Angina pectoris

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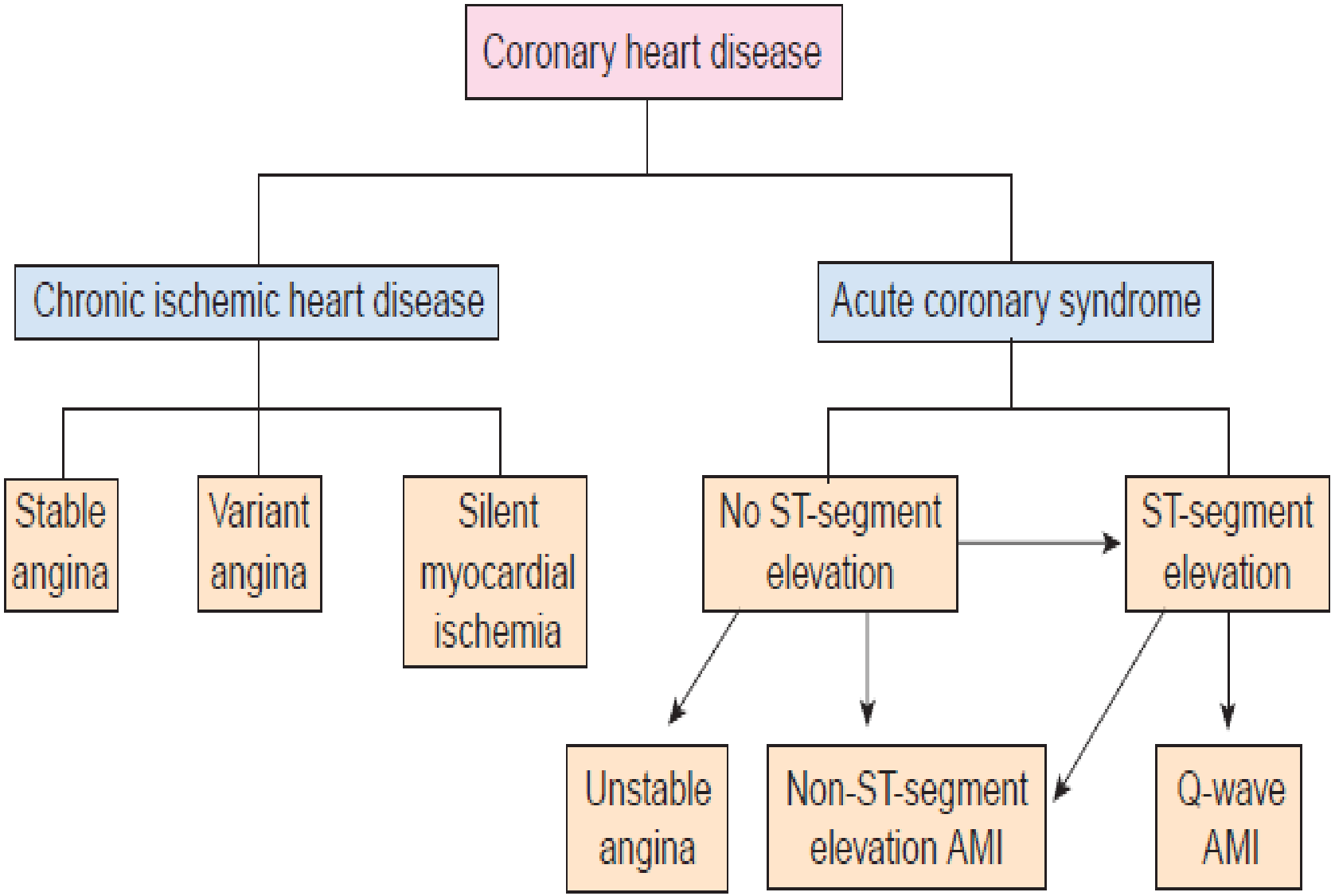

Coronary heart disease
Chronic ischemic heart disease
Acute coronary syndrome
Stable angina
Variant angina
Silent myocardial ischemia
No ST-segment elevation
ST-segment elevation
Unstable angina
Non-ST-segment elevation AMI
Q-wave AMI

Introduction

- *Coronary artery disease is caused by impaired coronary blood flow*

- *Ischemic heart disease (IHD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium; it typically occurs when there is an imbalance between myocardial oxygen supply and demand.*

- *Angina pectoris is a "clinical syndrome characterized by discomfort in the chest, jaw, shoulder, back, or arm."*

Types of angina

Stable Angina

- *Stable angina is the most common type of angina. It occurs when the heart is working harder than usual. Stable angina has a regular pattern refers to how often the angina occurs, how severe it is, and what factors trigger it*
- *The pain usually goes away a few minutes after rest or medication use.*

Unstable Angina

- *There is no predictable pattern to this kind of angina; it can just as easily occur during exercise and in rest.*
- *It should always be treated as an emergency.*
- *People with unstable angina are at increased risk for heart attacks, cardiac arrest, or severe cardiac arrhythmias*

Variant angina

- ***Also known as Prinzmetal's angina.***
- ***It often occurs in rest***
- ***It has no predictable pattern .***
- ***This kind of angina may cause severe pain, and is usually caused by a spasm in a coronary artery.***

Microvascular angina ***—sometimes referred to as Syndrome X— occurs when tiny vessels in the heart become narrow and stop functioning properly, even if the bigger arteries are not blocked by plaque.***

Atypical angina

- ***Often doesn't cause pain, but you may feel a vague discomfort in your chest, experience shortness of breath, feel tired or nauseous, have indigestion, or pain in your back or neck.***

ETIOLOGY of Angina

- ***Reduced coronary blood flow***
  - ***Coronary atherosclerosis***
  - ***Vasospasm***
  - ***Thrombosis.***
  - ***Arteritis,***
  - ***Emboli,***
  - ***Hypotension***
- ***Increased myocardial demand***
  - ***Tachycardia,***
  - ***Hypertrophy***
- ***Hypoxia due to diminished oxygen transport***
  - ***Anemia,***
  - ***Lung disease,***
  - ***Cyanotic CHD,***
  - ***Carbon monoxide (CO) poisoning,***
  - ***Cigarette smoking.***

ETIOLOGY for MI

- *Intraplaque hemorrhage,*
- *Plaque erosion, or*
- *Plaque rupture with superimposed thrombosis*
- *Vasospasm*
- *Embolization*
- *smaller vessel obstruction*
    - *Vasculitis,*
    - *Amyloidosis,*
    - *Sickle cell disease*

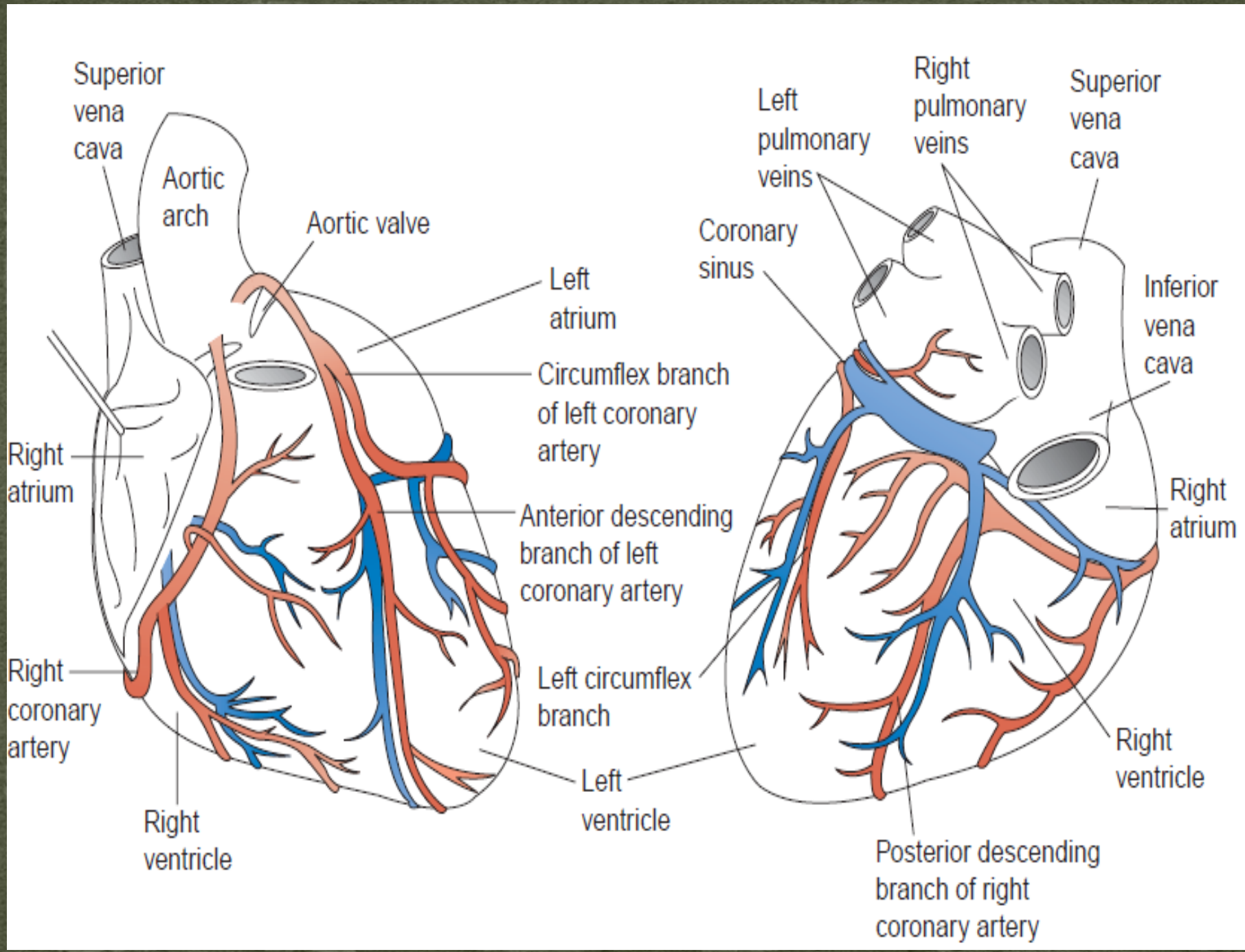
Acute coronary syndrome

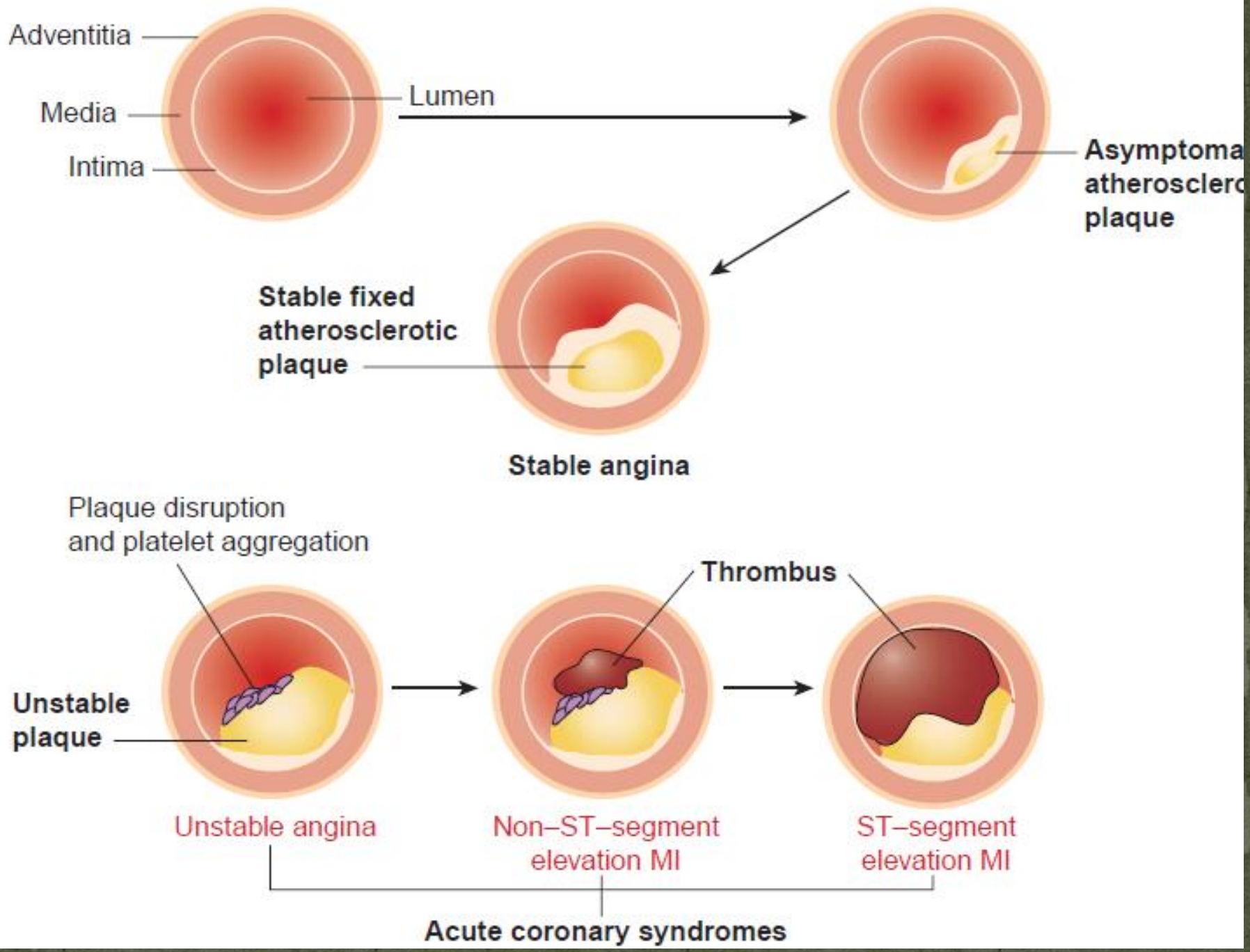
- ***ACS represents a spectrum of ischemic heart diseases ranging from unstable ischemia to acute MI***

- ***Myocardial infarction (MI) (ie, heart attack) is the irreversible necrosis of heart muscle secondary to prolonged ischemia.***

OR

- ***Myocardial infarction (MI) is myocyte cell death caused by vascular occlusion.***


Superior vena cava
Aortic arch
Aortic valve
Left atrium
Circumflex branch of left coronary artery
Right atrium
Anterior descending branch of left coronary artery
Right coronary artery
Left circumflex branch
Left ventricle
Right ventricle
Left pulmonary veins
Right pulmonary veins
Superior vena cava
Coronary sinus
Inferior vena cava
Right atrium
Right ventricle
Posterior descending branch of right coronary artery

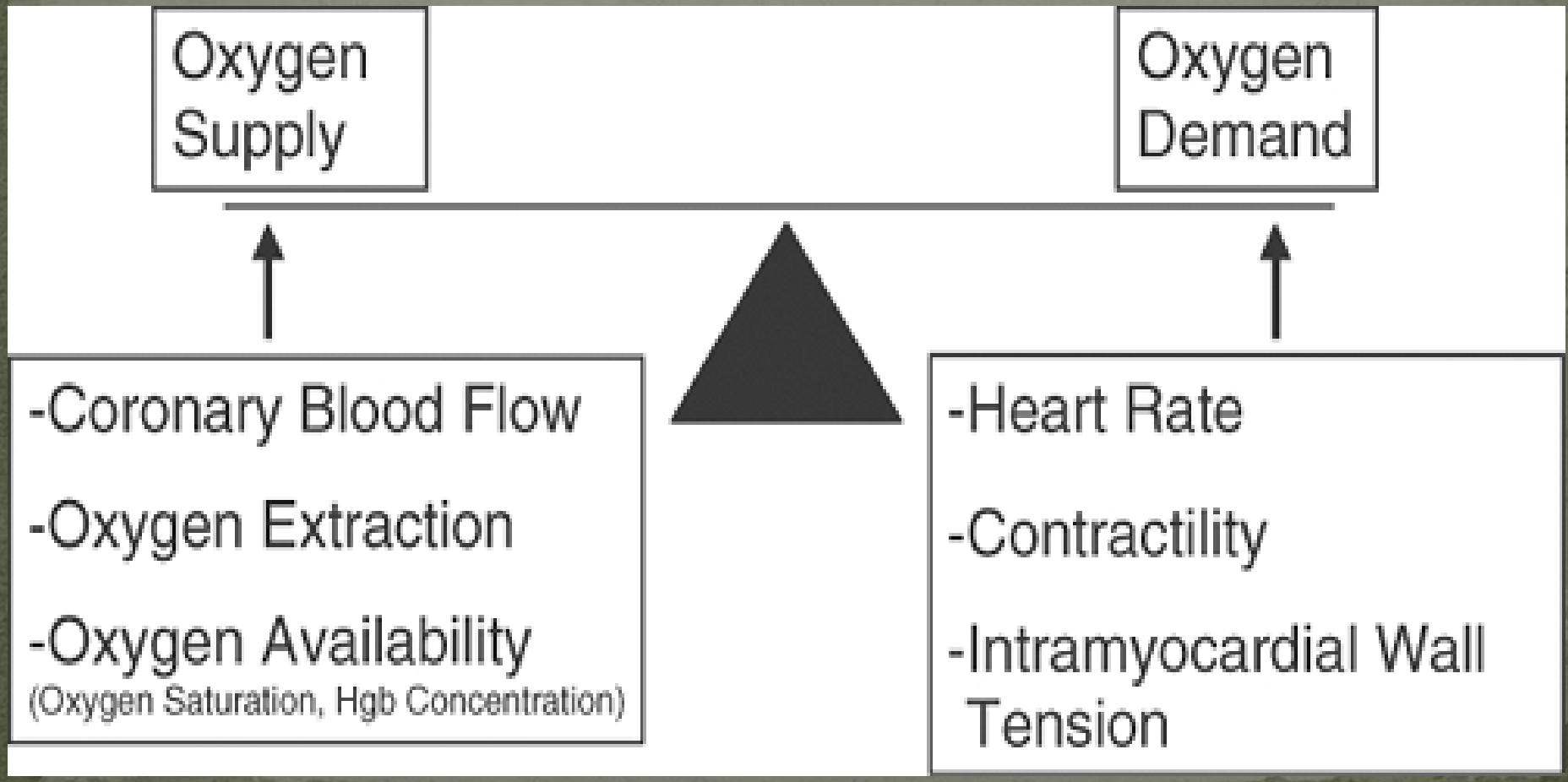

Adventitia
Media
Intima
Lumen
Asymptoma
atherosclero
plaque
Stable fixed
atherosclerotic
plaque
Stable angina
Plaque disruption
and platelet aggregation
Thrombus
Unstable
plaque
Unstable angina
Non–ST–segment
elevation MI
ST–segment
elevation MI
Acute coronary syndromes

Myocardial ischemia can result from

- *(1) A reduction of coronary blood flow caused by fixed and/or dynamic epicardial coronary artery (ie, conductive vessel) stenosis,*
- *(2) Abnormal constriction or deficient relaxation of coronary microcirculation (ie, resistance vessels), or*
- *(3) Reduced oxygen-carrying capacity of the blood.*

Pathophysiology

- Angina pectoris typically occurs when myocardial oxygen demand exceeds myocardial oxygen supply (perfusion).



- ***Under normal conditions, the myocardium will be supplied with oxygen-rich blood to prevent underperfusion of myocytes and the subsequent development of ischemia and infarction.***

- ***Blood flows through the coronary arteries in a phasic fashion, with the majority occurring during diastole.***

- *About 75% of the total coronary resistance to flow occurs across three sets of arteries: (1) large epicardial arteries (Resistance 1 = R_1), (2) prearteriolar vessels (R_2), and (3) arteriolar and intramyocardial capillary vessels (R_3).*

- *Under normal circumstances, the resistance in R 2 is much greater than that in R 1.*

- *Myocardial blood flow is inversely related to arteriolar resistance and directly related to the coronary driving pressure.*

- *Atherosclerotic lesions occluding R 1 increase arteriolar resistance, and R2 can vasodilate to maintain coronary blood flow*

- *The ability of the coronary arteries to increase blood flow in response to increased cardiac metabolic demand is referred to as coronary flow reserve (CFR)*

- *With greater degrees of obstruction, the coronary flow reserve afforded by R2 vasodilation is insufficient to meet oxygen demand.*

- *If the diameter of an epicardial artery is reduced by 50%, there is a limitation in increasing the flow to meet increased myocardial demand.*

- *When the diameter is reduced by ~80%, it can decrease coronary flow dramatically and cause myocardial ischemia.*

- *Patients with a fixed coronary atherosclerotic lesion of at least 50% or more show myocardial ischemia during increased myocardial metabolic demand as the result of a significant reduction in CFR and hence, angina pectoris*

- *Fixed atherosclerotic lesions of at least 90% almost completely abolish the flow reserve; patients with these lesions may experience angina at rest thus unstable angina.*

- *Coronary spasm can also reduce CFR significantly by causing dynamic stenosis of coronary arteries. Prinzmetal angina is defined as resting angina associated with ST-segment elevation caused by focal coronary artery spasm.*

- ***Myocardial ischemia can also be the result of factors affecting blood composition, such as reduced oxygen-carrying capacity of blood, as is observed with severe anemia (hemoglobin, < 8 g/dL), or elevated levels of carboxyhemoglobin.***

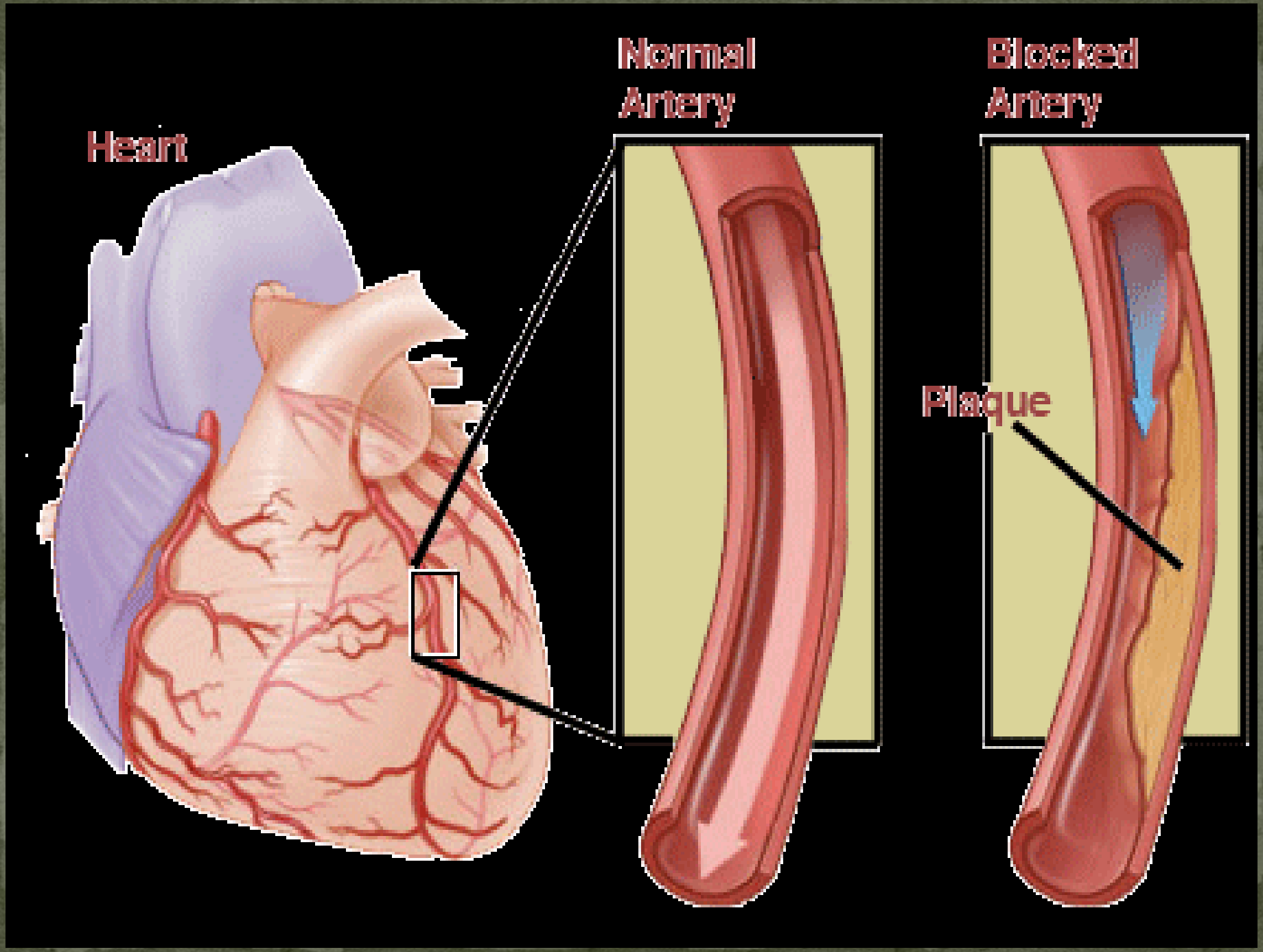
- ***The latter may be the result of inhalation of carbon monoxide in a closed area or of long-term smoking.***

- ***If ischemia is prolonged it can lead to myocyte necrosis results in myocardial infarction.***

- *Pathophysiology continues for Myocardial infarction*
- *Severe ischemia leads to ATP depletion and loss of contractile function (but not cell death) within 60 seconds.*
- *This may precipitate myocardial failure long before there is cell death.*
- *Complete deprivation of blood flow for 20 to 30 minutes leads to irreversible myocardial injury*

- *Pathophysiology continues for Myocardial infarction*
- *Severe compromise of flow for prolonged periods (i.e., 2 to 4 hours) can also cause irreversible injury*
- *Necrosis is usually complete within 6 hours of severe ischemia.*
- *The distribution of myocardial necrosis depends on the vessel involved*

- *Pathophysiology continues for Myocardial infarction*
- *The distribution of a single coronary artery and are transmural (the full thickness of the ventricular wall)-show "ST elevation MI"*
- *Subendocardial MIs are limited to the inner 30% to 50% of the ventricle-show "Non ST elevation MI"*


Heart
Normal Artery
Blocked Artery
Plaque

Grading of Angina Pectoris by the Canadian Cardiovascular Society Classification System

Class I

- *Ordinary physical activity does not cause angina. Angina occurs with strenuous, rapid, or prolonged exertion at work.*

Class II

- *Slight limitation of ordinary activity. Angina occurs On walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, or in cold, or in wind, or under emotional stress, or only during the few hours after wakening.*

Class III

- ***Marked limitations of ordinary physical activity**. Angina occurs on walking 1 to 2 blocks on the level and climbing flight of stairs in normal conditions and at a normal pace.*

Class IV

- ***Inability to carry on any physical activity without discomfort**— anginal symptoms may be present at rest.*

- *Reduction in the oxygen-carrying capacity of blood, such as elevated carboxyhemoglobin or severe anemia (hemoglobin, < 8 g/dL)*

- *Congenital anomalies of the origin and/or course of the major epicardial coronary arteries*

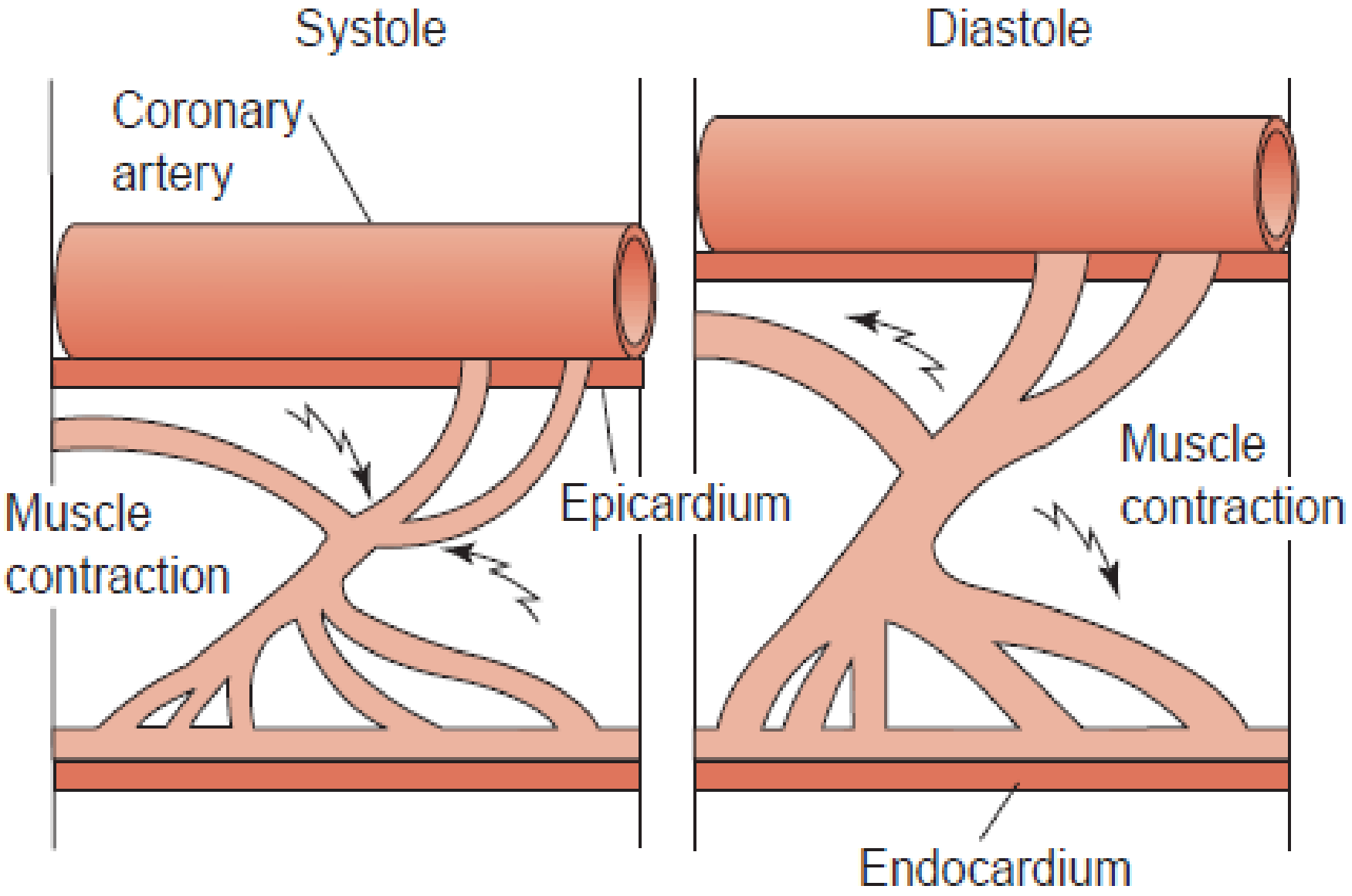
Symptoms for angina

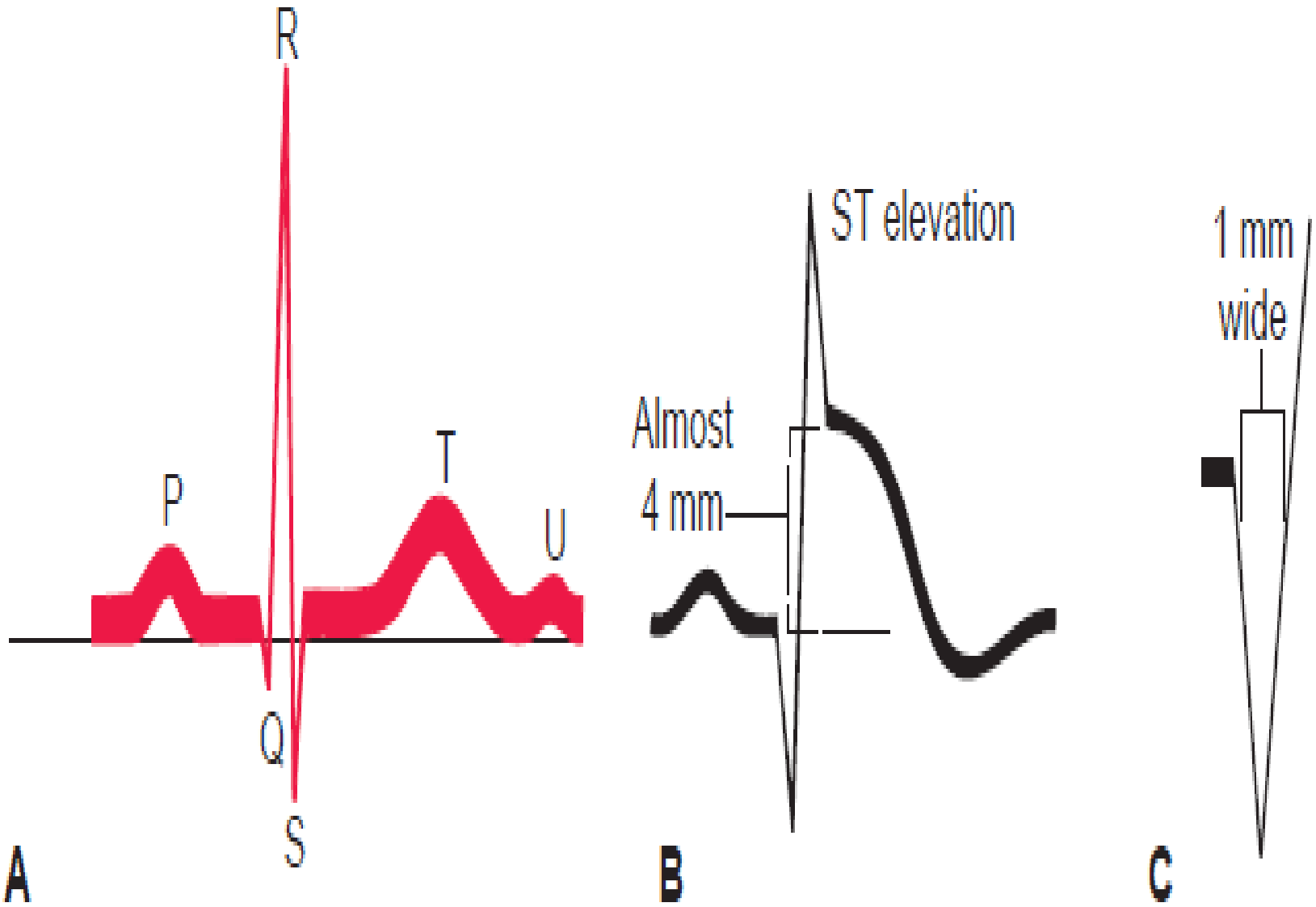
- *Reproducible pattern of pain after specific amount of exertion.*
- *Pain radiation patterns include anterior chest pain (96%), left upper arm pain (83.7%), left lower arm pain (29.3%), and neck pain at some time (22%).*
- *Increased frequency, severity, duration, or symptoms at rest suggest an unstable angina*
- *Sensation of pressure or burning over the sternum or near it, often but not always radiating to the left jaw, shoulder and arm*

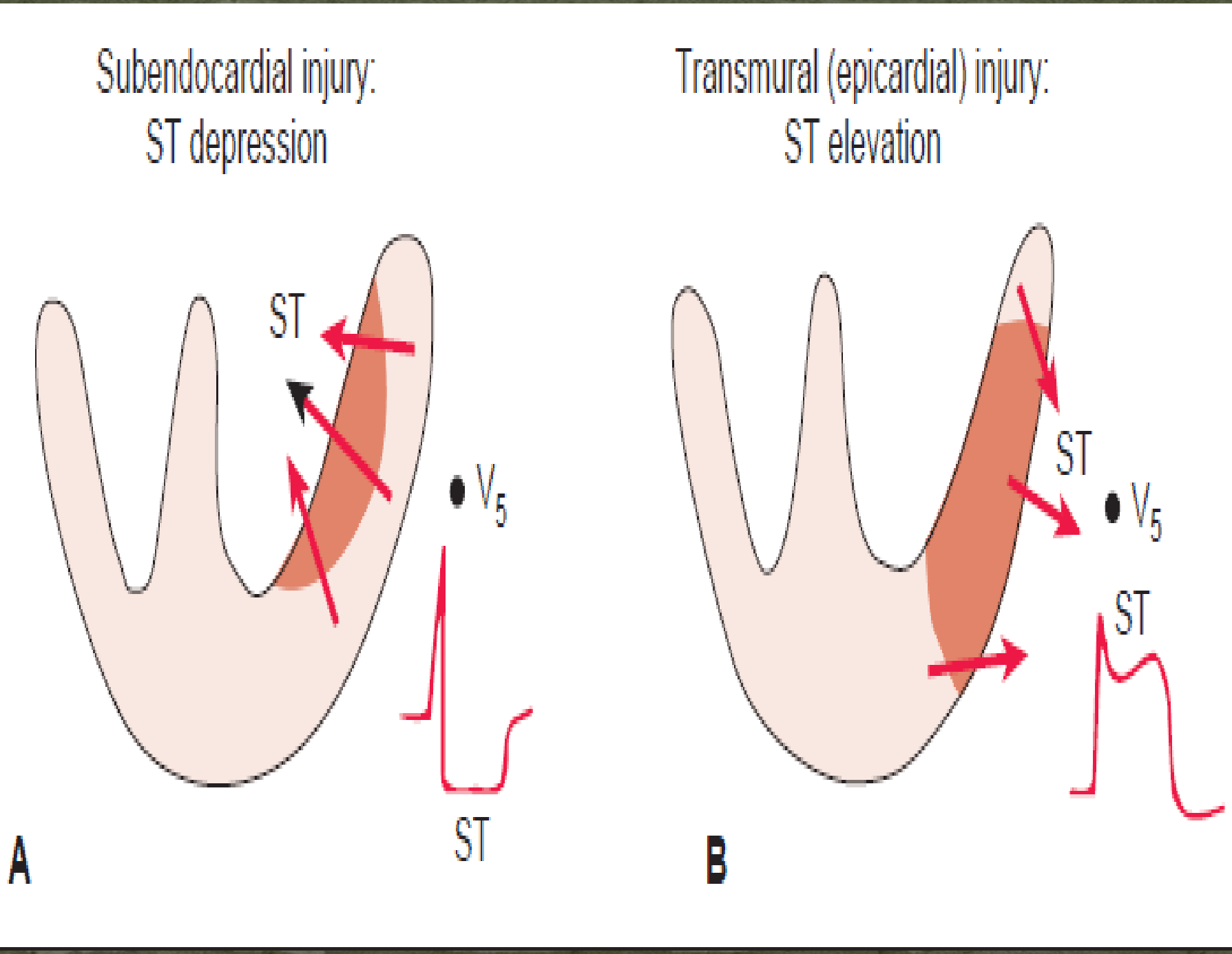
- *Pain usually lasts from 0.5 to 30 minutes*
- *Palpitations*
- *Fatigue*
- *Chest tightness*
- *Shortness of breath*

Signs

- *Abnormal precordial (over the heart) systolic bulge*
- *Abnormal heart sounds*


Systole
Diastole
Coronary artery
Muscle contraction
Epicardium
Muscle contraction
Endocardium


R
P
T
U
Q
S
A
ST elevation
Almost
4 mm
B
1 mm
wide
C


Subendocardial injury:
ST depression
ST
V5
ST
A
Transmural (epicardial) injury:
ST elevation
ST
V5
ST
B

- ***NSTEMI differs from UA in that ischemia is severe enough to produce myocardial necrosis, resulting in release of detectable amounts of biochemical markers, primarily troponin I or T and creatine kinase myocardial band (CK-MB) from the necrotic myocytes into the bloodstream.***

Clinical Presentation

Symptoms

- *Anterior chest discomfort.*
- *Arm, back, or jaw pain,*
- *Nausea,*
- *Vomiting, or*
- *Shortness of breath.*

Signs

- *Patients with ACS may present with signs of acute HF including jugular venous distention and an S3 sound on auscultation.*
- *Patients may also present with arrhythmias, and therefore may have tachycardia, bradycardia, or heart block*

Complications