Disorders of Cardiac Function

Objectives:
• Pericardial and endocardial disorders.
• Cardiomyopathies and cardiac arrhythmias.
• Valvular and congenital heart disorders.
• Coronary artery disease, myocardial ischemic disease and myocardial infarction.

Part 1: Pericardial and Endocardial Disorders
Acute Pericarditis:
Inflammation of the pericardium

Etiologies:
• Bacterial/viral infections
• Autoimmune diseases
• Renal failure
• Radiation
• Idiopathic

Acute Pericarditis:
Manifestations:
• Chest pain
• Pericardial friction rub
• ECG changes
Constrictive Pericarditis: characterized by the development of fibrous scar tissue between visceral and parietal pericardium

- Over time, the scar tissue may shrink and interfere with the filling ability of the heart leading to reduced cardiac output.

Constrictive Pericarditis:

Manifestations:
- Inflammatory indicators in blood
- Ascites and edema in extremities
- Jugular venous distention
- Kussmaul sign: increased jugular venous pressure during inspiration
- Exercise intolerance, dyspnea
- Muscle wasting, weight loss

Pericardial Effusion: accumulation of fluid in the pericardial cavity

Etiologies:
- Malignancies
- Cardiac Surgery
- Trauma
- Cardiac rupture
- Dissecting aneurysm
- Infections
- Autoimmune Disease
Pathogenesis:
- The left ventricle becomes compressed from within by fluid in the pericardium and the interventricular septum
- Increases intercardiac pressure
- Decreased ventricular diastolic filling
- Decreased SV and CO
- Increased central venous pressure
- Jugular venous distention
- Decreased blood pressure, could lead to circulatory shock

Cardiac Tamponade: a life-threatening compression of the heart due to fluids in the pericardial cavity.
Pathogenesis:
- The left ventricle becomes compressed from within by fluid in the pericardium and the interventricular septum
- Increases intercardiac pressure
- Decreased ventricular diastolic filling
- Decreased SV and CO
- Increased central venous pressure
- Jugular venous distention
- Decreased blood pressure, could lead to circulatory shock
Cardiac Tamponade:

Diagnosis:
- Heart sounds distant and muted
- **Pulsus paradoxus**: accentuated decrease in blood pressure during inspiration
- Echocardiogram
- ECG (nonspecific T-wave changes)
- Aspiration (laboratory analysis of fluid biopsy)
Cardiac Tamponade:

Treatment:
- Anti-inflammatories, pericardiocentesis, surgery

Infective Endocarditis:

infection of the endocardium and heart valves

Pathogenesis:
- Vegetative formations develop that are made up of infectious organisms, cellular debris and clotted blood.
- These vegetations have the ability to release bacteria into the blood causing other infections in other parts of the body as well as immune disorders.
- As vegetations grow, they destroy heart tissues and valves causing heart arrhythmias, pericarditis, emboli and aneurysms.
Infective endocarditis is an infection of the heart chambers or valves.
Infectious Endocarditis:

Etiology:
- Primarily bacterial infections (staphylococcal, streptococcal)
Risk Factors:
- Previously damaged endocardial surfaces
- IV drug use
- Immunodeficiencies
- Diabetes
- Cardiovascular devices
**Infectious Endocarditis:**

**Manifestations:**
- Fever
- Immune responses
- Petechiae and splinter hemorrhages

**Treatment:**
- Antibiotics
- Care for emboli and damaging heart effects
- Surgery

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**Rheumatic Heart Disease:** results from rheumatic fever and subsequent chronic rheumatic heart disease due to complications of a Strep-A throat infection.

- Antibodies produced against pathogen also attack similar antigens on heart endocardium, joints and other tissues.

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**Rheumatic Heart Disease:**

**Manifestations:**
- Usually occur 2-3 weeks after infection
- Acute, recurrent or chronic
  - Aschoff body: local lesion of tissue necrosis surrounded by immune cells
  - Migratory polyarthritis of large joints
  - Subcutaneous nodules
  - Erythemia margination
  - Sydenham chorea
- Chronic phase: characterized by permanent deformity of heart valves possibly leading to mitral valve stenosis or other valvular issues.
Part 2: Cardiomyopathies and Cardiac Arrhythmias

Diseases of the Myocardium:
- Primary Cardiomyopathies: confined to myocardium
- Secondary Cardiomyopathies: changes that occur to myocardium as a result of systemic disorders.

Primary Cardiomyopathies:
Hypertrophic Cardiomyopathy (HCM): left ventricular hypertrophy
- Most common cause of sudden cardiac death (SCD) in young athletes.
- Idiopathic or autosomal dominant
- Mainly asymptomatic, if symptoms: dyspnea, chest pain with exertion, syncope, exercise intolerance
- Tx: beta blockers, Ca2+ blockers, anti-arrhythmia drugs, cardioverter defibrillators (ICDs)
Dilated Cardiomyopathy (DCM):
• Caused by a combination of genetic and acquired etiologies
• Heart becomes dilated, flabby and hypertrophic, loses contractility
• Most common cause for heart transplantation
**Primary Cardiomyopathies:**

**Myocarditis:** Inflammatory cardiomyopathy

Manifestations:
- Fever, chest pain, exertional dyspnea, if severe: circulatory collapse and sudden death.

Etiologies:
- Viral, bacterial, fungal infections
- Drug hypersensitivities
- Autoimmune disorders
- Immunodeficiencies

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**Primary Cardiomyopathies:**

**Tako-Tsubo Cardiomyopathy:**

- Transient and reversible left-ventricular dysfunction in response to profound psychological or emotional stress

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**Primary Cardiomyopathies:**

**Peripartum Cardiomyopathy:**

- Can occur at end of pregnancy or several months after delivery
- Rare inflammatory response that can potentially lead to heart failure
Heart Conduction Disorders (Arrhythmias):

- Some arrhythmias can cause palpitations (patient can feel heart beat) while many are asymptomatic
- Some are relatively harmless, some can lead to serious conditions
- Can be persistent or transient
- Due to genetics and/or heart damage
- Treatment: anti-arrhythmia drugs, ablation, heart devices (pacemakers, implantable cardioverter debrillators) and portable debrillators.

Supraventricular Arrhythmias:

- atrial origin
- Premature Atrial Contraction (PACs)
- Atrial Fibrillation
- Atrial Flutter
Ventricular Arrhythmias: ventricular origin

- Premature Ventricular Complex (PVC)
- Ventricular Fibrillation (V-Fib)

PREMATURE VENTRICULAR CONTRACTION
A single impulse originates at right ventricle
Sinus Node Arrhythmias:

- Sinus Tachycardia
- Sinus Bradycardia

Atrioventricular Block:
heart conduction issue

Heart Blocks between SA node and AV node:
- First degree
- Second degree
- Third degree
First degree AV block

- Prolonged PR interval

A-V BLOCK, SECOND DEGREE

Sudden dropped QRS-complex

Intermittently dropped ventricular beat
Part 3: Valvular and Congenital Heart Disorders

Third degree AV block or complete AV block

*None of P waves conduct to ventricles (P-P and QRS-QRS are independent)
*Slow, regular ventricular escape rhythm
Valvular Disorders:

- Produce abnormal heart sounds because they reflect defective movement of blood through the valve (heart murmur)
- Some are harmless, others are severe and need treatment
- Defective valves over time lead to heart muscle dilation and hypertrophy, ultimately failure

Etiologies:

- Congenital defects, infections, heart damage

Valvular Disorders:

**Regurgitation:** (insufficiency/incompetence) valves do not completely close and blood flows backwards.

**Stenosis:** valves become narrowed so heart must pump against additional resistance
Valvular Disorders:

**Mitral Valve Stenosis:** narrowing of mitral valve  
Pathogenesis:  
• Heart sound: a snap is heard before the second heart sound (dub) and turbulence during diastole (diastolic murmur)  
• Left ventricle receives less blood, reduced CO  
• Left atrium accumulates blood  
• If untreated: the left atrium stretches over time, works harder, thickens, hypertrophies and can eventually fail

Valvular Disorders:

**Mitral Valve Regurgitation:** weakness, leakage of mitral valve  
Pathogenesis:  
• Heart sound: systolic murmur  
• Valve does not close properly during systole so when the ventricle contracts, blood can go backwards and forwards at the same time  
• Same general pathogenesis as mitral valve stenosis

Valvular Disorders:

**Mitral Valve Prolapse:** mitral valve cusps degenerate, become enlarged and lose tone, eventually prolapsing (balloon push back into the left atrium during systole)  
• Genetics (collagen defects)  
• Asymptomatic, but if symptomatic: angina, fatigue, palpitations, dizziness
Valvular Disorders:

Diagnosis:
- Cardiac auscultation (heart sounds)
- Echocardiography
- Cardiac catheterization

Treatment:
- Surgery to repair or replace defective valves
- Percutaneous balloon valvuloplasty
- Management of symptoms and heart failure if it has occurred

Mitrail Stenosis

[Diagram of narrowed mitral valve]

Regurgitation

[Diagram of normal blood flow]

Normal blood flow

Regurgitation
Congenital Heart Disorders:
- Occur during 4-7th week of embryonic development
- Multifactorial etiologies: genetics, environment
- Diagnosis: ultrasound, echocardiography
- Manifestations: cyanosis, respiratory difficulty, fatigue, failure to thrive, exercise intolerance, angina

Congenital Heart Disorders:
- Patent Ductus Arteriosus: persistence of ductus arteriosus
- Atrial and Ventricular Septal Defects
- Tetralogy of Fallot: ventricular septal defect, shift of aorta position, abnormal pulmonary trunk, hypertrophy of right ventricle
- Coarctation of the aorta
- Transposition of the great arteries
Pathogenesis:

- Shunting: Blood is diverted from one circulatory system to another leading to cyanosis
  - Left to right shunt (arterial to venous)
  - Right to left shunt (venous to arterial)
- Severity based on the location of the abnormal openings between the circulatory pathways and the amount of resistance to blood flow in these openings.
- Most severe issues can lead to heart failure.
Part 4: Coronary Artery Disease, Myocardial Ischemic Disease and Myocardial Infarction

Myocardial Ischemic Disease: a disease process in which blood flow to heart muscle is reduced over time.

- Coronary Artery Disease (CAD): accumulation of plaque along the coronary arteries (atherosclerosis)
- Other etiologies: coronary spasm, cardiac arrhythmias, anemia, hypertension, valvular heart disease
Clinical manifestations of Myocardial Ischemic Disease:

Reversible:
- Stable Angina
- Prinzmetal Angina
- Silent Ischemia

Acute Coronary Syndromes:
- Unstable Angina
- Myocardial Infarction

Reversible MID manifestations:

Stable Angina:
- Chest pain with transient myocardial ischemia
- Radiating pain (relieved by rest and nitroglycerin)
- May be mistaken for indigestion
- Perspiration, difficulty breathing
Reversible MID manifestations:

**Prinzmetal Angina:**
- Chest pain with transient myocardial ischemia
- Pain is caused by vasospasm of one or more coronary arteries.

**Silent Ischemia:**
- Vague symptoms such as fatigue, feeling of unease, breathlessness
- More often experienced by females

Acute Coronary Syndrome manifestations:

**Unstable Angina:**
- Resulting from reversible myocardial ischemia
- Atheroma has become complicated and infarction may soon follow
- Angina occurs at rest and during activity.
Acute Coronary Syndrome manifestations:
Myocardial Infarction:
• Non-ST elevation MI (non-STEMI): less severe, only a portion of a blood vessel/s occluded and limited portions of heart tissue are lost.
• ST-elevation MI (STEMI): more severe, blood vessel/s are completely occluded and there is greater loss of heart tissue.
Pathophysiology of myocardial ischemic disease that leads to myocardial infarction:

- Partially occluded vessels lead to increased resistance, decreasing blood flow to heart muscle. Hypoxia starts to develop in heart muscle cells. Normally, blood vessels vasodilate in hypoxic conditions, but because of fibrous changes the coronary blood vessels stiffen and can’t effectively vasodilate.
- Episodes of myocardial ischemia occur especially during exertion with or without angina.

Pathophysiology of myocardial ischemic disease that leads to myocardial infarction:

- As heart muscle cells become more and more ischemic, the cardiovascular reflex occurs (sympathetic nervous system). Heart rate is increased and there is increased vasoconstriction of blood vessels.
- This leads to increased peripheral resistance (TPR) and increased afterload, increased venous return and increased stroke volume and cardiac output. These compensations work temporarily.
Pathophysiology of myocardial ischemic disease that leads to myocardial infarction:

- The heart hypertrophies to deal with increased work load, but it also requires more oxygen and energy.
- Cardiac muscle continues to become more hypoxic, cells utilize anaerobic respiration, pH decreases and contractility is less and less effective. This leads to reduced SV, Bp and CO.
- Cardiovascular reflex continues to try to compensate.
- Eventually, the renin loop kicks in due to reduced blood flow and blood pressure to the kidney. Renin ultimately causes the heart to work even harder.

Pathophysiology of myocardial ischemic disease that leads to myocardial infarction:

- Eventually, prolonged (30 minutes or longer) rather than periodic ischemia results as blockage of blood vessel/s is more complete and complicated. A myocardial infarction occurs when blood supply is cut off from heart muscle tissue and myocardial cells die.

Effects during a myocardial infarction (MI):

- Oxygen deprivation to heart muscle is usually accompanied by electrolyte disturbances.
- Heart cells release catecholamines which affect the autonomic nervous system. Heart rate can accelerate and cardiac arrhythmias may occur. This also causes the release of glycogen, glucose and fat into the blood approximately one hour after a myocardial infarction. Hyperglycemia is present around 72 hours after an acute myocardial infarction.
- Angiotensin II is released during an MI, causing increased peripheral vasoconstriction, coronary artery spasm and fluid retention.
Effects during a myocardial infarction (MI):

- MI results in abnormal ventricular function leading to decreased ejection fraction and increased end diastolic volume (EDV), reduced cardiac output.
- When cardiac muscle cells die, muscle components such as creatinine, phosphokinase and troponin leak into the blood.
- Cardiac tissue surrounding the area of the infarction is vulnerable, oxygen-deficient and undergoes structural changes (hypertrophy and reduced contractility).
- In the area of myocardial necrosis, a severe inflammatory response occurs and wound repair begins as damaged cells are degraded and fibroblasts produce scar tissue.
Possible Clinical Manifestations of Myocardial Infarction:

- Some are silent
- Sudden, severe chest pain (heavy and crushing), persistent
- Radiating pain (neck, jaw, back, shoulder, left arm)
- Indigestion that does not go away
- Excessive perspiration, cool and clammy skin
- Difficulty breathing
- Sense of impending doom
- Nausea and vomiting

Diagnosis:

- Serum cardiac troponins (I and II)
- Serum creatine kinase (CK) and lactic dehydrogenase (LDH)
- ECG
- Leukocytosis
- Serum CRP
- ESR
- Elevated glucose level
- Hypoxemia
- Physical exam and medical history
Treatment (dependent on severity):
• Supplemental oxygen & aspirin (or ticlopidine if allergic to aspirin)
• Sublingual nitroglycerine and morphine sulfate for pain relief
• Fluids/electrolytes
• Continuous monitoring of cardiac rhythms and enzymatic changes in blood
• Thrombolytic and anti-arrhythmia drugs
• Possibly: ACE inhibitors and beta blockers
• Moderate MI: vasodilator drugs, severe MI: vasoconstrictor drugs
• Coronary intervention

Coronary Intervention:
• Angioplasty
• Arthrectomy
• Coronary Bi-pass
Possible Complications of MI:
- Dangerous heart arrhythmias
- Pericarditis/pericardial effusion/cardiac tamponade
- Cardiogenic shock
- Stroke
- Thromboemboli
- Cardiac rupture
- Sudden Cardiac Death (SCD)

Treatment (long-term):
- Bed rest followed by a slow return to daily activities
- Thrombolytic drugs
- Stool softeners
- Hyperlipidemia treatment (Statins)
- Diet, exercise, stress reduction
- Cessation of caffeine, smoking, alcohol