Pathophysiology: Coronary Artery Disease, Aortic Stenosis and Mitral Regurgitation

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Outline

• Coronary Artery Disease
  • Angina
  • Acute Coronary Syndrome (UA/NSTEMI, STEMI)

• Heart Failure
  • Left and right side

• Aortic Stenosis

• Mitral Regurgitation
Coronary Artery Disease

- **Arteriosclerosis** loss of elasticity of blood vessels.

- **Atherosclerosis** narrowing of vessels due to fatty deposits.
Risk Factors For CAD

- Family history
- Advancing age
- Male Gender
- Total cholesterol >200, LDL>100 HDL< 35
- Hypertension
- Smoking
- Overweight/obesity
- Sedentary life-style
- Stress
- Diabetes Mellitus
Evolution of Plaque

1. LDL enters
2. & Oxidizes
3. Monocytes adhere & 4. cross intima differentiating yielding macrophages
5. Macrophages engulf LDL yielding Foam cells
8. Muscle cells die & harden plaque - Calcium develops

Atherosclerosis

Two Processes

- First the fats:
  - Lipid accumulation & oxidation

- Then the vessel deforms:
  - Endothelial dysfunction
  - Spastic vessels
# Manifestations of Coronary Artery Disease

<table>
<thead>
<tr>
<th>Stable Angina</th>
<th>Unstable Angina</th>
<th>Myocardial Infarct</th>
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</thead>
<tbody>
<tr>
<td>• Predictable</td>
<td>• Unpredictable, new or occurs at rest</td>
<td>• Often related to occlusive thrombus &amp; plaque rupture</td>
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<tr>
<td>• Unbalanced Supply &amp; Demand</td>
<td>• Unbalanced Supply &amp; Demand</td>
<td>• Stasis of flow</td>
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<tr>
<td>• Reduced $O_2$</td>
<td>• Less/Unresponsive to Medication</td>
<td>• Loss of supply with increase in demand</td>
</tr>
<tr>
<td>• Reduced Blood Flow</td>
<td>• Often related to transient non-occlusive thrombus</td>
<td>• Irreversible Myocardial Damage</td>
</tr>
<tr>
<td>• Responsive to Medication</td>
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Other Angina Types

Prinzmetal’s / Variant
- Typically occurs at rest
- Caused by focal spasm of angiographically normal coronary arteries. In the majority of patients there is also atherosclerotic coronary artery obstruction. In cases where there is atherosclerotic obstruction the vasospasm occurs near the stenotic lesion.

Post infarct
- Secondary to ischemic tissue around the infarcted area.

Microvascular / Syndrome X
- Angina without angiographic evidence of stenosis or disease
- Thought to be microvasculature circulation deficiency
Angina Management

- Lifestyle
- Nitrates
- Statins
- Beta Blockers
- CCB’s
- Aspirin
- PCI or CABG (if indicated)
Acute Coronary Syndrome

Old Terminology:
- UA
- NQMI
- STE-MI

New Terminology:
- Non-ST-Segment Elevation Acute Coronary Syndrome (NSTEMI-ACS)
- ST-Segment Elevation Acute Coronary Syndrome (STEMI-ACS)

Plaque Disruption/Fissure/Erosion
Thrombus Formation
Patient Presentation of ACS

- Syncope or near syncope without other cause
- Impairment of cognitive function without other cause
- Chest pain described as pressure, fullness, or squeezing in the midportion of the thorax
- Radiation of chest pain into the jaw or teeth, shoulder, arm, and/or back
- Associated dyspnea or shortness of breath
- Associated epigastric discomfort with or without nausea and vomiting
- Associated diaphoresis or sweating
Cardiac Markers

- Creatine Kinase- MB
  - 4-6 hours after symptom onset
  - Normal in 48-72 hours

- Troponins (C, I, T)
  - 4-8 hours after symptom onset
  - Remain elevated as long as 7-10 days
  - I & T specific to cardiac muscle
Treatment of Acute Coronary Syndrome

Initial Treatment of ACS

STEMI*
- Antiplatelet, anti-ischemic, or anticoagulant therapy
- Thrombolytics
- PCI or CABG

UA/NSTEMI†
- Antiplatelet, anti-ischemic, or anticoagulant therapy
- PCI or CABG

Long-Term Medical Management

Complications of MI

- Dysrhythmias
  - Heart blocks
  - Bundle branch block
  - Ventricular
- Heart Failure
- LV Aneurysm
- LV Rupture
- VSD
- Mitral Regurgitation

Images: Courtesy of W D Edwards (Mayo Foundation)
Heart Failure

• Pathophysiological state in which cardiac function fails to pump blood at a volume commensurate with venous return to meet metabolic demands.

• Inadequate forward flow with volume back up to capillary beds “behind” the failing ventricle

• Can be left or right Ventricular in nature

• RV failure is most commonly due to chronic LV failure.
Etiology of Heart Failure

**Volume Overload:**
- Reguritate AO valve
- High output status

**Pressure Overload:**
- Systemic hypertension
- Outflow obstruction (AS)

**Loss of Muscle:**
- Post MI
- Chronic ischemia
- Connective tissue diseases
- Infection

**Restricted Filling:**
- Pericardial diseases
- Restrictive cardiomyopathy
Left Sided Heart Failure

**Acute:**
Increased LVEDP, LA, PCWP, PAP
Decrease: CO, CI

**Chronic:**
Increased RV, RA/CVP
Decrease: CO, CI
Left Ventricular Dysfunction

- Signs & Symptoms
  - Basilar Rales
  - Pulmonary Edema
  - Pulsus Alternans
  - S3, S4 Gallop
  - Pleural Effusion
  - Cheyne-Stokes Respiration
  - Enlarged heart-CTR > 50%

- Symptoms
  - Dyspnea on Exertion
  - Paroxysmal Nocturnal Dyspnea
  - Tachycardia
  - Cough
  - Hemoptysis
  - Weakness, confusion
  - Cool, pale extremities
Right Sided Heart Failure

Increase:
RVEDP, RA, CVP, JVP

Possible decrease:
PAP, PCWP, CO, CI
Right Ventricular Dysfunction
Systolic and Diastolic

• Symptoms
  • Abdominal Pain
  • Anorexia
  • Nausea
  • Bloating
  • Swelling

• Physical Signs
  • Peripheral Edema
  • Jugular Venous Distention
  • Abdominal-Jugular Reflux
  • Hepatomegaly
Assessing Heart Failure

- Echo
- ECG Monitor
- O2 Sat Monitor
- Chest X-ray
- Pulmonary Artery Catheter
  - SVR / PVR
  - CO/CI
  - PCWP
  - CVP
Functional Classifications
(New York Heart Association)

- **Class I**: No symptoms with activity.

- **Class II**: Slight limitations to physical activity, comfortable at rest, physical activity results in fatigue, palpitations, SOB, or angina.

- **Class III**: Marked limitations to physical activity, comfortable at rest, slight physical activity results in fatigue, palpitations, SOB, or angina.

- **Class IV**: Unable to carry out any physical activity without discomfort.
Pharmacologic/Medical Management

- Oxygen
- Digoxin
- Diuretics
- ACE Inhibitors
- Beta-Blockers
- Antagonists
- Angiotensin Receptor Blockers (ARBs)
- Dopamine/Dobutamine
- Intra Aortic Balloon Pump/LV assist
- Underlying Cause
Heart Failure Summary

• Heart failure is a chronic, progressive disease that is generally not curable, but treatable

• Most recent guidelines promote lifestyle modifications and medical

• It is estimated 15% of all heart failure patients may be candidates for cardiac resynchronization therapy

• Close follow-up of the heart failure patient is essential, with necessary adjustments in medical management
Valve Disorders
Aortic Stenosis

- Normal aortic valve area is 2.5 to 3.6 cm$^2$
- Critical severe aortic stenosis less than 0.7 cm$^2$ (can vary)
Chronic Aortic Stenosis

- Compensation
  - Pressure overload
  - Initially increased LVEDV and contractility
  - Starlings law
Aortic Stenosis

- LVOT obstruction
- LVH initially
- Pulsus parvus/tarvus-narrow pulse pressure
- Decreased systemic perfusion
- Inc. LVEDP, LA, PCWP, PA
- Pulmonary congestion
- LV-AO Systolic gradient
- Systolic murmur
- SAD-syncope, angina, dyspnea
You can Learn a lot From a Garden Hose

Richard E. Klabunde, PhD - Cardiovascular Physiology Concepts
AS can Lead to Pulmonary Edema
Normal

Aortic Stenosis

AO dec systolic & narrow pulse pressure
LV Inc systolic, diastolic, & LVEDP

Baim DS, Grossman’s Cardiac Catheterization, Angiography, and Intervention, 7th ed.
Hemodynamic Manifestations of AS

- Decreased SV, CO/CI, and AO
  - Pulsus Parvus et Tarvus
    - May be brisk in elderly with non-compliant vessels
- Increased PA and PCWP
- LV & AO systolic gradient
- Varies on the severity

Richard E. Klabunde, PhD - Cardiovascular Physiology Concepts
What gradient do we use???

- **Gradient**
  - Peak to Peak
  - Peak instantaneous gradient-Doppler flow velocity
  - Mean gradient (Simultaneous LV and AO)

- **Severity**
  - Mild \(>1.5 \text{ cm}^2, < 25 \text{ mmHg}\)
  - Moderate \(1.1-1.5 \text{ cm}^2, 25-40 \text{ mmHg}\)
  - Severe \(0.8-1.0 \text{ cm}^2, > 40-50 \text{ mmHg}\)
  - Critical \(<0.7 \text{ cm}^2, > 50 \text{ mmHg}\)

- **Combined severe AI and AS**
  - Underestimate AV Flow and valve area

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Do we Phase shift or Not???

- Gradient
  - Ideally simultaneous LV and Ascending AO
  - AO and FA-FA delayed, widened and amplified, record AO and FA together to establish relationship
Mitral Regurgitation

Class I+ - Trace amounts of contrast flow into the LA, but it clears during atrial contraction

Class II+ - Moderate insufficiency; contrast is visible within the entire LA, and does not clear

Class III+ - Moderately severe MR; contrast opacifies the LA, becomes more dense with each contraction of the LV

Class IV+ - Severe regurgitation; wide open MR, contrast completely opacifies the LA during first LV contraction
Mitral Regurgitation

- LV ejecting into LA, PV’S
- Decreased CO and systemic perfusion
- Inc. LA, PCWP, PA
- Systolic murmur
- Pulmonary congestion
- LA/PCPW inc V wave
- Left Atrial enlargement
- A Fib
LA/PCWP Hemodynamics

- **LA/PCWP:**
  - 4-12 mmHg
- **a wave:** LA contraction
  - 12-15 mmHg
- **c wave:** MV closes
- **x decent:** LA relaxation
- **v wave:** LA filling
  - 12-15 mmHg
- **y decent:** LA emptying after MV opens
Mitral Regurgitation

- Increased PA, LA/PCWP
  - V wave

- Decreased forward CO/CI

- S & S LHF and pulmonary edema
Mitral Regurgitation: Acute vs. Chronic
Baim DS, Grossman’s Cardiac Catheterization, Angiography, and Intervention, 7th ed.
How Serious is it?

• RF% Severity
  • Mild 20% to 40%
  • Moderate 40% to 60%
  • Severe Greater than 60%
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18th Annual Conference
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