HEART SOUNDS:
DO YOU HEAR WHAT I HEAR?

Seton Medical Center
Raising the Bar for Excellence in Cardiac Care

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"THE MOST IMPORTANT PRACTICAL LESSON THAN CAN BE GIVEN TO NURSES IS TO TEACH THEM WHAT TO OBSERVE…“

~FLORENCE NIGHTINGALE, 1859
We Begin With The Cardiac Cycle

TO UNDERSTAND HEART SOUNDS
CARDIAC DIASTOLE (ATRIAL & VENTRICULAR): EARLY PASSIVE VENTRICULAR FILLING
ATRIAL SYSTOLE & VENTRICULAR DIASTOLE: LATE ACTIVE VENTRICULAR FILLING

Atrial Kick
BEGINNING VENTRICULAR SYSTOLE: ISOVOLUMIC CONTRACTION
VENTRICULAR SYSTOLE: EJECTION

- RIGHT ATRIUM
- LEFT ATRIUM
- RIGHT VENTRICLE
- LEFT VENTRICLE
- AORTA
- Pulmonary Artery

Diagram showing the flow and parts of the heart during ventricular systole.
HEART SOUNDS – THE BASIS FOR THE SOUNDS

**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

**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
In 1816, I was consulted by a young woman laboring under general symptoms of diseased heart, and in whose case percussion and the application of the hand were of little avail on account of the great degree of fatness. The other method just mentioned [direct auscultation] being rendered inadmissible by the age and sex of the patient, I happened to recollect a simple and well-known fact in acoustics, ... the great distinctness with which we hear the scratch of a pin at one end of a piece of wood on applying our ear to the other. Immediately, on this suggestion, I rolled a quire of paper into a kind of cylinder and applied one end of it to the region of the heart and the other to my ear, and was not a little surprised and pleased to find that I could thereby perceive the action of the heart in a manner much more clear and distinct than I had ever been able to do by the immediate application of my ear.

Dr. R. Laennec (De l'Auscultation Médiate, August 1819)
Modern stethoscope with two ear pieces invented in 1851 by Arthur Leared.

George Cammann perfected the design of the instrument for commercial production in 1852.
“THE MOST IMPORTANT PART OF THE STETHOSCOPE IS THE PART BETWEEN THE EAR PIECES”

Dr. Terry Tegtmeier 1999
BASIC HEART SOUNDS

$S_1$

- Closure of the Mitral ($M_1$) valve and the Tricuspid ($T_1$) 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
DIASTOLIC FILLING SOUNDS
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
DIASTOLIC FILLING SOUNDS

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.
DIASTOLIC FILLING SOUNDS

$S_4$ - 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
DIASTOLIC FILLING SOUNDS $S_4$

- Patient position: left lateral decubitus position.
- Location
  - Left-sided $S_4$ – mitral area.
  - Right-sided $S_4$ – tricuspid area.
- Intensity
  - Left-sided louder on expiration.
  - Right-sided louder on inspiration.
- Duration: Short
- Quality: Thud like
- Pitch: Low

[C:\Users\Cynthia\Webner\Music\iTunes\iTunes Media\Podcasts\Heart Songs 3 - Video\1-07 07. Fourth Heart Sound.mp4]
d. Quadruple rhythm

e. Incomplete summation gallop

f. Summation gallop (SG)
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
MURMURS / BRUITS

- Forward flow through a septal defect or fistula
- Flow into a dilated chamber or portion of a vessel
MURMUR FUNDAMENTALS

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

**Location**
- Place heard the loudest

**Radiation**
- Direction in which murmur radiates
MURMUR FUNDAMENTALS

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

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

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

- **Grade 1**
  - Barely audible in a quiet room

- **Grade 2**
  - Quiet, but readily heard immediately after placing stethoscope on chest

- **Grade 3**
  - Moderate intensity, readily audible

- **Grade 4**
  - Loup with 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 the chest.
STENOTIC MURMURS
• Valve does not open properly
• Heard during the part of the cardiac cycle when the valve is open

REGURGE MURMURS
• Valve does not close properly
• Heard during the part of the cardiac cycle when the valve is closed
MURMURS THAT OCCUR DURING SYSTOLE

Systolic Filling Murmurs
- Forward flow across stenotic or obstructed valve
- Pulmonic and Aortic Valve Open
  - Pulmonic Stenosis
  - Aortic Stenosis

Systolic Regurgitant Murmurs
- Retrograde flow across an incompetent valve
- Tricuspid and Mitral Valve Closed
  - Tricuspid Regurgitation
  - Mitral Regurgitation

INNOCENT SYSTOLIC MURMURS
PULMONIC STENOSIS
SYSTOLIC EJECTION MURMUR

- **Timing:** Midsystolic
- **Location:** Best heard over pulmonic area
- **Radiation:** Left neck of left shoulder
- **Configuration:** Crescendo-decrescendo
- **Pitch:** Medium
- **Quality:** Harsh
Obstruction of flow at the level of the aortic valve.

AORTIC STENOSIS
AORTIC STENOSIS
PATHOPHYSIOLOGY
AORTIC STENOSIS SYMPTOMS

- Classic Triad
  - Angina
  - Syncope
  - Heart Failure
AORTIC STENOSIS
SIGNS (EXAMINATION)

• In addition to classic triad:
  • Decreased pulse sharpness
  • Systolic Ejection Murmur
  • S4
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
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
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 1968;38(Suppl V):V-61.
MITRAL VALVE REGURGITATION

- Valve cusps do not close completely

- Blood travels retrograde through the valve during ventricular systole
MITRAL REGURGITATION
PATHOPHYSIOLOGY
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
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

医疗服务

SVR

blood flow to area of least resistance (through non-functional MV)

cardiac output (forward flow) & atrial volume (fluid overload)

SVR and symptoms of volume overload

blood flow to area of least resistance

cardiac output (forward flow) & atrial volume (fluid overload)

acute pulmonary edema & shock
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
SYSTOLIC MURMURS
TRICUSPID REGURGITATION

- **Timing**: Holosystolic
- **Location**: Tricuspid area
- **Radiation**: To the right of sternum
- **Configuration**: Plateau
- **Pitch**: High
- **Quality**: Scratchy or blowing
DIASTOLIC MURMURS

Diastolic Regurgitant Murmurs

- Retrograde flow across an incompetent semilunar valve
- Pulmonic and Aortic Valves Close
  - Pulmonic Regurgitation
  - Aortic Regurgitation

Diastolic Filling Murmurs

- Forward flow across stenotic or obstructed AV valves
- Tricuspid and Mitral Valves Open
  - Tricuspid Stenosis
  - Mitral Stenosis

NO SUCH THING AS AN INNOCENT DIATOLIC MURMUR
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
AORTIC REGURGITATION
PATHOPHYSIOLOGY
AORTIC REGURGITATION
CLASSIFICATION

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

Acute
- Trauma
- Acute infective endocarditis
- Acute aortic dissection
CHRONIC AORTIC REGURGITATION SYMPTOMS

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

- Apical Impulse
- Diastolic Murmur of AR
- Systolic Flow Murmur
- Austin Flint Murmur
- Signs of Hyperdynamic Perfusion
Diastolic Murmur of AR

- Length of murmur correlates severity of AR
- Timing: Early diastole
- Location: left sternal boarder
  - $3^{rd}, 4^{th}$ 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 WITH CHRONIC AR

- 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
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
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
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
DIASTOLIC MURMURS
PULMONIC REGURGITATION

- **Timing**: Early diastole
- **Location**: Pulmonic area
  - Erb’s Point
- **Radiation**: Toward apex
- **Configuration**: Decrescendo
- **Pitch**: High
- **Quality**: Blowing
MITRAL VALVE STENOSIS

- Mitral Valve no longer opens normally
- Causing an obstruction of blood flow from the left atrium to the left ventricle
MITRAL STENOSIS
PATHOPHYSIOLOGY
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

• 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
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
MITRAL STENOSIS WITH 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
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 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
DIASTOLIC MURMUR
TRICUSPID STENOSIS

- **Timing:** Mid to Late diastole
- **Location:** Tricuspid area
- **Radiation:** None
- **Configuration:** Decrescendo
- **Pitch:** Low
- **Quality:** Rumbling
- **Increases during inspiration and decreases during expiration**
OTHER SOUNDS
PERICARDIAL FRICTION RUB

- **Timing**: Systolic, Early diastolic and late diastolic
- **Location**: Tricuspid area and Xyphoid area
- **Radiation**: None
- **Configuration**: Plateau
- May get louder during inspiration
- **Pitch**: High
- **Quality**: Grating, scratching
VENTRICULAR SEPTAL DEFECT OR RUPTURE

- **Timing:** Holosystolic Continuous
- **Location:** 3-4 LSB
- **Radiation:** Widely throughout the precordium
- **Configuration:** Plateau
- **Pitch:** High
- **Quality:** Harsh
HYPERTROPHIC CARDIOMYOPATHY
1 of every 500 (Maron et al, 2003)

Primary genetic cardiomyopathy

Effects men and women equally

Hypertrophy of myocardial muscle mass in the absence of increased ventricular afterload

Associated with decreased ventricular filling (diastolic dysfunction) and decreased cardiac output

Most common cause of sudden death in young adults

Cause unknown
  • 50% transmitted genetically
HYPERTROPHIC CARDIOMYOPATHY

- Disarray of cardiac myofibrils with hypertrophy of myocytes
- Cells take on a variety of shapes
- Myocardial scarring and fibrosis occurs
HYPERTROPHIC CARDIOMYOPATHY

- Usually only effects Left Ventricle
- Changes may be symmetrical
- Asymmetrical septal hypertrophy is more common
HYPERTROPHIC CARDIOMYOPATHY

- May involve entire septum or only a portion of septum
OBSTRUCTIVE HYPERTROPHIC CARDIOMYOPATHY

- 30-50% of HCM patients have obstruction
- Obstruction of outflow tract
- Septal wall enlarges into ventricular cavity
- Anterior leaflet of mitral valve drawn towards the septum during ejection
- Early closure of aortic valve, decreased ejection time, decreased cardiac output
Many asymptomatic for years
Incidence of sudden death often first presentation
Or identified during screening of relative of patient with HCM
Symptoms related to severity of diastolic dysfunction
Heart failure
Dyspnea #1 sign
• Syncope / palpitations with activity
Chest pain
Supraventricular arrhythmias
Development of mitral regurgitation
Hypertrophic Cardiomyopathy
Presentation

- Bisferiens Carotid Pulse (HOCM)
  - Brisk initial upstroke
  - Collapse of pulse then secondary rise
  - Must differentiate from AS – delayed upstroke
- PMI forceful and brisk
- S4
- MR murmur
- Systolic murmurs with obstructive disease process
  - Differentiating between HOCM and Aortic Stenosis
SUBVALVULAR LEFT VENTRICULAR OUTFLOW OBSTRUCTION SYSTOLIC MURMUR

- Timing: Mid systolic
- Location: best heard along left sternal boarder
- Radiation: usually does not radiate
- Configuration: crescendo-decrescendo
- Intensity: grade 3/6 to 4/6
- Pitch: medium
- Quality: harsh or rough
• HOCM murmur louder during Valsalva’s maneuver

• Decreases venous return to the heart
  • Decreased preload $\rightarrow$ ↓ left ventricular filling
  • Decreased left ventricular filling $\rightarrow$ ↑obstruction

• Any factor that decreases venous return to the heart increases the murmur in HOCM
  • Squatting increases venous return
  • Standing decreases venous return

• Aortic stenosis murmur becomes quieter during Valsalva’s maneuver
## Hypertrophic Cardiomyopathy Diagnosis

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<tr>
<th>ECHO</th>
<th>ECG</th>
<th>Cardiac Cath</th>
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</thead>
<tbody>
<tr>
<td>Wall thickness</td>
<td>LV hypertrophy</td>
<td>Not very helpful</td>
</tr>
<tr>
<td>LV size</td>
<td>Deep symmetrical T wave inversions</td>
<td>Do not often find CAD with HCM</td>
</tr>
<tr>
<td>Hyperdynamic LV function</td>
<td>P wave abnormalities</td>
<td></td>
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<tr>
<td>Atrial size</td>
<td>Arrhythmias</td>
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<tr>
<td>MV leaflets</td>
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<tr>
<td>LV outflow obstruction</td>
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</tbody>
</table>
Hypertrophic Cardiomyopathy
Treatment

• Goals
  • Relief of symptoms
  • Preventing complications
  • Preventing or reducing risk of sudden death
  • No evidence to support treatment of non-symptomatic patients
HYPERTROPHIC CARDIOMYOPATHY TREATMENT

- **Beta Blockers**
  - 1st choice (with or without HOCM)
  - Symptomatic benefit / improved exercise tolerance
  - Decreases HR
  - Improves LV relaxation
  - Helps control arrhythmias

- **Calcium Channel Blockers**
  - If Beta Blocker not effective
  - Decrease LV wall tension
  - Decreases HR
  - Diltiazem or Verapamil (no nifedipine D/T vasodilatation)
Hetertrophic Cardiomyopathy

**Treatment**

- **Disopyramide**
  - Negative inotrope
  - Class I antiarrhythmic
  - Use with BB to treat LV outflow track obstruction
  - Assists in HR control
  - May cause ventricular arrhythmias

- **Anti-arrhythmic Therapy**
  - **Atrial Fibrillation**
    - Most common arrhythmia
    - Poorly tolerated
    - Anticoagulation
  - Amiodarone or sotolol
    - Obstructive or non-obstructive OK
    - Ventricular or atrial arrhythmias
Other Medications
• Diuretics
  • With caution
• ACE Inhibitors and NTG
  • Avoided in HOCM
• Positive Inotropes
  • Strictly avoid any medication that increases contractility in HOCM

Pregnancy
• Not restricted in non-obstructive disease

Endocarditis Prophylaxis
• NO LONGER INDICATED (was previously indicated in obstructive disease only)

Non-Obstructive Disease Treatment
• More difficult to treat if no symptoms
• Ultimately evolves into dilated cardiomyopathy
Surgical Myectomy

- Marked outflow obstruction
- On maximum medical therapy
- NYHA Class III or IV
- MV Replacement or repair at same time (increases operative mortality)
- Improvement noted immediately and last 20-30 years
- Survival Rates 80% at 10 years
- May need pacemaker (2%)
Percutaneous Alcohol Septal Ablation

- Symptomatic with full therapy
- NYHA Class III or IV
- Not appropriate if MVR needed
- Cath Lab Procedure
- Catheter in septal perforator
- Ethyl alcohol injected
- Myocardial infarction occurs
- Enlarged septum eventually shrinks
- May need pacemaker (20%)
SOUNDS ASSOCIATED WITH GROIN COMPLICATIONS
ACCESS SITE COMPLICATIONS
BLEEDING: RETROPERITONEAL

• Bleeding into the Retroperitoneal Space
  • The retroperitoneal space is deep within the abdominal cavity
  • It can hold several liters of blood
  • More common with:
    • Punctures above the inguinal ligament
    • Double wall punctures & multiple sticks
  • Diagnosis by abdominal CT scan or ultrasound
ACCESS SITE COMPLICATIONS

BLEEDING: RETROPERITONEAL

- Manifestations
  - Tachycardia (? beta blockers)
    - Due to hypovolemia
  - Pain
    - Flank, leg, back or lower abdominal pain
    - Referred pain due to nerve compression
      - Ipsilateral shoulder
      - Feeling to have a bowel movement
  - Foot or leg palsy
  - Distension of the abdomen
  - Hypotension (late sign)
    - Body is no longer compensating
ACCESS SITE COMPLICATIONS

BLEEDING: PSEUDOANEURYSM

• An Unstable Pulsatile Pouch Attached to the Artery
  • The arteriotomy (punctured arterial wall) fails to seal
  • To and fro movement of blood into the pouch
  • The pseudoaneurysm may rupture resulting in exsanguination at the puncture site
• More common with puncture of superficial or deep femoral artery
ACCESS SITE COMPLICATIONS
BLEEDING: PSEUDOANEURYSM

- Causes
  - Inadequate compression of arteriotomy

- Manifestations
  - Groin pain
  - Pulsatile mass
  - Bruit (systolic)

- Management Strategies
  - Ultrasound guided thrombin injection
  - Ultrasound guided compression
  - Surgery
ACCESS SITE COMPLICATIONS
ARTERIOVENOUS FISTULA

- An Abnormal Communication Track Between an Artery and a Vein
  - Turbulent arterial blood flowing within the vein
    - Differentiation with a to and fro bruit
    - Thrill
  - A risk if both the artery and vein are in close proximity
    - Both the artery and vein are cannulated
    - Multiple punctures
    - The vein is “nicked” with arterial cannulation
ACCESS SITE COMPLICATIONS
ARTERIOVENOUS FISTULA

• Management Strategies
  • To reduce risk
    • Remove one catheter/sheath first
      • Remove arterial first then the venous
    • Obtain hemostasis before removing the other catheter/sheath
QUESTIONS
A DILIGENT ASSESSMENT BY A NURSE MAY UNCOVER A FINDING THAT WILL MAKE A DIFFERENCE IN A LIFE.

LISTEN CAREFULLY!!!!
BE THE BEST THAT YOU CAN BE EVERY DAY. YOUR PATIENTS ARE COUNTING ON IT!

THANK YOU!!!

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