AORTIC STENOSIS

TYPES OF AORTIC STENOSIS

1. Valvular AS
   - Congenital: bicuspid or unicuspid, young patients
     - Bicuspid valve
       - 1-2% population
       - 20% with CoAo (80% with CoAo have bicuspid valve)
       - Fusion of R and L leaflets
       - Root Ao dilatation
   - Rheumatic (co-exist MV ds)
   - Degenerative (1st cause) – atherosclerosis associated to AS
   - Aortic sclerosis – systolic murmur in elderly
     - Valve thickening without obstruction of LVOT
   - Radiation (mediastinal – breast, lymphoma, esophagus)
     - > 30 Gy
     - 15-20 years after exposure
     - Aortic valve (mixed disease) > mitral > tricuspid

2. Subvalvular AS
   - Tunnel of muscular tissue or membrane
   - Shone complex (multiple left-side obstructions: supravalvular MS, parachute mitral valve, subvalvular AS, bicuspic Ao, CoAo)
   - Associated with patent ductus, VSD
   - Jet lesion: 2o aortic regurgitation

3. Supravalvular AS
   - William Syndrome: child facies, pulmonary stenosis, hypercalcemia
   - Familial dyslipidemia
   - CoAo thoracic and abdominal, renal artery stenosis

PATHOPHYSIOLOGY

- Normal AVA = 3-4 cm²
- Pressure drop at AVA of 1.5
- Systolic murmur with even mild forms
- HD significant AS if 60-75% narrowing (AVA 0.7-0.8)
- Compensated pure AS does not have LV dilatation and enlargement
- LVH (↑ mass and LVEDP)
- ↓0.12 cm²/year; ↑ 7 mmHg men gradient/year

CLINICAL FINDINGS

- Dyspnea (diastolic dysfunction)
- Angina ➔ survival of 5 years
- Syncope ➔ survival of 3 years
- Heart failure ➔ survival of 2 years
- GI bleeding: AV malformations (Heyde Syndrome) – destruction of von Willebrand factor by the valve (acquired vW defect) = 20%

PHYSICAL FINDINGS

BP
- SBP normal – in absence of HTN or AR
- Advanced AS, pulse pressure ↓
- Systemic HTN in elderly

Carotid
- Hallmark – slow rising and weak (parvus), delayed peak (tardus), small-volume
- Systolic thrill or shudder in upstroke
- Anacrotic notch
- Mask severity
  - ↑ CO and elastic vessels in children and adolescents
  - ↑ stiff vessels in elderly (arteriosclerosis)
- assoc AI
- Systemic HTN
- ↓ stroke volume of CHF
- Exaggerate severity
  - ↓ EF
  - hypovolemia
  - MS
- Peripheral - B-R delay, may not be useful

JVP
- JVP elevated if biventricular HF
- A more prominent (rare)

Precordial
- Sustained with no displacement (displace if LV dilated
  - HD NB, compensated AS
- May have palpable A wave (S4) ** - means elevated LVEDP = significant AS
- Apical carotid delay
- Systolic thrill – RICS 1 or 2

S1
- Normal
- May be quieter, never louder
- if loud = Aortic ejection sound or assoc MS

Aortic Ejection Click
- After S1
- Aortic ejection murmurs and clicks heard best in apex
- More calc leaflets, click quieter and then gone
- 40-80ms after S1
- loudest at apex; heard though apex
- Click DOES NOT vary with resp (as does PS)

S2
- A2 intensity
  - More severe AS – A2 quieter
  - Severe AS – A2 absent
- A2 splitting
  - S2 physiologic - Mild AS, or severe congenital AS
  - S2 single (delayed A2, and/or A2 silent) - Mod AS
  - S2 paradoxical - Severe AS (if normal LV and no LBBB)

S3
- CHF or signif LV dysfunction

S4
- Palpable – must be significant AS

Murmur
- Loudest at RICS 2, radiate to bilat carotids
- Length, time of peak = severity
- Louder = AR, quieter = LV dysfct, CHF
- Gallavardin phenomenon – highest’ frequency components radiate to apex and may even sound musical at that site suggesting a murmur of MR
- Characteristic quality of the loud murmur of mod to severe AS
  - Harsh, rasping, grunting and coarse murmur
  - Sounds like person clearing their throat
  - Put palm on diaphragm, scratch dorsum with finger
  - If louder radiation to right carotid – think supravalvular AS
- Louder post PVC
- louder = severe murmur in children
- ddx – aortic sclerosis, MR
MANEUVERS
VALSALVA - STRAIN (ddx HOCM) – murmur decreased
SQUATTING (ddx HOCM) - increase murmur – increase venous return
INSPIRATION (ddx PS) - slightly decreases
VASOPRESOR (ddx HOCM)
– increase murmur (stimulate contractility, raise BP, increase gradient)
AMYL NITRATE (ddx HOCM) – immed BP drop, flow does not increase till 20 sec later
- murmur does not begin to increase for 20 sec (b/c it is dependent on flow)

DIFFERENTIAL DIAGNOSTIC

- **Aortic sclerosis** – classic systolic ejection murmur, < grade 4/6, A2 preserved, carotid upstroke brisk, No LV enlargement on palpation, overall length and peak intensity of murmur falls in first half of systole

- **MR** – note length of murmur esp late systole, radiate to axilla (AS loud over clavicle), normal carotid, no change with beat to beat (AS louder with post-PVC or following a long R-R cycle)

ECG

- LVH (compensatory mechanism to maintain normal LV function)
- Complete AV block

X-RAY

- Valvular calcifications
- LV enlargement (if advanced disease)
- Ao dilatation

EXERCISE TESTING

- To confirm changes with exercise and confirm the absence of symptoms in asymptomatic patients with calcified aortic valve and aortic velocity 4.0 m/s or greater or mean pressure gradient 40 mm Hg or higher
- **SHOULD NOT** perform in symptomatic patients with AS aortic velocity 4.0 m/s or greater or mean pressure gradient is 40 mm Hg or higher

ECHOCARDIOGRAPHY

LVH, diastolic dysfunction

Class I

1- Diagnosis and assess AS severity (level of evidence B)
2- Assess LV wall thickness, size and function (B)
3- Re-evaluation of patients with AS and change of symptoms and signs (B)
4- Changes in hemodynamic severity and LV function of AS during pregnancy (B)
5- TTE to re-evaluate asymptomatic patients: (B)
  a. 1 year: severe AS
  b. 1-2 years: moderate AS
  c. 3-5 years: mild AS

<table>
<thead>
<tr>
<th>Aortic sclerosis</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic jet velocity (m/s)</td>
<td>≤ 2.5</td>
<td>2.6-2.9</td>
<td>3.0-4.0</td>
</tr>
<tr>
<td>Mean gradient (mm Hg)</td>
<td>&lt; 20</td>
<td>20-40</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>AVA (cm²)</td>
<td>&gt; 1.5</td>
<td>1.0-1.5</td>
<td>&lt; 1.0</td>
</tr>
<tr>
<td>Indexed AVA (cm²/m²)</td>
<td>&gt; 0.85</td>
<td>0.60-0.85</td>
<td>&lt; 0.60</td>
</tr>
<tr>
<td>Velocity ratio</td>
<td>&gt; 0.50</td>
<td>0.25-0.50</td>
<td>&lt; 0.25</td>
</tr>
</tbody>
</table>


Severe AS

1- **True severe AS** (D1): high gradient and high velocity
2- **Low flow-low gradient (D2)**: low EF ≤ 40% – **5-10% AS**
3- Paradoxal low flow/low SV/small cavity (D3): normal EF – 10-25% AS
   a. Restrictive physiology (reduced size, compliance and filling LV, concentric remodeling/fibrosis), old women, HTA
   b. EOA < 1.0 cm², gradient < 40 mm Hg but SV ≤ 35 cc/m²

Dobutamine stress TTE
Used to distinguish true versus pseudosevere AS
   - 20 µg/kg/min increased gradually
   - No LV flow reserve = increase SV < 20% with dobutamine
   - Pseudosevere AS: increase EOA with little increase of gradient in response to increase flow
   - True severe AS: little or no increase EOA with increase in gradient with increase of flow

★ Suggested reference:
   • Pibarot & Dumesnil, J AM Coll Cardiol 2012;60:1845-53
   • Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. J Am Coll Cardiol 2014;63:e57-185. – Table 8

CATHETERIZATION

Pullback gradient and AVA is determined by Gorlin formula

\[
\text{Aortic Valve Area = Cardiac Output} \\
\text{SEP x HR x 44.3/Vx}
\]

Usually done when there is discrepancy between physical exam, symptoms and echocardiography measurements (gradient, AVA)

MANAGEMENT


• Medical treatment
• Surgical management – timing of intervention
  o Surgical risk higher in patients with EF < 35%
• Indications for aortic valve replacement
• Indications for balloon dilatation (lasts less than 6 months)
• Indications for TAVI

Valve replacement options
   - Mechanical valves
     o Anticoagulation (INR 2-3)
     o < 65 years-old, renal disease (calcium)
   - Biological valves
     o No anticoagulation
     o > 65 years-old, woman in child-bearing years
     o Porcine or bovine pericardial tissue – life expectancy 10-15 years, < 10 years in young patients
     o ASA 81 mg DIE
   - Homograft
     o Cadaveric valves
     o For patients with active endocarditis
     o No anticoagulation
     o Durability not better than a tissue valve
   - Ross procedure
     o Pulmonary valve transplanted in aortic position + homograft in pulmonary position
**TAVI**

**Partner B study:** non operable patients (risk ≥ 50%)

- TAVI versus medical treatment
- Femoral access only (SAPIENS)
- Mean age 83 y-o
- Mortality 5% with TAVI and 2.8% medical treatment (P=0.41) – after 1 year: 30.7% TAVI vs 50.7% medical group (P<0.0001)
- NNT = 5 – improvement in functional status, reduced hospitalization, cost benefit, follow-up at 2 years with further benefit


**Partner A study:** high-risk operable patients

- Operative mortality ≥ 15% (STS ≥ 10)
- TAVI non inferior to SAVR
- Femoral and apical access
- Mortality at 30 days with TAVI 3.4% vs 6.5% SAVR (P=0.07) and no difference at 1 year, but TAVI non inferior
- **TAVI:** quality of life markedly improved, lower major bleeding (P<0.001), shorter intensive care and hospitalization, rate PMP similar, major vascular complications (11% vs 3.2%), more major strokes at 30 days (3.8% vs 2.1% - P = 0.20) – no difference of strokes event at 2 year follow-up between TAVI and SAVR


**CCS RECOMMENDATION (2012)**


1- Transfemoral recommended if (Strong recommendation, high evidence)
   a. Risk of SAVR is prohibitive +
   b. A significant improvement in duration or quality of life is expected +
   c. Life expectancy with treatment expected >1-2 years

2- Patients not candidate for SAVR or TAVI using femoral arterial access may be considered for other alternative access procedures – transapical, transaxillary or transaortic (Conditional recommendation, Low evidence)

3- TAVI is reasonable alternative to SAVR for patients at high risk of mortality and major morbidity (high risk = risk of mortality ≥ 8% or major morbidity > 50% within 30 days of surgery – STS risk calculator)
   a. Duration and quality of life is likely to improve significantly with treatment
   b. Life expectancy with treatment expected >1-2 years
   c. Consensus among the heart team (Strong recommendation, moderate evidence)

4- SAVR is the treatment of choice for patients with severe symptomatic AS at intermediate or low surgical risk (Strong recommendation, moderate evidence)

5- TAVI may be offered to selected patients with severe symptomatic AS considered intermediate to low risk of mortality with consensus with Heart team that have significantly increased risk of morbidity of mortality due to frailty, very advanced age, patent bypass grafts, multivalve disease, etc. (Conditional recommendation, low evidence)

**VALVE-IN-VALVE IMPLANTS**

1- Surgical valve replacement is the treatment of choice for non-high risk patients with failure of biosprothetic surgical valve (Strong recommendation, low evidence)

2- Reasonable in patients with failed surgical biosprothetic valves in whom the Heart team consensus is:
   a. Risk of SAVR is prohibitive +
   b. Significant improvement in duration and quality of life is likely +
   c. Life expectancy with treatment expected >1-2 years
   d. Dimensions and characteristics of failed valve are understood and compatible with good transcatheter valve function (Conditional recommendation, low evidence)
3- Transcatheter valve implants is reasonable in carefully selected patients with Transcatheter valve failure (Conditional recommendation, low evidence)

TAVI EVALUATION
1- Screening for TAVI should include all of the following:
   a. Comprehensive assessment of medical history
   b. Complete physical exam with special attention to signs and severe AS, lung disease, peripheral artery disease; objective neurocognitive function and frailty; exercise testing and standardized walk test
   c. ECG, chest X-Ray, FSC, electrolytes, creatinine, liver function, BNP
   d. TTE: annulus diameter – low dose dobutamine may be helpful in patients with severely reduced LV function and low transaortic gradients
   e. TEE to assess annulus diameter is recommended in the absence of MSCT measurements
   f. Coronary angiography
   g. Aortography and ilio-femoral invasive angiography or MSCT angiography, preferably both
   h. Accurate measurements of aortic annulus size by TEE and/or MSCT or MRI is key to appropriate selection of prosthesis size
   (Strong recommendation, weak evidence)

ANATOMY REQUIREMENTS
• Annulus to ostial distance must be ≥ 12 mm
  o Low lying ostia will increase risk of coronary ostial occlusion by TAVI
• Imaging of the entire path from femoral access to aortic valve is important to assess aortic diameter, degree of calcification and tortuosity of arterial vasculature
  o Dilated ascendant Ao may be C-I to implantation of prosthesis relying on fixation in ascendant Ao but not those with annular fixation alone
  o Severe aortic tortuosity, aneurysm and protruding atheroma or thrombus are relative C-I to transarterial access
  o Porcelain Ao is not a particular concern but lumen must be sufficiently large
• Delivery system is 7 mm (external diameter) – artery with diameter > 6 mm is required (more if ASO, calcification of severe tortuosity)

POSTPROCEDURE MANAGEMENT
Complications: airway compromise, bleeding, heart failure, neurologic events, peripheral artery complications
• ICU first 24 hours
• AV block – prophylactic temporary pacing
  o Monitoring x 48 hours
• Low ASA for 1-3 months (no evidence)
• Patients with ACO – adjunctive antiplatelet agents is controversial = triple therapy should be avoided
• Endocarditis prophylaxis according to guidelines
• Follow up with Heart tem at 1 month and annually with cardiologist
• TTE before discharge, at 1 month and yearly

Content of this summary from these references: