Valvular Heart Disease:
Volume Versus Pressure and the Hemodynamic Compromise

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AHA / ACC Guidelines for the Management of Patients with Valvular Heart Disease

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Chronic Valve Disease

Basic Tenants

- Early recognition is key to treatment
- Identified through routine cardiac auscultation
- Compensatory changes occur over years to decades
- No treatment until symptoms or severe disease by echocardiogram
- Careful routine follow up

Recommendations for Echocardiography

Class I Recommendations

- Asymptomatic patients with diastolic murmurs, continuous murmurs, holosystolic murmurs, late systolic murmurs, murmurs associated with clicks or murmurs that radiate to the neck or back (Level of evidence: C)
- Patient with heart murmurs and symptoms or signs of heart failure, myocardial ischemia/infarction, syncope, thromboembolism, infective endocarditis, or other clinical evidence of structural heart disease. (Level of evidence: C)
- Asymptomatic patients with grade III or louder midpeaking systolic murmur (Level of evidence: C)
Cardiac Valves
“The Simple Facts”

Prevent Backward Flow  Permit Forward Flow

Valve Dysfunction

Impedance of forward flow

Backward flow of volume

Decreased Cardiac Output

Compensatory Changes
Cardiac Valves

- AV Valves
  - Tricuspid
  - Mitral (bicuspid)
- Semilunar Valves
  - Pulmonic
  - Aortic

AV (Atrioventricular) Valves

- Tricuspid
  - Lies between right atrium and right ventricle
  - Larger, but thinner
  - 3 cusps
- Mitral
  - Lies between the left atrium and left ventricle
  - Smaller
  - 2 cusps
AV Valves

- Fibrous Ring (Annulus)
- Cordae Tendineae
- Papillary Muscles

Semilunar Valves

- Pulmonic
  - Lies between right ventricle and pulmonary artery
- Aortic
  - Lies between left ventricle and aorta
Semilunar Valves

- Three cusps
- Annulus (ring)
- Commissures
- The leaflets are smaller and thicker than the AV valves
- Openings are smaller than the AV valves
- The velocity of ejected blood is higher than AV valves.

Normal Valve Function in Relation to the Cardiac Cycle
Cardiac Diastole (Atrial and Ventricular): Early Passive Ventricular Filling

Atrial Systole & Ventricular Diastole: Late Active Ventricular Filling

Atrial Kick
Beginning Ventricular Systole:
Isovolumic Contraction

Ventricular Systole:
Ejection
Heart Sounds - Basics

- **Systole**
  - Isovolumic contraction
  - Ejection of LV Contents
  - Valves Open:
    - Aortic
    - Pulmonic
    - Don’t open well
      - Stenosis
  - Valves Closed
    - Mitral
    - Tricuspid
    - Don’t close well
      - Regurgitation

- **Diastole**
  - Passive Ventricular Filling
    - S3
  - Active Ventricular Filling
    - Atrial Kick – S4
  - Valves Open
    - Mitral
    - Tricuspid
    - Don’t open well
      - Stenosis
  - Valves Closed
    - Aortic
    - Pulmonic
    - Don’t close well
      - Regurgitation

Basic Heart Sounds

**S1**

- Closure of the Mitral (M1) valve and the Tricuspid (T1) valve
- Beginning of Ventricular Systole and Atrial Diastole
- Location: Mitral area
- Intensity: Directly related to force of contraction
- Duration: Short
- Quality: Dull
- Pitch: High
Basic Heart Sounds

\( S_2 \)

- Closure of Aortic (A\(_2\)) and Pulmonic (P\(_2\)) Valve
- End of Ventricular Systole
- Location: Pulmonic area
- Intensity: Directly related to closing pressure in the aorta and pulmonary artery
- Duration: Shorter than \( S_1 \)
- Quality: Booming
- Pitch: High

Murmurs

- High blood flow through a normal or abnormal valve
- Forward flow through a narrowed or irregular orifice into a dilated chamber or vessel
- Backward or regurgitant flow through an incompetent valve
Systolic vs Diastolic Dysfunction

Systolic Dysfunction

- Impaired wall motion and ejection
- Dilated chamber
- 2/3 of HF Population
- Hallmark: Decreased LV Ejection Fraction < 40%
- Coronary artery disease is cause in 2/3 of patients
- Remainder – other causes

Enlarged atria

Normal heart
(cross section)

Enlarged ventricles

Dilated heart
Diastolic Dysfunction

- Filling impairment
- Normal chamber size
- 20 to 40% of patients with HF have preserved LV function
- Normal EF or elevated
- Caused by
  - Hypertension
  - Restrictive myopathy
  - Ischemic heart disease
  - Ventricular hypertrophy
  - Valve disease
  - Idiopathic

Diastolic Dysfunction

- Diagnosis is made when rate of ventricular filling is slow
- Elevated left ventricular filling pressures when volume and contractility are normal
Pathophysiology

Remember:
- Stenosis = Pressure
- Regurgitation = Volume

AORTIC STENOSIS

Obstruction of flow at the level of the aortic valve.
### Aortic Stenosis Classifications

<table>
<thead>
<tr>
<th>Congenital</th>
<th>Acquired</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Most common cause of AS in men under 70</td>
<td>• Most common form of AS in those &gt;70</td>
</tr>
<tr>
<td>• More prevalent in men</td>
<td>• RHD</td>
</tr>
<tr>
<td>• Abnormal number of valve cusps</td>
<td>• Senile Degenerative Calcification</td>
</tr>
<tr>
<td>• Symptoms appear between age of 40 and 60</td>
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</tbody>
</table>

### Aortic Stenosis Classification

- RHD
  - Fibrosis of valve leaflets with commissure fusion
  - Occasionally calcification
  - Fibrotic leaflets unable to open fully
  - RHD impacts mitral valve as well
Aortic Stenosis

Classification

• Senile Degenerative Calcification
  • Most common for AS patients >70
  • Calcified nodules on valve leaflets
  • Thickened leaflets
  • Concomitant aortic regurgitation not uncommon
  • More common in men
  • Most common reason for AVR
  • Higher incidence in patients with:
    • elevated lipoprotein and low-density lipoprotein cholesterol levels.
    • hypertension.
    • diabetes.
    • elevated serum calcium levels.
    • elevated serum creatinine levels.

Aortic Stenosis

Pathophysiology
Compensatory Mechanisms

Aortic Valve Orifice Narrows

$\rightarrow$ ____Afterload

$\rightarrow$ ____LV Workload

$\rightarrow$ ____LV Wall Mass

$\rightarrow$ ____LV Hypertrophy

$\rightarrow$ ___________ Dysfunction

Works well for years – even decades.

Compensatory system ultimately fails $\rightarrow$ Symptoms

Aortic Stenosis
Pathophysiology
Compensatory Mechanisms

Aortic Valve Orifice Narrows

- ↑ Afterload
- ↑ LV Workload
- ↑ LV Wall Mass
- ↑ LV Hypertrophy
- **Diastolic** Dysfunction

Works well for years – even decades.
Compensatory system ultimately fails ➜ Symptoms

http://www.marvistavet.com/assets/images/aortic_stenosis.gif
Aortic Stenosis

Symptoms

• Classic Triad
  • Angina
  • Syncope
  • Heart Failure

Angina

• Left ventricular hypertrophy
• Imbalance in supply and demand
Syncope

- Normal hemodynamic response to exercise
  - Arterial vasodilatation – decreased systemic vascular resistance
  - Increased heart rate will increase cardiac output

- Hemodynamic response to exercise with aortic stenosis
  - Arterial vasodilatation – decreased systemic vascular resistance
  - Increased heart rate does not work to increase cardiac output -> syncope

Congestive Heart Failure

- Diastolic Failure
  - Concentric Hypertrophy

- Poor fill
  - Small chamber

- Systolic failure
  - Pump fails
### Aortic Stenosis

#### Signs (Examination)

- In addition to classic triad:
  - Decreased pulse sharpness
  - Systolic Ejection Murmur
  - S4

#### Systolic Ejection Murmur

- May be present before any significant hemodynamic changes occur
- More severe AS $\rightarrow$ longer murmur
- **Timing**: Midsystolic
- **Location**: Best heard over aortic area
- **Radiation**: Toward neck and shoulders
  - May radiate to apex
- **Configuration**: Crescendo
- **Pitch**: Medium to high
- **Quality**: Harsh
- D:\Track03.cda
**S₄ - Atrial Gallop**

- Late diastolic filling sound
- Caused by atrial contraction and the propulsion of blood into a noncompliant (stiff) ventricle.
- Most frequently associated with diastolic dysfunction
- Associated with:
  - Fluid overload state
  - Systemic hypertension
  - Restrictive cardiomyopathy
  - Ischemia
  - Aortic stenosis
  - Hypertrophic cardiomyopathy
- May be normal in athletes
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**Left or Right Sided S₄**

- Patient position: left lateral decubitus position.
- Location
  - Left-sided S₄ – mitral area.
  - Right-sided S₄ – tricuspid area.
- Intensity
  - Left-sided louder on expiration.
  - Right-sided louder on inspiration.
- Duration: Short
- Quality: Thud like
- Pitch: Low
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Diagnosis
Cardiac Echocardiography

- Primary tool utilized to confirm the diagnosis of aortic stenosis
- Quantifies severity of stenosis
- Evaluation of **Pressure Gradient**
  - Pressure difference from one side of the valve to the other.
  - As disease progresses peak aortic systolic pressure becomes lower than peak left ventricular pressure
  - **Mild AS** Mean Gradient < 25 mmHg
  - **Moderate AS** Mean Gradient 25-40 mmHg
  - **Severe AS** Mean Gradient > 40 mmHg

Cardiac Echocardiography

- Evaluation of **Valve Area**
  - Normal valve area ranges from 3 cm² to 4 cm²
  - Change in flow across the valve occurs when valve area is one fourth the normal size
  - **Normal** Valve area 3.0-4.0 cm²
  - **Mild AS** Valve area >1.5 cm²
  - **Moderate AS** Valve area 1.0 to 1.5 cm²
  - **Severe AS** Valve area <1.0 cm²

- Evaluation of **Jet Velocity**
  - Directly related to valve gradient and valve area
  - Increases as gradient increases and valve area decreases
  - **Mild AS** <3.0 m/second
  - **Moderate AS** 3.0-3.0 m/second
  - **Severe AS** >4.0 m/second
Diagnosis
Cardiac Echocardiography

- Evaluation of **Left Ventricular Function** for:
  - Concentric Hypertrophy
  - Left Ventricular Chamber Size
  - Diastolic Dysfunction
- Evaluation of ** Valve Leaflets** for
  - **Thickening** of valve leaflets
  - Decreased **mobility** of valve leaflets
  - **Calcification** of valve cusps

Diagnosis

- **Stress Test**
  - Assess for exercise capacity
  - Determine abnormal blood pressure response to exercise
  - Induce symptoms
  - NOT INDICATED in symptomatic patients (Class III)
  - OK in asymptomatic patients with severe AS to induce symptoms (Class Iib)

- **Cardiac Cath**
  - Verify absence/presence of CAD before AVR (Class IB)
  - Evaluate pressure gradients (Class 1C)
  - Not recommended to assess severity of disease when noninvasive test are adequate (Class III)
  - Injection of dye ➔ arterial vasodilatation

- **ECG**
  - Left Ventricular Hypertrophy
  - LBBB

- **Chest x-ray**
  - Heart: Normal size
  - Concentric hypertrophy: rounded left ventricular border
Medical Treatment

- Rarely needed
- HR/rhythm control
- ACE Inhibitors: Not in severe AS
  - Development of hypotension and syncope
- Nitroglycerin: With Caution
  - Low dose: Impact on preload
  - High dose: Impact on afterload
- Beta blockers: Contraindicated in severe AS
  - Blocks normal adrenergic response of increased HR
- Statin use in calcific aortic stenosis

Medical Treatment

- Volume Management
  - Precarious
- Exercise
  - No restriction in asymptomatic mild AS
  - Asymptomatic patients with moderate or severe AS
    - Avoid competitive sports
    - Evaluate tolerance to exercise per stress test
- Continuous physician follow up
  - Annual exams
    - History and physical
    - Serial echocardiogram
- Endocarditis prophylaxis
  - 2007 AHA Guidelines
Endocarditis Prophylaxis

**ALL Patients with Valve Disease**

- Only for those with the highest risk for the development of infective endocarditis.
- Population at the highest risk for the development of infective endocarditis:
  - 1. Prosthetic cardiac valve
  - 2. Previous infective endocarditis
  - 3. Congenital heart disease
  - 4. Cardiac transplant recipients who develop cardiac valvulopathy

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Severe Aortic Stenosis Prognosis

*Figure 8-5.* The natural history of aortic stenosis. There is little change in survival until the symptoms of angina, syncope, or heart failure develop. Then the decline is precipitous. Adapted, with permission, from Ross J Jr, Braunwald E. *Circulation* 1966;38(Suppl IV):41.
### ACC/AHA Recommendations for Aortic Valve Replacement (AVR) in Aortic Stenosis

<table>
<thead>
<tr>
<th>Class</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class I</strong></td>
<td>Symptomatic patients with severe AS.</td>
</tr>
<tr>
<td></td>
<td>Severe AS undergoing coronary artery bypass graft surgery</td>
</tr>
<tr>
<td></td>
<td>Severe AS undergoing surgery on the aorta or other heart valves.</td>
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<tr>
<td></td>
<td>Severe AS and LV systolic dysfunction (ejection fraction less than 0.50).</td>
</tr>
<tr>
<td><strong>Class IIa</strong></td>
<td>Moderate AS undergoing CABG or surgery on the aorta or other heart valves.</td>
</tr>
</tbody>
</table>

**Class IIb Recommendations**

- Asymptomatic patients with severe AS and abnormal response to exercise.
- Severe asymptomatic AS if there is a high likelihood of rapid progression.
- In patients undergoing CABG who have mild AS when there is evidence, such as moderate to severe valve calcification, that progression may be rapid.
- Asymptomatic patients with extremely severe AS when the patient’s expected operative mortality is 1.0% or less.
Mechanical Valve

**ALL** Valve Replacements

- More durable than tissue valves
- Require life-long anticoagulation
- Post-op mortality related to valve
  - Thromboembolism, hemorrhage,
  - Endocarditis, periprosthetic leak
- Ball and cage
- Single Leaflet Tilting Disc
- Bileaflet Valve

Tissue Valves

**ALL** Valve Replacements

- Bioprosthetic
- Less durable than mechanical valves
- Do not have risk of mechanical failure
- No anticoagulation with warfarin required
- Homograft (Allograft)
- Heterograft (Xenograft)
  - Stented
  - Stentless
### Surgical Treatment Options

- AV Replacement with mechanical or bioprosthetic valve
- Aortic Valve Repair

### Aortic Valve Replacement

- Replacement is procedure of choice
- Mortality increase as LV dysfunction decreases
- Bileaflet valve most common choice for mechanical valve in aortic position
- Stented heterograft most common aortic valve prosthetic in US
- Homograft common with total aortic root repair
Aortic Valve Repair

- Limited use
- Decalcification of valve
- High rate of recalcification and restenosis
- Repair of aortic root may repair aortic valve
- No need for anticoagulation

Percutaneous Balloon Valvotomy

- Balloon placed across aortic valve
- Fractures calcium deposits in the leaflets
- Separate fused or calcified commissures
- Considered palliative in the aortic position (Class IIB)
- May be used as a bridge to surgery (Class IIB)
- All benefit gone in 6 months
- Development of or increased severity of AR
  - Not appropriate if AR > 2+ prior to procedure
Percutaneous Aortic Valve Replacement

• The Future
  • Percutaneous aortic valve replacement

Anticoagulation For All Valve Replacements

• Mitral valve replacement highest risk for embolization
• Mechanical Valve
  • Warfarin for all patients
  • Warfarin and ASA if high-risk for thromboembolism
  • Consider warfarin and clopiogrel if allergy to ASA
• Tissue Valve
  • Warfarin for 3 months post op in all patients (especially MVR)
  • ASA for all patients with no risk for thromboembolism
Endocarditis Prophylaxis
For All Valve Replacements

- Appropriate for all patients with prosthetic valves
- Procedures requiring prophylaxis
  - Dental procedures involving manipulation of gingival tissue or periapical region of teeth or perforation of oral mucosa
    - Biopsies
    - Sutures – placement or removal
    - Placement of orthodontic bands
  - Respiratory procedures
    - Involving incision or biopsy of respiratory mucosa
- No longer required
  - GI or GU procedures
- Single dose 2 hours prior to procedure
- Amoxicillin or cephalexin (if penicillin or amoxicillin allergy)
- KEY TO PREVENTION – Good oral hygiene

Post Operative Considerations

- LV Hypertrophy regresses
- Diastolic dysfunction never returns to normal
- Patient experiences improvement immediately after surgery
- Follow Up (common to all valve surgeries)
  - Repeat echocardiogram for patients with tissue valve
    - Annually after 1st 5 years even if no symptoms
  - Repeat echocardiogram for patients with mechanical valve if:
    - Evidence of prosthetic valve dysfunction
    - Evidence of LV dysfunction
    - Evidence of new murmur
    - Change in clinical status
Aortic Regurgitation
(Insufficiency / Incompetence)

- Occurs when valve cusps do not close completely and blood is allowed to travel retrograde through the valve during ventricular systole.

Aortic Valve and Aortic Recoil
Pathophysiology

Remember:
Stenosis = Pressure
Regurgitation = Volume

Aortic Regurgitation
Pathophysiology
Compensatory Mechanisms In Aortic Regurgitation

During diastole there is backward filling of the LV from the aorta and forward filling from the left atrium

- ___LV Volume
  - LV ____________ (size)
    - LV ____________ (muscle mass)
  - stroke volume __________

Works well for years – even decades. Compensatory system ultimately fails -> _________ Dysfunction

- Heart Failure

Aortic Regurgitation Pathophysiology
Compensatory Mechanisms In Aortic Regurgitation

During diastole there is backward filling of the LV from the aorta and forward filling from the left atrium

- \( \uparrow \) LV Volume
- LV \textbf{dilates} (size)
- LV \textbf{hypertrophies} (muscle mass)
- \( \rightarrow \) stroke volume \textbf{increases}

Works well for years – even decades.
Compensatory system ultimately fails

- \( \rightarrow \) \textbf{Systolic} Dysfunction \( \rightarrow \) Heart Failure

Aortic Regurgitation Classification

<table>
<thead>
<tr>
<th>Chronic</th>
<th>Acute</th>
</tr>
</thead>
<tbody>
<tr>
<td>• RHD</td>
<td>• Trauma</td>
</tr>
<tr>
<td>• Congenital</td>
<td>• Acute infective endocarditis</td>
</tr>
<tr>
<td>• Infective endocarditis</td>
<td>• Acute aortic dissection</td>
</tr>
<tr>
<td>• Marfan’s</td>
<td></td>
</tr>
<tr>
<td>• Inflammatory diseases</td>
<td></td>
</tr>
<tr>
<td>• Syphilis</td>
<td></td>
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<tr>
<td>• Severe systemic Hypertension</td>
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</tbody>
</table>
**Chronic Aortic Regurgitation**

**Symptoms**

- Exertional dyspnea
- PND
- Orthopnea
- Angina
- Aware of heart beat – especially when lying
- Pulsatile sensation in head

**Physical Examination**

- Apical Impulse
- Diastolic Murmur of AR
- Systolic Flow Murmur
- Austin Flint Murmur
- Signs of Hyperdynamic Perfusion
**Chronic Aortic Regurgitation**  
**Physical Examination**

<table>
<thead>
<tr>
<th>Event</th>
<th>Details</th>
</tr>
</thead>
</table>
| Diastolic Murmur of AR | Length of murmur correlates severity of AR  
Timing: Early diastole  
Location: left sternal boarder  
3rd, 4th ICS  
Radiation: Towards apex  
Configuration: Decrescendo  
Pitch: High  
Quality: Blowing  
Patient Position: sitting and leaning forward at end expiration  
Intensity: Increases with increased peripheral vascular resistance  
Squatting, exercising, hand gripping  
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<table>
<thead>
<tr>
<th>Systolic Flow Murmur</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Result of turbulent flow across valve during systolic</td>
<td></td>
</tr>
</tbody>
</table>
Large volumes of blood from hyperdynamic perfusion causes turbulence  
Timing: Mid systolic  
Location: Along left sternal boarder  
Configuration: Crescendo-decrescendo  
Pitch: Medium (best with diaphragm)  
Quality: Soft  
Intensity: May increase after coughing or when elevating legs while in lying position |
Austin Flint Murmur

- Very severe chronic AR or acute AR
- Diastolic murmur: functional mitral stenosis
- Severe AR
  - blood flow back through the aortic valve
  - regurgitant volume presses on open anterior leaflet of mitral valve
  - moves the leaflet towards the closed position
  - functional Mitral Stenosis
- Timing: Mid diastolic
- Location: cardiac apex
- Configuration: Plateau
- Pitch: Low pitch
- Quality: Rumbling
- Intensity: Soft

Signs of Hyperdynamic Perfusion

- Warm, flushed, reddish mucous membranes
- Wide pulse pressure (>100mmHg)
- De-Musset Sign
  - Head bobbing with each heart beat
- Water-Hammer pulse
  - Rapid rise and collapse of the pulse upon palpitation
- Corrigan’s Pulse
  - Large carotid pulsation in the neck
- Traube’s Sign
  - Loud, sharp “pistol-shot-like” sound heard over the femoral pulse
- Duroziez’s Sign
  - Murmur heard over the femoral artery when compressed
- Quinke’s Sign
  - Pulsatile blanching and reddening of the fingernails when light pressure is applied
## Acute Aortic Regurgitation

### Pathophysiology

- Sudden decrease in cardiac output
- Increased LV afterload (increases regurgitation)
- Increased LV preload
- Pulmonary edema, cardiogenic shock and acute decompensation

![Diagram of heart](image)

### Diagnosis

#### Cardiac Echocardiogram

- Emergently for acute cases
- Determination of **Grade of Severity**
  - **Mild AR**  
    - 1+
  - **Moderate AR**  
    - 2+
  - **Severe AR**  
    - 3-4+
- Evaluation of **Regurgitant Jet Width**
  - **Mild AR**  
    - Width < 25% of LV outflow track
  - **Moderate AR**  
    - Width > mild but no signs of severe AR
  - **Severe AR**  
    - Width > 65% of LV outflow track
- **Regurgitant Volume**  
  - Evaluation of (amount returned to LV each beat)
  - **Mild AR**  
    - < 30 ml / beat
  - **Moderate AR**  
    - 30-59 ml / beat
  - **Severe AR**  
    - > 60 ml / beat
Diagnosis
Cardiac Echocardiogram

- Evaluation of Left Ventricular Function
  - Eccentric Hypertrophy
  - Concentric Hypertrophy
  - Left ventricular ejection fraction
    - Assessment for systolic dysfunction
  - *EF evaluates that which is ejected*

- Evaluation of Valve Leaflets
  - Valve thickening
  - Loss of coaptation of commissures
  - Presence of vegetation on the valve
  - Dilation of aortic root

Aortic Regurgitation - Diagnosis

- Stress Testing
  - Reasonable for assessment of functional capacity and symptomatic response in patients with history of equivocal symptoms (Class IIIB)
  - Reasonable for evaluation of symptoms and functional capacity before participation in athletic activities (IIC)

- Cardiac Cath: Verify absence or presence of CAD (Class I C)

- ECG
  - Increased QRS voltage with LV hypertrophy
  - Left axis deviation may be present with moderate/severe AR

- Chest x-ray
  - Enlarged heart (Cow’s Heart)
  - Failure in chronic decompensated AR
  - Failure in acute AR
### Chronic Aortic Regurgitation

#### Medical Treatment

- If normal LV function no treatment
- Arterial Vasodilators in symptomatic patients with severe AR and symptoms of LV dysfunction and not a surgical candidate (Class IB)
  - Symptom relief preop (Class IIB)
  - Decrease afterload ➔ decrease regurgitation
  - Not indicated in asymptomatic patients (Class III)
- Digoxin and diuretics helpful with HF symptoms
- Avoid arterial vasoconstrictors
- Intra-aortic balloon pump
  - *Contraindicated* in all patients with AR
- Continuous physician follow up
  - Annual exams
    - History and physical
    - Serial echocardiogram

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### Acute Aortic Regurgitation

#### Treatment

- Urgent Surgical Intervention
- STAT ECHO
- Reduce afterload
  - Nitroprusside
- Reduce preload
  - Help reduce fluid overload
- Beta blockers
  - With caution
  - Block sympathetic response of increased HR
- Inotropes
  - Increase contractility for forward flow
Surgical Treatment

• Mortality rates increase as EF decreases
• Once symptomatic 50% will not survive > 3-5 years without surgery
• Valve repair reasonable alternative to replacement in this population
• Valve replacement options the same as with AS
• Goal should be quality of life not longevity
• Looking for symptom relief
• Acute AR requires acute intervention

ACC/AHA Recommendations for Aortic Valve Replacement / Repair (AVR) in Aortic Regurgitation

• Class I Recommendations
  • Symptomatic patients with severe AR irrespective of LV systolic function.
  • Asymptomatic patients with chronic severe AR and LV systolic dysfunction (ejection fraction ≤ 0.50).
  • Patients with chronic severe AR while undergoing CABG or surgery on the aorta or other heart valves.

• Class IIa Recommendations
  • Asymptomatic patients with severe AR with normal left ventricular systolic function but with severe left ventricular dilatation.
ACC/AHA Recommendations for Aortic Valve Replacement / Repair (AVR) in Aortic Regurgitation

- Class IIb Recommendations
  - Moderate AR while undergoing surgery on the ascending aorta.
  - Moderate AR while undergoing CABG.
  - Asymptomatic patients with severe AR and normal left ventricular function at rest when the degree of dilatation exceeds an end-diastolic dimension of 70mm or end-systolic dimension of 50 mm, when there is evidence of progressive left ventricular dilatation, declining exercise tolerance, or abnormal hemodynamic responses to exercise.

Aortic Regurgitation Outcomes

- LV function improves within first 10-14 days
- LV Function may improve for up to 2 years
- If LV function does not improve or only minimally then symptomatic treatment will be required.
- Endocarditis the same as with AS
- Anticoagulation the same as with AS
- Follow up the same as with AS
Mitral Valve Disease

Normal Function of Mitral Valve

- Annulus
- Leaflets
- Chordae Tendineae
- Papillary Muscles
Normal Mitral Valve

Mitral Valve Regurgitation

- Valve cusps do not close completely
- Blood travels retrograde through the valve during ventricular systole
Classification of MR

- **Organic Processes**
  - Involve the structure of the valve itself
  - Mitral Valve Prolapse
  - Rheumatic Heart Disease
  - Infective Endocarditis
  - Collagen Vascular Diseases

- **Functional Abnormalities**
  - Changes in other structures
  - Results in changes in the valve function
  - Left ventricular or atrial dilatation
  - Papillary muscle ischemia

Mitral Valve Prolapse

Pathophysiology

- Most common form of valvular heart disease
- Most commonly effects young women
- Abnormal function in MVP
  - Lengthened chordae tendineae cannot keep the valve leaflet in its proper place
  - Valve leaflet is forced into the atrial chamber.

**MVP DOES NOT = MR**
Mitral Valve Prolapse

Changes with Rheumatic Heart Disease

- Valve leaflets become fibrotic and shorten
- Inflammation of valve may also lead to calcification
- Leaflets become stiff
- Remain in fixed open position.
Other Changes

Bacterial Endocarditis
- Organic
  - Vegetation growing on leaflets prevent proper closure
- Functional
  - Causes chordae tendineae or papillary muscle dysfunction
  - Chordae tendineae can rupture
  - Papillary muscle can rupture

Changes in the size of the left atrium or left ventricle
- Affects the mitral valve annulus
- Dilatation of the left ventricular or the left atrium stretches the mitral annulus
- Stretching of the annulus prevents leaflets from closing properly
Pathophysiology

- Remember:
- Stenosis = Pressure
- Regurgitation = Volume
Compensatory Mechanisms In Mitral Regurgitation

During systole as the LV contracts blood is ejected from left ventricle through the open aortic valve AND some is diverted retrograde through dysfunctional mitral valve

- ______ left atrial volume and pressure AND
- left atrium responds by ________
  - atrium sends ________ volume to ventricle
  - LV adjusts by ____________ AND
  - LV increases ____________ to assure forward flow

Works well for years – even decades.
Compensatory system ultimately fails ______ Dysfunction

- Symptoms
Mitral Regurgitation
Pathophysiology

During systole as the LV contracts blood is ejected from left ventricle through the open aortic valve AND some is diverted retrograde through dysfunctional mitral valve.

- ↑ left atrial volume and pressure AND
- left atrium responds by dilating
  - atrium sends ↑ volume to ventricle
  - LV adjusts by dilating AND
  - LV increases contractility to assure forward flow

Works well for years – even decades.
Compensatory system ultimately fails → Systolic Dysfunction
- Symptoms
Clinical Presentation

- Remain asymptomatic for years
- Most frequent
  - Fatigue
  - Dyspnea on exertion
- Progress to include
  - Paroxysmal nocturnal dyspnea
  - Orthopnea
  - Palpitations from atrial fibrillation
- Initial diagnosis sometimes made with new onset AF
- Mitral valve prolapse patients early on report symptoms of tachycardia, orthostatic hypotension or panic attacks

Physical Exam

- Increased heart rate with atrial fibrillation or heart failure
- Pulse pressure narrows with decreased stroke volume
- Decreased carotid pulse volume
- Apical impulse displaced due to the dilation of the left ventricle
- Signs of heart failure
- Signs of pulmonary hypertension – advanced disease
- Systolic Murmur of Mitral Regurgitation
- S3
- Large V Waves on RA or PAWP trace
Systolic Murmurs
Mitral Regurgitation

- **Timing:** Holosystolic
- **Location:** Mitral area
- **Radiation:** To the left axilla
- **Configuration:** Plateau
- **Pitch:** High
- **Quality:** Blowing, harsh or musical
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Left or Right Sided S₃

- **Patient position:** left lateral decubitus position
- **Location:**
  - Left-sided S₃ – mitral area.
  - Right-sided S₃ – tricuspid area.
- **Intensity**
  - Left-sided heard best during expiration.
  - Right-sided heard best during inspiration.
- **Duration:** short.
- **Quality:** dull, thud like.
- **Pitch:** low.
- May be normal in children, young adults (up to 35–40) and in the 3rd trimester of pregnancy.
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"I Believe"
Large V Waves

PCWP Tracing with Large V Waves

Acute Mitral Regurgitation
Pathophysiology

• Acute MI
  • Impairment or rupture of a papillary muscle
  • Damaged to myocardial wall → damage to attachment of the papillary muscle to that ventricular wall
  • Papillary muscle continues to contract with each cardiac cycle
  • Attachment of papillary muscle to ventricular wall becomes weaker with each contraction
  • With enough damage to the myocardial wall or papillary muscle the papillary muscle will actually disconnect from the ventricular wall
  • Acute mitral regurgitation state
  • Emergency measures are necessary to preserve the patient’s life
Acute Mitral Regurgitation

Acute decrease in cardiac output

- ↑ SVR
  - blood flow to area of least resistance (through non-functional MV)
  - ↓ cardiac output (forward flow) and ↑ atrial volume (fluid overload)

- ↑ SVR and symptoms of volume overload
  - blood flow to area of least resistance
  - ↓ cardiac output (forward flow) and ↑ atrial volume (fluid overload)
  - acute pulmonary edema & shock
Diagnosis
Cardiac Echocardiogram (TEE)

- Primary tool utilized to confirm diagnosis
- Determine **Grade of Severity**
  - Mild MR 1+
  - Moderate MR 2+
  - **Severe MR** 3-4+
- Evaluation of **Regurgitant Volume**
  - Mild  < 30 ml / beat
  - Moderate 30-59 ml / beat
  - **Severe** > 60 ml / beat
- Evaluate **Regurgitant Jet**
  - Mild MR Width < 4CM² or <20% of LA area
  - Moderate MR Width > mild but no sign of severe MR
  - **Severe MR** Width > 40% of Left Atrial area or with a wall impinging jet of any size, swirling in left atrium

Diagnosis
Cardiac Echocardiogram

- **Evaluation of Valve** for:
  - Thickening valve leaflets
  - Loss of coaptation
  - Presence of vegetation on valve
  - Papillary muscle dysfunction
  - Lengthening chordae tendineae
- Evaluation of Left Ventricle for:
  - Eccentric Hypertrophy (dilated left ventricle)
  - Left ventricular ejection fraction – systolic function
    - **EF evaluates that which is ejected**
- Evaluation of Left Atrium for
  - Left atrial **dilation**
Diagnosis

• Stress Test
  • Not helpful in diagnosis
• Cardiac Cath
  • Assessment for CAD
  • Right heart cath helpful in assessing pulmonary hypertension
• Chest X-ray
  • Enlarged left atrium
  • Enlarged left ventricle
  • Enlarged right ventricle of PHTN present
• ECG
  • Left atrial hypertrophy
  • Left ventricular hypertrophy
  • AF

Medical Treatment

• No treatment for asymptomatic patient with normal ventricular function
• Continuous physician follow up
  • Annual exams
    • History and physical
    • Serial echocardiogram
• Rhythm Control
  • Atrial fibrillation
• Anticoagulation in patients
• ACE Inhibitors
  • Useful in non-surgical candidates
  • No benefit in asymptomatic patients
**Treatment for Acute MR**

- STAT Echo
- Surgery emergently
- IABP
- Afterload Reduction
  - Nitroprusside
- Antibiotics

**Surgical Treatment**

- EF < 60% considered abnormal
- Surgical options include:
  - Mitral valve repair
  - Mitral valve replacement with preservation of mitral apparatus
  - Mitral valve replacement with removal of mitral apparatus
- Mortality rates in those >75 higher with mitral valve surgery than aortic valve
- Mortality rates less with repair than replacement
### ACC/AHA Recommendations for Mitral Valve Repair /Replacement in Mitral Regurgitation

#### Class I Recommendations
- Symptomatic patients with acute severe MR.
- Chronic severe MR and NYHA functional class II, III, or IV symptoms in the absence of severe LV dysfunction.
- Asymptomatic patients with chronic severe MR and mild to moderate LV dysfunction (ejection fraction 30%-60%).
- Repair is recommended over replacement for the majority of patients with severe chronic MR who require surgery, and patients should be referred to surgical centers experienced in MV repair.

#### Class IIa Recommendations
- Repair in experienced surgical centers for asymptomatic patients with chronic severe MR and preserved LV function (ejection fraction >60% and end-systolic LV dimensions > 40 mm) in whom the likelihood of successful repair without residual MR is > 90%.
- Asymptomatic patients with chronic severe MR, preserved LV function and new onset atrial fibrillation.
- Asymptomatic patients with chronic severe MR and preserved LV function and pulmonary hypertension.
- Chronic severe MR due to a primary abnormality of the mitral apparatus and NYHA functional class III-IV symptoms and severe LV dysfunction (ejection fraction <30% and/or end-systolic dimension > 55 mm in whom MV repair is highly likely.)
Mitral Valve Repair

• Operation of choice
• Avoids anticoagulation
• Preserves natural function
  • Preserves shape and function of LV
• Technically complex
• Surgeon expertise directly related to success
• Longer pump time

Annular ring placement for dilated annulus
Chordae Repair

Mitral Valve Clip - Evolve
Mitral Valve Replacement

- Preservation of part or all of the apparatus
- Better post operative outcomes
- Improved mortality over replacement
- Requires prosthetic valve
- Removal of mitral apparatus
  - Required if native apparatus is damaged beyond the ability to repair
Outcomes

- Symptoms improve post operatively if no LV dysfunction
- Those with MVP have best outcomes
- Endocarditis the same as with AS
- Anticoagulation the same as with AS
- Follow up the same as with AS

Mitral Valve Stenosis

- Mitral Valve no longer opens normally
- Causing an obstruction of blood flow from the left atrium to the left ventricle
### Causes

- **Rheumatic Heart Disease**
  - Fibrosis and calcification of the valve leaflets
  - Valve commissures fuse together
  - Chordae tendineae thicken and shorten
  - Combination of some or all of these things results in a valve orifice that is much smaller than normal
  - Normal mitral valve area is 4.0 to 5.0cm²

- **Other causes**
  - Rare
  - Congenital mitral stenosis
  - Atrial myxoma
  - Systemic lupus erythematosus
  - Bacterial endocarditis

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### Mitral Stenosis

**Pathophysiology**
Normal Compensatory Mechanisms in Mitral Stenosis

Valve opening narrows
- Passive filling from left atrium to left ventricle ________________
- Left atrial pressure ________ in attempt to maintain normal flow across the valve
  - ________ left atrial pressure transferred back to the pulmonary vascular bed
- Pulmonary pressures subsequently _________
- Left atrium ________ as forward flow ________
- More difficult to empty atrium
- Chronic _________ in left atrial pressure
- Pulmonary hypertension develops

Compensatory system ultimately fails ➔ Right ventricular failure

Mitral Stenosis
Pathophysiology
Normal Compensatory Mechanisms in Mitral Stenosis

Valve opening narrows
- Passive filling from left atrium to left ventricle **slows**
- Left atrial pressure **↑** in attempt to maintain normal flow across the valve
  - ↑ left atrial pressure transferred back to the pulmonary vascular bed
  - Pulmonary pressures subsequently **↑**
  - Left atrium **↑** in attempt to maintain normal flow **slows**
- More difficult to empty atrium
  - Chronic **↑** in left atrial pressure
  - Pulmonary hypertension develops

Compensatory system ultimately fails ➔ Right ventricular failure

Symptoms

- 40 years before symptoms develop
  - Valve area ½ normal (2.0 -2.5cm$^2$)
- 10 more years for the development of serious symptoms
- Symptoms at rest
  - Valve area < 1.5cm$^2$
### Symptoms

- Dyspnea with exertion
- Pulmonary symptoms increase
- Development of orthopnea and paroxysmal nocturnal dyspnea
- Valve orifice less than 1.0 cm²
  - Dyspnea at rest
  - Confined to the bed or chair
- Develop cough and hemoptysis
- Ultimately RV Failure

### Symptoms

- Often discovered with conditions that increase heart rate
  - Pregnancy
  - New onset atrial fibrillation
  - Hyperthyroidism
  - Fever
- Stroke
  - Enlarged atrium
  - High risk for development of thrombi
- Atrial Fibrillation
  - 50% of patients with MS
  - Enlarged atrium
Physical Exam

- Heart Sounds
  - Low-pitched rumbling diastolic murmur
  - Murmur of mitral regurgitation
    - Systolic murmur
    - May also be present

Diastolic Murmurs
Mitral Stenosis

- **Timing:**
  - Holodiastolic if severe MS
  - Mid to Late diastole if moderate MS
- **Location:** Apex
- **Configuration:** Crescendo
- **Pitch:** Low
- **Quality:** Rumbling
- Best heard with patient in left lateral position
- Increases with isometric exercise, and expiration
### Physical Exam

**Opening Snap**
- Not present if heavily calcified
- Location: Cardiac apex
- Timing: Just after S2
- Pitch: High
- Radiation: across precordium
- Often confused with S3
  - S3 better heard with bell of stethoscope
  - S3 louder during expiration than inspiration (OS does not change)
  - OS occurs closer to S2 than S3
  - Diastolic murmur helps confirm OS

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### Physical Exam

**Signs of right ventricular failure if disease process is severe**
- Jugular venous distension
- Hepatomegaly
- Peripheral edema
- Ascites

**Mitral Facies**
- Pinkish-purple discoloration of the cheeks
- Common with severe mitral stenosis
Diagnosis

Echocardiogram

- Evaluation of Gradient Pressure
  - Mild < 5mmHG
  - Moderate 5-10mmHG
  - Severe > 10mmHG

- Evaluation of Valve Area
  - Normal 4-5cm²
  - Mild > 1.5 cm²
  - Moderate 1.0-1.5 cm²
  - Severe < 1.0 cm²

- Evaluation of Atrial Size
- Evaluation of Pulmonary Artery Pressures
  - Mild < 30mmHG
  - Moderate 30-50mmHG
  - Severe > 50 mmHG

- Evaluation of Valve Leaflets
  - Mobility
  - Calcification
  - Valve Thickening
  - Subvalvular Thickening

Diagnosis

- Stress Test
  - Helpful in evaluation of symptoms

- Chest X-ray
  - Fluid overload if in failure
  - Increased prominence of the pulmonary arteries
  - Elevation of the left main stem bronchus
    - Noted with left atrial enlargement

- Cardiac Catheterization
  - Assessment of need for coronary artery revascularization if valve replacement is needed.
  - Measure pulmonary pressures
  - Measure left atrial pressure
    - Assists in the evaluation of the progression of the disease
Diagnosis

- ECG
  - Left atrial enlargement
    - Abnormal P waves
    - Wide, notched P waves in Lead II
  - Right ventricular hypertrophy
    - Axis shift to the right may be present
  - Atrial fibrillation
    - Alert for new onset atrial fib

Medical Treatment

- Is of limited use in asymptomatic patients in NSR
- Atrial Fibrillation Treatment
  - Beta blockers or calcium channel blockers
    - Maintain a ventricular rate of less than 100 beats per minute
  - Since atrial fibrillation is poorly tolerated it is reasonable to attempt to return the patient to normal sinus rhythm with cardioversion
- Heart Rate Control
  - Calcium channel blockers, beta-blockers helpful if experiencing exercise intolerance
- Other Benefits of Beta-blockers and Calcium Channel Blockers
  - Decrease ventricular wall tension
  - Improve filling from the atria
Medical Treatment

- **Preload Reduction**
  - Diuretics and sodium restriction if fluid overloaded

- **Anticoagulation**
  - High risk due to LA enlargement
  - Class I ACC/AHA Recommendations
    - MS with atrial fibrillation
    - MS and prior embolic event
    - MS and left atrial thromus
  - Class IIb ACC/AHA Recommendations
    - Consider in asymptomatic patients with severe MS and LA dimension > 55 mm by echocardiogram

- **Continuous Follow Up for Asymptomatic Patients**

Surgical Treatment

- Once symptoms occur surgery should occur
- Valve area < 1.5 cm²
  - Symptoms at rest
  - Lifestyle affected

- **Surgical Options**
  - Percutaneous mitral balloon valvotomy
  - Closed surgical commissurotomy
  - Open surgical commissurotomy
  - Mitral valve replacement
ACC/AHA Recommendations for Balloon Valvotomy

• Class I Recommendations
  • Symptomatic patients (NYHA functional class II, III, or IV) with moderate or severe MS and valve morphology favorable (noncalcified pliable valves, mild subvalvular fusions and no calcium in the commissures) for PMBV in the absence of left atrial thrombus or moderate to severe MR.
  • Asymptomatic patients with moderate or severe MS and valve morphology that is favorable for PMBV in the absence of left atrial thrombus or moderate to severe MR.

Surgical Treatment Valvotomy

• Better long-term results for MV than AV
• Inflated balloon causes fused leaflets to split
• Best results in patient with no valve calcification and strictly a fusion of the commissures
• If LA is greatly dilated or the valve is very calcified the results will be suboptimal
• Should not be performed if also have mitral regurgitation of 2+ or more
### Commissurotomy

- Commissures are cut apart
- Allows for increased movement of the leaflets
- Beneficial to patients with pliable leaflets and no calcification
- Closed repair or open repair
- **Closed repair**
  - Cannot visualize the valve
  - No cardiopulmonary bypass needed
  - Valvotomy becoming more common
- **Open repair** is preferred method
  - Can remove calcium deposits and left atrial clots
  - Amputation of left atrial appendage
  - Open chest procedure requiring the use of cardiac bypass

### Surgical Treatment

#### Mitral Valve Replacement

**ACC/AHA Recommendations for MVR with MS**

- **Class I Recommendations**
  - Symptomatic moderate or severe MS when PMBV is unavailable, PMBV is contraindicated due to left atrial clot, or the valve morphology is not favorable for PMBV in a patient with acceptable operative risk. Repair over replacement if able.
  - Symptomatic moderate to severe MS who also have moderate to severe MR should receive valve replacement unless repair is possible.
- **Class IIa Recommendations**
  - Replacement for patients with severe MS and severe pulmonary hypertension and NYHA functional class I-II symptom who are not considered candidates for PMBV or surgical repair.
- **Class IIb Recommendations**
  - Considered for asymptomatic patients with moderate or severe MS who have had recurrent embolic events while receiving adequate anticoagulation and who have morphology favorable for repair.
## Mitral Valve Replacement

- Extensive calcification, fibrosis, mitral regurgitation
- MAZE procedure may be done
- Amputation of left atrial appendage
- Many receive mechanical valve as they are already on warfarin

## Outcomes

- Excellent outcomes with valvuloplasty and commissurotomy
- Symptom improvement occurs as soon as procedure is complete
- Endocarditis the same as with AS
- Anticoagulation the same as with AS
- Follow up the same as with AS
QUESTIONS??

Thank You!

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