Valvular Heart Disease: Volume Versus Pressure and the Hemodynamic Compromise

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Mastery is not something that strikes in an instant, like a thunderbolt, but a gathering power that moves steadily through time, like weather.

- John Champlain Gardner Jr. (1933-1982)
Cardiac Valves

Prevent Backward Flow

Permit Forward Flow

Valve Dysfunction

Impedance of forward flow

Decreased Cardiac Output

Backward flow of volume

Compensatory Changes
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

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.
Aortic Valve and Coronary Artery Perfusion

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
Cardiac Auscultation
The Key to Discovery

Auscultatory Areas

- Aortic Area (2RSB)
- Pulmonic Area (2LSB)
- Tricuspid Area (5LSB)
- Mitral Area (5MCL)
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

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Basic Heart Sounds

**S₂**

- Closure of Aortic (A₂) and Pulmonic (P₂) Valve  
- End of Ventricular Systole  
- **Location:** Pulmonic area  
- **Intensity:** Directly related to closing pressure in the aorta and pulmonary artery  
- **Duration:** Shorter than S₁  
- **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

Murmur Evaluation

- Systolic murmurs
  - Innocent vs. pathological
  - Pathological
- Diastolic Murmurs
- Cardiac Echocardiogram
Murmur Fundamentals

- **Timing**
  - Systolic
    - Holosystolic
    - Ejection (midsystolic)
    - Late
  - Diastolic
    - Early
    - Middiastolic
    - Late

- **Location**
  - Place heard the loudest

- **Grade**
  - Grade 1
    - Barely audible in a quiet room
  - Grade 2
    - Quiet, but heard immediately after placing stethoscope on chest
  - Grade 3
    - Moderate intensity, readily audible
  - Grade 4
    - Loud palpable thrill
  - Grade 5
    - Very loud, with thrill. Audible with stethoscope tilted slightly off the chest.
  - Grade 6
    - Very loud with thrill. Audible with stethoscope lifted off chest.

- **Configuration**
  - Crescendo
    - Gets louder
  - Decrescendo
    - Gets softer
  - Crescendo – Decrescendo
    - Louder then softer
  - Plateau
    - Even intensity throughout
Murmur Fundamentals

- **Pitch**
  - High Pitched - diaphragm
  - Low Pitched – bell

- **Quality**
  - Soft
  - Harsh
  - Blowing
  - Musical
  - Rumbling
  - Rough

Murmur Fundamentals

- **Stenotic Murmurs**
  - Valve does not open appropriately
  - Heard during the part of the cardiac cycle when the valve is open

- **Regurgitant Murmurs**
  - Valve does not close appropriately
  - Heard during the part of the cardiac cycle when the valve is to be closed
Systolic Murmurs

• Systolic regurgitant murmurs
  – Retrograde flow across an incompetent AV valve
  – Tricuspid and Mitral Valve Closed
  – Tricuspid Regurgitation
  – Mitral Regurgitation

• Systolic filling murmurs
  – Forward flow across stenotic or obstructed semilunar valve
  – Pulmonic and Aortic Valve Open
  – Pulmonic Stenosis
  – Aortic Stenosis

Diastolic Murmurs

• Diastolic regurgitant murmurs
  – Retrograde flow across an incompetent semilunar valve
  – Pulmonic and Aortic Valves Closed
  – Pulmonic Regurgitation
  – Aortic Regurgitation

• Diastolic filling murmurs
  – Forward flow across stenotic or obstructed AV valves
  – Tricuspid and Mitral Valves Open
  – Tricuspid Stenosis
  – Mitral Stenosis
Cardiac Output and Normal Cardiac Function

Let’s Review the Basics

The Heart as a Pump

**Goal:** Forward propulsion of blood to perfuse the body.

- Heart
- Arteries
- Veins
- Volume

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Terms

- **Cardiac Output**
  - Volume of blood ejected by the ventricle each minute

- **Stroke Volume**
  - Volume of blood ejected with each beat
  - Normal 60-120 ml / beat

- **Ejection Fraction**
  - Percent of blood ejected from the ventricle each beat
  - Normal: 55% to 60%

Basic Hemodynamic Formula

Cardiac Output = Heart Rate X Stroke Volume

Preload  Afterload  Contractility

Same four components also determine myocardial oxygen demand
Preload

• **End-diastolic stretch on myocardial muscles fibers**

• Determined by:
  - Volume of blood filling the ventricle at end of diastole
  - Greater the volume the greater the stretch (muscle fiber length)
  - Greater the stretch the greater the contraction
  - Greater the contraction the greater cardiac output

**TO A POINT**

Factors Influencing Preload

• Distribution of Volume
  - Venous Tone
  - Body Position
  - Intrathoracic pressure
  - Intrapericardial pressure

• Volume
• Atrial Kick
• Heart Rate
• LV Function
Afterload

- Pressure ventricle needs to overcome to eject blood volume
- Blood pressure is major component of afterload but it does not equal afterload
- Other components
  - Valve compliance
  - Viscosity of blood
  - Arterial wall compliance

Factors Influencing Afterload

- Right ventricular afterload is impacted by
  - Pressure in the lungs
  - Pulmonic valve function
Factors Influencing Afterload

- Left Ventricular afterload impacted by
  - Systemic vascular resistance
  - Aortic compliance
  - Aortic valve
- Diastolic BP is closest noninvasive measurement
- Left ventricular afterload does not equal blood pressure
- Increased diastolic BP = arteriole vasoconstriction.

Contractility

- Ability of myocardium to contract independent of preload or afterload
  - Velocity and extent of myocardial fiber shortening
  - Inotropic state
- Related to degree of myocardial fiber stretch (preload) and wall tension (afterload).
- Influences myocardial oxygen consumption
- ↑ contractility
  ⇒ ↑ myocardial workload
  ⇒ ↑ myocardial oxygen consumption
Factors Altering Contractility

- Causes of decreased contractility
  - Excessive preload or afterload
  - Drugs – negative inotropes
  - Myocardial damage
  - Ischemia
  - Cardiomyopathy
  - Hypoxia
  - Acidosis
  - Electrolyte imbalance
  - Hypothyroidism

- Causes of increased contractility
  - Drugs
    - Positive inotropes
  - Hyperthyroidism

Heart Rate

- Mathematically heart rate increases cardiac output

- Physiological limit where increased heart rate will decrease cardiac output due to decreased filling time (decreased preload)
Constant Hemodynamic Balance

Diastolic Dysfunction Versus Systolic Dysfunction
## Diastolic Dysfunction
- Filling Impairment
- Rate of LV filling is slow
- Elevated LV filling pressure when volume and contractility are normal
- Concentric Hypertrophy
- Thickened myocytes
- Pressure Overload
- Often elevated LV EF

## Systolic Dysfunction
- Impaired Contractility
- Decreased LV Ejection Fraction < 40%
- Eccentric Hypertrophy
- Elongated myocytes
- Volume overload

### Basic Hemodynamic Considerations Ventricular Dysfunction

#### Diastolic Dysfuncion
- Adequate preload needed
- Afterload not directly impacted
- Ejection fraction may be elevated
- Diastolic filling times important
  - Low heart rates
  - Atrial Kick

#### Systolic Dysfunetion
- Adequate preload needed
- Afterload can have major impact
- Contractility decreased
- Heart Rate elevates to compensate for decreased cardiac output
Aortic Valve Disease

Aortic Stenosis

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

Congenital
- Most common cause of AS in men under 70
- More prevalent in men
- Abnormal number of valve cusps
- Symptoms appear between age of 40 and 60

Acquired
- Most common form of AS in those >70
- RHD
- Senile Degenerative Calcification

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.
    ◆ smokers

Pathophysiology

Remember:
Stenosis = Pressure
Regurgitation = Volume
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
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)

- Decreased pulse sharpness
- Systolic Ejection Murmur
- S4
**Systolic Ejection Murmur**

- May be present before any significant hemodynamic changes occur
- More severe AS → longer murmur
- **Timing:** Midsystolic
- **Location:** Best heard over aortic area
- **Radiation:** Toward neck and shoulders
  - May radiate to apex
- **Configuration:** Crescendo-decrescendo
- **Pitch:** Medium to high
- **Quality:** Harsh

**S₄**

**Atrial Gallop**

- Occurs during late ventricular diastole
- Caused by atrial contraction and the propulsion of blood into a noncompliant (stiff) ventricle.
- Associated with systemic hypertension, restrictive cardiomyopathy, ischemia, aortic stenosis.
- May be normal in athletes
- Left- or right-sided, depending on ventricle affected.
  - Left-sided louder on expiration.
  - Right-sided louder on inspiration
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

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 cm$^2$ to 4 cm$^2$
  - Change in flow across the valve occurs when valve area is one fourth the normal size
  - Normal Valve area 3.0–4.0 cm$^2$
  - Mild AS Valve area >1.5 cm$^2$
  - Moderate AS Valve area 1.0 to 1.5 cm$^2$
  - **Severe AS** Valve area <1.0 cm$^2$

- **Evaluation of Jet Velocity**
  - Directly related to valve gradient and valve area
  - Increases as gradient increases and valve area decreases
  - Mils AS <3.0 m/second
  - Moderate AS 3.0–4.0 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
  - CAUTION in severe AS by ECHO

- **Cardiac Cath**
  - Verify absence/presence of CAD
  - Evaluate pressure gradients
  - Injection of dye $\rightarrow$ 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
  - Development of 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**
  - New 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

AHA April 2007
Severe Aortic Stenosis Prognosis

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

• **Class I Recommendations**
  - Symptomatic patients with severe AS.
  - Severe AS undergoing coronary artery bypass graft surgery
  - Severe AS undergoing surgery on the aorta or other heart valves.
  - Severe AS and LV systolic dysfunction (ejection fraction less than 0.50).

• **Class IIa Recommendations**
  - Moderate AS undergoing CABG or surgery on the aorta or other heart valves.
ACC/AHA Recommendations for Aortic Valve Replacement (AVR) in Aortic Stenosis

• 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.

Surgical Treatment Options

• AV Replacement with mechanical or bioprosthetic valve

• Aortic Valve Repair
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
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
- May be used as a bridge to surgery
- 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
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)
  - 1st visit 3-4 weeks
  - Subsequent visits
    - 6-12 months then annually
    - Repeat echocardiogram for patients with tissue valve
      - Annually after 1st 5 years even if no symptoms
    - Repeat echocardiogram for patients with mechanical valve
      - Evidence of prosthetic valve dysfunction
      - Evidence of LV dysfunction
      - Evidence of new murmur
      - Change in clinical status

Endocarditis Prophylaxis

For All Valve Replacements

- Appropriate for all patients with prosthetic valves
- Procedures requiring prophylaxis
  - Dental procedures involving manipulation of gingival tissue 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
Anticoagulation
For **All** Valve Replacements

- Mitral valve replacement highest risk for embolization
- Mechanical Valve
  - Warfarin for all patients – INR 2.5-3.5
  - 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

Linking Knowledge to Practice

- *Fluid balance* can be critical.

- Development of *atrial fibrillation* results in loss of atrial kick and therefore preload. Will result in decreased diastolic filling. Fluid balance can be critical.

- *Angina* may occur even without coronary artery disease. Need to assure good oxygen delivery too meet demand. (Oxygen/ hemoglobin)

- In severe AS avoid ACE Inhibitors and beta blockers
Aortic Regurgitation

Failure of the aortic valve to close tightly causes back flow of blood into the left ventricle.

Aortic Regurgitation
(Insufficiency / Incompetence)

- Valve cusps do not close completely
- Blood travels retrograde through the valve during ventricular diastole
Aortic Valve and Aortic Recoil

Aortic Regurgitation Classification

Chronic
- RHD
- Congenital
- Infective endocarditis
- Marfan’s
- Inflammatory diseases
- Syphilis
- Severe systemic Hypertension

Acute
- Trauma
- Acute infective endocarditis
- Acute aortic dissection
Pathophysiology

- Remember:
  - Stenosis = Pressure
  - Regurgitation = Volume

Aortic Regurgitation Pathophysiology

[Diagram of cardiac anatomy with labels such as LA, LV, Mitral valve, Pulmonary vein, Aorta, Capillaries, LUNG, LV (LVEDP)]
Compensatory Mechanisms In Aortic Regurgitation

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

\[ \text{LV \text{ dilates}} \text{ (size)} \]
\[ \text{LV \text{ hypertrophies}} \text{ (muscle mass)} \]
\[ \text{stroke volume \text{ increases}} \]

Works well for years – even decades.
Compensatory system ultimately fails
\[ \text{Systolic \text{ Dysfunction}} \to \text{Heart Failure} \]

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

- Length of murmur correlates severity of AR
- Timing: Early diastole
- Location: left sternal boarder
  - 3rd or 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
Systolic Flow Murmur

- Result of turbulent flow across valve during systolic
- 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
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

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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**
  - Not needed to confirm diagnosis
  - May test functional capacity and response to exercise
- **Cardiac Cath**
  - Verify absence or presence of CAD
- **ECG**
  - Increased QRS voltage with LV hypertrophy
  - Left axis deviation may be present with moderate to 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
  - Decrease afterload ➔ decrease regurgitation
  - Not indicated in asymptomatic patients
- 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
**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 may be a 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.

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**• 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

Linking Knowledge to Practice

- *Intra aortic balloon pumping is contraindicated* in patients with AR especially acute AR – assess for diastolic murmur of AR

- Remind patients to follow physicians recommended *follow up* plan so replacement can occur before there is loss of LV function

- Avoid medications that will arterial vasoconstrict

- Nipride may be helpful in the acute setting
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

Mitral Regurgitation Pathophysiology
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 Murmur

- **Timing:** Holosystolic
- **Location:** Mitral area
  - May be louder in aortic area depending on leaflet involved
- **Radiation:** To the left axilla or posteriorly over lung bases
- **Configuration:** Plateau
- **Pitch:** High
- **Quality:** Blowing, harsh or musical
S₃
Ventricular Gallop

- Early diastolic filling sound
- Caused by increased pressure and resistance to filling.
- Most frequently associated with systolic dysfunction
- Associated with:
  - Fluid overload state
  - Right or left ventricular failure
  - Ischemia
  - Aortic regurgitation
  - Mitral regurgitation

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.
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

- Primary tool utilize to conform 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

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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
- 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

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**Affect of Afterload Reduction**
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.
ACC/AHA Recommendations for Mitral Valve Repair /Replacement in Mitral Regurgitation

• Class IIa Recommendations
  – Repair 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.

• Class IIb
  – Repair may be considered for patients with chronic severe MR due to severe LV dysfunction (ejection fraction <30%) that has persistent NYHA functional class III-IV symptoms despite optimal therapy for heart failure, including biventricular pacing.

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

ARTIFICIAL CHORDAE → NATURAL CHORDAE
**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
Linking Knowledge to Practice

- Patients with mitral valve prolapse should avoid stimulants such as caffeine as they may increase the incidence of tachycardia.
- Acute papillary muscle ruptures do not generally occur at the onset of infarction but may develop 48-72 hours later.
- Post mitral valve replacement for mitral regurgitation the patient often feels “draggy” for the first four to six weeks due to the ventricles sudden increase in workload. These patients may require short term diuretics due to increased left ventricular volume and distension.

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

Pathophysiology

- Remember:
- Stenosis = Pressure
- Regurgitation = Volume
Mitral Stenosis Pathophysiology

Valve opening narrows
- Passive filling from left atrium to left ventricle slows
- Left atrial pressure increases in attempt to maintain normal flow across the valve
  - Left atrial pressure transferred back to the pulmonary vascular bed
  - Pulmonary pressures subsequently dilates
  - Left atrium as forward flow slows
  - More difficult to empty atrium
  - Chronic increase 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 -2.5cm²)
• 10 more years for the development of serious symptoms
• Symptoms at rest
  – Valve area < 1.5cm²

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

- 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
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

• 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 Pressure
  – Mild <5mmHG
  – Moderate 5-10mmHG
  – Severe > 10mmHG
• 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

• Medical Treatment
  – 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 thrombus
  – 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 (NHYA functional class II, III, or IV) with moderate or severe MS and valve morphology favorable (non calcified 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 commisurotomy
- 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

Mitral Valve Replacement
Linking Knowledge to Practice

- Patients with severe mitral stenosis are at increased risk of developing pulmonary edema with any increase in heart rate.

- While unusually stressful exercise should be avoided in those with mild to moderate mitral stenosis a low level of aerobic exercise is important to maintain cardiovascular fitness.

- Patients with mitral stenosis are dependent on atrial kick for ventricular filling. Therefore, atrial fibrillation may result in a fairly quick loss in perfusion.

- Clinicians should be particularly alert for the development of atrial fibrillation because the incidence of stroke is high with this population.

QUESTIONS

- In the patient with severe aortic stenosis which class of medications is contraindicated?
  - Beta Blocker
  - ACE Inhibitor
  - Low Dose Nitrate
  - Diuretic
QUESTION

• The heart’s primary compensatory response to chronic aortic stenosis includes
  – Left atrial hypertrophy
  – Left ventricular hypertrophy
  – Left ventricular dilation
  – Left atrial dilation

QUESTION

• A prominent V wave on the pulmonary artery wedge tracing will be noted in the patient with:
  – Aortic Stenosis
  – Aortic Regurgitation
  – Mitral Stenosis
  – Mitral Regurgitation
QUESTION

• Primary pulmonary hypertension is the primary end result of long term chronic compensation in which type of valve disease?
  – Aortic Stenosis
  – Aortic Regurgitation
  – Mitral Stenosis
  – Mitral Regurgitation

Let’s Review
Aortic Stenosis
Pathophysiology

Aortic Regurgitation
Pathophysiology
Mitral Regurgitation Pathophysiology

Mitral Stenosis Pathophysiology
A Final Thought:
We must not, in trying to think about how we can make a big difference, ignore the small daily differences we can make which, overtime, add up to big differences that we often cannot foresee.

-Marian Wright Edelman

WITH CONFIDENCE

Thank You!

Handouts will be available on the internet at www.cardionursing.com on Monday.