

# Mitral Stenosis

Narrowing of the mitral valve prevents LV filling with a resultant decrease in SV and CO; it also prevents emptying of the LA with implications of increased PA pressure, AFib and atrial thrombus formation.

## ANESTHETIC CONSIDERATIONS:

- Cardiac physiologic changes
  - Obstruction to LV inflow (limited ability to increase CO in response to metabolic demands)
  - Cardiomyopathy, pulmonary HTN / edema, RV failure, hepatic dysfunction
  - AFib (CO depends on atrial kick)
  - Risk of systemic embolization
- Medications
  - Anticoagulation, diuretics
- Co-existing disease
  - Thromboembolic events (CVA, systemic)
  - Rheumatic fever
- Associated valvular disease (TR, AI, AS)

## ANESTHETIC GOALS:

- Preload – maintain; avoid hypovolemia as flow across stenotic MV requires higher pressure gradient; avoid hypervolemia which may precipitate CHF
- PVR – avoid ↑PVR; avoid hypercapnia, hypoxia, pain, high airway pressures, high PEEP, pulm edema
- Rate – low-normal to allow enough time for diastolic filling of LV; avoid tachycardia
- Rhythm – sinus; atrial contribution to stroke volume may be elevated in early MS
- Contractility – maintain; LV chronically underloaded
- Afterload (SVR) – avoid hypotension; reflex tachycardia not tolerated

## HISTORY

- MV area and cause of valvular dysfunction
- CHF (fatigue, dyspnea, orthopnea, PND, hemoptysis)
  - Pulmonary edema precipitated by pain, AFib, sepsis, pregnancy
- Hoarseness (L RLN compressed by enlarged LA and PA)
- Angina
- Pulmonary HTN (dyspnea, fatigue, chest pain, hemoptysis)
- AFib (palpitations, syncope)
  - Anticoagulation
  - Thromboembolic complications (neuro deficits, TIA)

## PHYSICAL

- **CVS**
  - MS: low pulse pressure, loud S1 (high closing pressure across MV), S4, opening snap, diastolic rumble murmur (accentuated by hand grip)
  - Pulmonary HTN: large a wave, L parasternal lift, loud P2
- **RESP**
  - Tachypnea, crackles and wheezes
- **GI**
  - Hepatomegaly
- **RENAL**
  - Pedal edema
- **CNS**
  - Focal neurologic deficits

## INVESTIGATIONS

- **Labs**
  - CBC, lytes, Cr, urea
  - INR, PTT if anticoagulation
  - LFTs
- **Imaging**
  - EKG: LAE (p mitrale), AFib, RVH
  - CXR: LAE, pulmonary edema
  - Echo: thickened MV, anterior movement of posterior leaflet during systole, and decreased mid-diastolic mitral closure and presence of a thrombus

Degree of MS	MV area (cm <sup>2</sup> )	Mean TVPG (mmHg)	Pressure half-time (ms)	Symptoms
Normal	4.0 – 6.0	< 6	<100	None
Mild	1.5 – 2.5	≤ 6	≤100	With exertion
Moderate	1 – 1.5	6 – 10	100 – 220	At rest
Severe	< 1.0	> 10	> 220	At rest

Note: When MVA is <1cm<sup>2</sup>, mean LAP of 25mmHg required to maintain adequate LV filling; PHT develops when LAP chronically >25mmHg

- **Special**
  - Cardiac catheterization: to assess coronaries and R-sided pressures
  - Head CT

## OPTIMIZATION

- **Tx symptoms**
  - Cardiology consult
    - ?MVR prior to elective non-cardiac surgery if progressive Sx or pulmonary HTN
      - Percutaneous valvotomy, valve repair, valve replacement
  - Medical Tx should not involve vasodilators or inotropes
  - CHF – diuretics (watch for hypokalemia, hypovolemia)
  - Afib – rate control, anticoagulation, cardioversion
- **Manage medications**
  - Anticoagulation – hold warfarin 5d preop, heparin bridge

## ANESTHETIC OPTIONS

- **Regional**
  - Peripheral nerve blocks
  - Neuraxial
    - Avoid hypotension and reflex tachycardia
    - Mild-mod MS may tolerate spinal or slowly titrated epidural; caution if severe MS
- **General anesthesia**

## ANESTHETIC SETUP

- **Drugs**
  - Vasopressors – phenylephrine preferred over ephedrine
  - Drugs for rate control – B-blockers
  - Pulmonary vasodilators – milrinone, dobutamine, inhaled NO
- **Equipment**
  - Standard CAS, 5 lead EKG, Bair Hugger
  - Consider artline, central line, PAC, TEE
    - PAC: PCWP prominent A wave (unless AFib), risk PA rupture with wedging in pulmonary HTN
    - TEE: MS preload dependent however overload leads to pulmonary edema
  - Crash cart for cardioversion of intraop AFib

## MANAGEMENT OF ANESTHESIA

- **Induction**
  - Avoid tachycardia (atropine, ketamine, pancuronium, meperidine)
  - Avoid drugs which cause histamine release/hypotension (morphine, atracurium)
  - Ensure adequate depth of anesthesia prior to airway manipulation and surgical incision (tachycardia)
  - Avoid precipitants of pulmonary HTN (hypoxia, hypercarbia, N2O)
- **Maintenance**
  - Judicious fluids
    - AFib, excess fluids, and Trendelenberg position can precipitate CHF
  - Avoid tachycardia
    - Adequate depth of anesthesia
    - Hypotension leads to reflex tachycardia
  - Avoid ↑PVR (avoid hypoxemia, hypercarbia, hypothermia, high airway pressures, high PEEP)
- **Emergence**
  - Avoid tachycardia
    - Administer anticholinergic component of reversal agent slowly
    - Ensure adequate analgesia

## DISPOSITION & MONITORING

- **Analgesia**
  - Ensure adequate analgesia, avoid meperidine (tachycardia) and morphine (histamine)
  - Neuraxial opioids useful in selected patients
- **Oxygenation**
  - Avoid hypoxemia and hypercapnia
- **Positioning**
- **Monitoring**
  - Continue CV monitoring due to ongoing risk of pulmonary edema and R heart failure

## COMPLICATIONS

- Limited ability to increase CO
  - Acute pulmonary edema precipitated by ↑HR, pregnancy, anxiety, fluid overload, exercise, and postoperative mobilization of sequestered (third space) interstitial and extracellular fluid
- Thromboembolism

## PREGNANCY

- Surgical correction of significant MS recommended before pregnancy
- MS not well tolerated in pregnancy due to ↑blood volume and ↑HR
  - B-blockers useful to prevent tachycardia during pregnancy
  - May require surgical intervention during pregnancy (T2 ideal timing)
    - Percutaneous valvuloplasty lower risk to fetus than open commissurotomy, similar maternal efficacy

- Acute AFib requires aggressive Tx
  - Rate control → cardioversion
  - LLD position and diuretic for associated acute pulmonary edema
  - Anticoagulation with heparin + ASA (warfarin teratogenic)
- Obstetric management
  - Avoid valsalva in 2<sup>nd</sup> stage of labour as sudden ↑ venous return not tolerated
  - Uterine contractions ↑ central blood volume → may precipitate CHF
  - Sudden ↑ preload after delivery → risk of severe pulmonary edema
    - Continue epidural into postpartum period to reduce preload
    - Continue hemodynamic monitoring 24hrs post-partum
- Anesthesia management
  - Invasive hemodynamic monitoring for labour and c-section with symptomatic MS
  - Epidural preferred for labour and c-section
    - Judicious fluids (PCWP 14), careful titration, small bolus phenylephrine
  - For GA avoid drugs which cause tachycardia (atropine, ketamine, pancuronium, meperidine)
    - B-blocker and opioid during induction
  - Caution with oxytocin, PGF2α and methylergonovine, as all ↑PVR

## PATHOPHYSIOLOGY

- **Etiology**
  - Rheumatic heart disease (most common)
  - Structural – congenital, LA myxoma, thrombosis, calcification, cor triatriatum
  - Connective tissue disease – RA, SLE
  - Carcinoid disease
- **Pathophysiologic changes**
  - ↓LV filling → ↓SV → ↓C.O.
    - Reflex SNS activation
      - Vasoconstriction prevents hypotension
      - Tachycardia worsens LV filling/hypotension → precipitates pulm edema
      - ↑HR → ↑TVPG (*Bernoulli equation  $P=4v^2$*  explains why ↑flow across valve ↑s pressure)
    - Arrhythmias further ↓LV filling → pulmonary edema
  - ↑LA volume → ↑LAP (maintains LV filling) → LA enlargement
    - Afib → thromboembolism (embolic stroke risk 7-15%/yr)
  - ↑LAP transmitted to pulmonary vasculature → ↑pulmonary venous pressures (PVP) → ↑PAP
    - ↑PVP → pulmonary edema + hemoptysis
      - Excess fluids, Trendelenberg, and uterine contractions ↑ central blood volume → further ↑LAP → pulmonary edema
    - ↑PVP → RV strain
      - Eventual RV dilation → RV dysfunction/failure
- LV contractility N in isolated M

## REFERENCES

- Miller Chpt 60, Barash Chpt 28 and 41, Co-Existing Chpt 2, Chestnut Chpt 41

## Extra notes from previous versions:

- Chronic:
  - Stage 1: mild mitral stenosis-asymptomatic with physiologic compensation
  - Stage 2: moderate MS-symptomatic impairment
    - MV area: 1-1.5 cm<sup>2</sup>
  - Stage 3: critical mitral stenosis
    - MV area: < 1 cm<sup>2</sup>, MV area 0.3-0.4 cm<sup>2</sup> is the smallest area compatible with life
    - Severe congestive failure
    - pHTN eventually leads to RV dilation
    - Dilated RV can cause a leftward shift of the intraventricular septum impairing LV ejection

