Pathophysiology of Ischemic Heart Disease

Etiology of Ischemic Heart Disease

- Heart rate
- Afterload
- Preload
- Contractility

Supply
- Coronary Artery
- O₂ extraction
- Diastolic filling time
Ischemic Heart Disease

- Coronary artery disease – leading cause of death in industrialized countries – type of ischemic heart disease
  - Leads to angina, myocardial infarction, sudden cardiac death, and chronic heart failure

- Causes – Modifiable and non-modifiable risk factors
  - Arteriosclerosis – natural changes in the intima, connective tissue, and diameter of artery
  - Atherosclerosis – pathologic phenomenon occurring in the coronary, carotid, iliac, and femoral arteries as well as the aorta (coronary artery disease)
This is a normal coronary artery. The lumen is large, without any narrowing by atheromatous plaque. The muscular arterial wall is of normal proportion.

How atherosclerosis develops
(www.merck.com)
The atherosclerotic process

Response to Injury hypothesis
- inflammatory response resulting in proliferation of tissue within the arterial wall which may result in obstruction of blood flow

Causes:
- elevated levels of cholesterol and triglyceride in the blood,
- high blood pressure – turbulent blood flow
- tobacco smoke
- glycosylated substances

Response to Injury Hypothesis (pg. 52-55, Brubaker text)

1. Injury to endothelium causing to platelets adhere to endothelium then release of growth factors
2. Monocytes attach to endothelium and penetrate (also LDL receptor activation) – monocytes become macrophages and take up LDL and SMC’s
3. Smooth muscle cell proliferation and migrate from medial to intimal layer
4. Foam cells are formed - migration to the intima smooth muscles with lipids form fatty streaks
5. Fibromuscular plaque – fibromuscular layer with cholesterol core
High cholesterol → Insulin resistance
Hypertension → Endothelial damage
Smoking → Monocyte
Monocyte adhesion and penetration → Fatty streak
Smooth muscle proliferation and migration → Foam cells (macrophages and smooth muscles containing lipid) and fatty streaks appear
Prominuous plaque

Figure 2.14 A cross section of a diseased coronary artery (eccentric lesion of the arterial wall).
Atherosclerotic Plaque

Blocked Coronary Artery

Cross Section of a Partially Blocked Coronary Artery

Coronary artery
Heart muscle deprived of oxygen
Fatty deposits

Schematic Area
Ischemic Heart Disease

- A result of CAD (atherosclerosis)
- Imbalance between supply and demand
- Narrowing and hardening of the arteries leads to imbalance between the supply and demand of blood for cardiac muscle = Ischemia
- Ischemia is either detected by a symptom (angina) or indirectly by electrocardiogram and other non-invasive and invasive diagnostic techniques

Classic symptoms of IHD:

**Angina pectoris**

- **Angina pectoris** – transient, referred cardiac pain resulting from myocardial ischemia
  - Usually in substernal region, jaw, neck, or arms, may also be in epigastrum and interscapular regions
  - Symptomology – pressure, heaviness, fullness, squeezing, burning, aching, choking, or even dyspnea
Types of Angina and Associated Pathophysiology

- **Typical Angina** – evoked by exertion, emotions, cold/heat exposure, meals, and sexual intercourse; relieved by rest or nitroglycerin
  - **Stable Angina** – reproducible and predictable in onset

- **Atypical Angina** – no relationship to exertion
  - **Unstable Angina** – new onset of typical angina, increasing in intensity or occurs at rest
  - **Variant (Prinzmetal’s angina)**

IHD: Demand > Supply

An ischemic state may result in one or more of the following symptoms or conditions
- Angina
- Myocardial infarction (heart attack)
- Silent Ischemia
  - Asymptomatic episodes of myocardial ischemia in those with CAD Dx
- Syndrome X
  - Symptoms of angina pectoris with no evidence of significant atherosclerosis.
  - May be due to problems in smallest of coronary arteries that are not visualized by angiographic techniques