Mitral Regurgitation and MVP

Mitral regurgitation manifests with LA /LV overload and eventually dilatation due to a faulty MV and lack of forward flow; it can be complicated by LV / RV failure & dysrhythmias (esp. atrial fibrillation); goals for management are to promote forward flow by afterload reduction and maintenance of rate, rhythm, contractility & preload

ANESTHETIC CONSIDERATIONS:

- Hemodynamic changes associated with mitral regurgitation
  - Volume overload: dynamic, varies with rate, valve area & pressure gradient across valve
  - Hypertrophied ventricle
    - Hidden systolic dysfunction
    - Frequent arrhythmias from LA dilation
    - Back pressure into pulmonary and R heart
    - Less angina than stenotic lesions
  - Underlying physiologic reserve and severity of MR
  - Acute MR may require emergent surgical / anesthetic management
- Associated co-morbidities including:
  - Other valvular pathology (MS, AR), IHD, endocarditis, SLE, etc
- Increased risk of perioperative cardiovascular complications (CHF, MI)

ANESTHETIC GOALS:

1. General – improve forward LV stroke volume and reduce the regurgitant fraction
2. Preload – high-normal; hypovolemia may worsen MR, excess volume further distends LV which worsens regurgitation and CHF
3. PVR – avoid ↑PVR; avoid hypercapnia, hypoxia, pain, high airway pressures, high PEEP
4. Rate – high-normal (80-100); bradycardia increases duration of systole (regurgitation) and diastole (excessive LV filling and dilation)
5. Rhythm – sinus
6. Contractility – maintain; EF overestimates LV function
7. Afterload (SVR) – low SVR to reduce regurgitation and maximize forward C.O.; avoid ↑SVR

HISTORY

- CHF (Sx of LHF, RHF)
- Ischemic heart disease (angina, previous MI)
- AFib (palpitations)
- Decreased renal perfusion (oliguria), diuretic induces electrolyte abnormalities (palpitations)
- Previous investigations for any of the above

PHYSICAL

- **CVS**
  - Volume status, signs of LV/RV failure
  - Holosystolic apical murmur radiating to axilla (mid-systolic click with MVP)
  - AFib
- **RESP**
  - Pulmonary edema

INVESTIGATIONS

- **Labs**
  - ABG – possible hypoxemia in chronic MR (increased PAP, edema)
  - CBC – possible hemolytic anemia, platelet consumption chronically
  - Lytes/Cr – hypokalemia with diuretics, renal function with ACE inhibitors
  - LFTs, PTT, INR – congestive hepatopathy, anticoagulation
- **Imaging**
  - ECG – LAE (biphasic P), LVH, AFib
  - CXR – cardiomegaly, pulmonary edema
  - ECHO – LAE, LA thrombus, LVH, MR severity (LVEF is unreliable)

Severity	Vena contracta (mm)	Pulmonary blood flow	EROA (cm2)	Regurgitant fraction (%)
Mild	< 3	Normal	< 0.2	20 – 30
Moderate	3 – 7	S blunted (S/D < 1)	0.2 – 0.4	30 – 50
Severe	>7	S reversed (S < 0)	≥ 0.4	> 50

Vena contracta = size of narrowest part of MR jet, EROA = effective regurgitant orifice area
Note: MR jet velocities are low in acute MR as MR occurs in a noncompliant chamber

- PA catheter
  - V wave in PCWP with wide pulse pressure
- Cardiac catheterization
  - Useful with concomitant ischemic heart disease

OPTIMIZATION

- Continue preop meds (esp B-blockers; consider holding ACE-I, diuretics)
- Acute MR – vasodilators, emergency MVR
- Chronic MR
  - Asymptomatic – no Tx

- Symptomatic
  - Cardiology consult
  - Consider need for biventricular pacing
  - Surgical intervention indicated for symptomatic MS even if EF normal
    - EF < 30% will not improve after MV surgery
    - Repair preserves LV geometry and is preferable to replacement
      - MV replacement can result in SAM and postop decline in LVEF
- Patients with previous MVR require careful management of perioperative anticoagulation – hold warfarin, bridge with LMWH or unfractionated heparin

ANESTHETIC OPTIONS

- **Regional anesthesia**
  - Peripheral nerve block
  - Neuraxial techniques
    - Decrease in SVR may be beneficial
- **General anesthesia**
  - Avoid ketamine (↑SVR)

ANESTHETIC SETUP

- **Drugs**
  - Vasodilators (nitroprusside, CCB), vasopressors (ephedrine preferred over norepinephrine), inotropes (milrinone, dobutamine, dopamine), atropine for bradycardia
- **Equipment**
  - CAS monitors, 5 lead EKG
  - Invasive monitors for severe or symptomatic MR
    - Consider pre-induction artline, central line
    - Consider TEE, PA catheter
      - Acute MR – large V wave, PCWP correlates well with LAP and LVEDP
      - Chronic MR – PCWP has poor correlation with LVEDV due to ↑LA compliance
  - Intraaortic balloon pump in setting of cardiogenic shock

MANAGEMENT OF ANESTHESIA

- **Induction**
  - Remember anesthestic goals (full, fast, vasodilated)
    - Avoid sudden decreases in heart rate (sux, high-dose opioids)
      - Consider preinduction anticholinergic
      - Consider ketamine to increase HR although watch SVR
    - Avoid sudden increases or decreases in SVR
      - Adequate depth of anesthesia for airway manipulation and surgical incision
    - Minimize drug induced myocardial depression
- **Maintenance**
  - Volatile agents help to achieve hemodynamic goals (↑HR, ↓SVR)
    - Reduced contractility may not be tolerated if severe myocardial dysfunction
  - Opioids have minimal effect on myocardial contractility, bradycardia poorly tolerated
  - Ventilation to avoid hypoxia, hypercarbia, excessive PEEP, ↑PVR (worsen RV failure)
  - Ensure adequate intravascular volume, avoid overload
- **Emergence**
  - Avoid HTN (→ MR and CHF)
    - Adequate analgesia
  - Risk of hypoventilation/hypoxemia with extubation (→ RV dysfunction)

DISPOSITION & MONITORING

- Analgesia adequate to avoid HTN
- Monitor symptomatic patients x 24h postop (volume status, ventilation, hemodynamics)

COMPLICATIONS

- **MVP**: similar to MR except too much LV emptying worsens MVP, leading to acute MR (i.e. increased SNS activity, large decrease in SVR, upright position, hypovolemia)
- **TR**: RV failure or decreased venous return (PPV or venodilation) can cause LV underload leading to intracardiac shunt and decreased CO
  - Avoid precipitants of pulmonary HTN, will reduce LV stroke volume
- **Systolic Anterior (leaflet) Motion (SAM)**: may occur in MR
  - Tx is increased SVR, volume expansion and decreased contractility, phenylephrine, fluid and B-blocker
- **Ischemia**: CAD vs. hypotension
- **Arrhythmia**: cardioversion vs. IV drugs
- **Hypotension**: preload vs. afterload vs. contractility
- **Air embolism**: keep lines free of air bubbles
- **Pulmonary edema and CHF**: precipitated by increased LV afterload (HTN) rapidly worsens MR
- Depressed LVEF, severe pulmonary HTN and RV dysfunction may be best predictors of **high perioperative risk**

PREGNANCY

- MR well tolerated in pregnancy (↑blood volume and ↓SVR promote forward flow across regurgitant valve)
- Symptomatic MR

- Consider vasodilator and diuretic
- MVR during pregnancy associated with ↑risk of fetal loss

- Anesthesia management
  - Hemodynamic goals as detailed above
  - Avoid aortocaval compression
  - Invasive monitors if severe MR (artline, CVP +/- PAC)
  - Epidural preferred for labour and c-section
    - Minimizes ↑ in SVR from pain
    - May SVR, promoting forward flow
    - Caution as may also venous return – judicious fluids, L uterine displacement
  - For GA – ketamine and pancuronium consistent with hemodynamic goals

PATHOPHYSIOLOGY

- **Classification**
  - **Primary (structural)**
    - Myxomatous degeneration (MVP)
    - Rheumatic heart disease (restricted leaflet motion)
    - Congenital, AVSD
    - LA myxoma
    - Endocarditis (leaflet perforation)
    - Ischemic heart disease (papillary muscle/chordal rupture)
    - Infiltrative systemic disease
    - Trauma
  - **Secondary (functional)**
    - Ischemic heart disease (annular dilation)
    - Eccentric LVH (annular dilation)
    - Dilated cardiomyopathy (annular dilation)
    - Connective tissue disease – RA, AS, SLE
    - Carcinoid syndrome
- **Severity**
  - Magnitude of regurgitant volume depends on
    - Size of regurgitant orifice (LV size)
    - Pressure gradient b/w LA and LV vs LV and aorta
    - Duration of regurgitant cycle (HR)
    - Compliance of receiving chamber

Modified Carpentier Classification of Mitral Regurgitation

	Leaflet Motion	Description
Ia	Normal	Annular dilatation
Ib		Leaflet perforation
IIa	Increased	Chordal elongation
IIb		Chordal rupture
IIc		Papillary muscle infarction or scarring
IId		Papillary muscle rupture
IIIa	Restricted	Commissural or chordal fusion and shortening
IIIb		Leaflet tethering by dyskinetic or aneurysmal LV segments
IV	Variable	Dynamic papillary muscle dysfunction

- **Pathophysiologic changes**
  - Basic problem is volume overload of both LA and LV
  - **Chronic MR** – LA and LV compensate for increase volume by gradually dilating, so patients remain asymptomatic until late in disease (when LVF, and biventricular HF can happen)
  - **Acute MR** – LA and LV can't compensate, and rapid pressure overload ensues, possibly leading to LV and RV failure, and cardiogenic shock
- Chronic MR commonly associated with mitral stenosis (progressive volume AND pressure overload → disease evolves faster)
- AFib occurs in roughly ⅓rd of patients with chronic MR, but of little hemodynamic consequence until LV compliance is decreased (late)
- Treatment of chronic MR consists of:
  - Afterload reduction (ACE inhibitors, diuretics)
  - Inotropy (digoxin)
  - Surgery (prior to symptoms: i.e. prior to LV dysfunction)
- Treatment of acute MR consists of
  - Afterload reduction (SNP, milrinone)
  - Inotropy (milrinone, dobutamine)
  - IABP, and possible emergent repair / replacement of mitral valve

- **Mitral Valve Prolapse**
  - Etiology
    - Inherited CT disorder
    - Myxomatous degeneration (replacement of collagen / elastin by mucopolysaccharides)
    - Associated with: Marfan's, Ehlers-Danlos, pseudoxanthoma elasticum, scoliosis, pectus excavatum, hyperadrenergic state, anxiety d/o
  - Mechanism
    - Structural weakness of valve apparatus
    - Displacement or prolapse of MV into LA during ventricular systole
    - Progressive annular dilatation, cordae elongation and stretching of valve leaflets
    - Development of MR
    - ↑ risk of endocarditis and embolic stroke due to surface abnormalities
  - Manifestations: dysrhythmias, atypical chest pain, SOBOE, MVR, TIAs, CVA
  - Complications: infectious endocarditis, embolic stroke, MVR (acute or chronic), CHF, sudden cardiac death
  - Assessment
    - Atypical chest pain, SOBOE, palpitations, syncope, neurological deficits
    - Mid and late apical non-ejection systolic click, focal neurological signs
  - Investigations: ECG (dysrhythmias, +/- nonspecific ST-T wave changes), ECHO (MVP, MVR)
  - **Goals**
    - **Rate - low normal**
    - **Rhythm - sinus if possible**
    - **Preload - high normal**
    - **Afterload - maintain**
    - **Contractility - maintain (or slightly decrease)**
    - MVP worsened by any conditions that decrease ventricular size (i.e. promote emptying)
  - Beneficial: Fluid, phenylephrine, β blockers
  - Avoid
    - MVP and MR worsened by conditions that decrease LV size
      - ↓ preload, ↓ afterload, ↑ HR, ↑ contractility
    - Anxiety, HTN
    - Atropine, ketamine, ephedrine, dehydration
    - Histamine-releasing drugs, NMB which cause tachycardia (pancuronium)
  - Management: Asymptomatic followed every few years with ECHO
  - Anesthetic implications
    - Preoperatively - antibiotic prophylaxis if MVR; anxiolysis preoperatively (midazolam or lorazepam)
    - Monitors - routine (unless significant MR)
    - Options
      - Local/sedation
      - Regional
        - Peripheral nerve block acceptable
        - Cautious with epidural & spinals b/c can cause abrupt ↓ preload and SVR
      - GA – avoid agents that cause tachycardia, drop preload or afterload significantly

REFERENCES

- Miller Chpt 60, Barash Chpt 28 and 41, Anesthesia and Co-exisiting Disease Chpt 2, Chestnut Chpt 41