size of defect (< 5mm = minimal hemodynamic consequences
    - Relative ventricular compliances
    - Relative systemic and pulmonary venous capacitance and pressure
    - Degree of TR
  - Infants have less compliant RV – resistance to emptying of RA → RAP → minimal shunting
    - With PVR, L-R shunt develops
  - L-R shunt
    - Occurs when LAP > RAP
    - Results in R-sided volume overload → RA/RV dilation, ↑pulmonary blood flow → pulmonary HTN
      - Dilated PA's may compress bronchi
      - ↓Forward CO; eventual R heart failure
    - Accentuated by ↓venous return
  - R-L shunt
    - Occurs when RAP > LAP
    - Results in hypoxemia
    - Precipitated by ↑PVR (pulmonary HT, Eisenmenger's syndrome, high PEEP, hypoxemia, vasoconstrictors), PE, RV infarction, TR
    - Reduced by nitroglycerin

REFERENCES

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