Overview of Cardiac Structure and Function

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Learning Objectives

1. To know the major anatomic parts of the heart
2. To understand the function of the parts of the heart
3. To understand how the heart accomplishes its pumping function
4. To understand what happens when heart function is impaired
The **Heart** resides in the Anterior Mediastinum, behind the Sternum, slightly left of midline, surrounded by the Lungs, in a sac called the Pericardium.

The heart is normally about the size of a clenched fist, and weighs 250-300 grams in females and 300-350 grams in males.
Heart (front view)

- Superior vena cava
- Pulmonary artery
- Base
- Right atrium (behind and lateral)
- Right atrial appendage
- Inferior vena cava
- Aorta
- Pulmonary veins (4 total)
- Left atrial appendage
- Left anterior descending coronary artery (LAD)
- Apex
Two Pumps

From Body

To Lungs

From Lungs

To Body
Two Pumps

From Body

Right Atrium

To Lungs

Right Ventricle

From Lungs

Left Atrium

To Body

Left Ventricle
Two Pumps

To Body

To Lungs

From Lungs

From Body

Right Atrium

Right Ventricle

RV outflow tract

Left Atrium

Left Ventricle

Netter Atlas of Human Anatomy
Parts of the Adult Heart

- Coronary Arteries
- Heart Valves
- Conducting System
- Myocardium
Parts of the Adult Heart

• Coronary Arteries
• Heart Valves
• Conducting System
• Myocardium
Coronary Arteries

Left
- Left Main
- LCx
- LAD

Right
- RCA
- PDA
Major Coronary Artery Supplies

- **LAD**
  - ~50% of LV

- **LCX**
  - ~20% of LV
  - Posteriolateral LV
  - Anterolateral papillary muscle

- **Right CA**
  - 30% LV
  - RV
Coronary Artery Dominance

• Whichever of the LCA or RCA gives rise to the posterior descending artery (PDA) and posterior lateral artery (PLA) is said to be “dominant.”

• ~70-80% of hearts are right side dominant

• ~10-20% “co-dominant”

• ~10% left side dominant
Coronary Artery Dominance

“Right Dominant”

“Left Dominant”

LCX

RCA

Posterior Descending Artery
Coronary artery angiogram (slice through the ventricles)
Parts of the Adult Heart

• Coronary Arteries
• Heart Valves
• Conducting System
• Myocardium
Heart Valves

- Tricuspid
  - Septal
  - Posterio-lateral

- Pulmonic
  - Left
  - Right

- Mitral
  - Anterior
  - Posterior
  - Left Coronary
  - Right Coronary

- Aortic
  - Anterior
  - Non-Coronary
Aortic Valve

Coronary Orifice
Mitral Valve
Mitral Valve
Parts of the Adult Heart

- Coronary Arteries
- Heart Valves
- Conducting System
- Myocardium
Conduction System

- SA Node
- AV Node
SA and AV nodes
Parts of the Adult Heart

• Coronary Arteries
• Heart Valves
• Conducting System
• Myocardium
Myocardial Wall

- Left Ventricle – 1.0-1.4 cm thick
- Right Ventricle – 0.3-0.5 cm thick
Myocardium
Intercalated discs

MYOFIBER

Myocyte

Na⁺ exchange

Ca²⁺ pump

Ca²⁺ enters

Ca²⁺ 'trigger'

Ca²⁺ leaves

FREE Ca²⁺

Contract

Relax

Systole

Diastole

Titin

Actin

M

Myosin

Head

43 nm
Membrane potential $\sim$-90 mV

$[\text{Na}^+] = \sim 10 \text{ mM}$

$[\text{Ca}^{++}] = \sim 100 \text{ nM}$

$[\text{Na}^+] = \sim 145 \text{ mM}$

$[\text{Ca}^{++}] = \sim 1.5 \text{ mM}$
Cardiac Myocyte Action Potential

![Cardiac Myocyte Action Potential Diagram]
Stroke Volume
LV End-Diastolic Volume
Normal
Failing
(or End-Diastolic Pressure)

ESPVR
Normal
Failing

EDV

Pressure vs Volume

Stroke Volume vs LV End-Diastolic Volume (or End-Diastolic Pressure)
Any Questions so far?
Diseases of the Heart

Heart disease can be classified by anatomy or by etiology, and I will use a combination of both. And I want to emphasize the relative frequency of each, so we will go from most common to least common, by etiology, and then by anatomy.
Leading Causes of Death in US, 2003

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVD</td>
<td>426,772</td>
<td>483,842</td>
</tr>
<tr>
<td>Cancer</td>
<td>286,741</td>
<td>267,902</td>
</tr>
<tr>
<td>Accident</td>
<td>76,923</td>
<td>65,672</td>
</tr>
<tr>
<td>COPD</td>
<td>60,456</td>
<td>45,058</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>35,217</td>
<td>38,748</td>
</tr>
<tr>
<td>Alzheimer</td>
<td>45,058</td>
<td></td>
</tr>
</tbody>
</table>

Source: CDC/NCHS, NHLBI, and AHA (Circulation 2006;113:e85-e151)
Deaths from Cardiovascular Diseases, US: 2003

Source: CDC/NCHS, NHLBI, and AHA (Circulation 2006;113:e85-e151)
US Deaths from Diseases of the Heart

Source: CDC/NCHS, NHLBI, and AHA (Circulation 2006;113:e85-e151)
ISCHEMIC HEART DISEASE

Although atherosclerosis of the coronary arteries is the most common mechanism responsible for myocardial ischemia, other less common mechanisms can also cause ischemia. These include:

- Coronary emboli
- Coronary spasm
Natural History of Atherosclerosis

Age in years

0-10: Fatty streak
10-20: Fibrous plaque
20-30: Complicated lesion—hemorrhage, ulceration, thrombosis
30-40: Calcification
40-50: Myocardial infarct, Cerebral infarct
50-60: Gangrene of extremities, Abdominal aortic aneurysm
60-70: Clinical horizon
MAJOR SYNDROMES

ANGINA PECTORIS

STABLE ANGINA

UNSTABLE ANGINA

MYOCARDIAL INFARCTION

SUDDEN CARDIAC DEATH

ISCHEMIC CARDIOMYOPATHY
PREVALENCE OF ISCHEMIC HEART DISEASE

13.5 million Americans (7% of adult population) have symptomatic IHD evidenced by:
   Angina Pectoris (50%)
   Previous MI (>50%)
   or both

500,000 deaths/year (one-third of all U.S. deaths)
   one-third are premature, i.e. before age 75

50% of deaths are complications of MI
50% of deaths are sudden cardiac death, often as first manifestation of IHD
Artery with fatty streak
Fibrous Plaque
Eccentric atherosclerotic plaque with lipid core
Ruptured atherosclerotic plaque with hemorrhage and thrombus on the surface
MYOCARDIAL INFARCT SIZE AFTER ISCHEMIA (I) AND REPERFUSION (R)

15 min I + R

3 hrs I + R

40 min I + R

4 days I; no R

Nonischemic

Occluded Vascular Bed (area at risk)

Infarct
### Morphologic Stages of Myocardial Infarction: Inflammatory Response and Repair

<table>
<thead>
<tr>
<th>Time Period</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 6 hours</td>
<td>No Change (Gross or Microscopic) unless reperfused</td>
</tr>
<tr>
<td>6 - 24 hours</td>
<td>“Wavy-fiber Change”; hemorrhage possible (Dark Mottling). Early features of Coagulative Necrosis (Cytoplasmic eosinophilia; Nuclear pyknosis followed by karyolysis)</td>
</tr>
<tr>
<td>1 - 4 days</td>
<td>Coagulative Necrosis with Acute Inflammatory Response (mostly neutrophils) - maximum at 2-3 days; neutrophils intact at first, disintegrating by 3-4 days; color shifts from dark to green to yellow as hemoglobin/myoglobin breaks down</td>
</tr>
<tr>
<td>4 - 7 days</td>
<td>Macrophage Activity (start of phagocysis of dead myocytes beginning on periphery); Hyperemic border, center yellow-tan</td>
</tr>
<tr>
<td>7 - 10 days</td>
<td>Developing peripheral rim of Granulation Tissue, removal of dead myocytes, capillary ingrowth, infarct begins to shrink</td>
</tr>
<tr>
<td>10 - 40 days</td>
<td>Progressive Organization (fibrosis) of infarct; gray-white color</td>
</tr>
<tr>
<td>1 - 3 months</td>
<td>Progressive Collagen Deposition; infarct is white, shrunken</td>
</tr>
</tbody>
</table>
Organizing anteroseptal MI; healed posteroseptal MI with aneurysmal thinning
Healed posteroseptal MI with aneurysmal thinning
INFLAMMATORY HEART DISEASES

ENDOCARDITIS

MYOCARDITIS

PERICARDITIS

RHEUMATIC HEART DISEASE
ENDOCARDITIS

INFECTIVE

NON-INFECTIVE
Acute bacterial endocarditis of aortic valve
NON-INFECTIVE ENDOCARDITIS

Nonbacterial Thrombotic Endocarditis (NBTE)

Marantic Endocarditis

Predisposing Factors

• Malignancy or debilitating chronic disease
• Hypercoagulable state
• Scarred valves

Libman-Sacks endocarditis - occurs in lupus
Nonbacterial Thrombotic Endocarditis - aortic valve
MYOCARDITIS

INFECTIONOUS

Most commonly thought to be due to viruses in the US (esp Coxsackie B)
Probably commonly occurs with many systemic viral infections, but often subclinical

NON-INFECTIONOUS

Hypersensitivity to various drugs
Rheumatic fever
Sarcoid
Interstitial lymphocytic infiltrate, consistent with viral myocarditis
PERICARDITIS

MORPHOLOGY

Acute Phase
  Serous
  Fibrinous ("Bread & Butter")
  Suppurative or Purulent
  Hemorrhagic

Chronic Phase
  Constrictive
  Adhesive
PERICARDITIS

ETIOLOGY

Infective
  Primary - Viral
  Secondary - almost any organism

Non-infective
  Metabolic, i.e. uremic
  Neoplastic
  Acute MI
  Hypersensitivity, i.e. rheumatic fever or post-MI
  Radiation-induced
  Traumatic, Idiopathic
Fibrinous pericarditis
Inflammation and Valvular Disease

Infection of normal valve $\rightarrow$ acute or subacute regurgitation

Chronic inflammation and scarring $\rightarrow$ stenosis $\pm$ regurgitation
RHEUMATIC HEART DISEASE

A non-suppurative inflammatory disease that may involve the joints, heart, blood vessels, skin, and CNS; it usually follows a group A beta-hemolytic streptococcal pharyngitis; it often recurs

Pathogenesis involves cross-reactivity between the immune response to cell surface antigens of the strep and antigens on cardiac myocytes and with heart valve glycoprotein
RHEUMATIC HEART DISEASE

Acute Rheumatic Fever is a PANCARDITIS, involving all layers of the heart. Pericarditis and myocarditis often responsible for initial symptoms.

PERICARDITIS - fibrinous

MYOCARDITIS

Aschoff bodies: Perivascular nodules of inflammatory cells including multinucleated Aschoff cells, Anitschkow cells, lymphocytes, and plasma cells

Myocarditis can lead to CHF and even death

ENDOCARDITIS - initially results in tiny vegetations along lines of closure of mitral and aortic valves, with little functional significance
With repeated episodes of Rheumatic Fever, the valve damage progressively increases. Nevertheless, it requires many years or decades before valvular damage becomes functionally significant. The latency may reflect slowly progressive, cumulative effects of turbulence created by relatively mild deformity as well as the direct effect of inflammation. Valvular deformities consist of thickening, fusion, and shortening of chordae tendineae, and fibrosis and fusion of commissures. Calcification is also common.

Valves become stiff and neither open fully nor close completely; therefore, often there is a combination of stenosis and insufficiency, stenosis often more severe.
Rheumatic aortic valve disease with fusion of commissures, resulting in stenosis and insufficiency
Consequences of Valve Damage

Acute regurgitation produces worst symptoms

Aortic valve disease affects mostly left ventricle

   Stenosis $\rightarrow$ LV hypertrophy initially

   Regurgitation $\rightarrow$ LV dilation, then hypertrophy

Mitral valve disease affects left atrium, lungs

  Stenosis and regurgitation both cause increased left atrial pressure and pulmonary congestion
Common Etiologies of Valvular Disease in Adults

Mitral Stenosis - mostly rheumatic

Mitral Insufficiency (Regurgitation) - rheumatic, annular dilation, prolapse, acute endocarditis

Aortic Stenosis - rheumatic, congenital defect (bicuspid valve), degenerative (calcific)

Aortic Insufficiency - rheumatic, acute endocarditis, dilation of proximal aorta

Tricuspid and Pulmonic Valves can have similar abnormalities as above (except prolapse)
Other Categories of Heart Disease

Congenital Heart Disease

Cardiomyopathies
Congenital Heart Defects

Incidence: 0.3 - 1.0% of live births

Causes shunts or obstructions or both

Virtually any structure can form abnormally; most common is a Ventricular Septal Defect

Shunts classified as right-to-left or left-to-right
Ventricular Septal Defect

Normal anatomy

VSD
Congenital Heart Defects

Left-to-right shunts $\rightarrow$ pulmonary congestion

Right-to-left shunts $\rightarrow$ cyanosis

Congenital defects can also cause murmurs
Cardiomyopathies

Intrinsic diseases of the myocardium

Classified as:
  - dilated
  - hypertrophic
  - restrictive
Dilated Cardiomyopathy

Typically biventricular dilation, due to poor contractile function of individual myocytes

Dilation allows cardiac output to be maintained with low ejection fraction

Usually some degree of compensatory hypertrophy
Hypertrophic Cardiomyopathy

Due to various genetic mutations in contractile proteins, resulting in synthesis of abnormal proteins that get incorporated into myofilaments

Autosomal dominant inheritance with variable penetrance; also can be spontaneous mutation

Asymmetric hypertrophy of interventricular septum; good contractility but high risk of exercise-induced arrhythmias and stiff ventricle
Restrictive Cardiomyopathy

Heterogeneous group of diseases characterized by a stiff ventricle (diastolic dysfunction) with initially good contractility

Example is amyloid: proteinaceous interstitial deposits reduce ventricular compliance

Left atrium is dilated and LV end-diastolic pressure is increased; generalized thickening of ventricular wall
Any questions about heart diseases?
Clinical Scenario to Illustrate Principles of Normal Heart Function and Dysfunction

A 55 year old man comes to the hospital because he feels severe crushing chest pain, like someone is standing on his chest, which started very suddenly.

Most likely explanation for these symptoms is?
He says that he has had similar symptoms before. It occurs when he is exercising, and in the past, it went away after a few minutes when he stopped exercising. He had seen a physician about this 6 months ago, and was given a prescription for pills to take when the symptoms occur. He puts a pill under his tongue, and now the symptoms subside even faster, within 1-2 minutes after he sits down and rests.

Why do the symptoms go away?
This time it is different. The symptoms did not go away when he took the pill and sat down. Instead, the discomfort continued. He felt his heart rate increasing and he became sweaty and felt nervous and jittery. He also felt like he was having trouble catching his breath and he was breathing more rapidly.

What might be different this time that kept the symptoms from going away?

What explains the new symptoms?
If the patient said that the chest discomfort had started 2 hours ago, what would be the logical approach for treatment?

If the patient did not come to the hospital until 10 hours after the discomfort started, what would be the logical approach?
Suppose the patient had seen a cardiologist earlier in the week, and some routine tests were done. At that time he felt fine. An echocardiogram was done, and it showed that all segments of the left ventricular wall were contracting normally and the ejection fraction was 55%. The left atrium, right ventricle, and right atrium were all normal size. The four cardiac valves were all functioning normally. An electrocardiogram was normal. Blood pressure is 120/80 (normal).
Suppose those same tests are repeated now. Suppose the echocardiogram show that the anterior and lateral wall of the left ventricle and anterior interventricular septum are contracting normally, but the posterior wall of the left ventricle and posterior third of the interventricular septum is not contracting, and is actually bulging during each heart beat and the ejection fraction is 35%.

What does this suggest?
The left atrium is now dilated, and there is mild mitral valve regurgitation. Blood pressure is 120/80 (unchanged).

Why does contractile function decrease?

Why does the ejection fraction decrease?

Why is there mitral valve regurgitation?

Why is blood pressure unchanged?
The electrocardiogram shows there is now a delay between atrial electrical activation and ventricular electrical activation and there are occasional atrial beats that are not followed by ventricular electrical activation. There are occasional premature beats.

What is responsible for these abnormalities?
A chest Xray is performed, showing a mild increase in heart size and a mild increase in lung density, interpreted as congestion and mild pulmonary edema.

What is responsible for these abnormalities?
What are possible outcomes for this patient?
If the infarct heals, the ejection fraction remains low, but nothing else detrimental happens, what impact will this have?
Stroke Volume
LV End-Diastolic Volume
Normal
Failing
Pressure
Volume
ESPVR
Normal
ESPVR
Failing
EDV
Stroke Volume
LV End-Diastolic Volume (or End-Diastolic Pressure)
Images from Netter’s *Atlas of Human Anatomy*, Sheppard and Davies’ *Practical Cardiovascular Pathology*, Harrison’s *Internal Medicine*, and Don Bers’ website
Additional reading

• Robbins Textbook of Pathology
• UpToDate (www.uptodate.com)
• American Heart Association (www.americanheart.com)