

The Nature of Disease
Pathology for the Health Professions

Thomas H. McConnell

Chapter 9

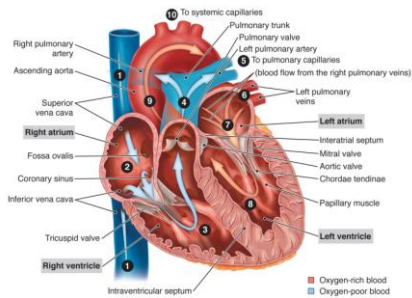
Disorders of the Heart

Lecture 9

Overview of Cardiac Lectures

- Review of Cardiac Physiology
- Heart Failure
- Coronary Artery Disease & Myocardial Infarction
- Valvular Heart Disease
- Diseases of the Myocardium (Heart Wall)
- Pericardial Disease
- Arrhythmias

Blood Flow Through the Heart



Coronary Blood Flow

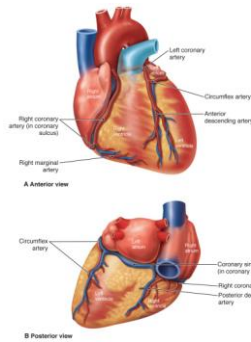
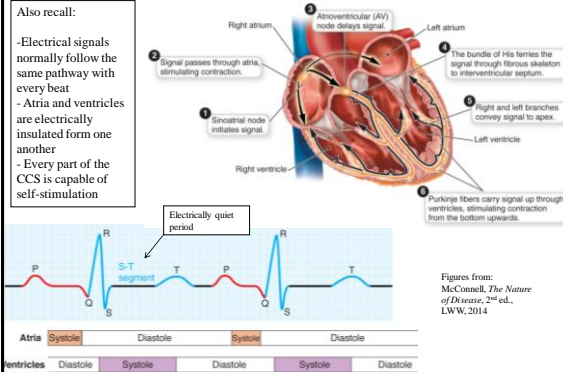


Figure from McConnell, *The Nature of Disease*, 2nd ed., LWW, 2014

Cardiac Conduction System & EKG

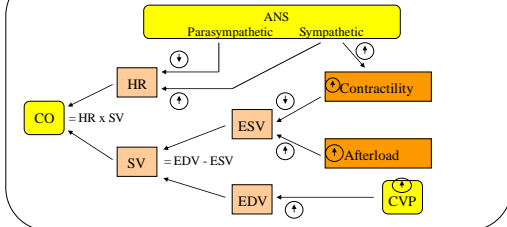
Also recall:

- Electrical signals normally follow the same pathway with every beat
- Atria and ventricles are electrically insulated from one another
- Every part of the CCS is capable of self-stimulation



Factors Affecting Cardiac Output

Figure adapted from: Aaronson & Ward, *The Cardiovascular System at a Glance*, Blackwell Publishing, 2007



CO – Cardiac Output (~5L/min). Dependent upon Stroke Volume (SV; ~70 ml) and Heart Rate (HR)

CVP – Central Venous Pressure; Pressure in vena cava near the right atrium (affects **preload**; **Starling mechanism**)

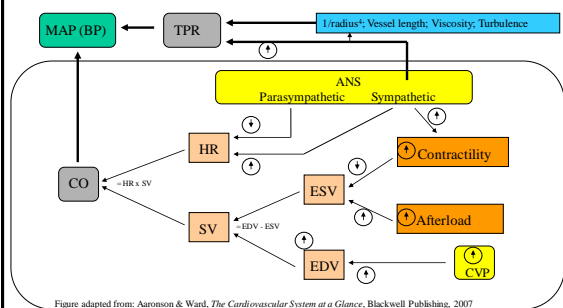
Contractility – Increase in force of muscle contraction *without* a change in starting length of sarcomeres

Afterload – Load against which the heart must pump, i.e., pressure in pulmonary artery or aorta

ESV – End Systolic Volume; Volume of blood left in heart *after* it has ejected blood (~50 ml)

EDV – End Diastolic Volume; Volume of blood in the ventricle *before* contraction (~120-140 ml)

Relationship of CO to Blood Pressure (MAP)



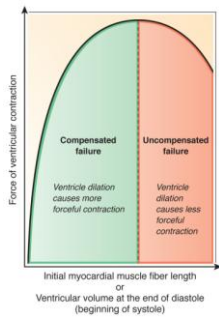
MAP - Mean Arterial Pressure = Average effective pressure driving blood flow through the systemic organs

**The MAP is dependent upon CO and TPR, i.e., $MAP = CO \times TPR$

TPR - Total Peripheral Resistance; depends upon *blood vessel radius, vessel length, blood viscosity, and turbulence

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Frank-Starling Law



- Amount of blood pumped by the heart each minute (CO) is almost entirely determined by the venous return
- Frank-Starling mechanism
 - Intrinsic ability of the heart to adapt to increasing volumes of inflowing blood
 - Cardiac muscle reacts to increased stretching (venous filling) by contracting more forcefully
 - Increased stretch of cardiac muscle causes optimum overlap of cardiac muscle (length-tension relationship)

What does the term "Ejection Fraction" mean?

Figure from: McConnell, *The Nature of Disease*, 2nd ed., LWW, 2014

General Mechanisms Leading to Heart Disease

1. **Pump failure**
 - Weak contraction
 - Reduced CO
2. **Obstructed flow**
 - Atherosclerosis
 - Valvular defects
3. **Abnormal Conduction**
 - Poorly timed, premature/late, or mechanically inefficient beats
 - Result is reduced CO
4. **Regurgitant flow (regurgitation)**
 - Valvular defects
 - Heart must re-pump blood
5. **Shunted flow**
 - Diversion by congenital defects, e.g., patent foramen ovale
 - Heart must work harder and re-pump blood

Heart Failure (HF)

- Definition: A syndrome of ventricular dysfunction in which
 - CO cannot meet metabolic demands, or
 - Ventricle must be dilated to meet metabolic demands (recall Frank-Starling Law and point of 'decompensation')
 - The endpoint for most serious heart diseases
- Causes of HF fall into two major groups
 1. Increased workload on the heart
 2. Muscle failure

Left/Right Sided Heart Failure

- Left heart failure (Congestive heart failure; more common)
 - Causes of increased workload on left ventricle
 - Hypertension
 - Mitral or aortic valve regurgitation
 - Aortic valve stenosis
 - Congenital disease
 - Reduced CO activates RAA System; ↑ fluid, BP
- Right heart failure
 - Causes of increased workload on right ventricle
 - *Most common cause: increased workload due to left ventricular failure
 - Increase in left ventricular filling pressure that is reflected back into the pulmonary circulation
- General causes of left & right ventricular muscle failure
 - Ventricular infarction (most common cause of left-sided failure)
 - Cardiomyopathy

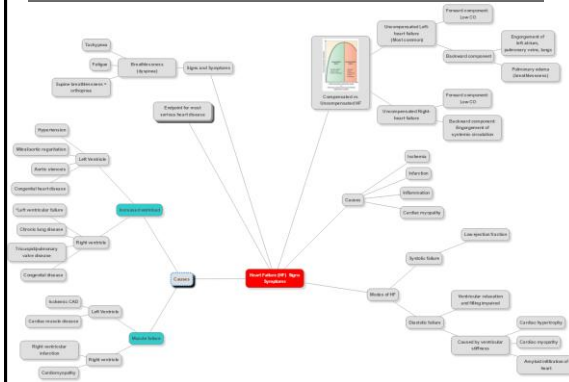
Heart Failure (HF)

- Before failing, heart tries to compensate
 - SNS and adrenal NE release
 - Cardiac Muscle Hypertrophy
- After failing
 - Systolic failure
 - Ventricle contracts poorly
 - Incomplete emptying of ventricle (↓ ejection fraction, CO)
 - Diastolic failure
 - Impaired ventricular relaxation
 - Impaired ventricular filling
 - Uncompensated failure (falling CO)
 - Forward failure (Low CO)
 - Backward failure (upstream venous congestion)
- Cor Pulmonale – Right HF due to pulmonary hypertension

Heart Failure (cont'd)

- Manifestations of **left heart failure**:
 - Associated with both forward and backward failure
 - Result of pulmonary vascular congestion and inadequate perfusion of the systemic circulation
 - Include dyspnea, orthopnea, cough of frothy sputum, fatigue, decreased urine output, and edema
- Manifestations of **right heart failure**:
 - Result of backward failure
 - Engorgement of system venous system
 - Hepatomegaly, splenomegaly
 - Edema of feet/legs (peripheral edema)
 - Ascites
 - Pleural effusion

Heart Failure



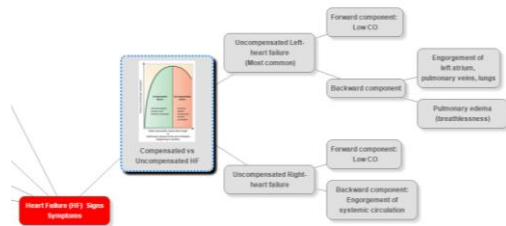
Heart Failure – General Causes and Classification



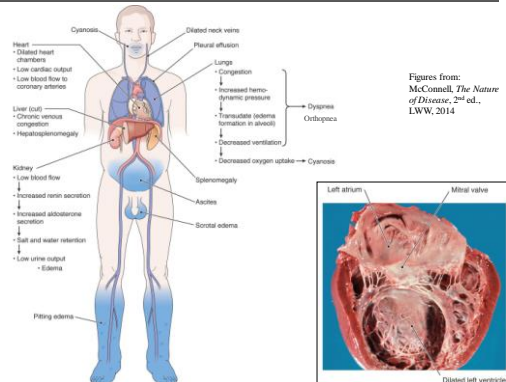
Heart Failure - Modes



Heart Failure – Compensated/Uncompensated



Signs and Symptoms of Heart Failure



General Mechanisms Leading to Heart Disease - Review

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CAD, Myocardial Ischemia, and Myocardial Infarction

- Continuum of diseases that narrows or occludes the coronary arteries leading to myocardial ischemia
- **Coronary Artery Disease (CAD)**
 - Usually occurs first; followed by MI or other heart damage
 - Typically caused by **atherosclerosis**
 - May lead to myocardial ischemia and infarction (MI) or irreversible heart damage (acute coronary syndromes)

-- Typical Risk factors for CAD and atherosclerosis --

- | | |
|--|--|
| <ul style="list-style-type: none"> - Major: <ul style="list-style-type: none"> • Increased age • Family history • Male gender or female gender post menopause | <ul style="list-style-type: none"> - Modifiable: <ul style="list-style-type: none"> • Dyslipidemia • Hypertension • Cigarette smoking • Diabetes mellitus • Obesity/sedentary lifestyle • Atherogenic diet |
|--|--|

Myocardial Ischemia

- Local, temporary deprivation of the coronary blood supply
- Some clinical manifestations
 - Stable angina - chest pain with gradual onset with exertion; relieved by rest
 - Unstable angina
 - Aggregation of platelets on an atherosclerotic plaque
 - Intensification of existing angina, new angina, nocturnal angina, prolonged angina
 - **Very serious: May indicate a MI is imminent
 - Not usually relieved by rest or medicine
 - Unremitting angina
 - **Caused by myocardial infarction
 - Doesn't fluctuate and can't be relieved by rest or medication
 - Prinzmetal angina (Transient, unpredictable, occurs at rest; vasospasm)
 - Silent/mental-stress ischemia (silent; more common in women)

Acute Coronary Syndromes

- Result of sudden coronary obstruction by thrombus or ruptured atherosclerotic plaque

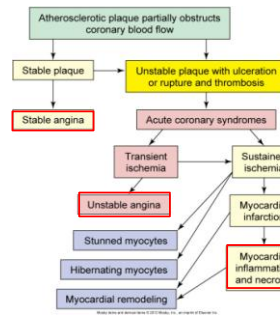
- Unstable angina

- Reversible
- Indicator of impending infarction
- Caused by platelet aggregation

- Myocardial infarction

- Prolonged ischemia
- Irreversible damage
- STEMI or non-STEMI
- Unremitting angina

- Sudden death can result from either



Myocardial Infarction

• Myocardial infarction

- Sudden and extended obstruction of the myocardial blood supply
- Subendocardial infarction
 - Only myocardium immediately beneath endocardium
 - Usually ST depression and T-inversion = non-STEMI
- Transmural infarction
 - Endocardium through the epicardium
 - ST segment elevation = STEMI
 - Highest risk for complications

• Pathophysiology

- Cellular injury
- Cellular death
- Structural and functional changes:
 - Myocardial stunning
 - Hibernating myocardium
 - Myocardial remodeling
- Repair

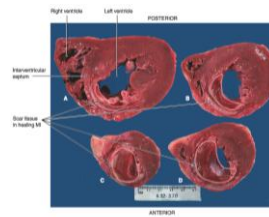


Figure 6-10: Microscopic view of myocardial infarction. The image shows four cross-sections of the heart wall. The top row shows the right ventricle and left ventricle. The bottom row shows the interventricular septum and the posterior wall. The images illustrate the extent of myocardial infarction, from subendocardial to transmural. Labels include: Right ventricle, Left ventricle, Interventricular septum, Posterior wall, and Anterior wall. A scale bar indicates 0.5 cm.

ECG Changes and Myocardial Ischemia

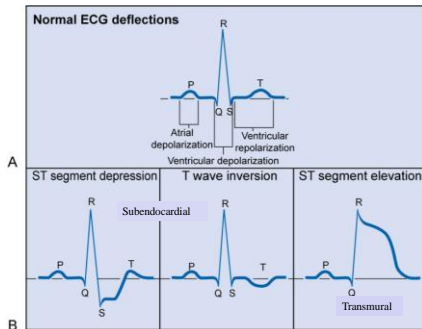
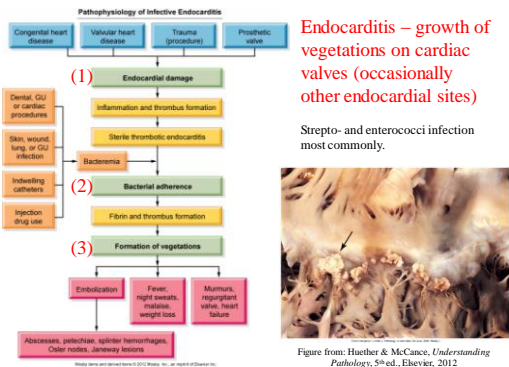


Fig 23-15: Electrocardiogram (ECG) and ischemia. A, Normal ECG. B, Electrocardiographic alterations associated with ischemia.

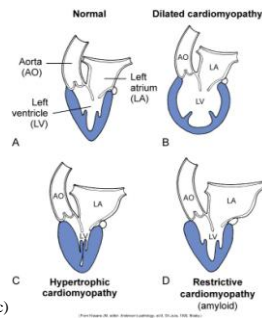
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Infective Endocarditis (cont'd)



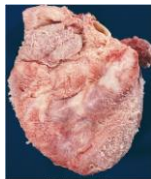
Disorders of the Myocardium: Cardiomyopathy

- Inflammatory = myocarditis
- Intrinsic muscle disease = cardiomyopathy
- Types of Cardiomyopathy:
 - Measurable dysfunction of the myocardium
 - Dilated cardiomyopathy (congestive cardiomyopathy)
 - Hypertrophic cardiomyopathy
 - Thickening of myocardium
 - Asymmetrical septal hypertrophy
 - Hypertensive (valvular hypertrophic) cardiomyopathy
 - Restrictive cardiomyopathy



Disorders of the Pericardium

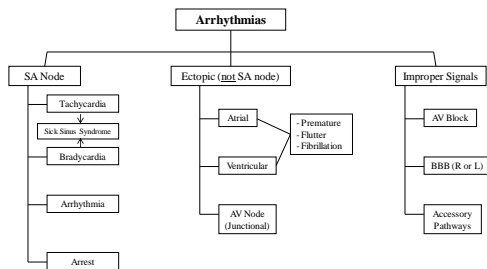
- Most common disorders of the pericardium
- Localized manifestation of another disorder:
 - Acute pericarditis (< 2 wks)
 - Acute inflammation of pericardium
 - Usual cause is viral infection or MI
 - Pericardial effusion
 - Tamponade (when effusion is rapid); Heart cannot fill
 - Hemopericardium (undiluted blood)
 - Constrictive (restrictive) pericarditis
 - Chronic scarring and obliteration of pericardial sac
 - Impaired diastolic filling



Arrhythmias (Dysrhythmias)

- Abnormalities of the heart rhythm
- Range from occasional “missed” or rapid beats to severe disturbances that affect the pumping ability of the heart
- Some definitions to know:
 - Escape rhythm – rhythms not initiated by the SA node
 - Ectopic beat – Originating at a site other than the SA node
 - Cardiac arrest – Sudden cardiovascular collapse and unconsciousness
 - Electroconversion – defibrillation
 - Sinus arrest – lack of any electrical discharge from the SA node
 - Premature atrial contractions (PACs) – ectopic, originate in atria
 - Premature ventricular contractions (PVCs) – ectopic, originate in ventricles (do not pass backwards to SA node)
 - Flutter – rapid, but regular and evenly spaced beats
 - Fibrillation – rapid, irregular and unevenly spaced (little/no CO)
 - Reentry loop – originates in CCS, but loops back into it again
 - Junctional arrhythmia – ectopic beat with origin near AV node

General Classification of Arrhythmias



Cardiac Arrhythmias (Dysrhythmias)

- Arrhythmias classified into three broad categories
 1. **Those associated with impulses arising from SA node**
 - Sinus brady- and tachycardia, sinus arrhythmia, sinus arrest
 2. **Those associated with impulses arising from OTHER than the SA node; Ectopic signals**
 - In atria: Premature, Flutter (rapid, regular), Fibrillation (irregular)
 - In ventricles: Premature, Flutter (tachycardia), Fibrillation
 - Ventricular tachycardia (≥ 3 consecutive ectopic beats; rate >120 bpm)
 - Ventricular fibrillation (CO is effectively zero)
 - At AV node junction (junctional arrhythmia)
 - Originate near the AV node
 - Sometimes called Supraventricular Tachycardia
 - Causes tachycardia

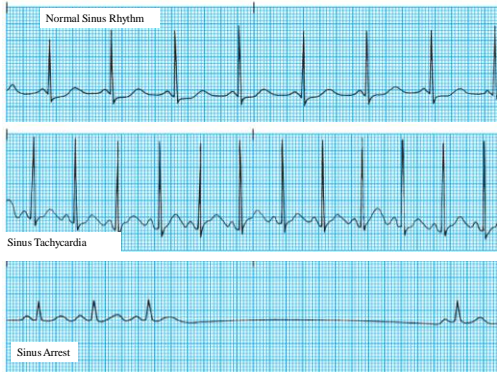
Cardiac Arrhythmias (Dysrhythmias)

- Arrhythmias classified into three broad categories (cont'd)

3. Those associated with impulses arising from OTHER than the SA node; Ectopic signals

- In atria
 - Premature atrial beats (usually not pathologic)
 - Atrial flutter (rapid, but regular)
 - Atrial fibrillation (rapid and irregular)
- In ventricles
 - Premature Ventricular Contractions (PVC)
 - » Early ventricular beat
 - » Interferes with next impulse from SA node
 - Ventricular tachycardia (≥ 3 consecutive ectopic beats; rate >120 bpm)
 - Ventricular fibrillation (CO is effectively zero)
- At AV node junction (junctional arrhythmia)
 - Originate near the AV node
 - Sometimes called Supraventricular Tachycardia
 - Causes tachycardia

EKGs



EKGs

