Aortic Stenosis

Progressive narrowing of the aortic valve leading to fixed LV output obstruction with limited ability to compensate for hypotension, with cardiac changes including LV hypertrophy and ultimately systolic dysfunction occurring in patients with significant co-morbid disease

ANESTHETIC CONSIDERATIONS:

- Cardiac physiologic changes
  - Fixed LVOT obstruction & limited ability to compensate for hypotension
  - Hypertrophied ventricle
    - Diastolic dysfunction (reduced ventricular compliance)
    - Altered myocardial O2 supply / demand
  - Systolic dysfunction (impaired contractility) occurs late
- Co-existing disease – elderly, CAD, coexisting valvulopathy
- Hemodynamic optimization
- Increased risk of perioperative CV complications (CHF, MI)

ANESTHETIC GOALS:

1. General – minimize pressure gradient across the aortic valve and maintain favorable myocardial O2 supply-demand balance
2. Preload – full; adequate LVEDV necessary to maintain SV
3. PVR – maintain
4. Rate – low-normal (60-70) to allow enough time for ejection of blood; avoid tachycardia which ↑s O2 demand, reduces time for C.O. in systole and LV filling and coronary perfusion in diastole; avoid bradycardia*
5. Rhythm – sinus; LV dependent on atrial kick to maintain LVEDV
6. Contractility – maintain; avoid negative inotropes
7. Afterload (SVR) – high-normal to maintain coronary perfusion pressure; avoid hypotension

*Bradycardia does not improve LVEDV in LVH, and ↓s C.O. in setting of fixed stroke volume with HR-dependent C.O.

HISTORY

- Aortic valve area and LV function (recent ECHO± current functional status); cardiac cath results
- Most patients asymptomatic until valve area <1.0 or mean gradient > 50
- Classic triad of AS:
  - **Angina**
    - May occur in absence of CAD due to ↑myocardial O2 demand and ↓myocardial O2 supply
    - Coexisting CAD in 50% of patients >50 y.o. with AS
  - **Syncope or pre-syncope**
    - May be exertional with fall of SVR with fixed CO
    - May also result from arrhythmias
  - **Dyspnea**
    - Due to diastolic dysfunction (early) and systolic dysfunction (late)

PHYSICAL

- **CVS**
  - General HF signs
    - Tachycardia, Cheyne-Stokes respirations, mental confusion, diaphoretic, cool extremities
    - LHF: tachypnea, rales in lower lung fields, wheezes, S3, pulsus alternans
    - RHF: elevated JVP, hepatosplenomegaly, peripheral edema, ascites, anasarca
  - Aortic stenosis:
    - Narrow pulse pressure, slow carotid upstroke (pulsus tardus) and low volume carotid pulse
    - Systolic crescendo-decrescendo murmur, maximal at 2nd R ICS, radiating to apex and neck
    - Soft S2
    - Stiff ventricle: S4 gallop
    - Artline waveform
      - Narrow pulse pressure (<50)
      - Delayed (slow) upstroke
      - Prominent anacrotic notch (occurs earlier in upstroke w/ ↑severity of AS)
      - ↓Size dicrotic notch w/ ↑severity of AS
  - Pulmonary HTN
    - Large jugular a wave, left parasternal lift, palpable left second intercostal space pulsations, loud P2, pulmonic ejection murmur
- **RESP**
  - CHF on pulmonary exam
- **CNS**
  - Neurological exam, carotid bruit (stroke, syncope)

INVESTIGATIONS

- **Labs**
  - **CBC, LFTs, Coags, BUN, Cr**
- **Imaging**
  - **EKG**
    - LVH +/- strain
    - LBBB
  - **CXR**

        - Calcification of aortic leaflets and aortic root in severe AS
        - Enlarged heart, post-stenotic aortic root dilatation
        - Evidence of CHF – pleural effusions, pulmonary congestions
    - **Echocardiography**
        - Severity of aortic stenosis (valve area, pressure gradient), LVH, LV function
        - Coexisting CAD

Severity	Aortic jet velocity (m/s)	Mean TVPG (mmHg)	Peak TVPG (mmHg)	AV area (cm2)	Symptoms
Normal	< 2	< 5		3 – 4	None
Mild	2 – 3	< 25	< 36	> 1.5	None
Moderate	3 – 4	25 – 50	> 50	1 – 1.5	With exertion
Severe	> 4	> 50	> 80	< 1	At rest
Critical				< 0.8	At rest

- Note: Associated findings may include concentric LVH, reduced EF, MR, LA dilation

- Exercise stress-test
    - Exercise-induced Sx may benefit from AVR
- Cardiac catheterization with angiography
    - Assess coronaries and AV gradient

OPTIMIZATION

- Asymptomatic
    - Ensure volume resuscitated and hemodynamics within goal range
- Symptomatic – Cardiology consult
    - Medical optimization
        - HTN – cautious vasodilators, cautious B-blockers (avoid if heart failure)
        - AFib – rate control, cardioversion
        - CHF – cautious diuretic + vasodilator
        - Tx co-existing CAD
    - Surgical management
        - Consider percutaneous balloon valvotomy or AVR +/- CABG

ANESTHETIC OPTIONS

- Any technique that avoids ↓SVR acceptable
- **Regional anesthesia**
    - Peripheral nerve block
    - Neuraxial techniques
        - ↑Risk hypotension with sympatholysis, extreme caution required
        - May be tolerated in mild-moderate AS; single-shot spinal relatively contraindicated in severe AS, although carefully titrated epidural may be considered
- **General anesthesia**
    - Preferable over neuraxial anesthesia (especially for severe AS); better control of hemodynamics

ANESTHETIC SETUP

- **Drugs**
    - Phenylephrine to maintain SVR
    - Atropine, glycopyrrolate, ephedrine for bradycardia
    - Esmolol for persistent tachycardia
    - Lidocaine/amiodarone for ventricular arrhythmias
    - Nitroglycerine (very low dose) for excessively elevated filling pressures
    - Nitroprusside (very low dose) for LV dysfunction
- **Monitors**
    - Standard CAS, 5 lead EKG for ischemia, artline, CVP; consider TEE, consider PA catheter
        - Consider artline and CVL prior to induction
        - PAC may induce arrhythmias, PCWP may overestimate LVEDV due to ↓LV compliance
    - Crash cart – apply external defibrillation pads for rapid cardioversion of AFib and ventricular arrhythmias

MANAGEMENT OF ANESTHESIA

- **Induction**
    - Avoid decrease in SVR, consider adding phenylephrine to induction drugs
    - Avoid hypotension and tachycardia
        - Judicious dosages of opioid, etomidate, benzodiazepine
        - Caution with propofol/thiopental (myocardial depression, hypotension), ketamine (tachycardia)
- **Maintenance**
    - Minimal inhalational anesthetic agent
        - Avoid excess myocardial depression and vasodilation; rapidly Tx hypotension
        - Volatile anesthetics associated with **junctional rhythm (loss of atrial kick →** ↓ SV, CO & BP)
    - Bradycardia can cause hypotension; thick ventricle of limited distensibility, and excessively prolonging diastole does not improve ventricular filling
    - Maintain intravascular fluid volume
- **Emergence**
    - Period of potential ischemia – smooth extubation with good pain control desirable
        - Pain relief helps prevent undesirable tachycardia

DISPOSITION & MONITORING

- Depends on severity of AS and procedure (ICU postop AVR)

COMPLICATIONS

- Prevent potential hemodynamic spiral of hypovolemia, hypotension & tachycardia by careful attention to fluid requirements, pain relief, resolution of any sympathetic block
- Development of supraventricular arrhythmias, including atrial fibrillation, deleterious; early identification and treatment can be crucial

PREGNANCY

- Asymptomatic/mild AS
  - Greater blood volume in pregnancy → tolerate pregnancy well
- Symptomatic/moderate-severe AS
  - Fixed SV → unable to achieve C.O. required in pregnancy
    - Inability to maintain coronary and cerebral perfusion with exertion
    - Risk of acute LV failure
  - AVR recommended prior to conception
    - Sx early in pregnancy may require termination of pregnancy or AVR
- Critical to maintain intravascular volume, venous return, and NSR; aortocaval compression, peripartum hemorrhage, and sympathectomy decrease C.O. and are poorly tolerated
- Obstetric management
  - Vaginal delivery preferred
- Anesthesia management
  - Hemodynamic management – see goals above
  - Avoid aortocaval compression
  - Arterial line for gradual titration of epidural or GA
    - Avoid using epinephrine in LA solutions
  - Central line for titration of fluids
    - High-normal intravascular volume to protect C.O. in case of peripartum hemorrhage

PATHOPHYSIOLOGY

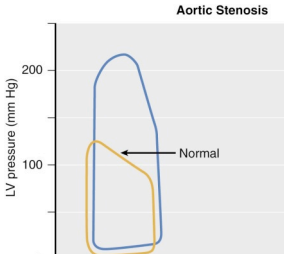
- **Classification**
  - Subvalvular, valvular (75%), supravalvular
- **Etiology**
  - Bicuspid AoV (1-2% of population, 5-15% become stenotic, usu < 60 y.o.)
  - Calcification
    - Risk factors as for atherosclerosis (age, male, smoking, HT, ↑lipids)**,** Paget disease, renal failure
      - Look for coexisting CAD
  - Rheumatic heart disease (rare, associated AI, MR, MS)
- **Severity**
  - Note: when CHF accompanies AS, transvalvular pressure gradient (TVPG) may be smaller (LV unable to generate large gradient)
- **Pathophysiologic changes**
  - Obstruction to ejection of blood into aorta → ↑intraventricular systolic pressure to maintain forward SV
  - ↑Intraventricular pressure → ↑afterload → compensatory concentric LVH
  - LVH results in:
    - ↑Myocardial O2 demand, ↓O2 supply
    - Ventricular arrhythmias
    - Diastolic dysfunction early
      - ↓LV compliance
      - ↑Impedance to early diastolic filling→ atrial kick essential to maintain LV filling and SV
    - Systolic dysfunction late (decompensated)
      - Further ↑s in afterload → LV dilation → ↓contractility → ↓C.O. → cardiogenic shock
  - Angina due to:
    - ↑Myocardial O2 demand – LVH, ↑afterload
    - ↓Myocardial O2 supply – ↑LV pressure compresses subendocardial coronaries
  - Syncope due to:
    - Arrhythmias
    - Exercise-induced vasodilation with inability to ↑C.O.
  - Dyspnea
    - Diastolic and systolic dysfunction
- **Prognosis**
  - ↑Risk perioperative cardiac complications independent of CAD
  - Onset of Sx correlates with life expectancy in absence of valve surgery
    - Angina 5 year survival, syncope 3 year survival, dyspnea 2 year survival
  - CPR unlikely effective in AS (chest compressions cannot generate adequate TVPG across stenotic valve)

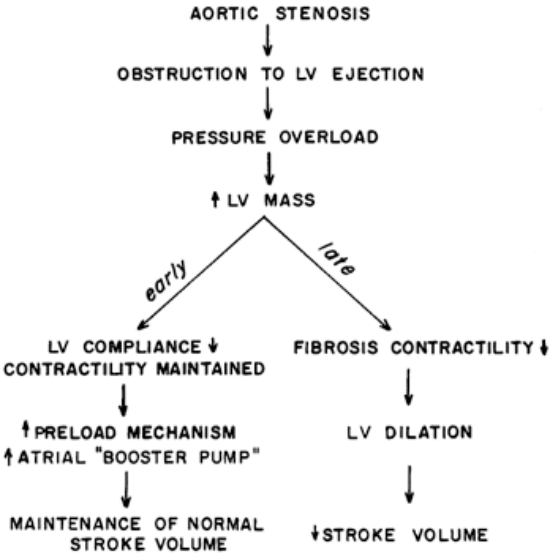
REFERENCES

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

Extra information from previous versions of notes:

- Do you know the **aortic valve area and LV function**?
  - Need for recent / current ECHO
- Have you ruled out co-existing CAD?
- Major valvulopathy is a major clinical risk factor
  - Does this patient require repair / replacement of their aortic valve?
    - Cardiology consult
    - Possible valvuloplasty, percutaneous AVR, AVR
- Is this patient medically optimized?
  - No evidence of CHF
  - NSR





- Three stages:
  - **Stage 1**: Mild AS-asymptomatic with physiologic compensation
    - Stroke volume maintained by compensatory concentric LVH
    - ↑ Stenosis = ↑ LV/aortic pressure gradient
    - Compensatory concentric LV hypertrophy
    - ↑ LVEDP = reduced compliance or ↓ LV diastolic function
  - **Stage 2**: Moderate AS-symptomatic impairment
    - AV area 0.7-0.9 cm^2
    - Concentric LVH → dilation
    - ↑ LVEDV / LVEDP = ↑ myocardial O_2 demand and ↓ O_2 supply
    - 40% ventricular filling dependent on atrial contraction
  - **Stage 3**: Critical AS-terminal failure
    - AV area: 0.5-0.7cm^2, AV valve index: 0.5 cm^2/m^2
    - ↑ LAP >25 mm Hg → CHF, sudden death or eventual RV failure