Aortic Insufficiency

Regurgitation of blood flow across the aortic valve resulting from failure of valve leaflet coaptation caused by disease of the aortic leaflets or the aortic root.

ANESTHETIC CONSIDERATIONS:

1. Acute vs chronic
    - Acute – rapid decompensation to cardiogenic shock and CHF
    - Chronic – hemodynamic compensation
2. Cardiac pathophysiologic changes
    - Decreased effective stroke volume
    - Pressure and volume overload of the LV
        - Altered myocardial oxygen supply / demand (subendocardial ischemia)
    - Systolic dysfunction (impaired contractility) occurs late in chronic disease
3. Co-existing disease associated with cause
    - Leaflets: endocarditis, rheumatic fever, bicuspid valve, anorexigenic drugs
    - Root: idiopathic, hypertension ectasia, dissection, syphilis, Marfans syndrome, Ehlers-Danlos, RA, ankylosing spondylitis, psoriatic arthritis
4. Increased risk of perioperative CV complications (CHF, MI)

ANESTHETIC GOALS:

1. General – avoid increasing LV wall stress, maintain forward LV stroke volume
2. Preload – high-normal to maintain filling of dilated LV
3. PVR – maintain
4. Rate – high-normal (80 – 90) to shorten diastolic time available for regurgitation*; avoid bradycardia (distends LV)
5. Rhythm – sinus
6. Contractility – maintain; myocardial depression can precipitate LV volume overload and decompensation
7. Afterload (SVR) – low-normal to reduce pressure gradient for regurgitation; avoid hypertension

* Results in a) ↓LV volume and wall tension b) ↑DBP and coronary perfusion

HISTORY

- Usually asymptomatic until LV dysfunction occurs
    - CHF (exercise intolerance, orthopnea, PND, edema)
    - Angina in absence of CAD
    - Palpitations, diaphoresis
    - Splanchnic ischemia (abdominal pain)
- Ischemic heart disease
- Symptoms of coexisting disease (RA, ankylosing spondilitis)
- Previous investigations for any of the above

PHYSICAL

- **CVS**
    - Volume status, signs of CHF (JVD, edema, S3, rales)
    - Hyperdynamic circulation
        - Wide pulse pressure (low diastolic BP), rapid upstroke and collapse of carotid pulse (waterhammer pulse), bounding pulses
    - Diastolic thrill along LSB
    - AI murmur – high-pitched blowing diastolic murmur along LSB in 3rd ICS
    - Austin flint murmur – low-pitched diastolic rumble (diastolic MR if LV pressure > LAP toward end of diastole)
- **RESP**
    - CHF
- **GI**
    - Abdominal pain, distended abdomen of splanchnic ischemia

INVESTIGATIONS

- **Labs**
    - CBC – possible hemolytic anemia, platelet consumption chronically
    - Lytes – hypokalemia if on diuretics
    - Cr – renal function if on ACE inhibitors
- **Imaging**
    - ECG – LVH, possible strain
    - CXR – cardiomegaly, pulmonary edema if late,chronic or acute
    - ECHO – LVEF is important to gage reserve, predict morbidity; degree of AR (mild, moderate, severe)

Severity	AI jet/LVOT diameter (%)	Vena contracta (mm)	PHT (ms)	Aortic diastolic flow reversal
Trace	< 25	< 3	-	None
Mild	25 – 45	-	> 500	None
Moderate	46 – 64	-	200 – 500	None
Severe	> 65	> 6	< 200	Holodiastolic

PHT = pressure half-time

- Cardiac catheterization and angiogram

OPTIMIZATION

- Is cardiogenic shock present?
    - IABP contraindicated prior to AVR
- **Cardiology consult**
    - Medical optimization; NSR, no CHF, HTN well-controlled
- **Acute AI**
    - Mild symptoms
        - Vasodilators (nitroprusside, milrinone) to reduce systolic HT and wall stress
        - Inotropes (dobutamine, milrinone) to improve LV function
    - Severe CHF requires emergency AVR
- **Chronic AI**
    - Asymptomatic – arterial vasodilators (nifedipine, amlodipine, hydralazine)
    - Symptomatic – AVR
    - Surgery recommended before LVEF < 55% and LVESV > 55 mL

ANESTHETIC OPTIONS

- **Regional anesthesia**
    - Peripheral nerve block
    - Neuraxial techniques – decreased SVR with sympathectomy may be beneficial
- **General anesthesia**

ANESTHETIC SETUP

- **Drugs**
    - Atropine for Tx of bradycardia or junctional rhythm
    - Vasodilators, inotropes, vasopressors pre-mixed
- **Monitors**
    - Standard CAS with 5-lead EKG
    - Invasive monitors (artline, CVL +/- PAC, TEE) in severe AI
        - To detect myocardial depression, facilitate volume replacement, assess response to vasodilators
        - Consider pre-induction artline in symptomatic patients
        - PCWP may underestimate LVEDP due to premature MV closure
        - PCWP may overestimate LVEDP in combined AR-MR

MANAGEMENT OF ANESTHESIA

- **Induction**
    - If emergency repair – considerations of full stomach
    - Avoid drugs which reduce HR or increase SVR
        - Ketamine, propofol, opioid, benzodiazepine
    - Adequate depth of anesthesia prior to airway instrumentation or surgical incision
- **Maintenance**
    - Volatile agents – properties consistent with hemodynamic goals
    - Opioid-based anesthetic – preferred if significant LV dysfunction
    - Pancuronium – tachycardia beneficial
    - Ventilation to normal oxygenation and CO2 elimination, with adequate time for venous return
    - Maintain normovolemia (preload)
    - Prolonged Trendelenberg position and arrhythmias poorly tolerated
- **Emergence**
    - Maintain hemodynamic goals

DISPOSITION & MONITORING

- Good analgesia to avoid HTN
- Maintain preload to preserve filling of dilated LV
- Symptomatic AI – need for monitored setting x 24h to follow volume, ventilation, analgesia

COMPLICATIONS

- Require retrograde cardioplegia for myocardial protection during cardiac surgery
  Supraventricular arrhythmias
- Intraoperative LV failure
    - Tx with afterload reduction (vasodilator) and inotrope to improve contractility
    - IABP contraindicated

PREGNANCY

- AI without LV dysfunction well-tolerated in pregnancy (↑blood volume, ↑HR and ↓SVR in pregnancy maintain forward flow and reduce regurgitant fraction)
- Anesthesia management
    - Hemodynamic management to achieve goals above
    - Avoid aortocaval compression
    - Symptomatic AI or associated LV dysfunction benefit from hemodynamic monitoring during labour
    - Acute ↑SVR with pain of labour can precipitate LV volume overload
    - Epidural beneficial for labour and c-section
        - Provides afterload reduction
        - Early epidural prevents pain-associated ↑SVR

PATHOPHYSIOLOGY

- **Etiology**
  - Chronic AI
    - Bicuspid valve
    - Calcification
    - Rheumatic heart disease
    - Connective tissue dz – Marfan's syndrome, Ehlers-Danlos syndrome, RA, AS, psoriatic arthritis
  - Acute AI
    - Endocarditis
    - Aortic dissection
    - Trauma
- **Magnitude of regurgitant fraction depends on**
  - Time available for regurgitation (depends on HR)
  - Pressure gradient across AoV (depends on SVR)
- **Pathophysiologic changes**
  - Basic pathology is volume overload of LV
  - **Chronic AI –** LVEDV (preload) is compensated for by gradual dilatation of LV (and LVH). Eventually the LV will fail, and a precipitous rise in LVEDP will follow
    - LVH (and increased myocardial oxygen demand) along with the characteristic low diastolic BP of aortic regurgitation can lead to angina in the absence of CAD
  - **Acute AI –** LVEDV and LVEDP rise concominantly (no gradual compensation is allowed) leading to LV failure and cardiogenic shock
- **Postoperative AVR**
  - Preexisting impaired LV fxn + mechanical AoV generates mild transvalvular P gradient
    - May require inotropes to improve LV fxn
    - Must continue preload augmentation to maintain filling of dilated LV
- **Prognosis**
  - Asymptomatic – mortality 0.2% /yr
  - Symptomatic – mortality >10% /yr

REFERENCES

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