# The Ins and Outs of Valve Disease

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#### **OBJECTIVES**

- Discuss the cardiac cycle in terms of valvular mechanics
- Understand the cardiac cycle where heart sounds and different murmurs fall during the cycle
- Outline an approach to a patient with valvular disease
- Discuss how valve disease can contribute to other cardiac conditions including but not limited to rhythm disturbances, CHF and angina.

## AORTIC VALVE STENOSIS

Most common cause of LV outflow obstruction due to restricted aortic valve leaflet motion

## **Aortic Stenosis**

#### Acquired AS

- Most common
- Degeneration and calcification of the aortic valve leaflets
  >50 yrs
  - Inflammatory process similar to atherosclerosis:
- Rheumatic
- Congenital AS
  - Bicuspid aortic valve
  - 30-40 yrs (cause in 50% of pts <70 yrs)</p>
  - Associated with coarctation of the aorta

## Normal Open Aortic Valve

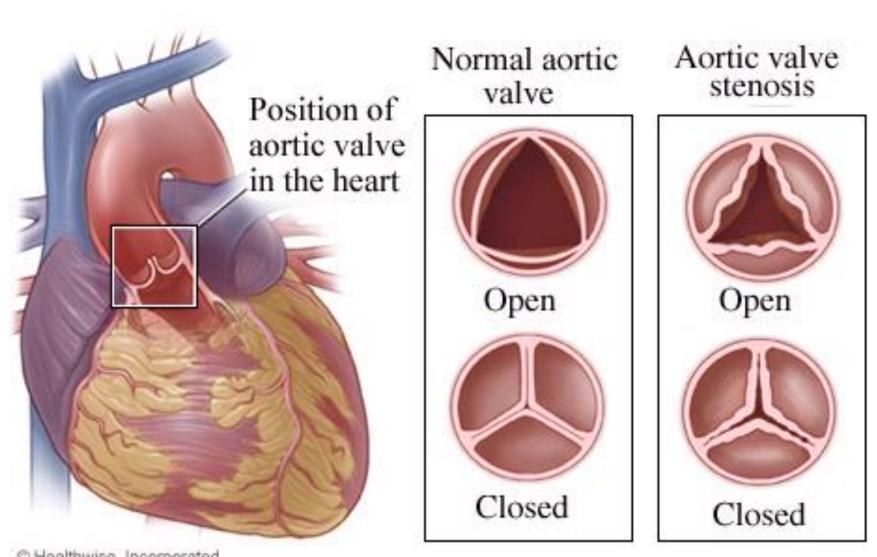


#### **Bicuspid Aortic Valve Stenosis**



#### **Calcific Tricuspid Aortic Stenosis**





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# EPIDEMIOLOGY

Prevalence

- Aortic sclerosis affects 25% of population > 65 yrs
- Critical AS in 2-3% of population > 75 yrs
- Bicuspid AV in 1% of population with 2:1 male to female ratio
- > 70% of bicuspid AV pts will develop AS

Insidious progression of LV outflow obstruction

Pressure gradient develops when effective AVA <1/2 baseline

Compensatory concentric LVH to maintain SV

Chronic LV pressure overload

Increase in LVEDP

Cause/exacerbate MR

Progression depends on functionally competent MV

Diastolic dysfunction and interstitial fibrosis

## PATHOPHYSIOLOGY

#### Presentation

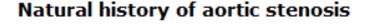
- Patients are often asymptomatic for many years
- Most common symptoms: DOE and exercise intolerance
- Progression of the stenosis results in symptoms
  - Usually occurs in 5<sup>th</sup> or 6<sup>th</sup> decade of life for acquired AS patients, about 10 years earlier for congenital AS patients

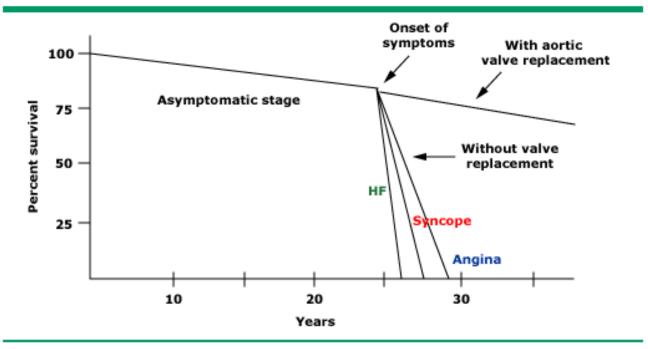
#### Complications

Remember: Aortic Stenosis Complications

- Angina
- Syncope
- Congestive heart failure

Symptoms are related to stenotic limitation to flow, increased left ventricular wall mass (therefore increased supply/demand mismatch), depending on symptoms





Schematic representation of the natural history of aortic stenosis and of the major impact of aortic valve replacement. Survival is excellent during the prolonged asymptomatic phase. After the development of symptoms, however, mortality exceeds 90 percent within a few years. Aortic valve replacement prevents this rapid downhill course.



# PHYSICAL EXAMINATION



- Turbulent flow across a stenotic aortic valve
- Heard best at RUSB, crescendo-decrescendo systolic murmur, late peaking, harsh, radiate to the neck, parvus et tardus\*, diminished S2\*
- Sustained apical impulse
- <u>AS murmur</u>

## Evaluation of AS

## EKG

CXR

Echo

Cardiac catheterization

Normal valve area = 3-4 cm<sup>2</sup>

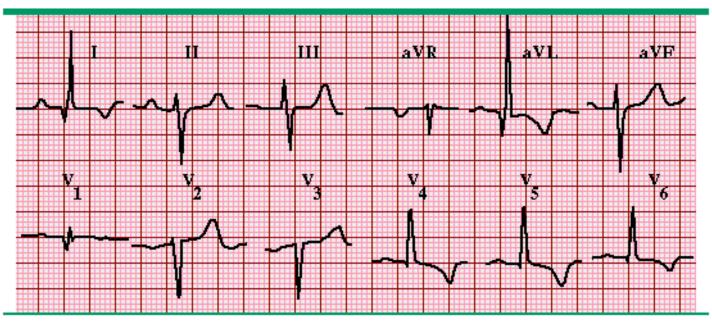
Severe AS < 1 cm<sup>2</sup>

Critical AS < 0.75 cm<sup>2</sup>

# **Treatment of AS**

- Aortic valve replacement is definitive
  - Balloon valvuloplasty may be used in young patients in which the patient has congenital AS
- Blood pressure control and gentle diuresis
- Statins if calcific aortic stenosis

#### Left ventricular hypertrophy with strain pattern



The ST-T wave abnormalities secondary to left ventricular hypertrophy (often termed "strain") are most often seen in the anterolateral leads (eg, I, aVL, V4-V6). Typical abnormalitites include a horizontal or downsloping ST segment and T wave inversions. In some cases there is concavity to the ST segment which has a final downward turn that blends into an inverted T wave.



#### Calcification of the aortic root



Lateral view of chest x-ray shows calcification at the aortic root (arrow). Courtesy of Jonathan Kruskal, MD.



## ACA/AHA GUIDELINE: Indications for AVR in AS

- Class I:
  - Symptomatic severe AS
  - Severe AS in pt undergoing CABG or surgery on the aorta or other heart valve
  - Severe AS w/ LVEF < 50%
- Class IIa:
  - Moderate AS in pt undergoing CABG or surgery on the aorta or other heart valve
- Class IIb:
  - Severe AS in asymptomatic pt with abnormal response to exercise
  - Severe AS in asymptomatic pt in whom surgery might be delayed at the time of symptom onset
  - Severe AS in asymptomatic pt with a high likelihood of rapid progression (age, valve calcification, CAD)
  - Mild AS in pt undergoing CABG in whom there is evidence (moderate or severe valve calcification) that progression may be rapid
  - Extremely severe AS (AVA < 0.6cm2, mean gradient >60mmHg, Jet velocity > 5.0m/sec) in asymptomatic pt in whom the expected operative mortality is <1%</li>

# BACKGROUND



- High mortality associated with untreated severe, symptomatic AS; i.e., 50% within 2 years
- AVR reduces symptoms and improves survival
- >30% severe, symptomatic AS untreated due to advanced age or comorbidities

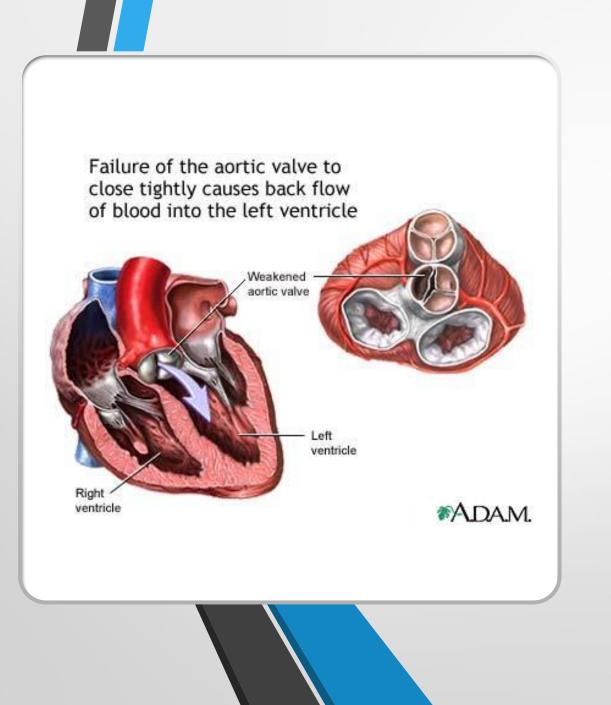
# Summary

#### Causes of aortic stenosis

Acquired

- Calcification vs. rheumatic
- Congenital
- Symptoms
- Angina, syncope, congestive heart failure
  Clinical findings
- Crescendo-decrescendo systolic murmur
- Possibly an S4
- *Parvus et tardus* ("weak" and "delayed" carotid upstrokes)
- Treatment

Surgery



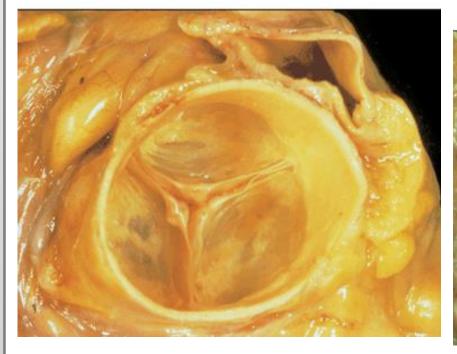
## Aortic Insufficiency

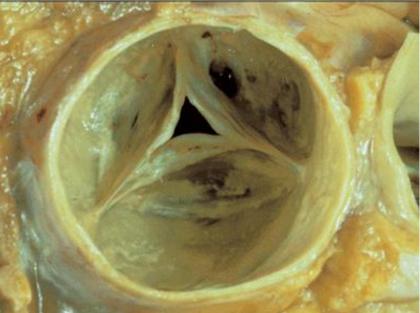
## Etiology of Al

- Causes:
  - Congenital abnormality
    - Bicuspid valve
  - Dilation of aortic root and ascending aorta
    - Aortic dissection
  - Infectious diseases
    - Rheumatic
    - Endocarditis

#### **Normal Closed Aortic Valve**

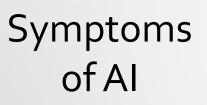
#### Non-Inflammatory Aortic Root Dilatation

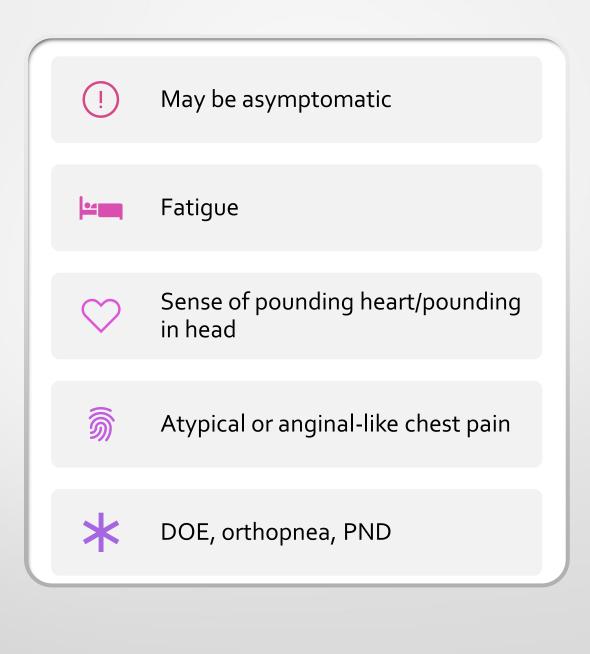




# **Causes of Aortic Insufficiency**

Leaflet abnormalities	Aortic root or ascending aorta abnormalities
Rheumatic fever	Systemic hypertension
Endocarditis	Aortitis (eg, syphilis)
Trauma	Trauma
Bicuspid aortic valve	Dissecting aneurysm
Marfan's syndrome	Ehlers-Danlos syndrome
Fenfluramin-	
phentermine	





# Physical Findings

#### Wide pulse pressure (SBP - DBP)

High systolic pressure due to large stroke volume

Low diastolic pressure due to rapid runoff

Rapid rise and fall of arterial pulses

Other findings due to hyperdynamic pulse

# Hyperdynamic Pulse Findings

- Corrigan's pulse "water-hammer" pulserapid rise and fall
- deMusset's sign head bob occurring with each heart beat.
- **Quincke's pulses** Capillary pulsations in fingernails
- **Mueller's sign** Systolic pulsations of the uvula.
- Rosenbach's sign Systolic pulsations of the liver.
- **Gerhard's sign** Systolic pulsations of the spleen.



## Al Murmur

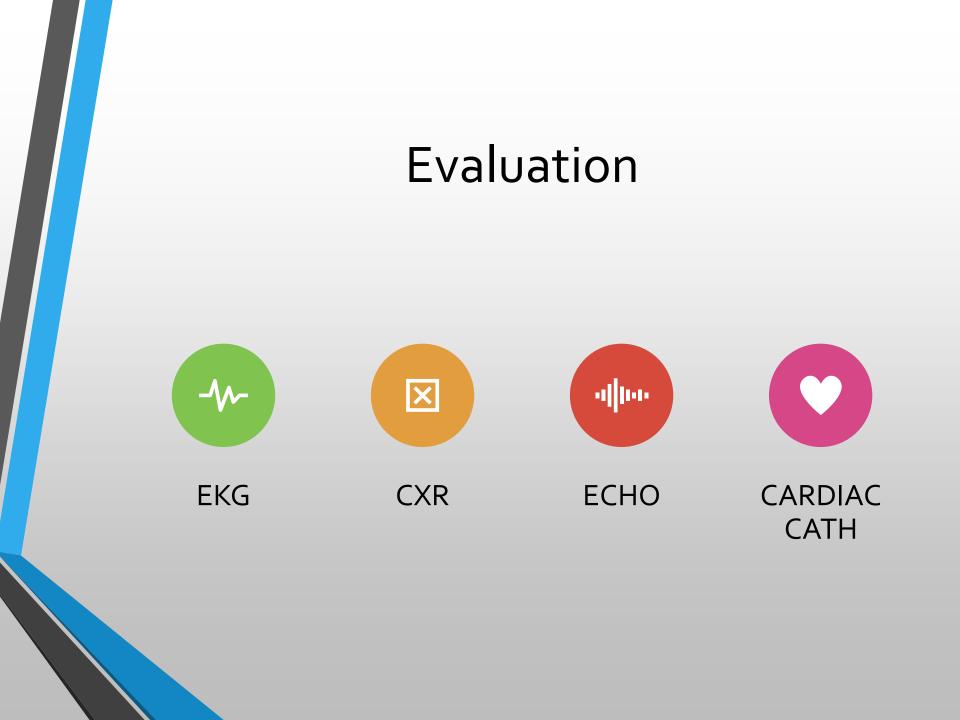
- Early diastolic murmur at lower left sternal border
- Blowing
- Sustained or decrescendo
- May be best heard with patient leaning forward and holding breath after exhalation
- Austin Flint murmur a second low-pitched diastolic murmur/"rumble" heard at the apex
- <u>Al murmur</u>

## Acute vs. Chronic Al

Chronic AI progresses over time so that the left ventricle has time to compensate by dilation and hypertrophy

#### Acute AI is a surgical emergency:

- Left ventricle has not had time to adapt
- Causes: Endocarditis, aortic root dissection, acute dysfunction of prosthetic valve, trauma
- Large regurgitant volume causes acute pulmonary edema
- Many physical exam findings are absent



# AORTIC INSUFFICIENCY

- Medical therapy-Vasodilators: Calcium channel blockers or ACE inhibitors for afterload reduction
  - Promotes forward flow in aorta
- Serial monitoring by echo of LV function
- Surgery- valve replacement if pt is
  - Symptomatic
  - has increasing LV size
  - decreasing LV function due to AI

# Summary

Causes of aortic insufficiency

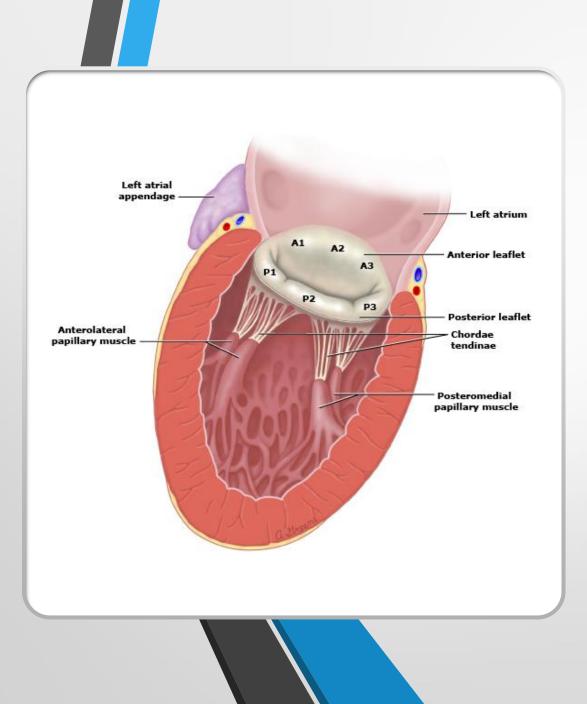
Aortic root dilation, bicuspid valve, rheumatic

## Symptoms

• "pounding" heart, atypical CP, pulmonary congestion

## **Clinical findings**

- Diastolic blowing murmur at the LLSB
- Possibly an S3
- Hyperdynamic pulses
- Treatment
  - Meds for vasodilation, Surgery



# MITRAL VALVE

## Etiology of MS

- Most have a rheumatic cause
  - Immobility and thickening of leaflets
  - Fusion of commissures
  - MS is not part of the clinical presentation of the first episode of acute rheumatic fever
- Rare cases of congenital abnormalities of MV
- Valvulitis (SLE or amyloid) or Infiltrative Dx

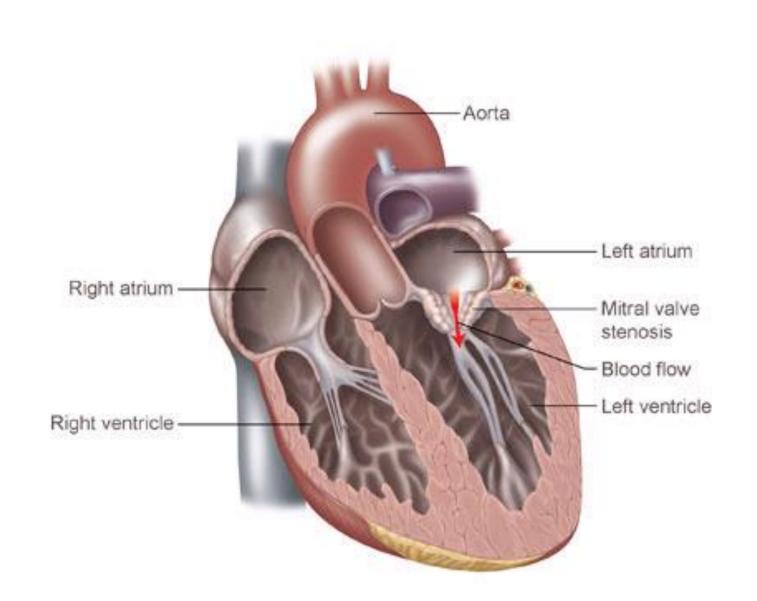
## Mitral Valve Stenosis

 Valvular obstruction results in increase in pressure in LA, pulm vasculature and right side of the heart

# Long Axis View

## of Normal Mitral Valve





## **Mitral Stenosis**

Looking Down on the Mitral and Tricuspid Orifice from the Atria



### **Mitral Stenosis**

Looking Down on a Mitral Valve with a Small Oval Aperture



- Dyspnea, orthopnea, PND
- Hemoptysis
- Chest pain
- Fatigue
- Atrial fibrillation

### Presentation

# Symptoms

- Interval between rheumatic fever and clinical manifestation of MS may be up to 20 yrs
- Mean interval between acute rheumatic fever and symptoms is 15-20 years
- Insidious onset of symptoms progression from mild to severe disability takes 8-9 years

### Physical Findings

- Opening snap follows S2
- Diastolic rumbling murmur heard best at the apex in left lateral decubitus position
  - Best heard with the bell
- <u>MS murmur</u>

### Evaluation



- Pharmacologic treatment
  - Diuretics to treat congestive symptoms
  - Maintenance of normal sinus rhythm-
    - Atrial fibrillation with RVR can be devastating and cardioversion is usually warranted

## Treatment

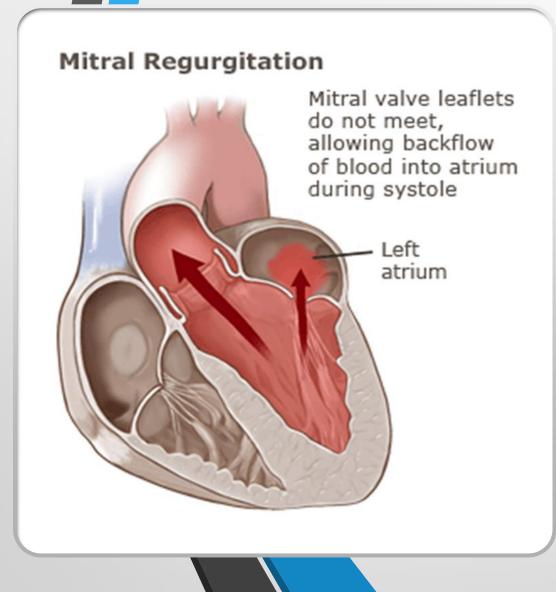
# Treatment

- Mitral valvuloplasty
  - Not a permanent fix
  - Echo criteria to determine suitability
    - Non-calcified, pliable leaflets
    - No significant mitral regurgitation
      - If present, valve replacement warranted
    - No LA thrombus
- MV replacement is the definitive treatment

# Summary

#### • Cause of mitral stenosis

- Usually rheumatic fever
- Exceedingly rare finding in western societies
- Symptoms
  - Dyspnea with slow, insidious onset
  - A-fib
- Clinical findings
  - Opening snap
  - Diastolic rumbling murmur
- Treatment
  - Surgery



# Mitral valve regurgitation

### Causes of MR

- Mitral valve prolapse is the most common cause of isolated severe MR
- Ischemia can cause dysfunction or rupture of papillary muscle, usually the posteromedial papillary muscle as it has a single blood supple (Posterior Descending Artery)

# Etiology

#### <u>Chronic</u>

- MVP
- LV dilation
- Posterior MI (scar)
- Rheumatic disease
- Endocarditis

#### <u>Acute</u>

- Posterior wall/ papillary muscle ischemia
- Rupture of chordae tendinae
- Endocarditis

#### Presentation

#### Chronic MR

- Prolonged asymptomatic period
- Fatigue/Dyspnea on exertion
- Atrial fibrillation
- Left heart failure: DOE, Orthopnea, PND
- Can lead to pulmonary hypertension and right heart failure

#### Acute MR

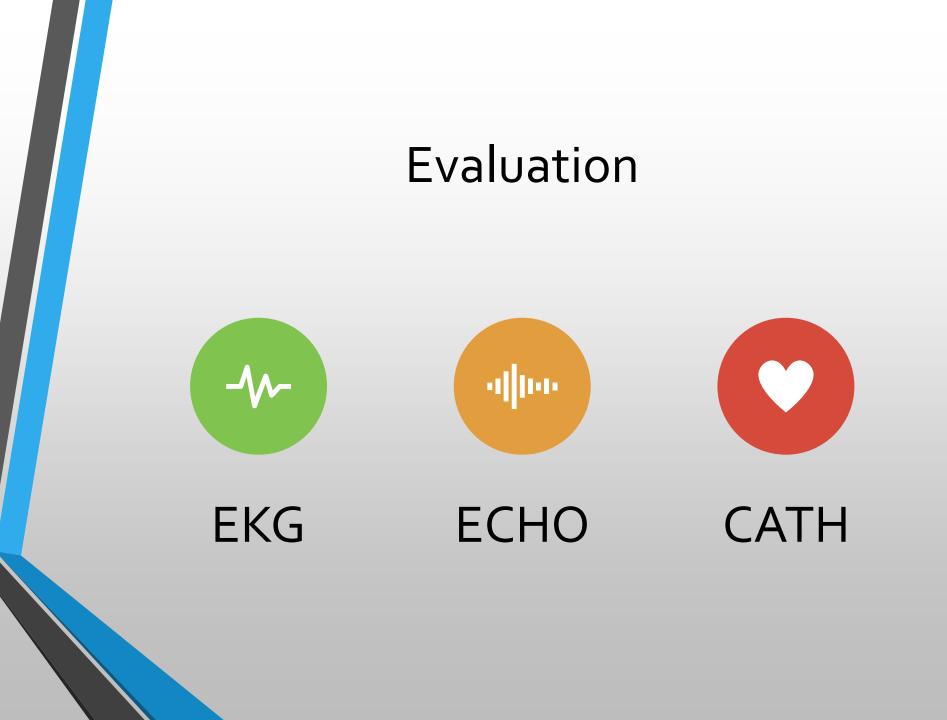
- Pulmonary edema
- Hypotension

### Physical Findings in MR

#### • Chronic:

- Blowing holosystolic murmur at apex radiating to the axilla and possibly the back
- S2 may be widely split
- Laterally displaced apical impulse (PMI)
- <u>MR murmur</u> (holosystololic)
- <u>MR murmur</u> (with a click)
- Acute MR
  - Systolic murmur may be short, soft or absent

S3



#### Management of Chronic MR

Often well tolerated

#### Afterload reduction-ACE inhibitors

Maintain sinus rhythm if possible

#### Decrease preloaddiuretics and nitrates

# Management of Chronic MR



- Serial echocardiograms in patients with moderate to severe MR – after EF falls, outcomes are worse
- Surgery indicated for symptomatic MR and reasonable EF
- Surgery indicated for asymptomatic patients if:
  - severe MR and EF < 55-60%</p>
  - Increase in LV end systolic dimension

#### MITRAL VALVE REGURGITATION

- Severe MR in 1-2% echocardiography
  - Primary
    - Intrinsic MV pathology
    - Degenerative MV disease is the most common cause
  - Secondary
    - Cardiomyopathy
    - Ischemic heart disease

 Acute severe MR is life-threatening with hemodynamic instability and requires rapid evaluation and surgery

# Management of Acute MR

- Treatment:
  - IV vasodilators for afterload reduction (nitroprusside)
  - Inotropes
  - IABP

# Summary

- MVP a most common cause also rheumatic disease
- Pt. may be asymptomatic or have classic "MVP" symptoms, additionally may have DOE, A-fib, CHF
- Commonly described as holosystolic murmur heard best at apex and lower left sternal border – may radiate to axilla
- Treatment reduce both preload and afterload (ACE and diuretics), surgery for symptomatic patients or asymptomatic patients with EF<55-60% or LV remodeling</li>

# THANK YOU!