

Aortic Stenosis

Calvin Madrigal, PGY 5

Objectives

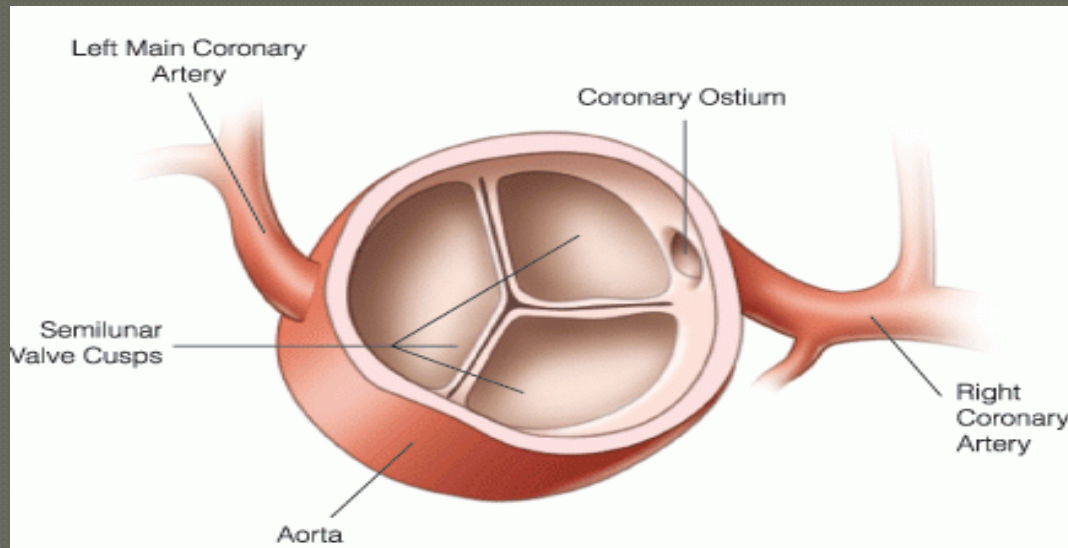
- ① 1. Prevalence of aortic stenosis in patients over the age of 65 in developed countries.
- ② 2. The prognosis between asymptomatic versus symptomatic disease.
- ③ 3. Describe the three symptoms of aortic stenosis and the pathophysiology associated with these symptoms.
- ④ 4. Describe the physical examination findings in patients with aortic stenosis and the reason for the absence of these findings.

Objectives

- 5. Define severe aortic stenosis and know the management for :
 - Severe symptomatic patient who is a candidate for Surgery.
 - Severe symptomatic patient who is not a candidate for surgery.
 - Moderate aortic stenosis who needs a CABG.
 - Severe asymptomatic aortic stenosis
- 6. Differentiate true aortic stenosis from “pseudostenosis and why it is important.

Aortic Valve

- Semilunar valve (Pulmonic and Aortic)
- Tricuspid (Left/right coronary cusp and noncoronary cusp)
- Normal area: 3-4cm²
- Normal transvalvular Velocity: <2m/s



Aortic Stenosis Epidemiology

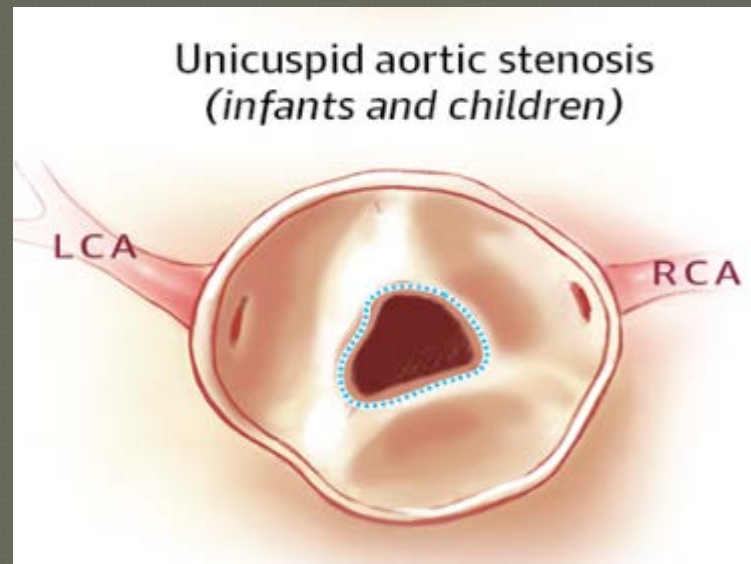
- The most common cause of left ventricular outflow obstruction in adults.
- Prevalence: Increases with age, 2-7% of all patients > 65 are reported to have AS.
- Valvular heart disease accounts for 10-20% of all cardiac surgical procedures in the US.
- Approximately 2/3 of all heart valve operations are for AS.
- Without intervention, mortality rates reach as high as 75% in 3 years once symptoms develop.

Aortic Stenosis Etiology

- Three primary causes :
 - I. Age associated calcific changes
 - II. Congenital aortic valve disease
 - a) Unicupsid
 - b) Bicuspid
 - III. Rheumatic valve disease

Aortic Stenosis Etiology

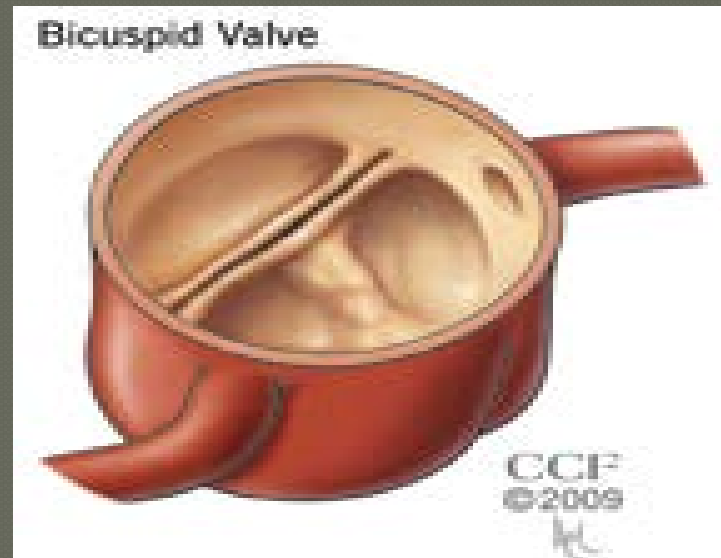
- Congenital aortic valve disease
 - Unicuspid: Typically produce severe obstruction in infancy and most frequent malformation found in fatal valvular AS in children <1 year.



Aortic Stenosis Etiology

○ Congenital aortic valve disease

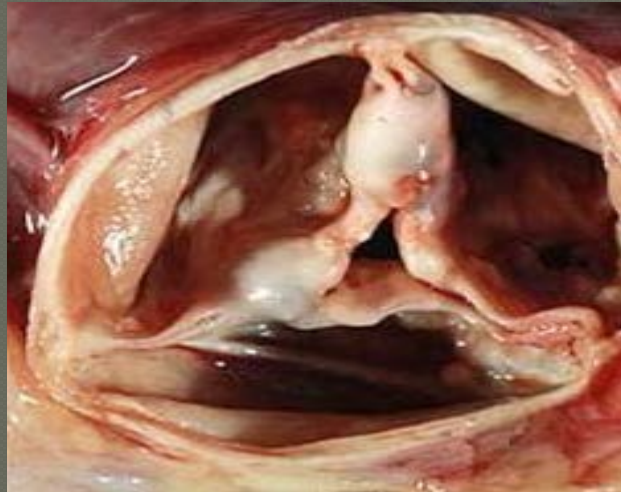
- Bicuspid: Present in 1-2% of the population with a greater prevalence for males. Commonly associated with coarctation of the aorta and aortic aneurysm. Earlier presentation of symptoms when compared to trileaflet valve.



Aortic Stenosis Etiology

○ Rheumatic valve disease

- Leads to adhesions and fusions of the commissures and cusps with subsequent stiffening.
- Orifice is reduced to a small round or triangular opening and often associated with concomitant aortic regurgitation.
- Remains a problem in developing countries



Aortic Stenosis Etiology

○ Calcific aortic valve disease (senile)

- Most common cause of AS in adults
- Once considered to represent normal mechanical stress on the valve.
- The evolving concept is that the disease is a mixture of genetic, proliferative and inflammatory changes resulting in calcification and subsequent immobilization of cusps.
- Linked to genetic polymorphisms, involving vitamin D receptors, interleukin-10 alleles and apolipoprotein E4 allele
- Risk factors include: Elevated LDL, lipoprotein(a), diabetes, smoking and hypertension.

Aortic Stenosis Etiology

○ Calcific aortic valve disease (senile)

TABLE 63-1 Strength of Associations in Observational and Epidemiologic Studies of Clinical Risk Factors and Calcific Aortic Valve Disease (CAVD)

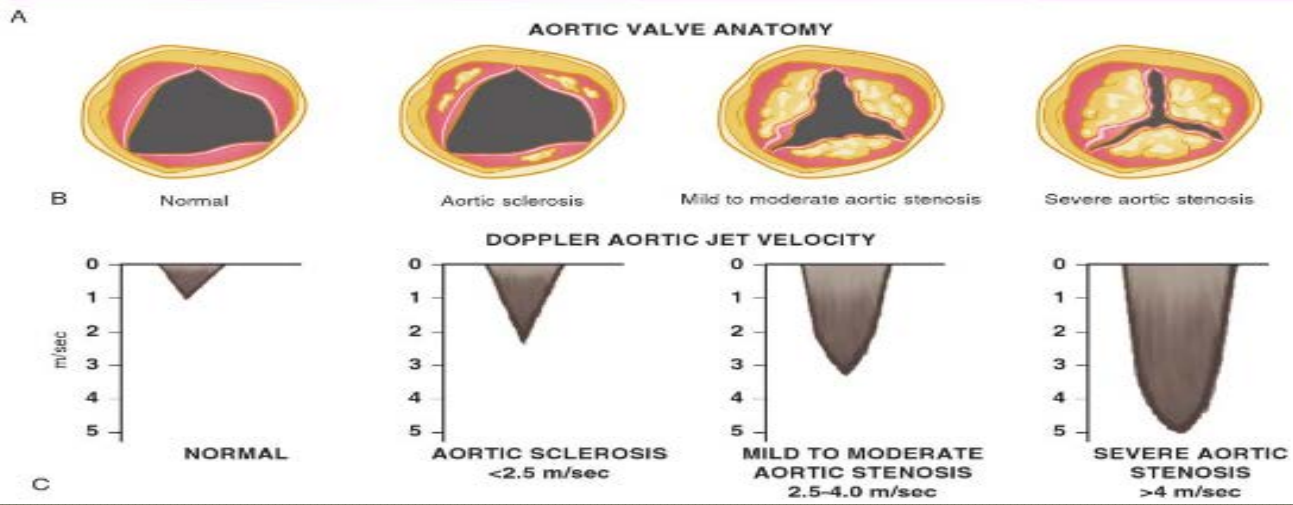
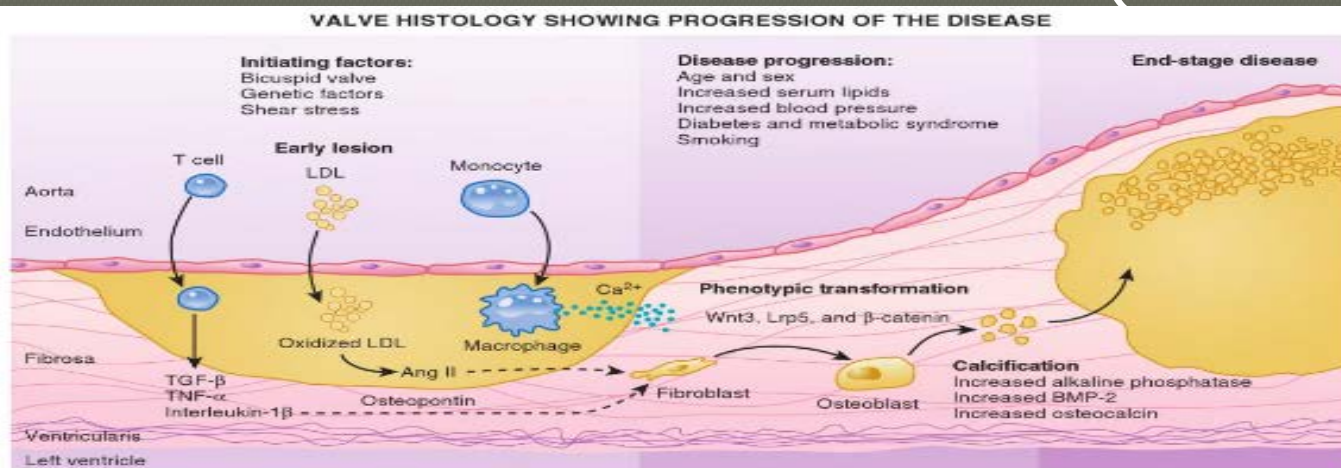
| RISK FACTOR | CAVD ANALYSIS | | |
|------------------------|-----------------|----------|-------------|
| | Cross-Sectional | Incident | Progression |
| Age | +++ | +++ | +++ |
| Male sex | ++/- | ++ | 0 |
| Height | ++ | ++ | 0 |
| BMI | ++ | ++ | 0 |
| Hypertension | ++ | ++ | 0 |
| Diabetes | +++ | +++ | 0 |
| Metabolic syndrome | ++ | ++ | + |
| Dyslipidemia | ++ | ++ | 0 |
| Smoking | ++ | ++ | + |
| Renal dysfunction | + | 0 | 0 |
| Inflammatory markers | + | 0 | 0 |
| Phosphorus levels | ++ | 0 | N/A |
| Calcium levels | 0 | 0 | N/A |
| Baseline calcium score | N/A | N/A | +++ |

TABLE 63-2 Candidate Gene Association Studies for Calcific Aortic Valve Disease

| STUDY (YEAR) | GENE | LOCATION | PHENOTYPE | CASES | RISK VARIANT | P VALUE |
|-----------------------|-----------------------------|-----------|---------------------------|-------|---------------------------------------|------------------|
| Ortlepp et al, 2001 | Vitamin D receptor | 12q12-q14 | Severe AS | 100 | B allele | .001 |
| Avakian et al, 2001 | ApoB | 2p24-p23 | Severe AS | 62 | X+ | .007 |
| | ApoE | 19q13.2 | AS | 43 | ApoE 2/4 + 3/4 genotypes | .03 |
| Nordström et al, 2003 | Estrogen receptor α | 6q25.1 | AVR | 41 | PvuII polymorphism | .03 |
| | TGF- β receptor type1 | 9q33-q34 | AVR | 41 | AocI polymorphism | * |
| Ortlepp et al, 2004 | Interleukin-10_ ENREF_95 | 1q31-q32 | Ex vivo atomic absorption | 187 | 3 promoter polymorphisms | .03 |
| | Chemokine receptor 5 | 3p21.31 | Ex vivo atomic absorption | 187 | 32-base pair deletion | .04 [†] |
| Moura et al, 2012 | Paraoxonase 1 | 7q21-22 | Moderate AS | 67 | Q192R polymorphisms | .03 |
| Kamstrup et al, 2014 | Lipoprotein (a) | 6q26-27 | AS with or without AVR | 454 | LPA genotypes rs10455872, rs3798220 | .001 |
| Mahmut et al, 2014 | LP PLA2 | rs1805017 | Ex vivo | 40 | Upregulation of PLA2G family of genes | .001 |

Aortic Valve Etiology

○ Calcific aortic valve disease (senile)

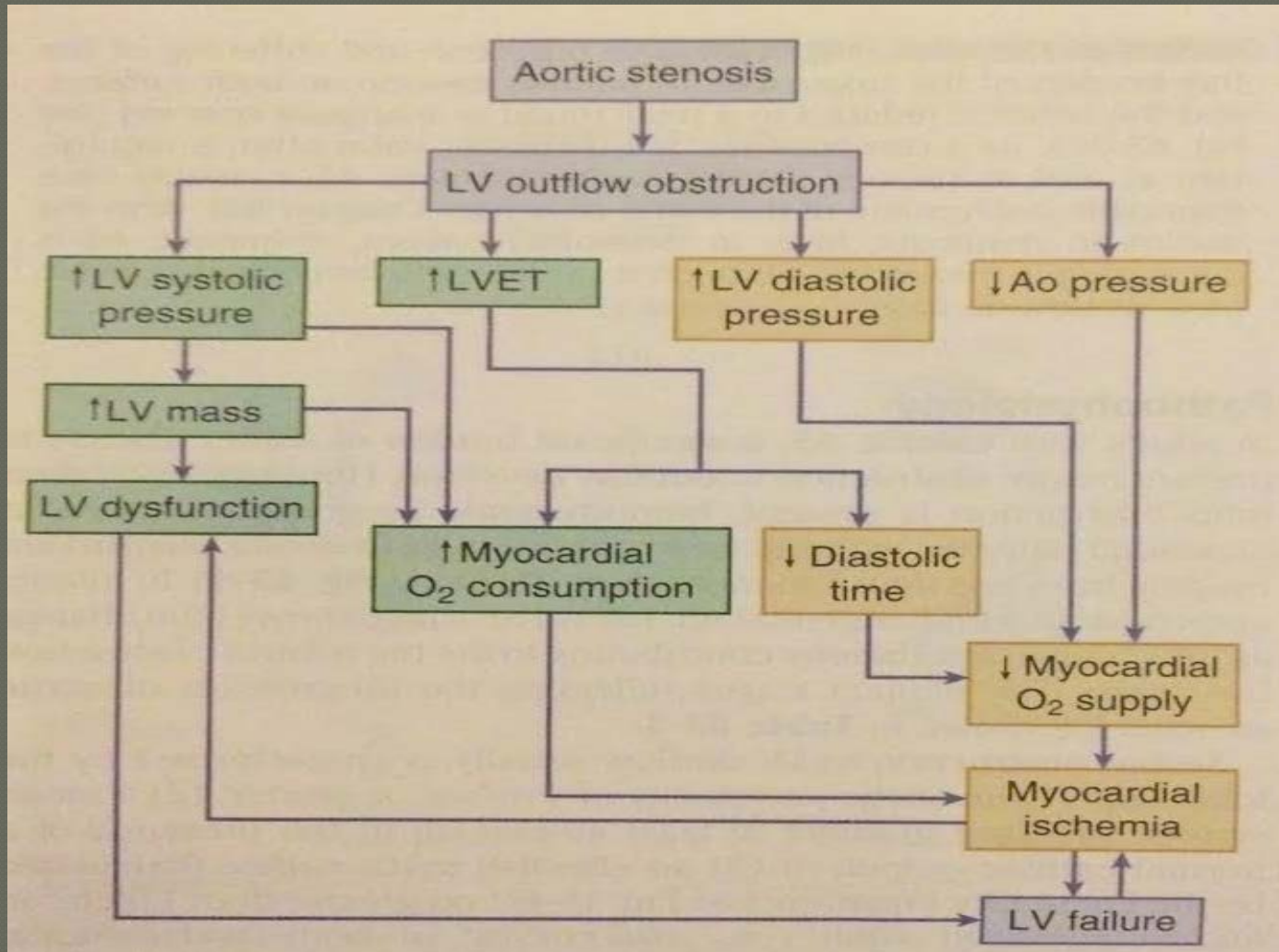


Aortic Stenosis Pathophysiology

● Outflow obstruction

- Increased LV systolic pressure leads to increased LV mass with subsequent diastolic dysfunction and increase in myocardial O₂ consumption.
- Increased LV ejection time leads to increased myocardial O₂ consumption and decreased diastolic time.
- Increased LV diastolic pressure leads to decreased myocardial O₂ supply
- Decreased aortic pressure leads to decreased myocardial O₂ supply
- Myocardial O₂ consumption exceeds myocardial O₂ supply and LV failure develops.

Aortic Stenosis Pathophysiology



Aortic Stenosis Symptoms

○ Cardinal Symptoms

- I. Exertional Dyspnea
- II. Angina
- III. Syncope

Aortic Stenosis Symptoms

○ Exertional Dyspnea

- Most common clinic presentation in severe AS.
- Associated with decrease in exercise tolerance and fatigue.
- Symptoms are due to the inability to increase cardiac output with exercise.
- LV diastolic dysfunction and subsequent rise in end-diastolic pressure can lead to pulmonary congestion.

Aortic Stenosis Symptoms

○ Angina

- Occurs in approximately 2/3 of patients with severe AS, approximately 50% of whom have significant CAD.
- In those without CAD, angina is due to increase oxygen requirement for the hypertrophied myocardium and reduction of O₂ delivery secondary to excessive compression of coronary vessels and decreased diastolic time.
- In those with CAD, angina is due to a combination of epicardial coronary compression and O₂ imbalance. Very rarely due calcific emboli to the coronary vessels.

Aortic Stenosis Symptoms

○ Syncope

- Systemic vasodilation during exertion leads to decline in arterial pressure. In the setting of a fixed cardiac output this leads to reduced cerebral perfusion.
- Malfunction of the baroreceptors via activation of the mechanosensitive vagal afferents in response to outflow obstruction leads to a vasodepressor response.
- Loss of atrial contribution to LV filling in the setting of transient Afib can also lead to decline in CO and subsequent syncope.
- Transient AV block due to extension of calcification into the conduction system can lead to syncope due to bradycardia or loss of atrial contribution to LV filling.

Aortic Stenosis Physical Exam

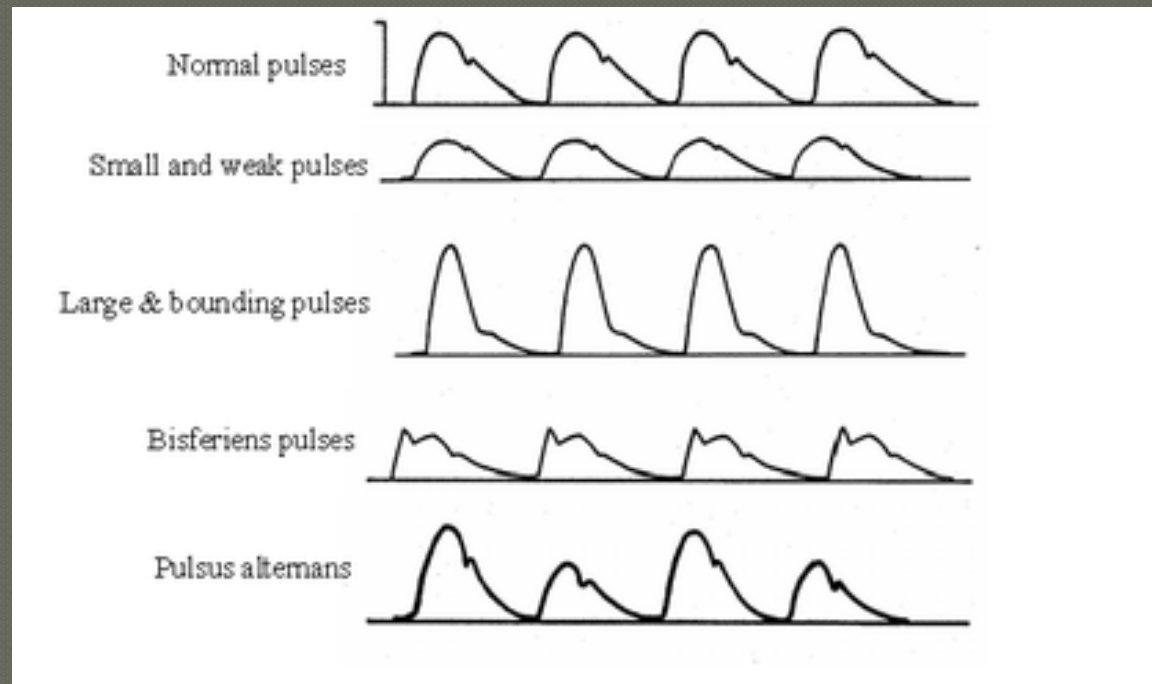
○ Key features of physical examination

I. Carotid upstroke – Pulsus parvus et tardus

- Reflects arterial pressure wave form
- Palpation of carotid pulse demonstrates a weak pulse (parvus)
- Simultaneous Palpation of the carotid upstroke while auscultating the heart will demonstrate peak carotid upstroke closer to S2 as opposed to S1 (tardus).
- Above findings specific for severe AS but not sensitive as concurrent conditions such as systemic hypertension can affect the carotid impulse.

Aortic Stenosis Physical Exam

- Key features of physical examination
 - I. Carotid upstroke – Pulsus parvus et tardus



Aortic Stenosis Physical Exam

○ Key features of physical exam

II. Murmur

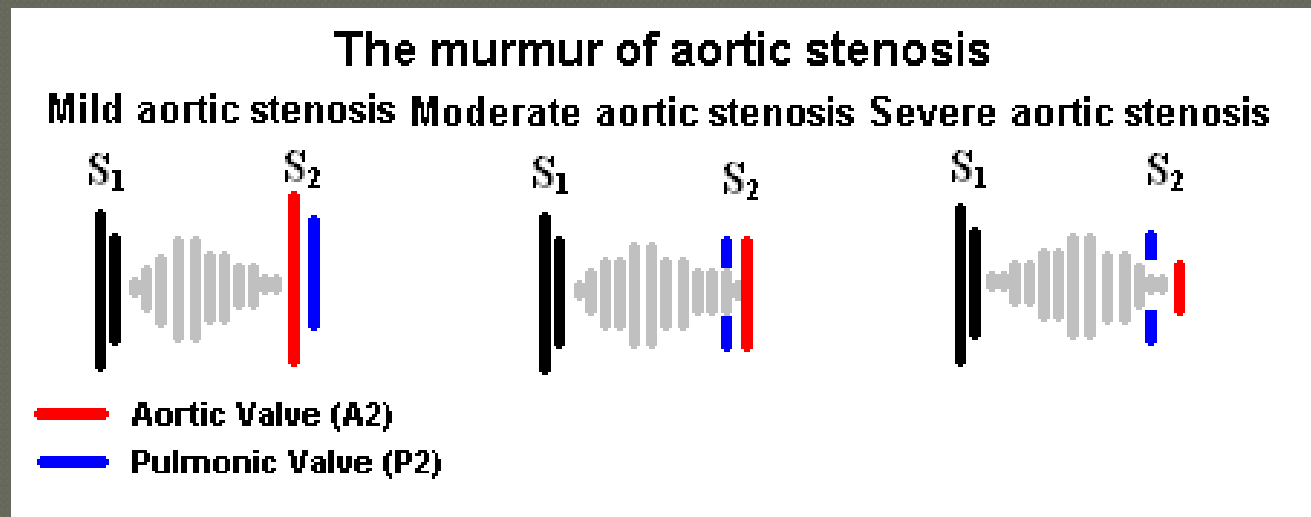
- Best heard over the 2nd RICS
- Harsh Crescendo-decrescendo systolic murmur
- Radiates to the carotids
- High frequency can radiate to apex mimicking MR – Gallavardian phenomenon.
- Intensity of the murmur is an insensitive marker for severity.
- Late peaking murmur indicates severe AS.
- Squatting augments murmur (increase SV)
- Valsalva and handgrip maneuver decrease murmur due to decreased preload and increased afterload, respectively

Aortic Stenosis Physical Exam

Key features of physical exam

III. Effects on S2

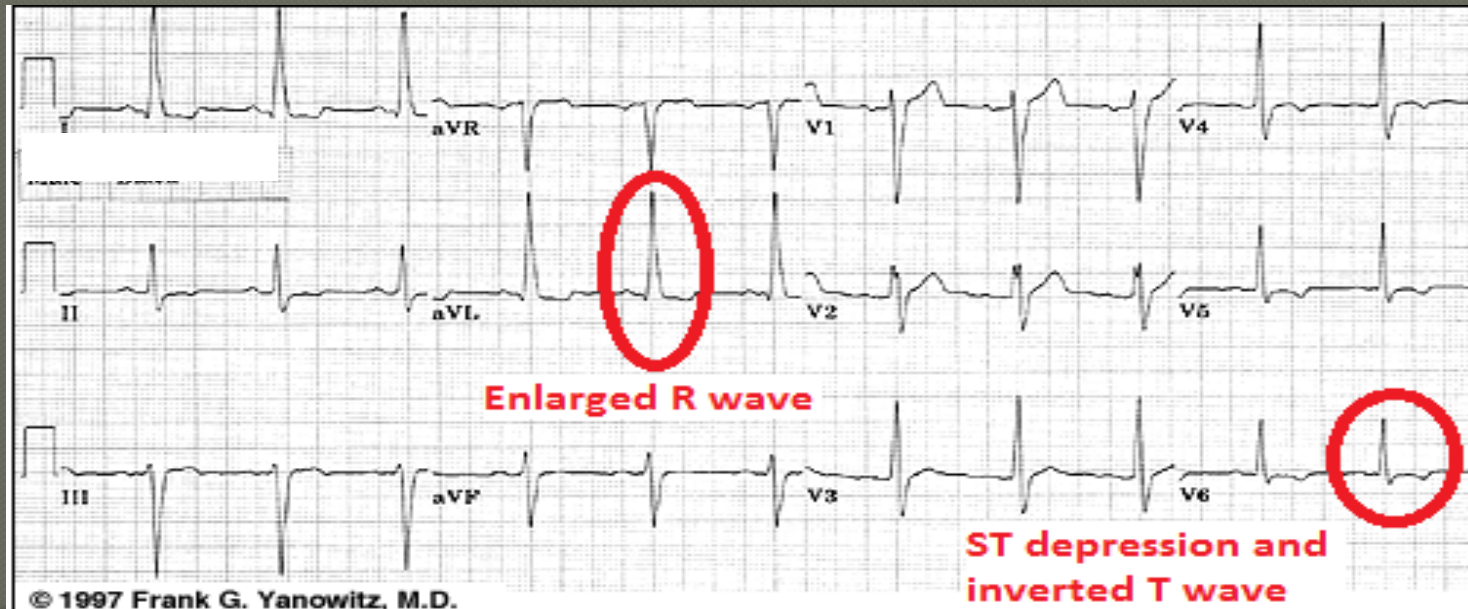
- Single S2, calcification and immobility of the aortic valve make A2 inaudible and P2 is only heard.
- Paradoxical split (LBBB or prolonged LV systole)
- Normally splitting excludes severe AS.



Aortic Stenosis Diagnostic Studies

○ EKG

- LV hypertrophy
- Left atrial enlargement
- AV or IVD depending on the extension of calcific infiltrates in to the conduction system.



Aortic Stenosis Diagnostic Studies

○ Echocardiography

- Standard approach for following and evaluating patients with AS.
- Accurate at assessing valve anatomy and severity of calcification.
- Assessment of LV function.
- Doppler allows measurements of transaortic jet velocity.
- Valve area and mean transaortic gradient can be calculated using continuity equation and Bernoulli equation, respectively.

Aortic Stenosis Diagnostic Studies

○ Echocardiography

| | Valve Area (cm ²) | Maximum Aortic Velocity (mmHg) | Mean Pressure Gradient (mmHg) |
|----------|-------------------------------|--------------------------------|-------------------------------|
| Mild | 1.5-2 | 2.5-3.0 | < 25 |
| Moderate | 1.0-1.5 | 3.0-4.0 | 25-40 |
| Severe | 0.6-1.0 | >4.0 | >40 |
| Critical | < 0.6 | | |

Aortic Stenosis Diagnostic Studies

○ Cardiac Catheterization

- Recommended when noninvasive testing is inconclusive , clinical and echocardiographic findings are discordant.
- Recommended prior to surgical intervention to assess coronary arteries.

Aortic Stenosis Stages

- In 2014, AHA/ACC published updated classification of VHD into 4 stages (A-D)

Table 3. Stages of Progression of VHD

| Stage | Definition | Description |
|-------|---------------------|---|
| A | At risk | Patients with risk factors for development of VHD |
| B | Progressive | Patients with progressive VHD (mild-to-moderate severity and asymptomatic) |
| C | Asymptomatic severe | Asymptomatic patients who have the criteria for severe VHD: C1: Asymptomatic patients with severe VHD in whom the left or right ventricle remains compensated C2: Asymptomatic patients with severe VHD, with decompensation of the left or right ventricle |
| D | Symptomatic severe | Patients who have developed symptoms as a result of VHD |

VHD indicates valvular heart disease.

Aortic Stenosis Stages

| Stage | Definition | Valve Anatomy | Valve Hemodynamics | Hemodynamic Consequences | Symptoms |
|-------|----------------|---|--|--|--|
| A | At risk of AS | <ul style="list-style-type: none"> Bicuspid aortic valve (or other congenital valve anomaly) Aortic valve sclerosis | <ul style="list-style-type: none"> Aortic $V_{\max} < 2$ m/s | <ul style="list-style-type: none"> None | <ul style="list-style-type: none"> None |
| B | Progressive AS | <ul style="list-style-type: none"> Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or Rheumatic valve changes with commissural fusion | <ul style="list-style-type: none"> Mild AS: Aortic V_{\max} 2.0–2.9 m/s or mean $\Delta P < 20$ mm Hg Moderate AS: Aortic V_{\max} 3.0–3.9 m/s or mean ΔP 20–39 mm Hg | <ul style="list-style-type: none"> Early LV diastolic dysfunction may be present Normal LVEF | <ul style="list-style-type: none"> None |

Aortic Stenosis Stages

C: Asymptomatic severe AS

| | | | | | |
|----|--|---|---|--|--|
| C1 | Asymptomatic severe AS | <ul style="list-style-type: none"> Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening | <ul style="list-style-type: none"> Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg AVA typically is ≤ 1.0 cm² (or AVAi ≤ 0.6 cm²/m²) Very severe AS is an aortic $V_{max} \geq 5$ m/s or mean $\Delta P \geq 60$ mm Hg | <ul style="list-style-type: none"> LV diastolic dysfunction Mild LV hypertrophy Normal LVEF | <ul style="list-style-type: none"> None: Exercise testing is reasonable to confirm symptom status |
| C2 | Asymptomatic severe AS with LV dysfunction | <ul style="list-style-type: none"> Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening | <ul style="list-style-type: none"> Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg AVA typically ≤ 1.0 cm² (or AVAi ≤ 0.6 cm²/m²) | <ul style="list-style-type: none"> LVEF $< 50\%$ | <ul style="list-style-type: none"> None |

Aortic Stenosis Stages

D: Symptomatic severe AS

| | | | | | |
|----|---|---|--|---|---|
| D1 | Symptomatic severe high-gradient AS | <ul style="list-style-type: none"> Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening | <ul style="list-style-type: none"> Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg AVA typically ≤ 1.0 cm² (or AVAi ≤ 0.6 cm²/m²) but may be larger with mixed AS/AR | <ul style="list-style-type: none"> LV diastolic dysfunction LV hypertrophy Pulmonary hypertension may be present | <ul style="list-style-type: none"> Exertional dyspnea or decreased exercise tolerance Exertional angina Exertional syncope or presyncope |
| D2 | Symptomatic severe low-flow/low-gradient AS with reduced LVEF | <ul style="list-style-type: none"> Severe leaflet calcification with severely reduced leaflet motion | <ul style="list-style-type: none"> AVA ≤ 1.0 cm² with resting aortic $V_{max} < 4$ m/s or mean $\Delta P < 40$ mm Hg Dobutamine stress echocardiography shows AVA ≤ 1.0 cm² with $V_{max} \geq 4$ m/s at any flow rate | <ul style="list-style-type: none"> LV diastolic dysfunction LV hypertrophy LVEF $< 50\%$ | <ul style="list-style-type: none"> HF Angina Syncope or presyncope |
| D3 | Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS | <ul style="list-style-type: none"> Severe leaflet calcification with severely reduced leaflet motion | <ul style="list-style-type: none"> AVA ≤ 1.0 cm² with aortic $V_{max} < 4$ m/s or mean $\Delta P < 40$ mm Hg Indexed AVA ≤ 0.6 cm²/m² and Stroke volume index < 35 mL/m² Measured when patient is normotensive (systolic BP < 140 mm Hg) | <ul style="list-style-type: none"> Increased LV relative wall thickness Small LV chamber with low stroke volume Restrictive diastolic filling LVEF $\geq 50\%$ | <ul style="list-style-type: none"> HF Angina Syncope or presyncope |

Vignette 1

- Mr A, 83 y/o male presents with shortness of breath and peripheral edema. His pulse is 64 bpm and his BP 110/90. Auscultation reveals an absent aortic second heart sound with a late peaking systolic murmur best heard over the 2nd RICS.

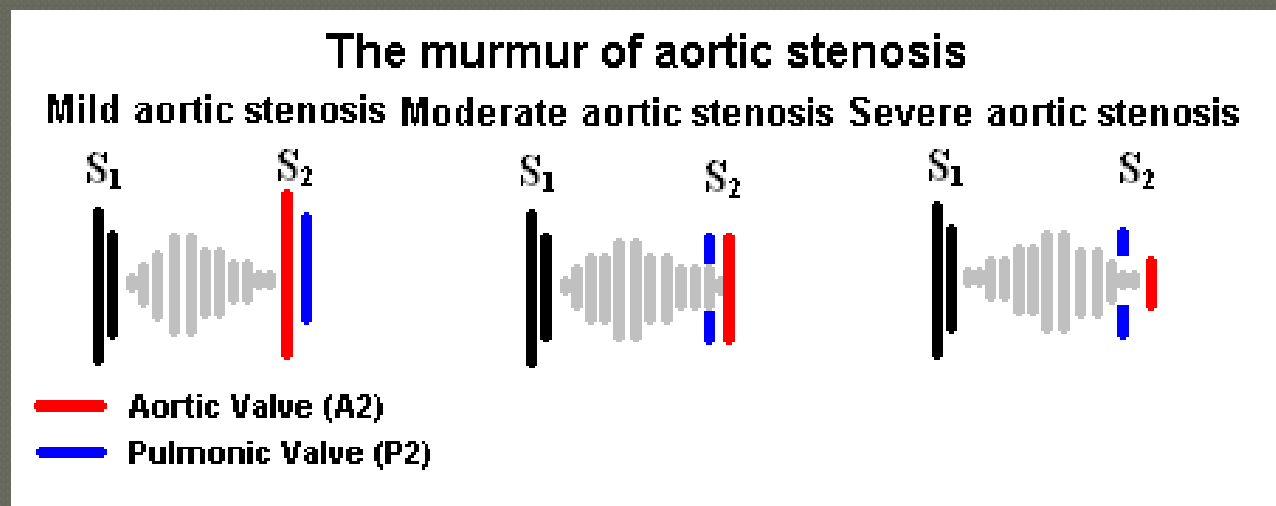
He has a pmh of HTN and HLD, otherwise his renal and pulmonary functions are normal.

Vignette 1 continued

- What features are concerning in regards to the patient physical exam?

Absent aortic second heart sound

Late peaking systolic murmur



Vignette 1 continued

- What diagnostic study would like to order?

Echocardiogram

- LV EF: 55%
 - Mean gradient: 60mmHg
 - AVA: 0.6cm²
 - Peak Velocity: 4.6 m/s
- What stage is the patient?
 - D1

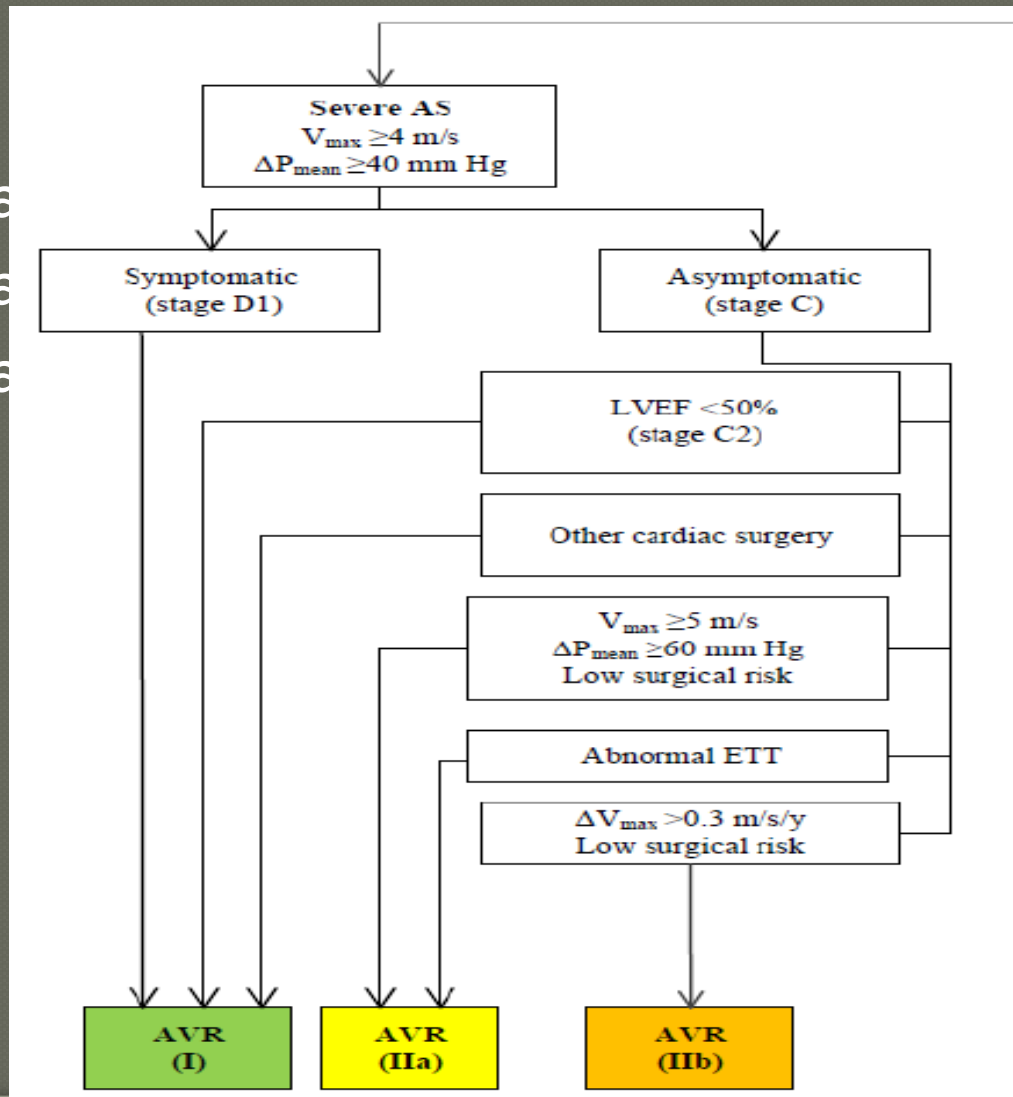
Vignette 1

- Mr. A is appreciative of your thorough evaluation and asks, “What is next?”
 - A. Tell Mr. A that his disease is severe and you will continue to monitor with surveillance echocardiograms.
 - B. Initiate guideline directed medical therapy.
 - C. Tell Mr. A that all is well and is free to go home.
 - D. Refer for surgical aortic valve replacement

Vignette 1

Class

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- Sta
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Vignette 2

Mr. B, 79 y/o male with HTN and HLD presents to the outpatient clinic with worsening shortness of breath and chest discomfort.

You order an EKG which demonstrates LVH and an echocardiogram which demonstrates an LV EF of 25%, a calcified aortic valve with a mean gradient of 28mmHg, AVA of 0.8cm² and peak velocity of 3.16m/s.

Vignette 2

- Does the patient's aortic stenosis account for his symptoms? Does the patient have severe AS? What would you like to do next?
 - A. The patient symptoms are secondary to heart failure. Optimize his medical regimen.
 - B. The patient has moderate aortic stenosis. Repeat echo in 6-12months.
 - C. Order a dobutamine stress echo to assess for severe AS versus pseudostenosis.

Vignette 2

- Differentiating true aortic stenosis from “pseudostenosis and its importance.
 - Dobutamine stress echo (DSE) has a class IIa indication in patient with stage D2 aortic stenosis.
 - It can differentiate true severe aortic stenosis (Stage D2) from “pseudostenosis”. This is paramount as the former is associated with improved long-term outcomes with AVR and the latter has no benefit from AVR.
 - Allows for assessment of contractile reserve, defined as an increase SV > 20%. Patient’s without contractile reserve have higher operative mortality.

Class IIa

- 1. Low-dose dobutamine stress testing using echocardiographic or invasive hemodynamic measurements is reasonable in patients with stage D2 AS with all of the following (46-48), (Level of Evidence: B):**
 - a. Calcified aortic valve with reduced systolic opening;**
 - b. LVEF less than 50%;**
 - c. Calculated valve area 1.0 cm² or less; and**
 - d. Aortic velocity less than 4.0 m per second or mean pressure gradient less than 40 mm Hg.**

Vignette 2

Dobutamine stress echocardiography in low-flow aortic stenosis

Low-flow, low-gradient aortic stenosis

Dobutamine stress echocardiography

Hemodynamic response

Aortic valve area $> 1.2 \text{ cm}^2$
Projected aortic valve area $> 1.0 \text{ cm}^2$

Aortic valve area $\leq 1.2 \text{ cm}^2$
Projected aortic valve area $\leq 1.0 \text{ cm}^2$

Pseudostenosis

Medical treatment

True severe aortic stenosis

Contractile reserve:
Increase in stroke volume $> 20\%$

Aortic valve replacement
with or without
coronary artery bypass grafting

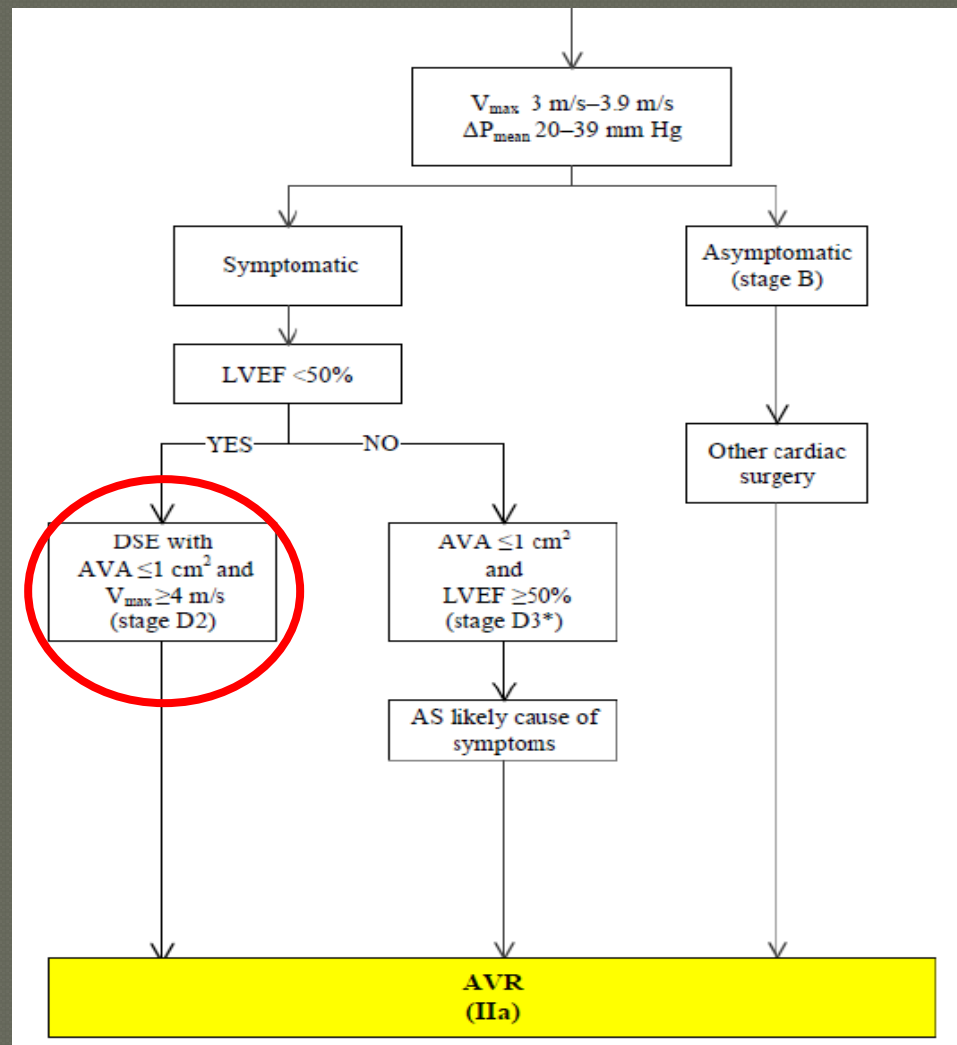
No contractile reserve:
Increase in stroke volume $\leq 20\%$

Options:
Transcatheter aortic valve replacement
Surgical aortic valve replacement
Heart transplantation
Heart failure therapy

Vignette 2

- Mr. B underwent DSE and results demonstrated increase in EF, SV and transvalvular gradient suggestive of true severe AS and contractile reserve. You subsequently refer him for AVR.

Vignette 2



Vignette 3

- Mr. C, 81 y/o male with HTN and HLD presents to the ER with chest pain. EKG demonstrates nonspecific ST changes with elevated troponin. He is diagnosed with NSTEMI. A LHC is performed and reveals triple vessel disease. Echocardiogram is ordered and demonstrates an EF of 55% and aortic stenosis with a AVA 1.2cm² and a mean gradient of 28mmHg and peak velocity 3.31 m/s

Vignette 3

○ What is the stage and the severity of the patient aortic stenosis?

- A. Stage A, mild AS
- B. Stage D, severe AS
- C. Stage C, moderate AS
- D. Stage B, moderate AS

| | | | | | |
|---|----------------|--|---|---|--|
| B | Progressive AS | <ul style="list-style-type: none">• Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or• Rheumatic valve changes with commissural fusion | <ul style="list-style-type: none">• Mild AS: Aortic V_{max} 2.0–2.9 m/s or mean $\Delta P < 20$ mm Hg• Moderate AS: Aortic V_{max} 3.0–3.9 m/s or mean ΔP 20–39 mm Hg | <ul style="list-style-type: none">• Early LV diastolic dysfunction may be present• Normal LVEF | <ul style="list-style-type: none">• None |
|---|----------------|--|---|---|--|

Vignette 3

- How would manage the patient's aortic stenosis?
 - A. Monitor with surveillance echocardiogram every 6-12 months.
 - B. Medical management with statin and ACE inhibitor.
 - C. Perform PCI to obstructive lesions and monitor AS as outpatient.
 - D. Refer CABG and AVR.

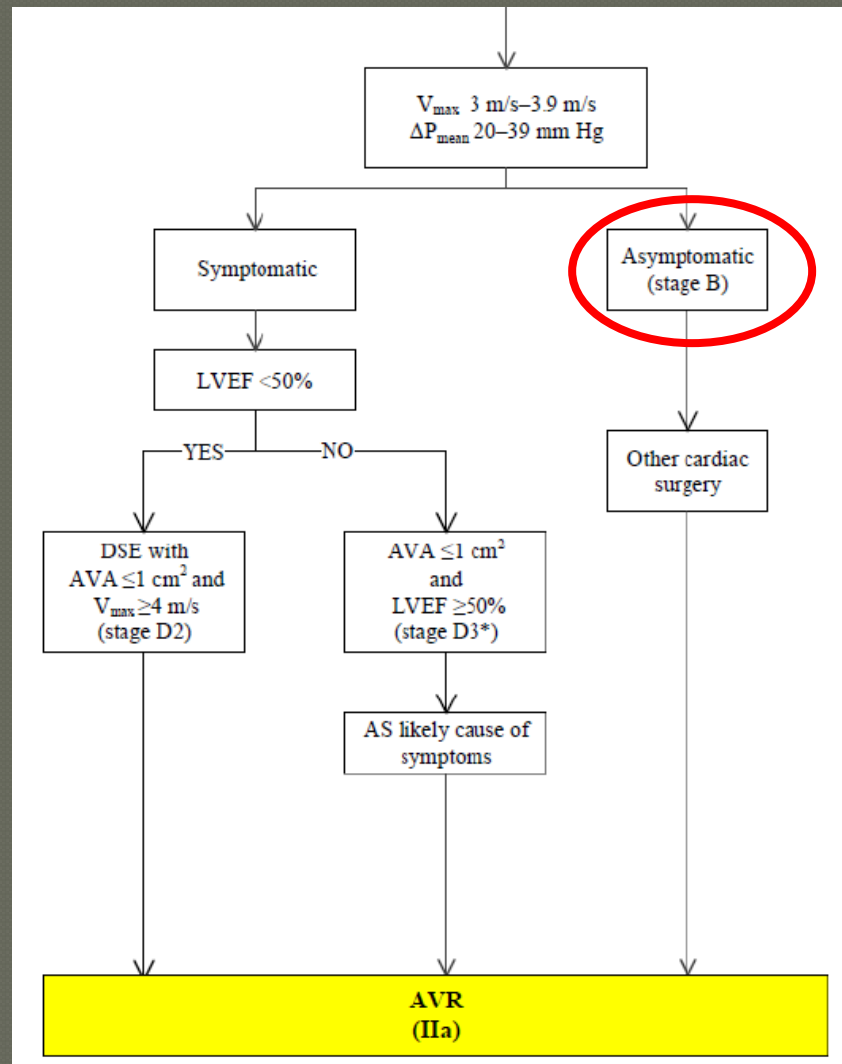
Vignette 3

- AHA/ACC guidelines for VHD state that AVR is a class IIa indication for moderate AS (stage B) undergoing cardiac surgery.
- Stage B patient, otherwise, follow the below surveillance regimen.

Table 4. Frequency of Echocardiograms in Asymptomatic Patients with VHD and Normal Left Ventricular Function

| Stage | Valve Lesion | | | |
|-----------------------|--|--|---|--|
| | Aortic Stenosis* | Aortic Regurgitation | Mitral Stenosis | Mitral Regurgitation |
| Progressive (stage B) | Every 3–5 y (mild severity V_{max} 2.0–2.9 m/s) every 1–2 y (moderate severity V_{max} 3.0–3.9 m/s) | Every 3–5 y (mild severity) Every 1–2 y (moderate severity) | Every 3–5 y (MVA >1.5 cm ²) | Every 3–5 y (mild severity) Every 1–2 y (moderate severity) |
| Severe (stage C) | Every 6–12 mo ($V_{max} \geq 4$ m/s) | Every 6–12 mo Dilating LV: more frequently | Every 1–2 y (MVA 1.0–1.5 cm ²) Once every year (MVA <1.0 cm ²) | Every 6–12 mo Dilating LV: more frequently |

Vignette 3



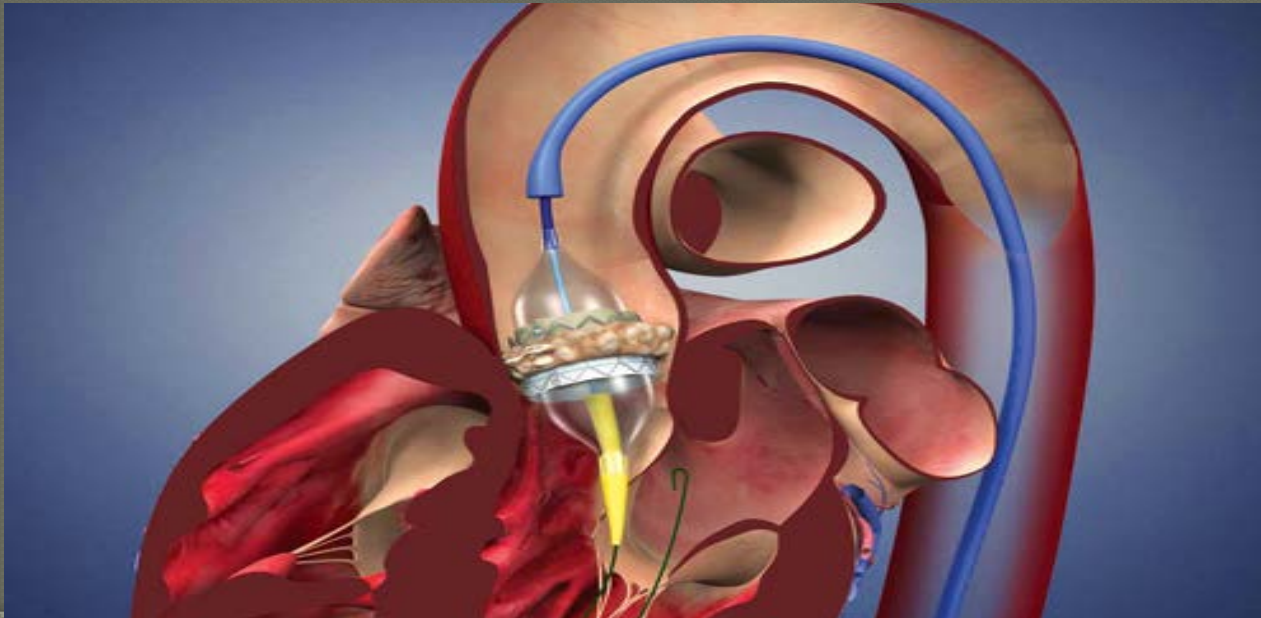
Vignette 4

- Mr. E, 84 y/o male has severe aortic stenosis (AVA 0.6cm² and mean gradient of 56mmHg), CAD s/p CABG, moderate COPD, CKD, HTN, HLD and DMII. He presents with worsening shortness of breath over the past month. Your impression is that he is very frail.
- How would you manage Mr. E's aortic stenosis?
 - A. Medical management as he is high risk.
 - B. Recommend balloon valvuloplasty.
 - C. Repeat echocardiogram.
 - D. Refer for transcatheter aortic valve replacement (TAVR)

Vignette 4

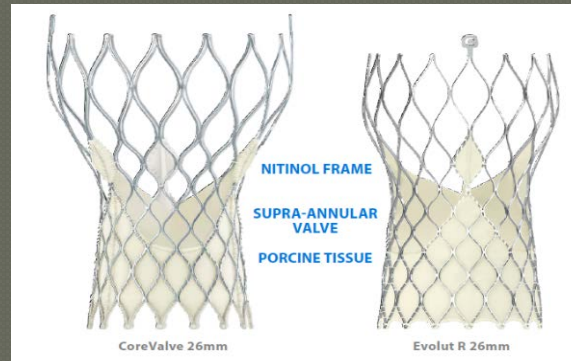
● TAVR

- Class I: TAVR is recommend in patients who have an indication for AVR with prohibitive surgical risk and a predicted post-TAVR survival greater than 12 months.
- Class IIa: TAVR is recommended in patients who have an indication for AVR with a high surgical risk.



Vignette 4

- FDA approved in 2011, Edward Sapien valve and Medtronic CoreValve are available in the US.



- Outcomes of TAVR in the PARTNER trial demonstrated that those treated with TAVR had significantly better survival compared to medical therapy and similar long term outcomes (2years) compared to SAVR in high risk surgical patients.

Vignette 4

- FDA approved, Edward Sapien valve for intermediate risk patient in August 2016 based on Partner II trial
- The Partner II trial demonstrated that TAVR in intermediate-risk patients is associated with low mortality, strokes, and regurgitation at 1 year. The propensity score analysis indicates a significant superiority composite outcome with TAVR compared with surgery

Vignette 4

- Partner III trial is currently in progress. The trial is evaluating TAVR in low risk patient with severe aortic regurgitation.

Vignette 5

- Mr. D, 74 y/o male with HTN, HLD and AS presents for his annual echocardiogram. He lives a sedentary lifestyle but feels well with performing his ADLs. He denies dyspnea on exertion, chest pain or syncope.
- His Echo demonstrates preserved EF, AVA of 0.7cm², mean gradient 70mmHg and peak velocity 4.7m/s. The prior year he had a mean gradient of 40 and peak velocity of 4.0m/s

How would you manage his aortic stenosis?

- A. Continue yearly surveillance echocardiograms.
- B. Refer for exercise treadmill test to elicit symptoms.
- C. Discontinue yearly surveillance echocardiograms.

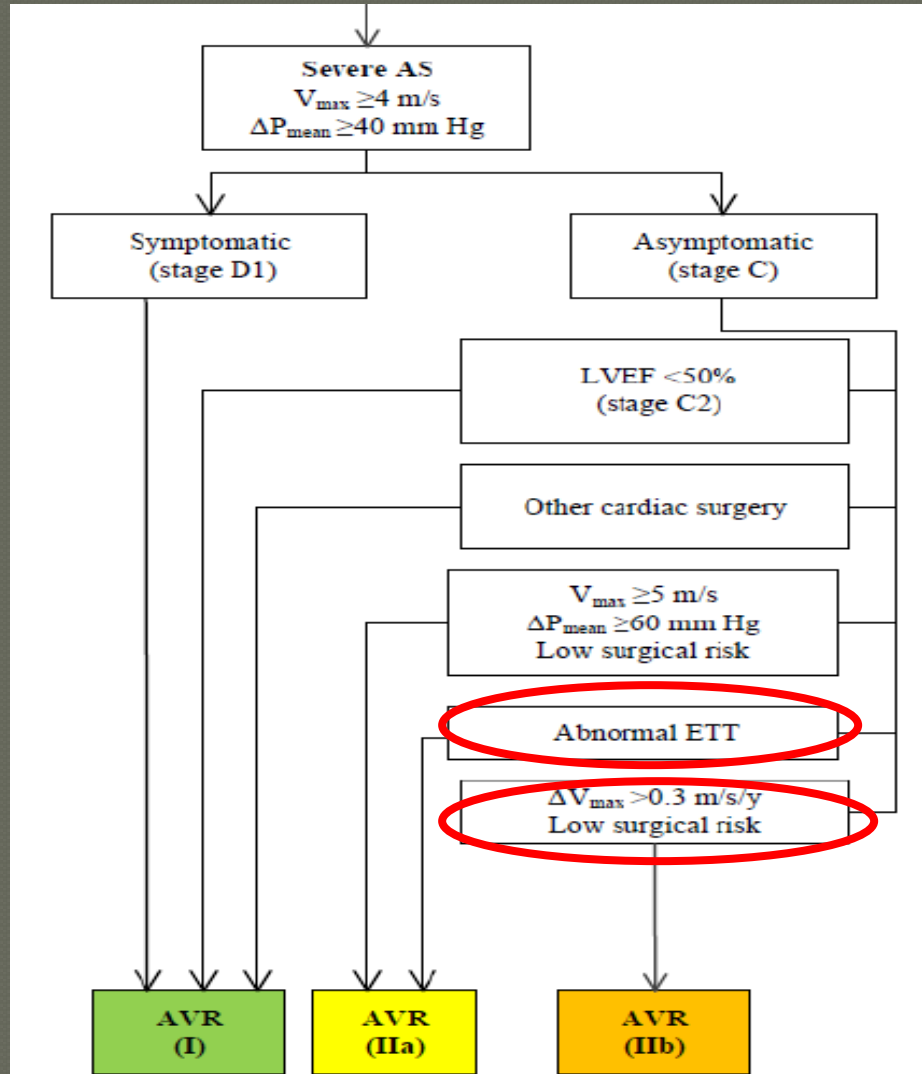
Vignette 5

- You refer Mr. D for an ETT which elicits symptoms. You subsequently refer him for AVR.

Class IIa

- 1. Low-dose dobutamine stress testing using echocardiographic or invasive hemodynamic measurements is reasonable in patients with stage D2 AS with all of the following (46-48), (*Level of Evidence: B*):**
 - a. Calcified aortic valve with reduced systolic opening;**
 - b. LVEF less than 50%;**
 - c. Calculated valve area 1.0 cm² or less; and**
 - d. Aortic velocity less than 4.0 m per second or mean pressure gradient less than 40 mm Hg.**
- 2. Exercise testing is reasonable to assess physiological changes with exercise and to confirm the absence of symptoms in asymptomatic patients with a calcified aortic valve and an aortic velocity 4.0 m per second or greater or mean pressure gradient 40 mm Hg or higher (stage C) (27, 37, 38, 49). (*Level of Evidence: B*)**

Vignette 5

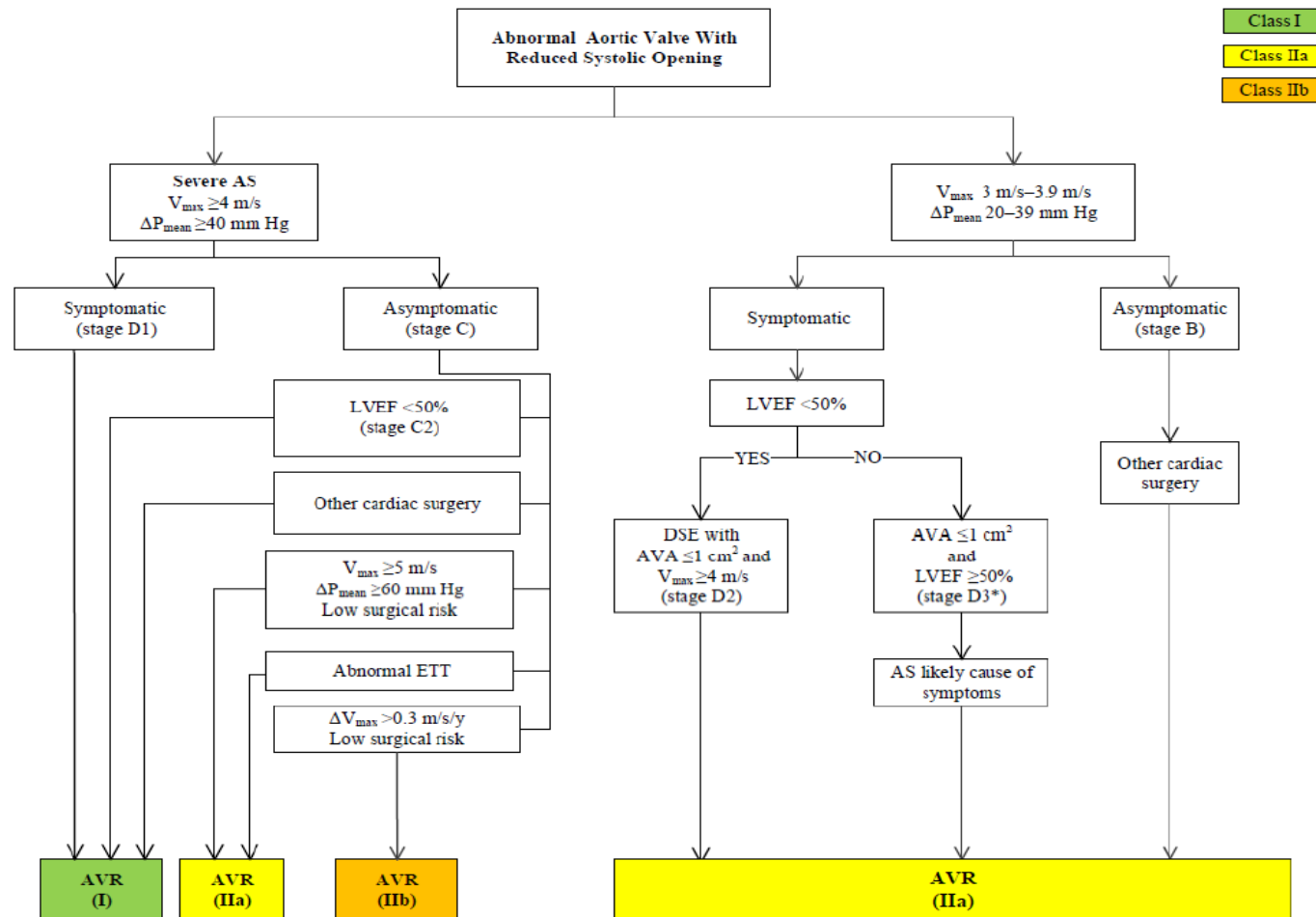


Aortic Stenosis Management

- In the case of life-limiting comorbidities consider aortic balloon valvuloplasty for palliation.
- Balloon valvuloplasty can also be used for bridge therapy
- Nitroprusside (vasodilator therapy) may be reasonable if used with invasive hemodynamic monitoring in the acute management of patients with stage D AS and NYHA stage IV heart failure symptoms. (IIb)

Aortic Stenosis Summary

Nishimura, RA et al.
2014 AHA/ACC Valvular Heart Disease Guideline



Surgical Risk

◉ Link to STS calculator:

<http://riskcalc.sts.org/stswebriskcalc/#/>

Table 5. Risk Assessment Combining STS Risk Estimate, Frailty, Major Organ System Dysfunction, and Procedure-Specific Impediments

| | Low Risk (Must Meet ALL Criteria in This Column) | Intermediate Risk (Any 1 Criterion in This Column) | High Risk (Any 1 Criterion in This Column) | Prohibitive Risk (Any 1 Criterion in This Column) |
|---|--|---|---|--|
| STS PROM* | <4% AND | 4% to 8% OR | >8% OR | Predicted risk with surgery of death or major morbidity (all-cause) >50% at 1 y OR |
| Frailty† | None AND | 1 Index (mild) OR | ≥2 Indices (moderate to severe) OR | |
| Major organ system compromise not to be improved postoperatively‡ | None AND | 1 Organ system OR | No more than 2 organ systems OR | ≥3 Organ systems OR |
| Procedure-specific impediment§ | None | Possible procedure-specific impediment | Possible procedure-specific impediment | Severe procedure-specific impediment |



Table 5. Risk Assessment Combining STS Risk Estimate, Frailty, Major Organ System Dysfunction, and Procedure-Specific Impediments

| | Low Risk (Must Meet ALL Criteria in This Column) | Intermediate Risk (Any 1 Criterion in This Column) | High Risk (Any 1 Criterion in This Column) | Prohibitive Risk (Any 1 Criterion in This Column) |
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