# CARDIOVASCULAR SYSTEM Part 1

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Ischemic Heart Disease
(Coronary Heart Disease)

* A group of closely related syndromes caused by an imbalance between the myocardial oxygen demand and blood supply.
* Angina pectoris (chest pain).
* Acute myocardial infarction.
* Sudden cardiac death.
* Chronic ischemic heart disease with congestive heart failure.

Ischemic Heart Disease: Epidemiology-(coronary atherosclerosis)

* Peak incidence: 60y for males and 70y for females.
* Men are more affected than women until the ninth decade.

Contributing factors:

* Hypertension.
* Diabetes mellitus.
* Smoking.
* High levels of LDL.
* Genetic factors (direct or indirect).
* Lack of exercise.

Pathogenesis of Ischemic Heart Disease

1) Role of Critical stenosis or obstruction:

(>=75% of the lumen of one or more coronary arteries by atherosclerotic plaque).

2) Role of Acute Plaque Change:

In most patients the myocardial ischemia underlying unstable angina, acute MI, and (in many cases) sudden cardiac death is precipitated by abrupt plaque change followed by thrombosis . Most often, the initiating event is disruption of previously only partially stenosing plaques with any of the following:

* Rupture/fissuring, exposing the highly thrombogenic plaque constituents
* Erosion/ulceration, exposing the thrombogenic subendothelial basement membrane to blood
* Hemorrhage into the atheroma, expanding its volume.

3) Role of Coronary Thrombus:

In acute transmural MI thrombus superimposed on a disrupted but previously only partially stenotic plaque converts it to a total occlusion. In unstable angina, acute subendocardial infarction, or sudden cardiac death, the extent of luminal obstruction by thrombosis is usually incomplete .

Thrombus in coronary artery can also embolize.

4) Role of Vasoconstriction:

Vasoconstriction compromises lumen size, and, by increasing the local mechanical forces, can potentiate plaque disruption.

5) Role of Inflammation:

Inflammatory processes play important roles at all stages of atherosclerosis.

To summarize, the acute coronary syndromes like angina, acute MI, and sudden death-share a common pathophysiologic basis which is coronary atherosclerotic plaque disruption and associated intraluminal platelet-fibrin thrombus formation. The critical consequence is downstream myocardial ischemia.

Stable angina results from increases in myocardial oxygen demand that outstrip the ability of markedly stenosed coronary arteries to increase oxygen delivery but is not usually associated with plaque disruption.

Unstable angina derives from a sudden change in plaque morphology, which induces partially occlusive platelet aggregation or mural thrombus, and vasoconstriction leading to severe but transient reductions in coronary blood flow. In MI, acute plaque change induces total thrombotic occlusion.

Finally, sudden cardiac death frequently involves a coronary lesion in which disrupted plaque and often partial thrombus and possibly embolus have led to regional myocardial ischemia that induces a fatal ventricular arrhythmia.

Angina pectoris

Angina pectoris is a symptom complex of IHD characterized by paroxysmal and usually recurrent attacks of substernal or precordial chest discomfort (variously described as constricting, squeezing, choking, or knifelike) caused by transient (15 seconds to 15 minutes) myocardial ischemia that falls short of inducing the cellular necrosis that defines infarction. There are three overlapping patterns of angina pectoris: (1) stable or typical angina, (2) Prinzmetal or variant angina, and (3) unstable or crescendo angina.

* Stable angina, the most common form and therefore called typical angina pectoris, appears to be caused by the reduction of coronary perfusion to a critical level by chronic stenosing coronary atherosclerosis; this renders the heart vulnerable to further ischemia whenever there is increased demand, such as that produced by physical activity, emotional excitement, or any other cause of increased cardiac workload. Episodic chest pain associated with exertion or some other form of stress.

The pain is described as a crushing or squeezing substernal sensation, which may radiate down the left arm. Typical angina pectoris is usually relieved by rest (thereby decreasing demand) or [nitroglycerin](http://www.robbinspathology.com/content/bookcontent.cfm?ID=HC012029##), a strong vasodilator.

Prinzmetal variant angina is an uncommon pattern of episodic angina that occurs at rest and is due to coronary artery spasm. Prinzmetal angina generally responds promptly to vasodilators, such as [nitroglycerin](http://www.robbinspathology.com/content/bookcontent.cfm?ID=HC012029##) and calcium channel blockers.

Unstable or crescendo angina refers to a pattern of pain that occurs with progressively increasing frequency, is precipitated with progressively less effort, often occurs at rest, and tends to be of more prolonged duration. It is induced by disruption of an atherosclerotic plaque with superimposed partia) thrombosis and possibly embolization or vasospasm (or both). Unstable angina is often the precursor of subsequent acute MI. Thus this referred to as preinfarction angina.