Heart Disorders

- Heart disease is ranked as a major cause of death in the U.S.
- Common heart diseases include:
  - Congenital heart defects
  - Hypertensive heart disease
  - Angina
  - Heart attacks
  - Cardiac arrhythmias
  - Congestive heart failure

Diagnostic Tests for CV Function

- ECG
- Auscultation
- Exercise stress tests
- Chest x-ray films
- Cardiac catheterization
- Angiography
- Doppler studies
- Blood tests
- Arterial blood gas determinations
Treatment for Cardiac Disorders

- Dietary modifications
- Regular exercise program
- Cessation of cigarette smoking
- Drug therapy:
  - Vasodilators
  - Beta-blockers
  - Calcium channel blockers
  - Digoxin

Coronary Artery Disease (CAD) or Ischemic Heart Disease (IHD)

- CAD includes:
  - Angina pectoris or temporary cardiac ischemia (partial obstruction leading to decreased blood flow)
  - Myocardial infarction or heart attack (total obstruction leading to necrosis)
- CAD may ultimately lead to:
  - Heart failure
  - Serious arrhythmias
  - Sudden death
- Basic problem in CAD is insufficient oxygen for the needs of the heart muscle
Arteriosclerosis and Atherosclerosis

- **Arteriosclerosis** is a general term for all types of arterial changes
  - Best applied to:
    - Degenerative changes in small arteries and arterioles commonly in older people and diabetics
    - Elasticity is lost, walls become thick and hard and the lumen gradually narrows
    - Leads to diffuse ischemia and necrosis in various tissues like kidneys, brain or heart

- **Atherosclerosis** is differentiated by the presence of atheromas, plaques consisting of lipids, cells, fibrin and cell debris, often with attached thrombi which form inside large arteries

- **Atheromas** form primarily in large arteries such as the aorta, iliac arteries, coronary arteries and the carotid arteries especially where they bifurcate
  - Lipids or fats, transported as lipoprotein (LDL) play important role in atheroma formation
  - Process appears to begin with injury to the endothelial lining of vessels, often at an early age
HDL vs LDL
Transport of Lipids

Development of an Atheroma

Consequences of Atherosclerosis
Coronary Artery Bypass

Angina pectoris
- This chest pain occurs when there is a deficit of oxygen for the heart muscle.
- Can occur when:
  - The blood supply (partial occlusion causing ischemia) or oxygen supply (anemia) to the myocardium is impaired.
  - When the heart is working harder than usual and needs more oxygen.
  - When a combination of these factors is present.
- Anginal pain is usually quickly relieved by rest and administration of vasodilators.

Angina Pectoris
Myocardial Infarctions

- A myocardial infarction (MI) or heart attack occurs when a coronary artery is totally obstructed leading to prolonged ischemia and cell death or infarction.
- MI is characterized by persistent chest pain radiating down the left arm, pallor and rapid, weak pulse.
- The most common cause is atherosclerosis, usually with thrombus attached
- Necrosis of cardiac tissue begins to occur after about 20 minutes of hypoxia.

Infarction may occur in three ways:

- The thrombus may build up to obstruct the artery
- Vasospasm may occur in the presence of a partial occlusion by an atheroma leading to total obstruction
- Part of a thrombus may break away, forming an embolus that flows through the coronary artery until it lodges in a smaller branch

Most MI's are transmural, involving all three layers of the heart
- Most involve the left ventricle
- The size and location of the infarct determine the severity of the damage
- At the point of obstruction, the heart tissue becomes necrotic and an area of injury and inflammation develops around the necrotic zone
With cell necrosis, specific enzymes are released into the tissue fluid and blood and are useful in diagnosis.

Since myocardial cells do not reproduce, the area of necrosis is gradually replaced by fibrous, nonfunctional tissue.

If thrombolytic therapy is administered within 20 minutes of the onset, blood flow can be restored and permanent damage averted.

Exercise may help prevent a second heart attack.
Cardiac Arrhythmias

- Cardiac arrhythmias are the most common cause of death immediately following MI.
- Alterations in the cardiac rate may be caused by:
  - Damage to the heart’s conduction system
  - Electrolyte abnormalities
  - Fever
  - Hypoxia
  - Stress
  - Infections
  - Drug toxicities

ECG provides a method of monitoring the conduction and detecting abnormalities

Arrhythmias reduce the efficiency of the cardiac cycle, reducing cardiac output and affecting the blood supply to vital organs and tissues.
Sinus Node Abnormalities

- Bradycardia-regular but slow heart rate, less than 60 beats per minute; usually due to vagal (parasympathetic) nerve stimulation
- Tachycardia-regular but rapid heart rate, 100-160 bpm due to:
  - Sympathetic stimulation
  - Exercise
  - Fever
  - Stress
  - Decreased blood volume

Sick sinus syndrome-a heart condition marked by alternating bradycardia and tachycardia often requires a pacemaker

Atrial Conduction Abnormalities

- Premature atrial contractions or beats (PAC/PAB) are ectopic beats of the atria usually caused by a focus of irritable atrial muscle cells outside the conduction pathway
- Atrial flutter refers to a heart rate of 160-350 bpm
Atrial Conduction Abnormalities

- Atrial fibrillation is a rate over 350 bpm
- Since the AV node delays conduction the ventricular rate is slower (80-180 bpm)

“Hot spots” in the atrial muscle or pulmonary veins

More that 2 million Americans have atrial fibrillation
- Sometimes electric shock or drugs can be used to reestablish a normal sinus rhythm
- If there are no serious symptoms, drugs to control the rate of ventricular contractions and a blood thinner to prevent clot formation and stroke may be used
- May experience lightheadedness, weakness, shortness of breath or even chest pain.
Radiofrequency energy used to treat atrial fibrillation

Heart block occurs when conduction is excessively delayed or stopped at the AV node or bundle of His.

Heart blocks may be:
- **First degree**, in which the conduction delay prolongs the PR interval.
- **Second degree**, in which a longer delay leads to a missed ventricular contraction periodically.
- **Third degree** or **total heart block** occurs when there is no transmission of impulses from atria to ventricles; spontaneous ventricular rate 30-45 bpm.
Ventricular Conduction Abnormalities

- **Bundle branch block** refers to interference with conduction in one of the bundle branches; usually does not alter cardiac output but appears on ECG.
- **Ventricular tachycardia** is likely to reduce cardiac output because of reduced filling time.

Ventricular Conduction Abnormalities

- In **ventricular fibrillation**, muscle fibers contract independently and rapidly without ejecting blood; the severe hypoxia to the myocardium causes contraction to stop.
Treatment for Cardiac Arrhythmias

- Problems caused by drugs like digitalis toxicity, beta blockers or potassium imbalance due to diuretics should be corrected
- Antiarrhythmic drugs (e.g. digoxin) can help in the case of heart damage
- SA nodal problems or total heart block requires a pacemaker

Cardiac Arrest or Standstill

- Cardiac arrest is cessation of all activity in the heart:
  - No conduction of impulses
  - ECG shows flat line
  - No cardiac output
  - Loss of consciousness due to lack of oxygen to brain
  - Respiration ceases
  - No pulse
Congestive Heart Failure

- CHF occurs when the heart is unable to pump sufficient blood to meet the metabolic needs of the body.
- CHF usually occurs as a complication secondary to another condition:
  - May present as an acute episode but is usually a chronic condition.
  - May result from a problem with the heart, such as an infarct or valve defect.
  - May be due to increased demands on the heart, such as in hypertension or lung disease.
  - May result from a combination of these factors.

Depending on the cause, one side of the heart usually fails first:

- An infarct in the left ventricle or essential hypertension (high blood pressure) affects the left ventricle first.
- Pulmonary valve stenosis or pulmonary disease affects the right side first.

Various compensatory mechanisms maintain cardiac output for a while:

- Reduced blood flow to the systemic circulation increases renin and aldosterone secretion.

The SNS response increases heart rate and peripheral resistance
- This may decrease efficiency of the heart by impeding filling
- Increases work load on heart muscle
- Hearts chambers tend to dilate and cardiac muscle hypertrophies (cardiomegaly)
  - Increases circulatory requirements for the enlarged heart muscle
  - Myocardial cells begin to die; replaced by CT

**Effects of CHF**

- Cardiac output or stroke volume decreases resulting in less blood reaching tissues
  - Decreased cell function
  - Fatigue
  - Lethargy
  - Mild acidosis develops
  - Venous return to that side of the heart impeded
- "Backup" congestion develops in the circulation behind the affected ventricle
Hypertension

- Hypertension is a common problem
  - Primary or essential hypertension is ideopathic (basic change is increased systemic vasoconstriction)
  - Secondary hypertension results from renal or endocrine disease
    - Renal nephrosclerosis causes renin release
    - Hyperaldosteronism causes excessive salt and water retention
    - Pheochromocytoma is benign tumor of adrenal medulla or SNS chain ganglia
  - Malignant hypertension is an uncontrollable, severe and rapidly progressive form

Study: Heart-failure treatment inadequate

By ANGELA V. HANLEY

ORLANDO, Fla. — Despite new drugs that lower blood pressure and improve heart function, many patients who have undergone surgery for heart failure continued to have trouble breathing and other symptoms of heart failure.

The report documented what doctors see as a common scenario: Patients who are being treated for heart failure with medications that are known to improve heart function and lower blood pressure, but who continue to experience symptoms of heart failure.

The report also highlights the challenges of treating heart failure patients, who often have multiple comorbidities and require complex medication regimens.

The study found that even with the best care, many patients continue to experience symptoms of heart failure, which can be managed with aggressive treatment and close monitoring.

Figure 18-29

Development of hypertension.
Classification of Hypertension

- May be classified as systolic or diastolic depending on which measurement is elevated:
  - Elderly people with loss of elasticity in their arteries often have high systolic and low diastolic
  - Essential hypertension develops when blood pressure is consistently above 140/90
  - The diastolic pressure is important because it indicates the degree of peripheral resistance and increased work load on the left ventricle
  - May be classified as mild, moderate or severe

Circulatory Shock

- Shock or hypotension results from a decreased circulating blood volume and tissue perfusion; in most cases cardiac output is low
- Several types of shock classified by cause:
  - Hypovolemic shock (hemorrhagic shock)
  - Cardiogenic shock
  - Anaphylactic shock
  - Neurogenic shock
  - Septic shock

Table 18-3

<table>
<thead>
<tr>
<th>Types of Shock</th>
<th>Mechanism</th>
<th>Specific Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>Loss of blood or plasma</td>
<td>Hemorrhage, burns, dehydration, peritonitis, pancreatitis, pulmonary embolus, cardiac tamponade</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>Decreased pumping capability of the heart</td>
<td>Myocardial infarction of left ventricle, cardiac arrhythmia, pulmonary embolus, cardiac tamponade</td>
</tr>
<tr>
<td>Anaphylactic</td>
<td>Systemic vasodilation and increased permeability due to severe allergic reaction</td>
<td>Insect stings, drugs, nuts, shellfish</td>
</tr>
<tr>
<td>Neurogenic or distributive</td>
<td>Vasodilation due to loss of sympathetic and vaso-motor tone</td>
<td>Pain and fear, spinal cord injury</td>
</tr>
<tr>
<td>Septic (septic)</td>
<td>Vasodilation due to severe infection, often with gram-negative bacteria</td>
<td>Virulent microorganisms or multiple infections</td>
</tr>
</tbody>
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Table 18.4

<table>
<thead>
<tr>
<th>Manifestations</th>
<th>Rationale</th>
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</thead>
<tbody>
<tr>
<td>Early signs</td>
<td>Anxiety and restlessness</td>
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<tr>
<td></td>
<td>Cool, pale, mold skin</td>
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<td></td>
<td>Oliguria</td>
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<td>Tachycardia</td>
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<td></td>
<td>Rapid respiration</td>
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<tr>
<td>Progressive</td>
<td>Lethargy, weakness, faintness</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
</tr>
</tbody>
</table>
Major defect in hemorrhagic shock is decreased cardiac output caused by decreased blood volume.

Cardiovascular Shock may also be cardiogenic in origin (also results in decreased cardiac output.)