Lectures: course of Pathophysiology for General medicine 3rd Year of Medical faculty; Dentistry 3rd Year

CARDIOVASCULAR PATHOPHYSIOLOGY 3
Ischemic heart disease

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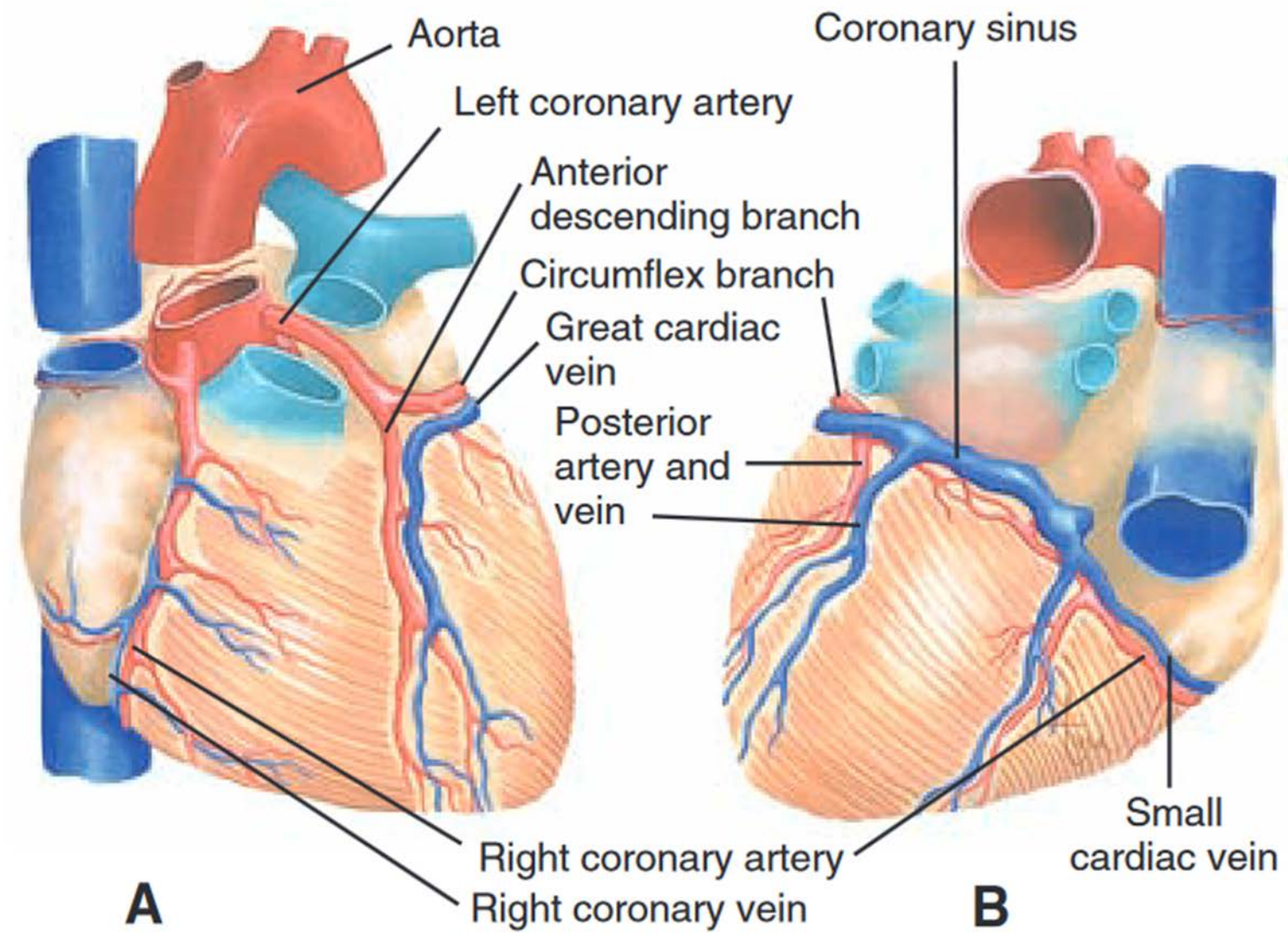
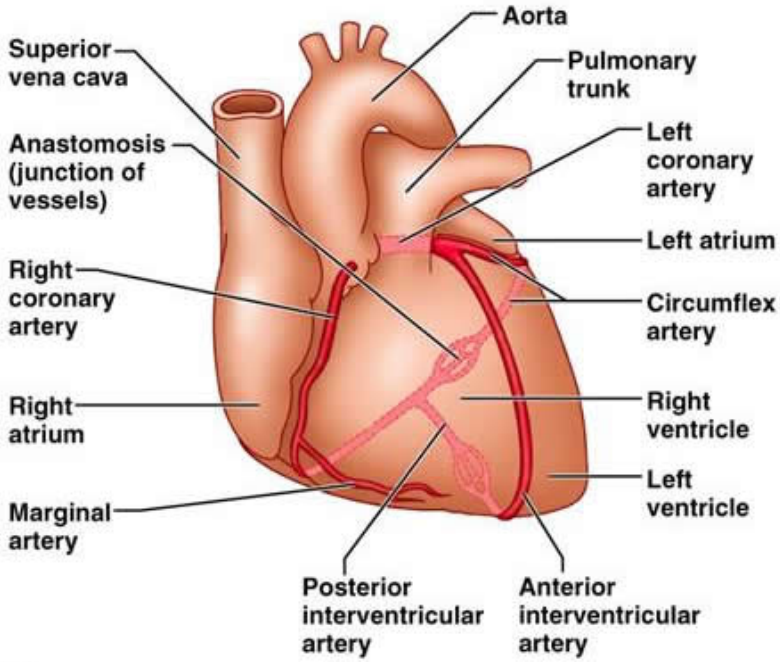
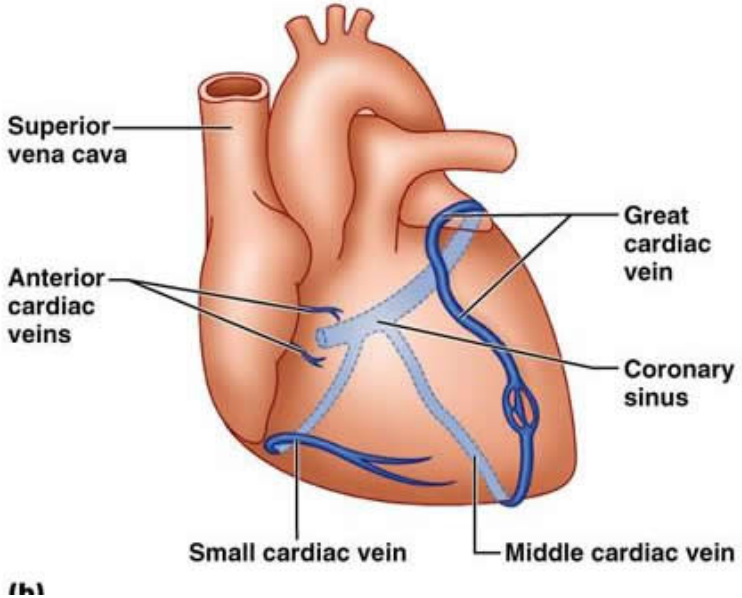
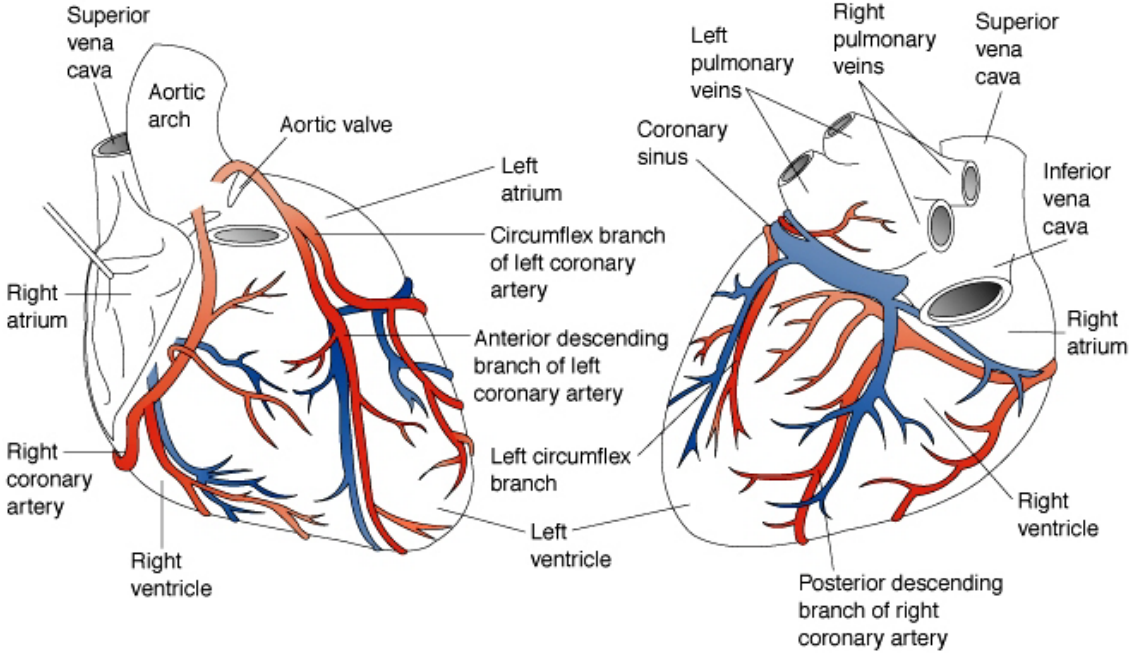
Coronary Arterial Circulation




Aorta
Superior vena cava
Pulmonary trunk
Anastomosis (junction of vessels)
Left coronary artery
Left atrium
Right coronary artery
Circumflex artery
Right atrium
Right ventricle
Left ventricle
Marginal artery
Posterior interventricular artery
Anterior interventricular artery
(a)


Superior vena cava
Great cardiac vein
Anterior cardiac veins
Coronary sinus
Small cardiac vein
Middle cardiac vein
(b)


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
Right ventricle
Left 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

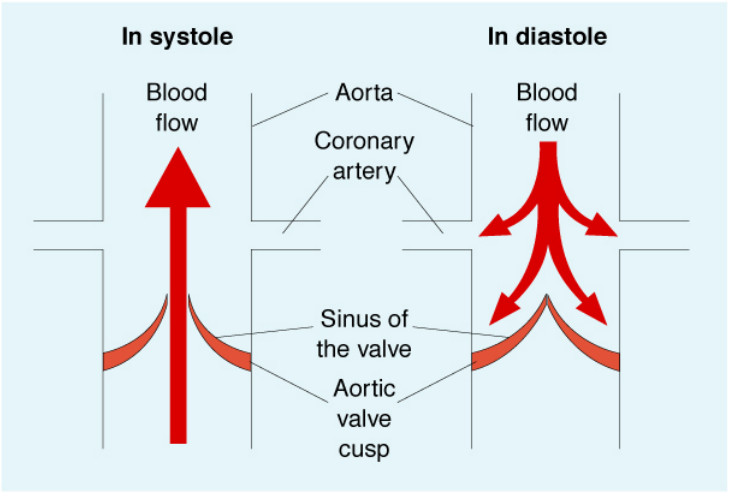
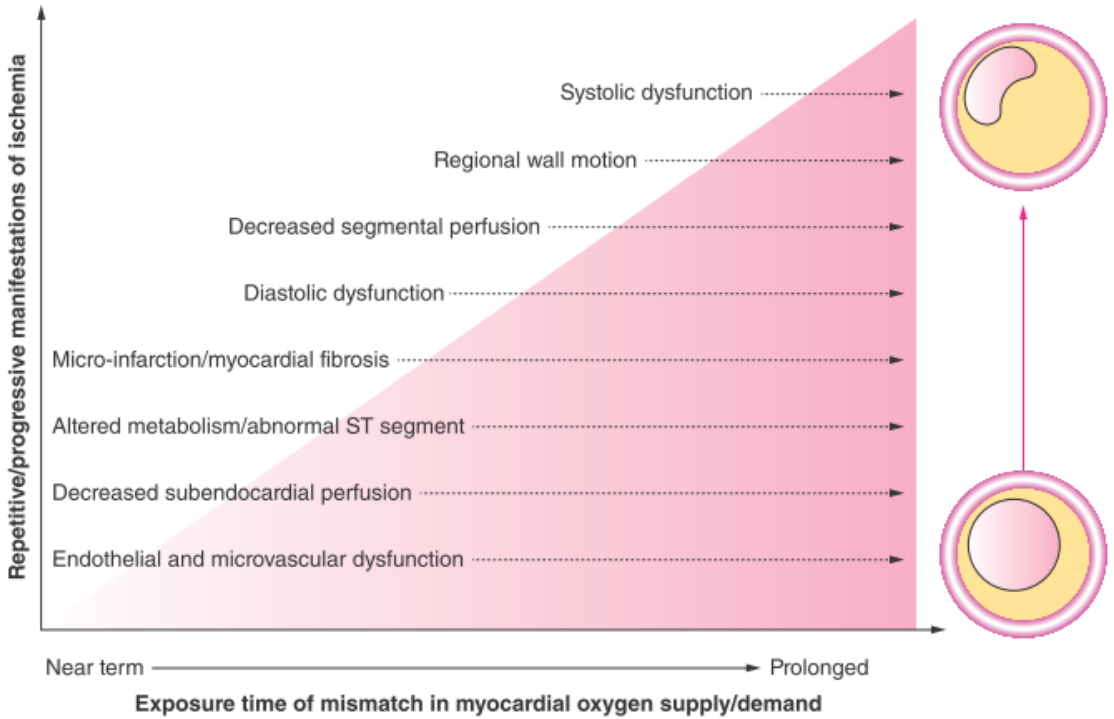
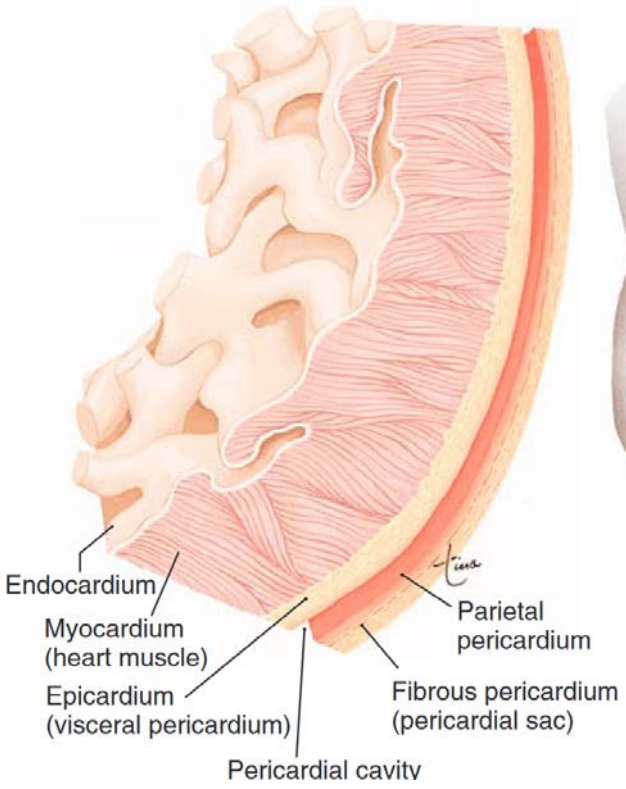
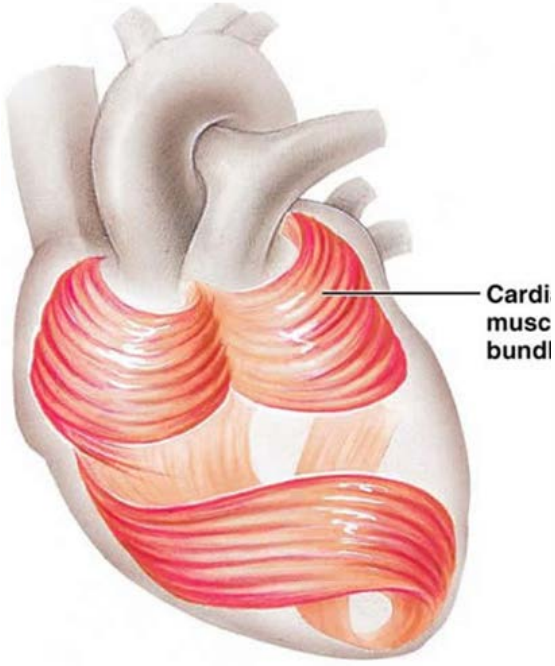
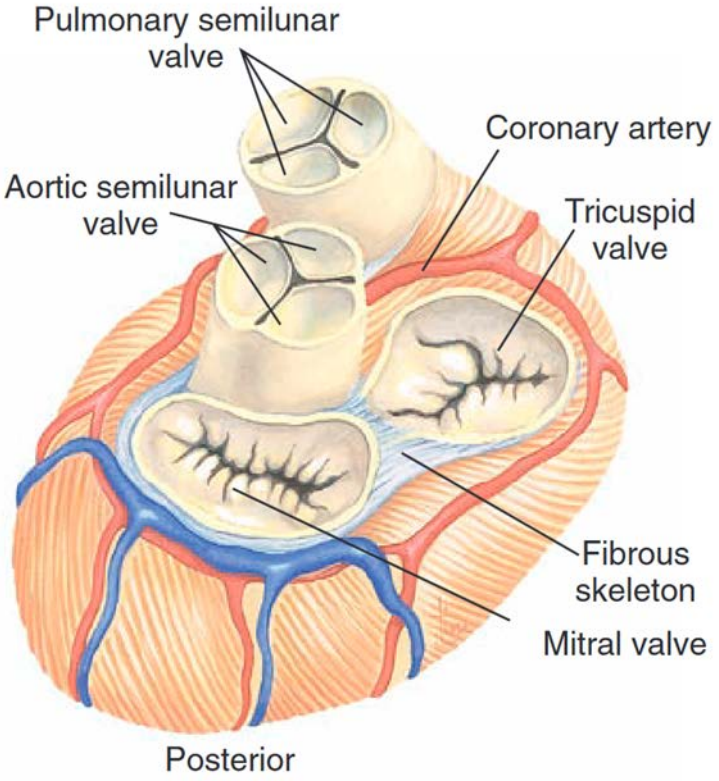


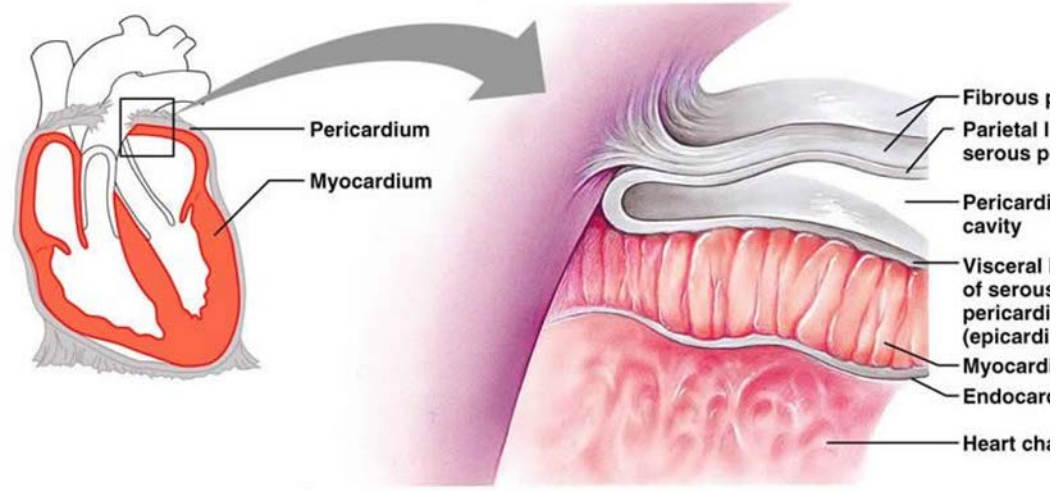
Figure 26-23 Location of the orifices for the coronary arteries in relation to the aortic valve and the direction of blood flow during systole and diastole.

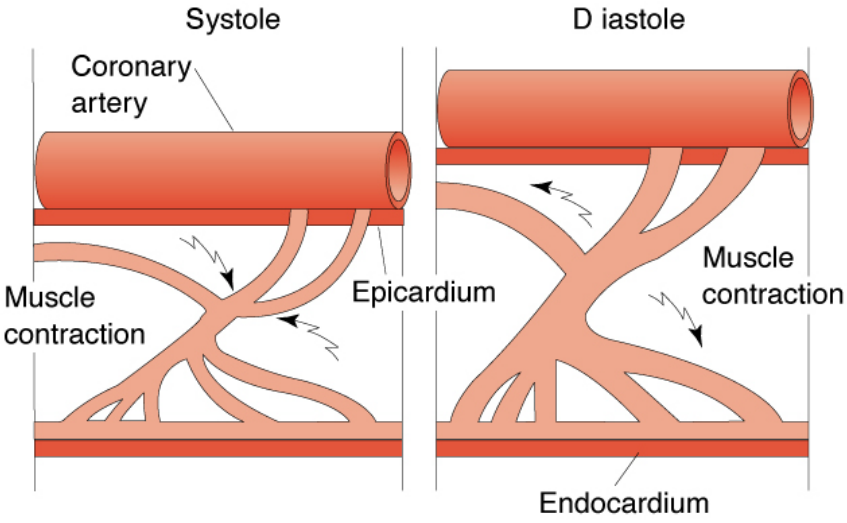



Endocardium
Myocardium
(heart muscle)
Epicardium
(visceral pericardium)
Pericardial cavity
Parietal
pericardium
Fibrous pericardium
(pericardial sac)


Cardi
musc
bundl


Pulmonary semilunar
valve
Coronary artery
Aortic semilunar
valve
Tricuspid
valve
Fibrous
skeleton
Mitral valve
Posterior


Pericardium
Myocardium
Fibrous
Parietal
serous p
Pericardi
cavity
Visceral
of serous
pericardi
(epicardi
Myocard
Endocar
Heart cha


Systole
D iastole
Coronary
artery
Muscle
contraction
Epicardium
Muscle
contraction
Endocardium

Ischemic heart disease

Major Risk Factors for Heart Disease

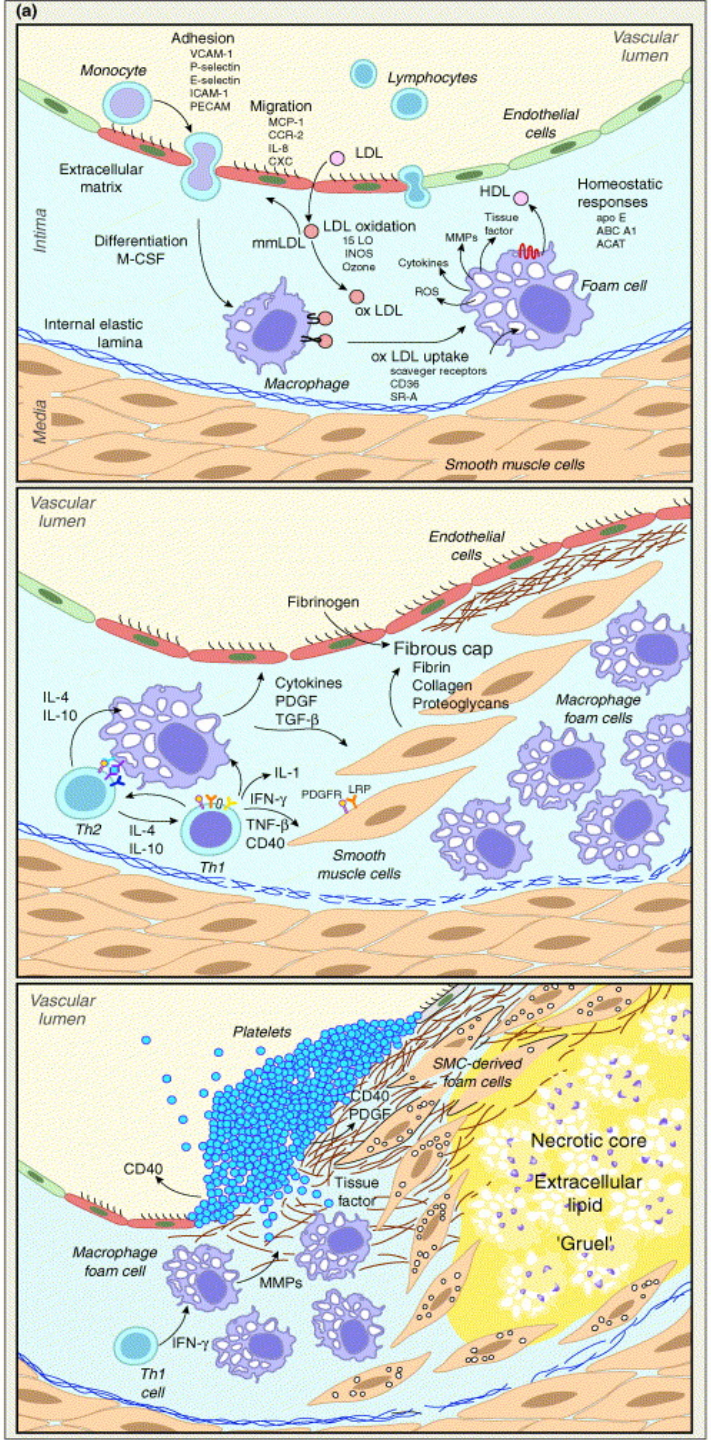
- Nonmodifiable: Age > 50 y, Sex male, Genetic , Race white,
- Modifiable: High blood pressure (ABP > 140/90 mm Hg), Cigarette smoking, High cholesterol, Obesity, inactivity Diabetes

Contributing Risk Factors

- Stress, unhealthy behaviors (rage, overwork)
- Alcohol with high BP, heart failure, and stroke
- Metabolic syndrome (3 or more factors): Glu higher, HDL low, TAG higher

Inflammatory Markers In Assessment of Risk for Heart Disease

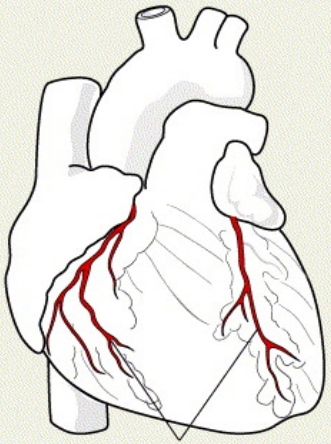
- Highly sensitive C-reactive protein (hs-CRP):
- hs-CRP <1.0 mg/dl: low risk
- hs-CRP 1.0 to 3.0 mg/dL: moderate risk
- hs-CRP >3.0 mg/dL: high risk



(b)

Cardiac hypertrophy

Hypertension
Hypertrophic cardiomyopathy

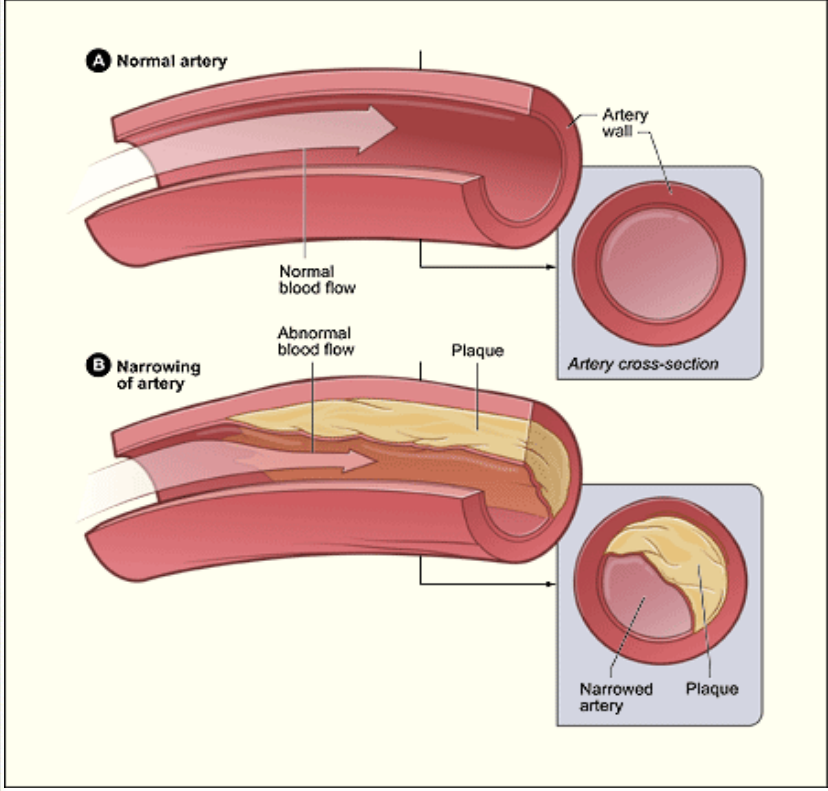


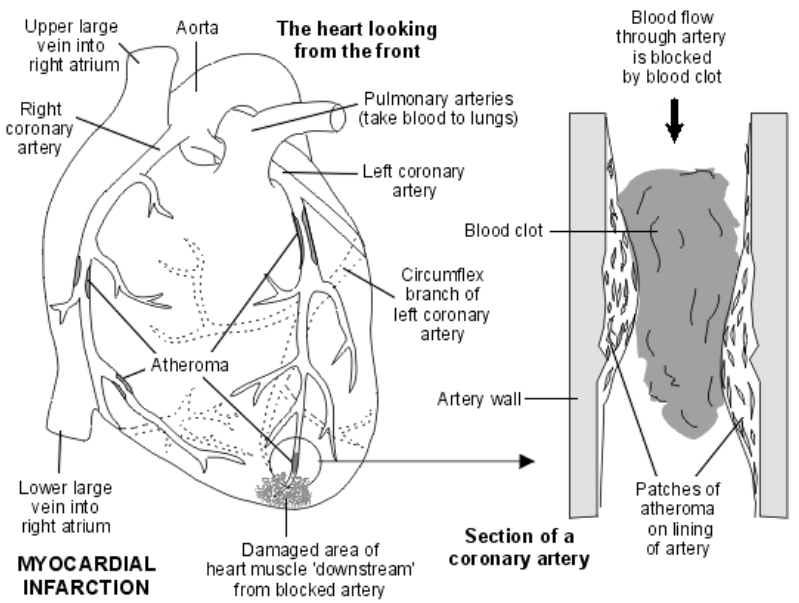
Coronary arteries

Cardiac dilatation and heart failure

Atherosclerosis
Hypertension
Metabolic syndrome
Dilated cardiomyopathy

Current Opinion in Genetics & Development

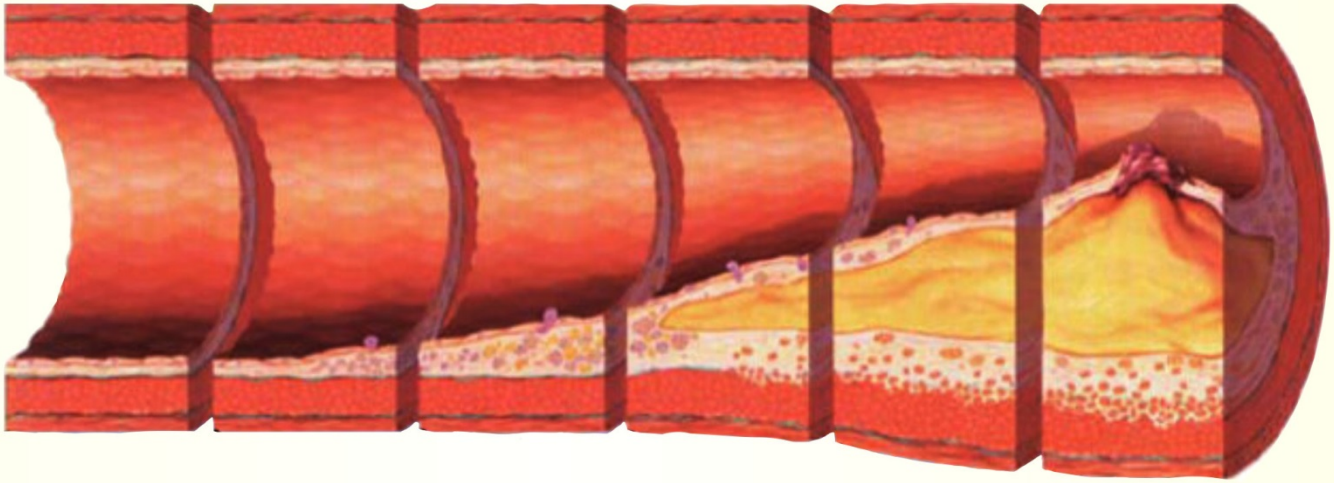




Atherosclerosis

CLINIC	Silent		Manifestant	
TIMING	Teenage - young adulthood	> 35 years	> 45 years	> 65 years
FH TIMING Aa	< Teenage	> 25 years	> 45 years	
FH TIMING AA	< Teenage		20 - 30 years	
COMPOSITION	extracell. oxidized cholesterol ester deposits;		Extracel. cholesterol deposits Lissomyocyte, Fibroblast burst	Thrombosis, calcification
ENDOTHELIAL DYSFUNCTION	Inflamm. reaction to endogenous deposits (chronic type)			

STAGES OF EVOLUTION



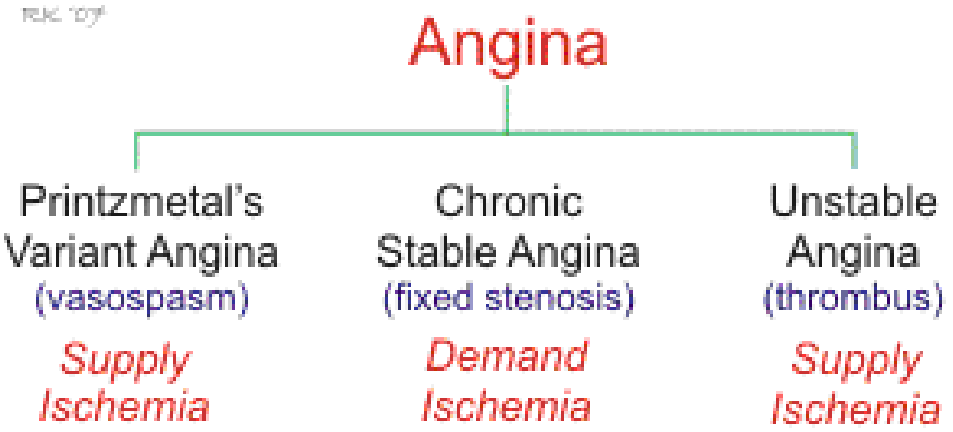
LESIONS

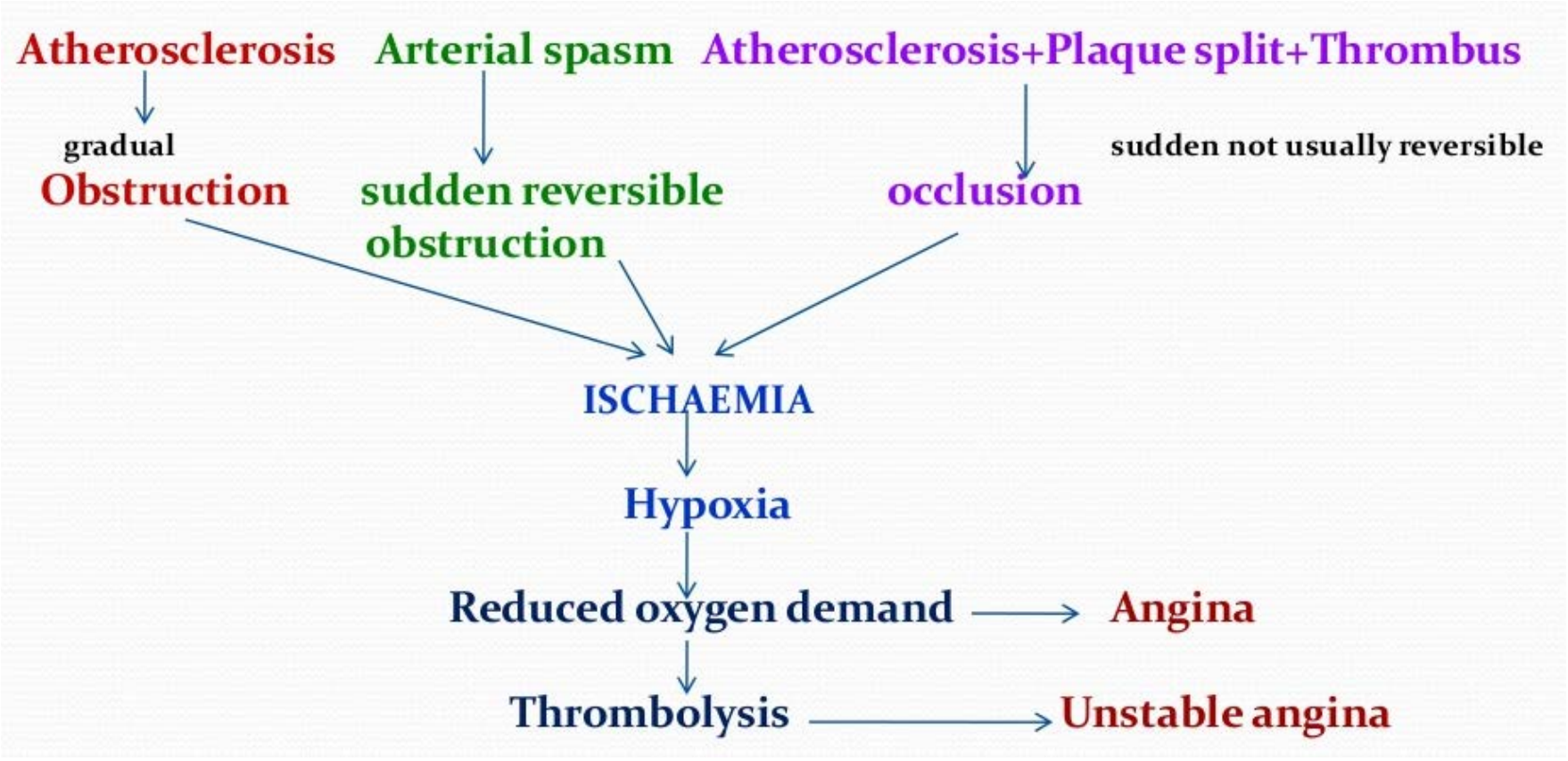
Initial lesion	**Fatty streak**	**Intermediate fatty streak**	**Atheroma (soft) plaque**	**Fibroatheroma Sclerotizing (hard) plaque**	**Complicated lesion/rupture**
Histology normal	Mo/Mf infiltration	Mo/Mf infiltration	Mo/Mf infiltration	Multiple lipid cores	Surface damage
Mo/Mf infiltration	Intracell. fat bodies	Intracell. fat dep.	Rich foam cells	Collagen fibromesh	Thrombosis
Normal cholest.	*Isolated* foam cells	*Heavy* foam cells	Extracell. lipids	Calcium crystals	Erosion of plaque
vessel passage					Atherothrombembolia

THERAPY	Life style modifications Low dose pharmacotherapy	Life style modifications High dose pharmacotherapy	Surgery, Pharmacotherapy Lifestyle modifications

Angina pectoris

- Stable Angina / Angina Pectoris.
- Unstable Angina.
- Variant (Prinzmetal) Angina
- Microvascular Angina





(1) Stable angina

- Def: Stable angina (**effort angina, classic angina, myocardial ischemia**) is chest discomfort, pain and associated symptoms (angina) precipitated by **some activity** (running, walking, sport, etc.) with minimal or no symptoms at rest or after administration of sublingual **nitroglycerin.**
- Etio: narrowing of the coronary arteries by atherosclerosis (buildup of fatty plaque + hardening of the arteries). Precipitated by cold weather, heavy meals, and emotional distress.
- **Risk factors**: Age (≥ 45 years for men, ≥ 55 for women), smoking, diabetes, dyslipidemia, high cholesterol, high blood pressure, family history of premature cardiovascular disease (men <55 years, female <65 years old), kidney disease (microalbuminuria or GFR<60 mL/min), obesity (BMI ≥ 30 kg/m2), physical inactivity, prolonged psychosocial stress
- Clin: Symptoms typically *abate several minutes after activity* and recur when activity resumes.
  - **chest discomfort** rather than actual pain: **pressure, heaviness, tightness, squeezing, burning**, or choking sensation.
  - **Radiating referred pain:** epigastrium, back, neck area, jaw, or shoulders, arms (inner left arm),
  - Pain is **precipitated** by exertion or emotional stress, full stomach, cold temperatures.
  - Accompanied by **breathlessness, sweating, and nausea** in some cases. In this case, the pulse rate and the blood pressure increases.
  - Chest pain lasting only a few seconds is normally not angina (such as precordial catch syndrome).

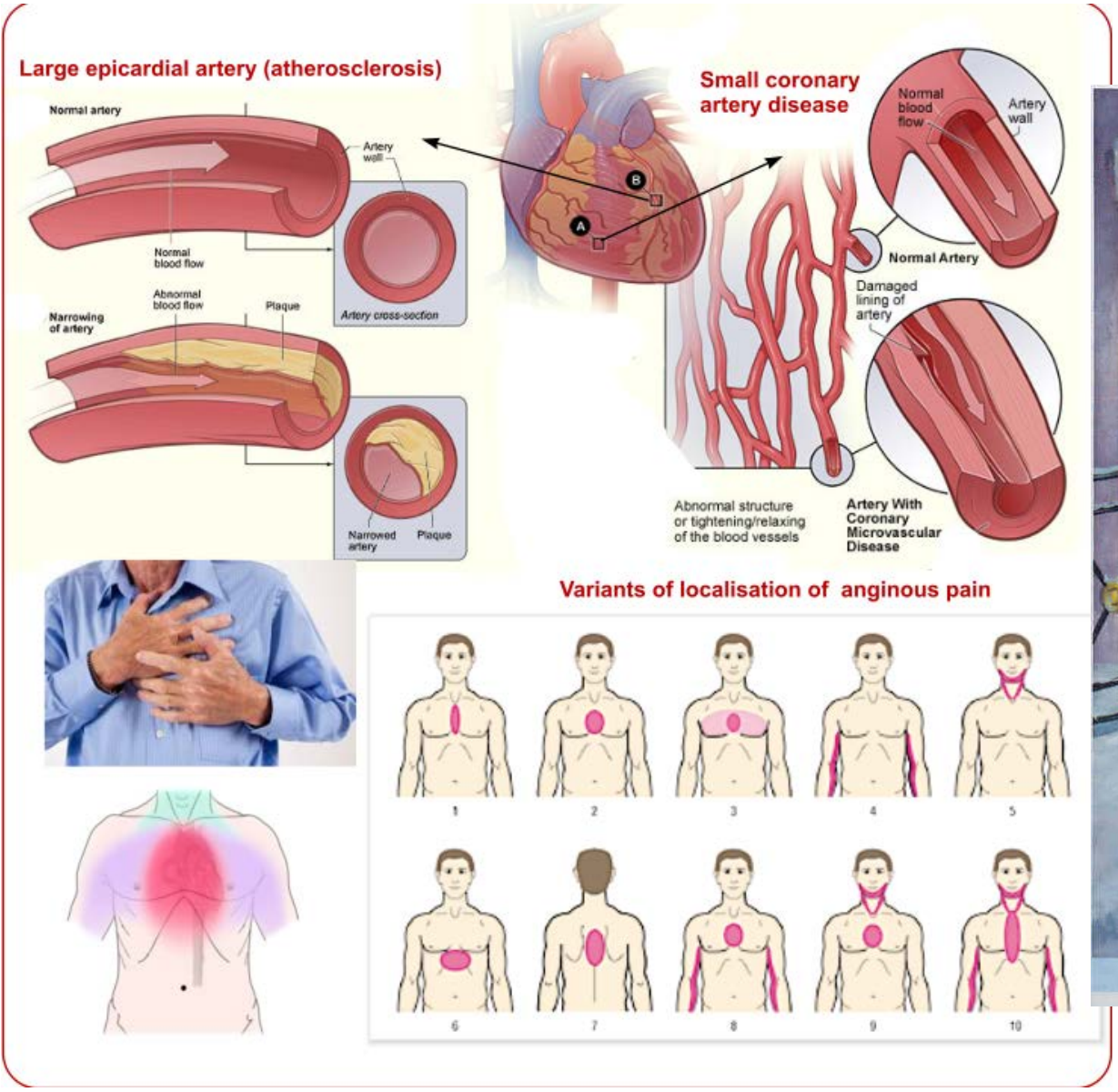
Stable angina

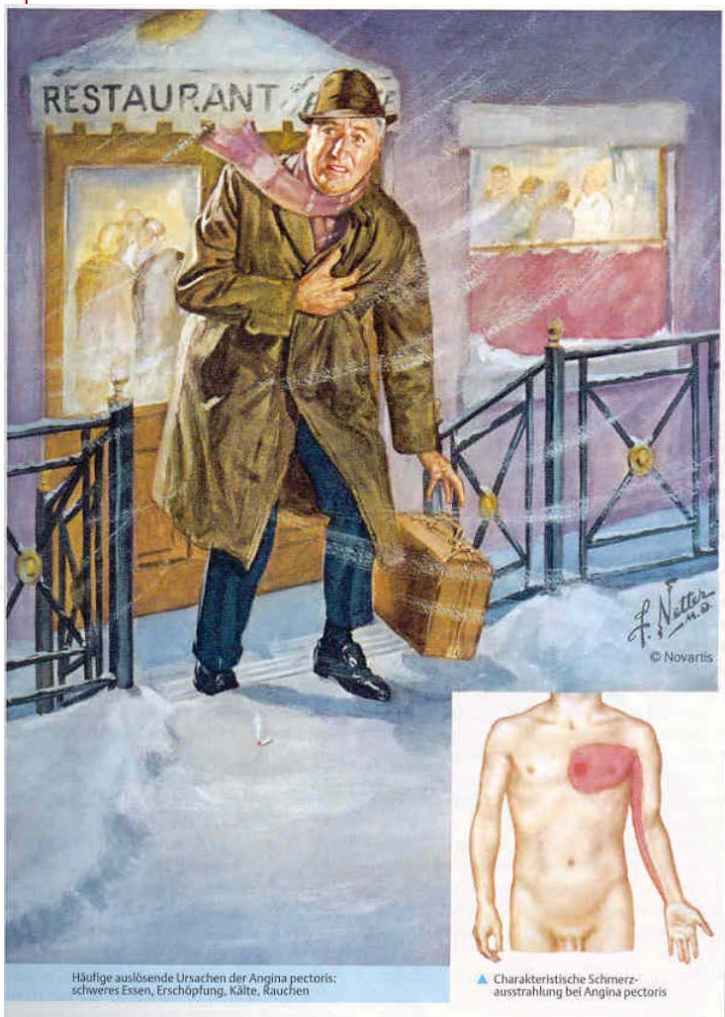
- Provoke angina: Medications (VasodilatorsExcessive thyroid hormone , Vasoconstrictors
  - Polycythemia, which thickens the blood, slowing its flow through the heart muscl
  - Hypothermia, Hypervolemia´, Hypovolemia

- Other medical problems: Esophageal disorders Gastroesophageal reflux disease (GERD),
  - Hyperthyroidism, Hypoxemiahypertension
  - Profound anemia, Bradyarrhythmia, Hypertrophic cardiomyopathy
  - Tachyarrhythmia Valvular heart disease

- Ptg:Myocardial ischemia ← insufficient blood and oxygen either because of
  - increased oxygen demand by the myocardium (work)
  - decreased supply to the myocardium. narrowed blood vesselsmor insufiicient return
- Some experience "autonomic symptoms" nausea, vomiting, and pallor.
- Coital angina (angina d'amour) angina subsequent to sexual intercourse. It is generally rare, in patients with severe coronary artery disease

Anginus pain



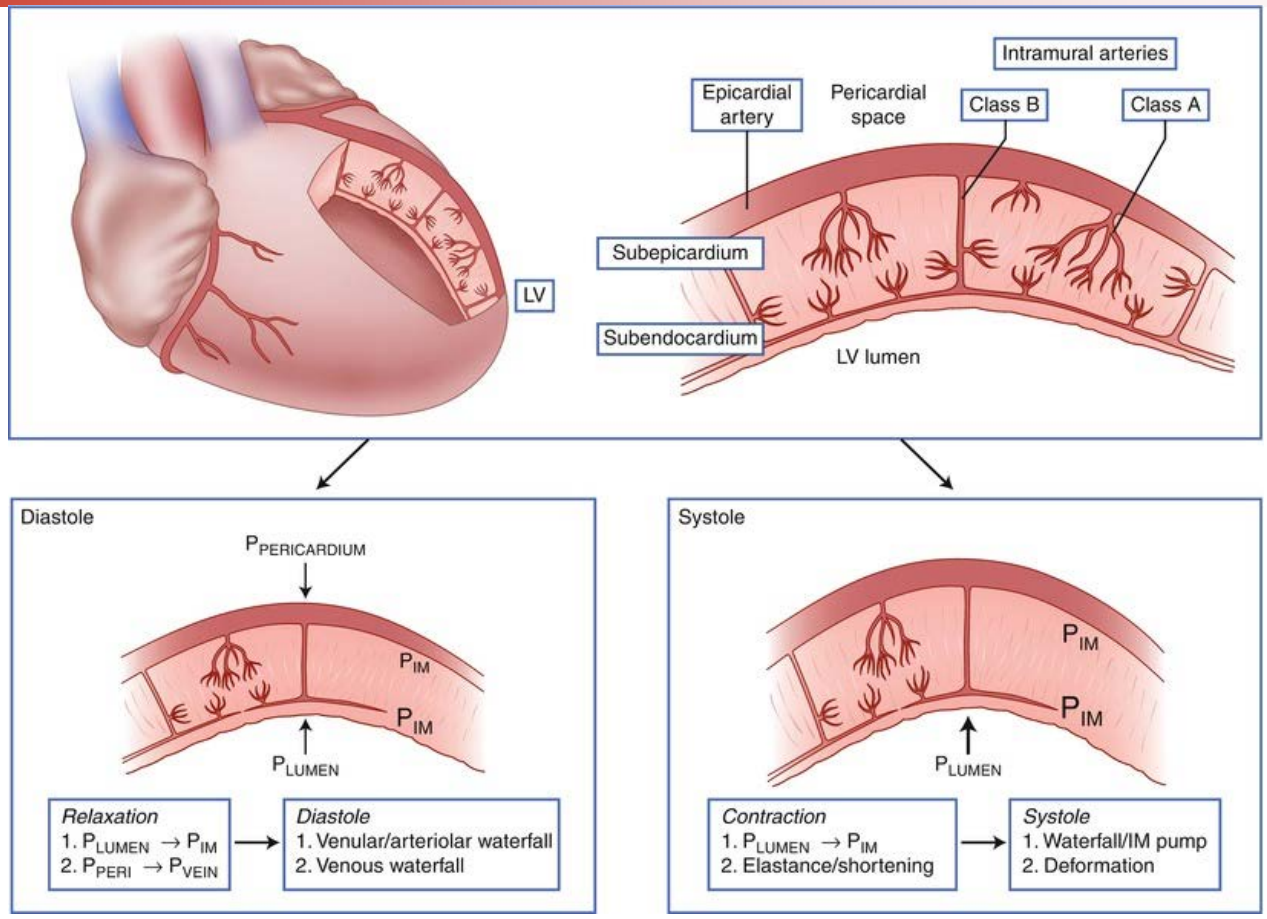


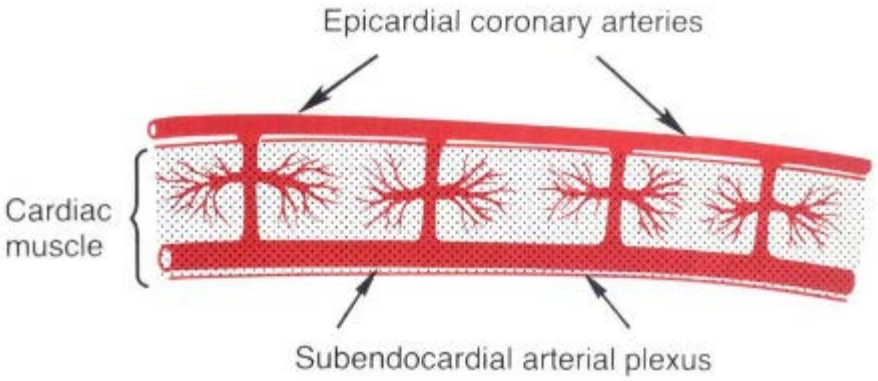
(2) Unstable angina

- Def.:Unstable angina (UA) (crescendo angina) emergency form included of ACS (acute coronary syndrome) as unpredicitive by occurence and different by manifestations as classic angina; has at least one of these three features:
  - it occurs unpredictably at rest (or with minimal exertion), usually lasting > 10 minutes
  - it is severe and of new onset (within the prior 4–6 weeks) resembles infarction
  - it occurs with a crescendo pattern (more severe and frequent as attack is prolonged).
- Occ: 64% of UA occur between 22:00 and 08:00 at rest, during sleep
- Etio: reduction of coronary flow due to *transient platelet aggregation* on apparently *normal endothelium,* coronary artery *spasms*, or coronary *thrombosis*
- Ptg: Atherosclerosis (stable angina protected with a fibrous cap) → progresses by inflammation → unstable plaque (cap may rupture) → thrombosis → acute myocardial ischemia; blood clots moves down the coronary vessel's lumen. (UA) develops independently of activity

(3) Cardiac syndrome X

- Def.: **Cardiac syndrome X (microvascular angina) =** angina-like chest pain (ischemic like) upon exercise (cardiac stress tests) in case of normal epicardial coronary arteries (by angiography).
- Occ:Women are more prone ; may explain the higher rates of angina in women
- Etio: The primary cause is **unknown**, but factors apparently involved are **endothelial dysfunction** and **reduced flow** (? due to spasm) in the **small resistance penetrating microvasculature** in the heart is dysfunctioned + enhanced pain perception
- **Risk factors:** abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, insulin resistance or intolerance to glucose, prothrombotic state or proinflammatory state. Older people are more at risk, genetic mutations that predispose to the syndrome, history of heart disease in the family
- Clin:**Angina:** can last longer more intense chest pain than individuals of pther angina
- **Abnormal cardiac stress test:** ST changes are typically similar to those of coronary artery disease, and the opposite of those of Prinzmetal's angina.
- Myocard perfusion imaging abnormal in 30% of patients. Coronary angiogram: Normal
- Other causes of chest pain must be ruled out, Prinzmetal's angina Esophageal spasm


LV
Intramural arteries
Epicardial artery
Pericardial space
Class B
Class A
Subepicardium
Subendocardium
LV lumen
Diastole
$P_{PERICARDIUM}$
P_{IM}
P_{IM}
P_{LUMEN}
Relaxation
1. $P_{LUMEN} \rightarrow P_{IM}$
2. $P_{PERI} \rightarrow P_{VEIN}$
Diastole
1. Venular/arteriolar waterfall
2. Venous waterfall
Systole
P_{IM}
P_{IM}
P_{LUMEN}
Contraction
1. $P_{LUMEN} \rightarrow P_{IM}$
2. Elastance/shortening
Systole
1. Waterfall/IM pump
2. Deformation


Epicardial coronary arteries
Cardiac muscle
Subendocardial arterial plexus

(4) Variant angina

- Def.: Variant angina (alt.vasospastic angina, Prinzmetal angina, angina inversa) also called coronary vasospasm, is an atypical angina syndrome (chest pain) which (angina incersa = opposite) i) *occurs in at rest or even asleep* and ii) is caused by sudden constriction of *coronary arteries* due to *vasospasm (*smooth muscle contraction)
- Occ: people are generally younger; have fewer risk factors for coronary artery disease; more often in women, high incidence in Japanese males as well as females
- Forms: typical, atypical (vasospastic angina), in portion of patients VA may be a manifestation of a more generalized episodic smooth muscle-contractile disorder such as migraine, Raynaud's phenomenon, or aspirin-induced asthma
- Clin: significant percentage of patients are asymptomatic
  - repeated episodes of chest pain in rest (late evening night, sleep,early in morning) occur in clusters, does not develop during treadmill stress testing (exercise tolerant) , no evidence of other forms of cardiac disease + elevations in the ST segment
  - many have serious arrythmias, individuals who smoke tobacco
- headeache, excessive sweating, tiredness; more serious s:fainting, shock
- typically does not progress to myocardial infarction .

Variant Angina

- **Risk factors** (triggers of an attack):
  - tobacco nicotine alcoholic beverages, marijuana, cocaine);Energy drinks
  - catecholamine-like stimulants (e.g. epinephrine, dopamine, amphetamines);
  - parasympathomimetic drugs (e.g. acetylcholine, methacholine);
  - anti-migraine drugs (e.g. various triptans), and;
  - the uterus-contracting drug, ergonovine;chemotherapeutic drugs (e.g. 5-fluorouracil, capecitabine).
  - Hyperventilation + virtually any stressful emotional or physical (e.g. cold exposure) causing significant rises of catecholamines

- Lab.: a) ECG: ST segment elevations > or depressions during angina attacks + widening of the R (broad QRS complex "monophasic curve".

  b) Enzymes: esmall elevations in cardiac marker enzymes, during long attack ther in the

Variant angina – Mechanisms

- Ptg: A) Mild coronary atherosclerosis in 2/3 VA(70% in a single coronary artery) spasms also occur in ST depression angina triggered by exertion, and/or who have atherosclerotic coronary artery disease
- B) Vasospasm:
  - 1) reduced nitric oxide which dilates smooth muscle cells (?? defect in the endothelial nitric oxide synthetase ?)
  - 2) Acetylcholine defect in dysfunctional epithelium Ach from nn.vagi predominantly induce dilation of the coronary arteries (stimulates endothel to produce NO). (Without NO in dysfunctional endothel Ach induces vasoconstriction.
  - 3) Abnormal platelet activation and excess effect of vasoconstrictors: *Thromboxane A2, serotonin, histamine, endothelin (abnormally activated platelets* by lipoprotein(a) competing with plasminogen
  - 4) Increased alpha-adrenergic effect receptor activity in epicardial coronary arteries or the excessive release of catecholamines (norepinephrine)
  - 5) intrinsic hypercontractility of coronary smooth muscles; existence of significant atherosclerotic coronary artery disease;
  - 6) reduced vagal activity

Acute coronary syndrome

ACUTE CORONARY SYNDROME (ACS)

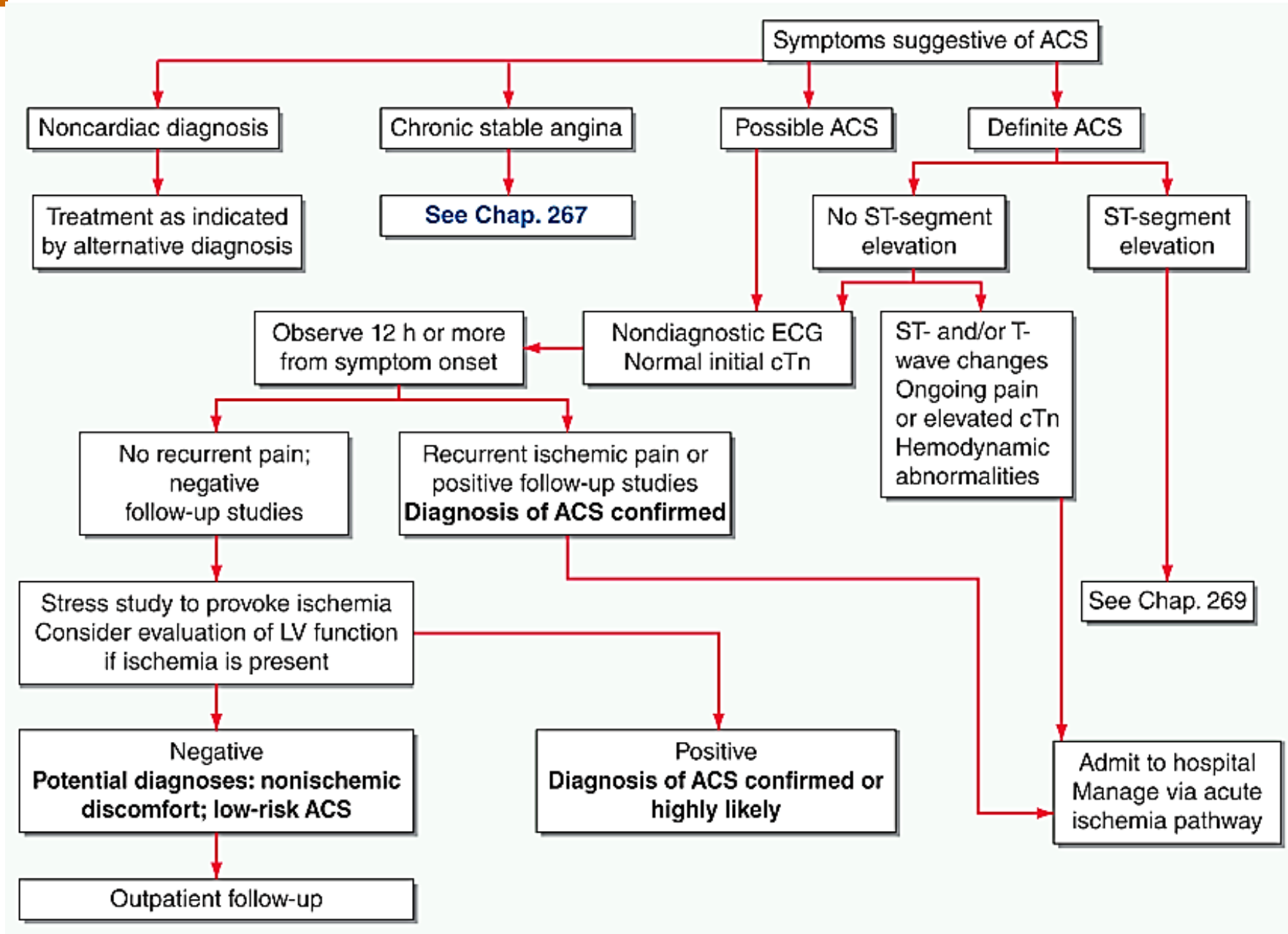
- **Def:** group of acute symptoms commonly known to occure due to sudden ischemia of the feart due to obstruction of the coronary arteries
- **Class:** according to ECG/EKG and cardiac markers
  - Unstable angina pectoris (38%)
  - Myocardial infarction - ST segment elevation myocardial infarction (STEMI) (30%)
    non-ST segment elevation myocardial infarction (NSTEMI) (25%)
  - Sudden cardiac death
- **Etio:** ischemia, tromboembolia, atherothromboembolia in coronary vessel, cocaine
- **Clin:** chest (mostly precordial) pain, often radiating to the left arm or angle of the jaw, *squeezing, pressure-like* in character, "atypical„ - pain experienced in different ways or even absent (more likely in females, diabetes).
  - nausea, vomiting , diaphoresis (sweating), dyspnoea (shortness of breath, palpitations),
  - anxiety or a sense of impending doom (angor animi)
- Dif.dg: Unstable angina - occurs suddenly, often at rest or with minimal exertion; "crescendo angina"). lesser degrees of exertion than the individual's previous angina; new onset angina is also considered unstable angina, since it suggests a new problem in a coronary artery.
  - Stable angina - exertion and resolves at rest.
  - Cardiac chest pain : anemia, bradycardias, tachycardias

MYOCARDIAL INFARCTION

- Type 1 MI (68%) (atherotromboembolic) atherosclerotic plaque disruption with plaque erosion or plaque rupture with thrombus formation (postmortem demonstration of acute atherothrombosis)
- Type 2 MI (28%) (exertional) coronary ischemia from oxygen supply-demand mismatch without acute plaque change (erosion or rupture; eg, sudden anemia, prolonged tachyarrhythmia, coronary artery spasm or dissection); coronary atherosclerosis is present.

Most types 1 and 2 occur spontaneously and present as NSTEMI, but some manifest as STEMI

- Type 3 MI (~ 3,5 %) (sudden cardiac death) postmortem atherothrombosis; new ischemic ECG or ventr. fibrillation; sy. suggestive of myocardial ischemia; death occurs before blood biomarkers can be obtained or before increases in biomarkers are identified
- Type 4 MI (0,2%) - procedure-related T1MI occurring within 48 h after: T4aMI percutaneous coronary intervention (PCI) ; T4bMI thrombosis after stenting or T4cMI after balloon angioplasty documented angiographically; the same criteria as type 1 MI; elevation in high-sensitivity cardiac troponin (hs-cTn)
- Type 5 MI (0.0004%) - T1MI occurring 48 h after coronary artery bypass graft (CABG)
- ~ 5.8 million new cases of IHD in %* coutries of European Society of Cardiology (ESC) in 2019. The median age-standardized incidence rate was 293.3 (195.8 - 529.5) /100,000 people. ~ 38% of deaths in women and 44% in men
- The estimated annual incidence of MI in US ~550,000 new cases + 200,000 recurrent cases.,. The average age of incidence of a first myocardial infarction is 65.1 for men and 72 for women.


Symptoms suggestive of ACS
Noncardiac diagnosis
Chronic stable angina
Possible ACS
Definite ACS
Treatment as indicated by alternative diagnosis
See Chap. 267
No ST-segment elevation
ST-segment elevation
Observe 12 h or more from symptom onset
Nondiagnostic ECG
Normal initial cTn
ST- and/or T-wave changes
Ongoing pain or elevated cTn
Hemodynamic abnormalities
No recurrent pain; negative follow-up studies
Recurrent ischemic pain or positive follow-up studies
Diagnosis of ACS confirmed
See Chap. 269
Stress study to provoke ischemia
Consider evaluation of LV function if ischemia is present
Negative
Potential diagnoses: nonischemic discomfort; low-risk ACS
Positive
Diagnosis of ACS confirmed or highly likely
Admit to hospital
Manage via acute ischemia pathway
Outpatient follow-up

ACUTE CORONARY SYNDROME (ACS)

(1) Acute precordial pain

ACUTE CARDIAC DEATH
(fatal arrhythmia)

sec/min

45 - 60 min

(2) ECG

ST- elevation

non ST- elevation
ST- depression

(3) Cardiac Markers

positive

Myocardial infarction (MI)

positive

negative

Unstable angina pectoris

Hours

STEMI

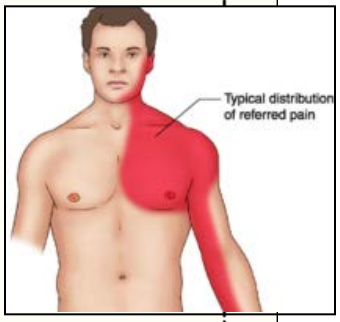
NSTEMI

Q-wave MI (QMI)

non-Q-wave MI (NQMI)

Days

Months



CRP

cTroponin T or I

CK-MB

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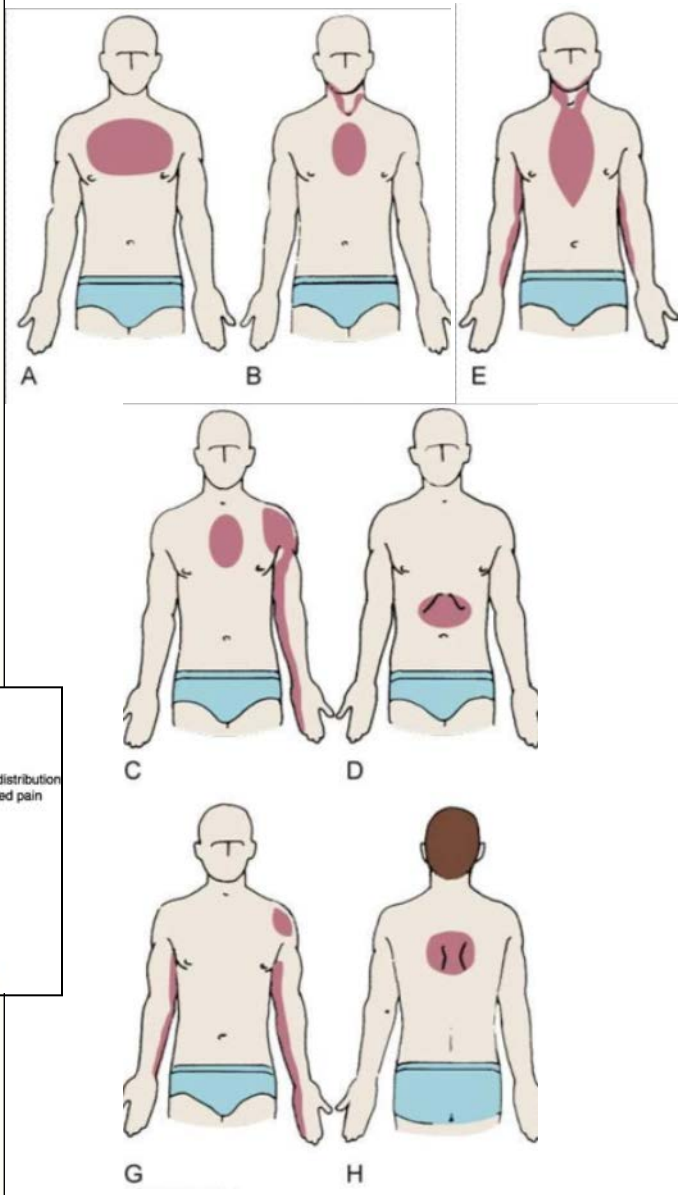
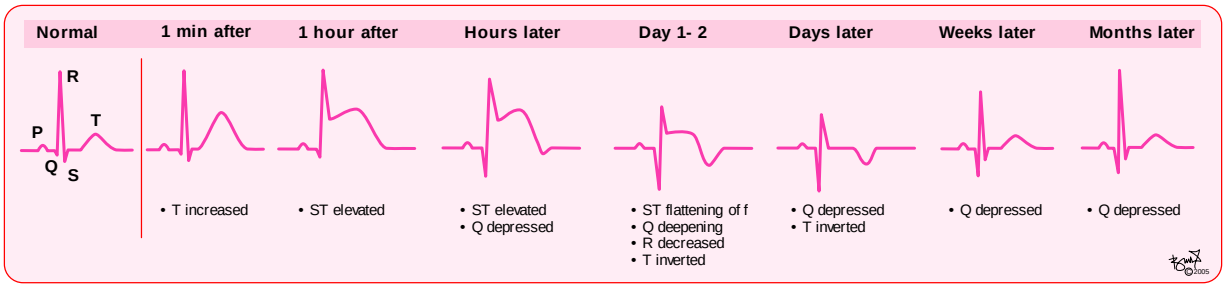


Pain projection in ACS

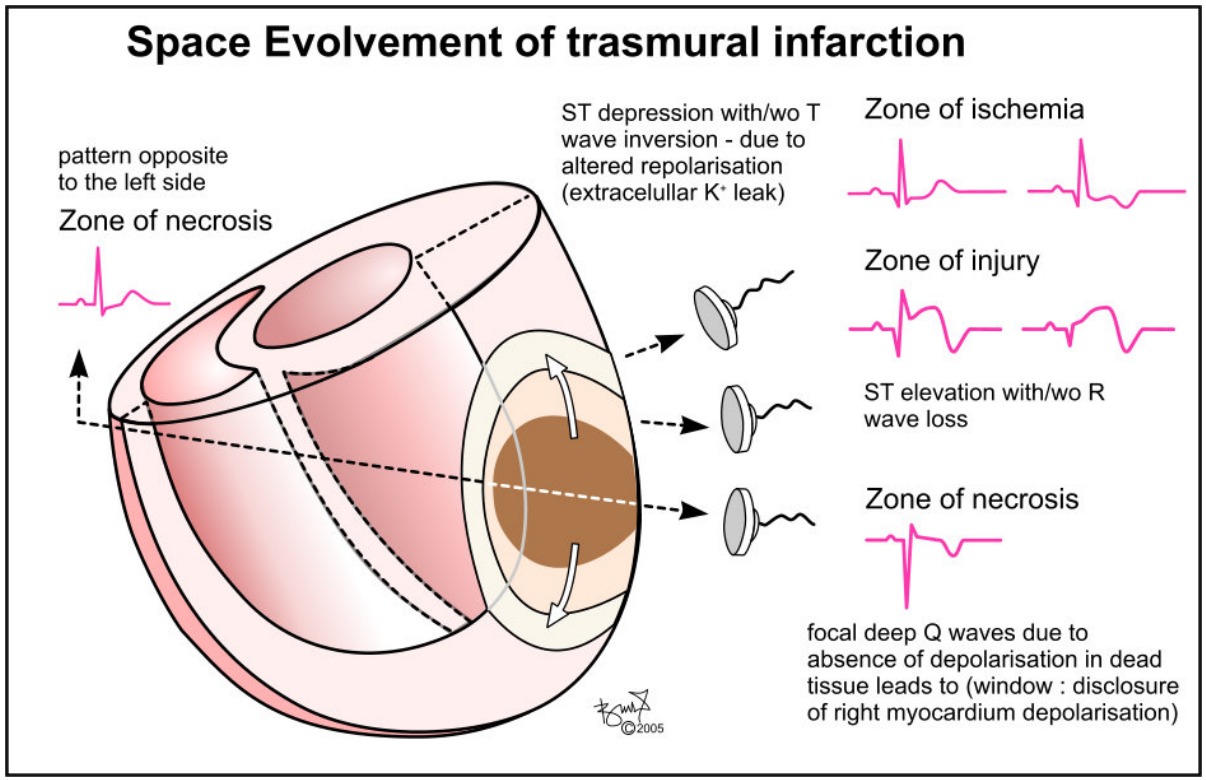
ST-Segment Elevation Myocardial Infarction (STEMI)

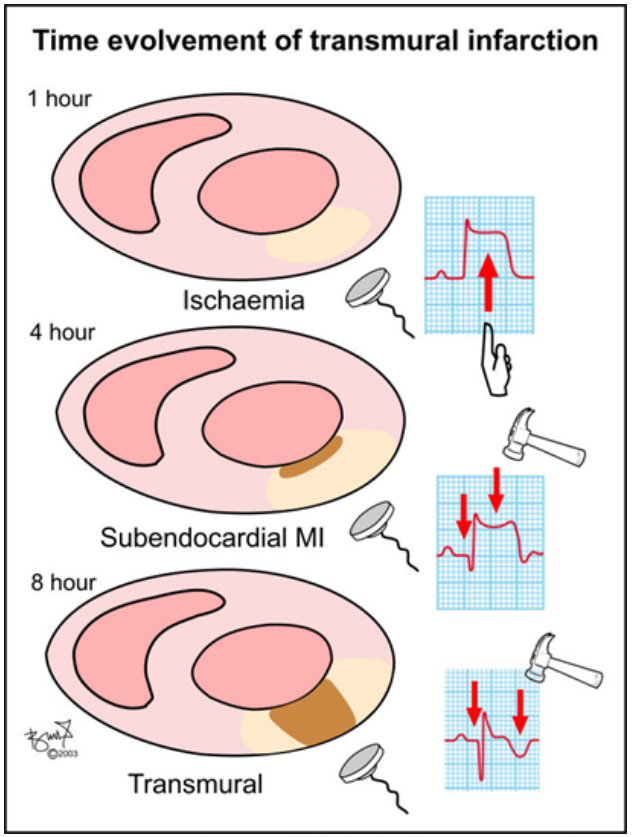
- Def.: Mostly transmural myocardial infarction (MI)
- Occ. App. ~38% of patients who present with ACS have STEMI
- Pa : Diminished cellular glycogen, relaxed myofibrils, sarcolemmal disruption, are the first ultrastructural changes and are seen ~10–15 min after the onset of ischemia. Mitochondrial abnormalities ~10 minutes after occlusion; myocyte necrosis progresses from the subendocardium to the subepicardium in man over several hours postmortem.
- Etio: occlusion of 1 or more coronary arteries; plaque rupture, erosion, fissuring, or dissection of coronary arteries with obstructive thrombus. Risk factors dyslipidemia, diabetes, hypertension, smoking, family history of coronary artery disease.
- Ptg: complete and persistent blood flow occlusion
  - .high-risk thin-cap fibroatheroma → sudden-onset plaque rupture cascade of platelet adhesion, activation, and aggregation, ultimately leading to thrombosis
  - "wave-front" of myocardial injury that spreads from the subendocardial myocardium to the subepicardial myocardium, resulting in a transmural infarction that produces ST-segment elevation on surface ECG.
- Lab: a transmural infarction that produces ST-segment elevation on surface ECG

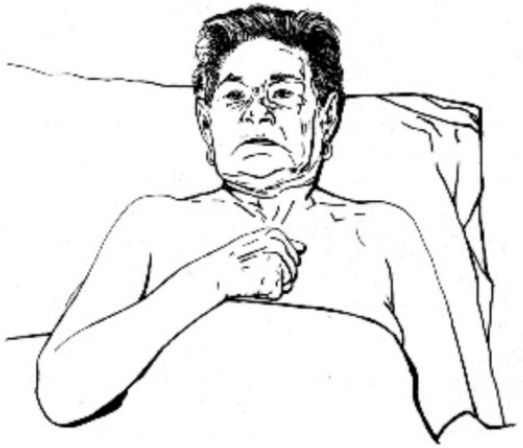
Typical temporal evolvement of STEMI



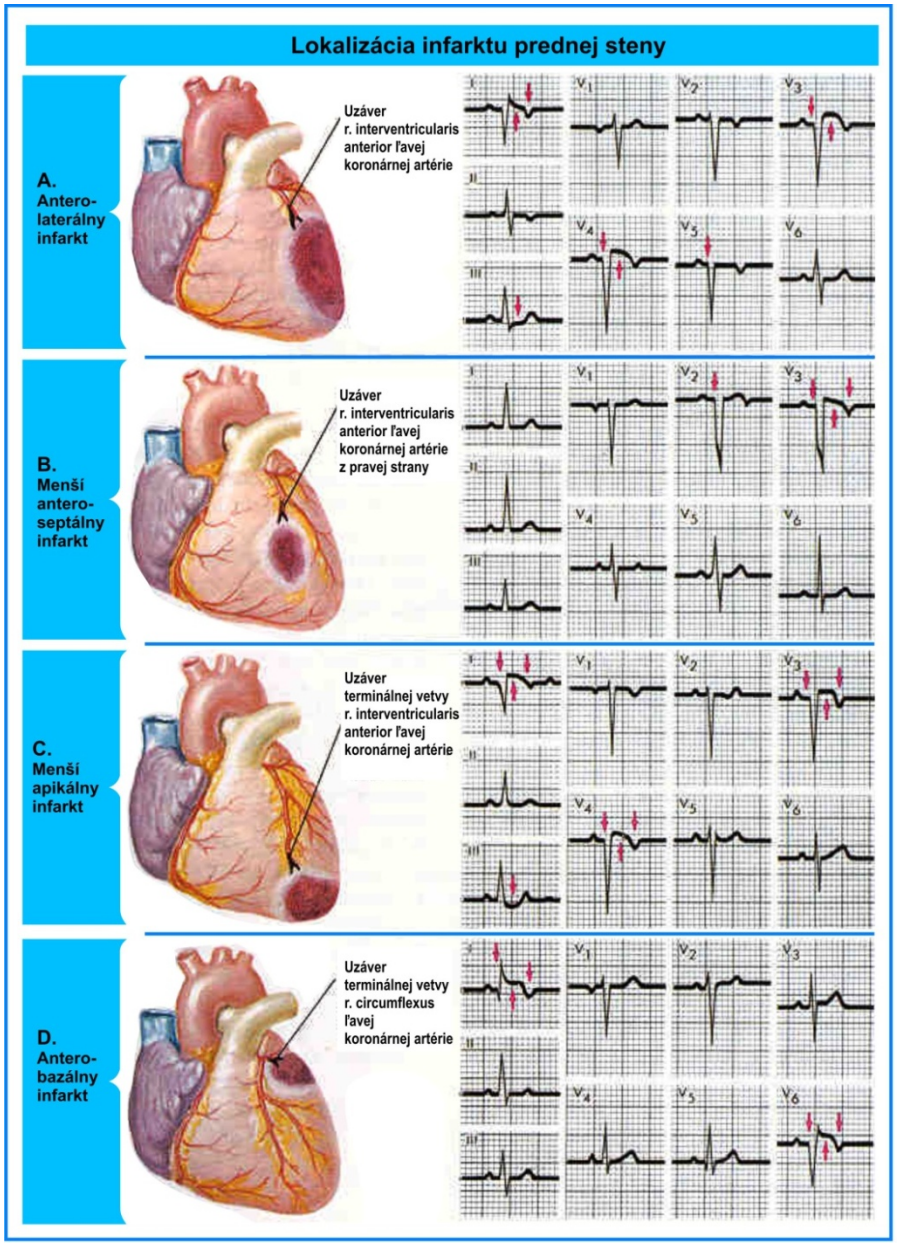
Evolution of ECG changes in trasmural MI in the lead over the necrosis. The earliest change emerge in minus as rise of T wave followed 30-60 min after by elevating whole ST segment ("the current of injury"). Progressive loss of R wave and deepening of Q wave signal trasmural necrotic window .

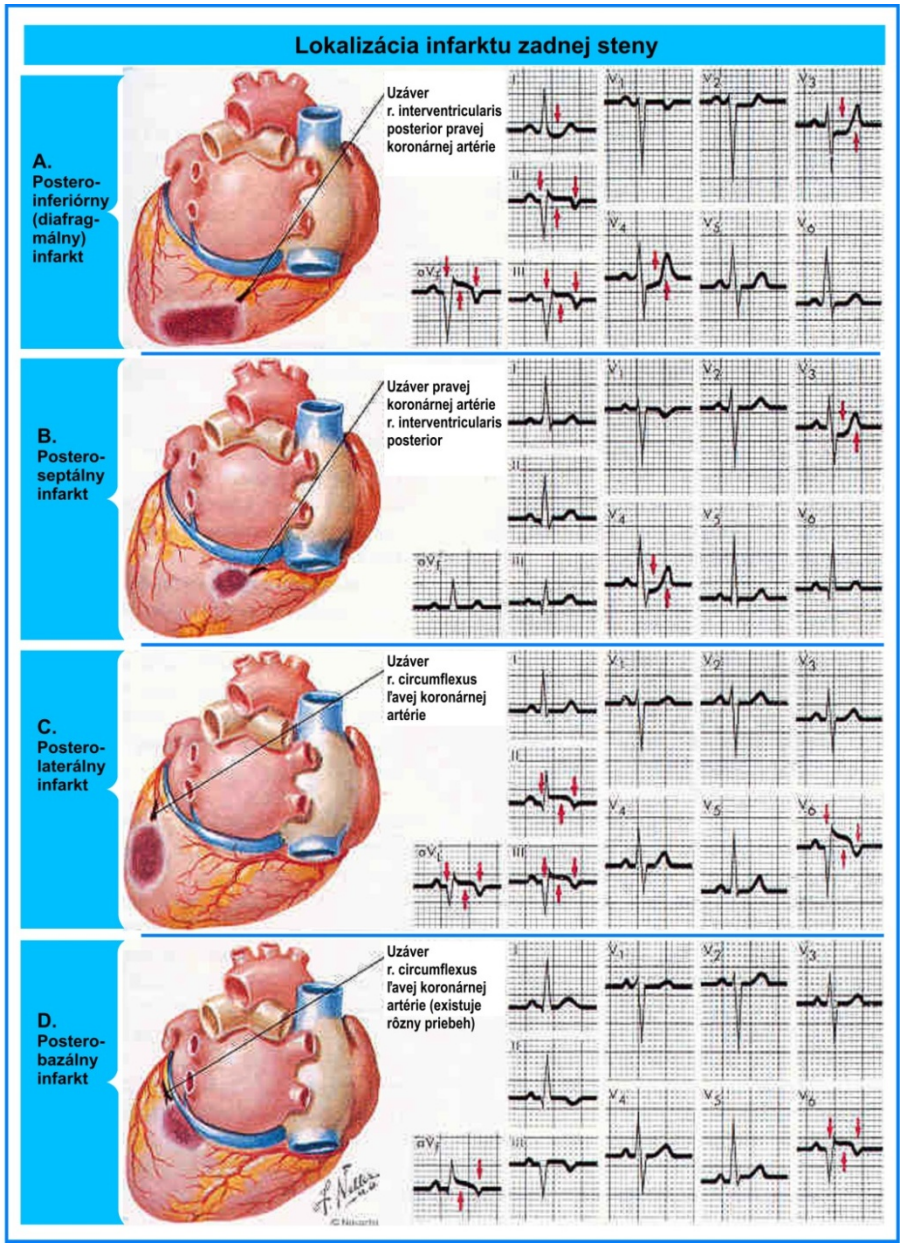






Myocardial infarction

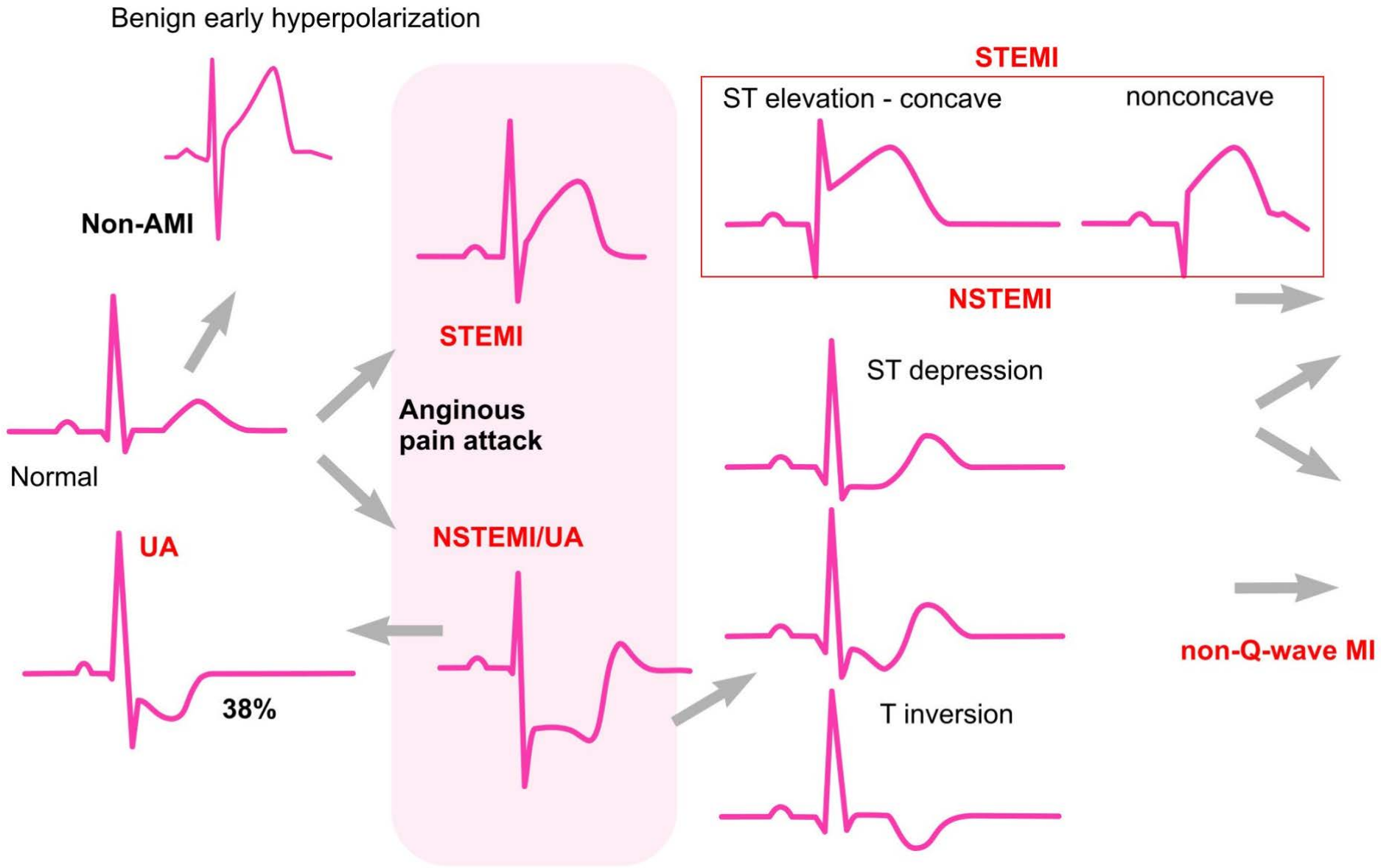




Non-ST-Segment Elevation Myocardial Infarction (NSTE-ACS)

- Occ. The relative incidence of NSTEMI is rising due to the increasing burden of *diabetes and chronic kidney disease in an aging population;* 65%–90% are type 1 MI; 10- 35% type 2 MI.
- Pa : ~10% stenosis of the left main coronary artery, **35% have three-vessel CAD**, 20% **have two-vessel disease**, 20% have single-vessel disease, and 15% have no apparent epicardial coronary artery stenosis; (studied at angiography),
- **Etio: Vulnerable plaques** at risk of disruption.= eccentric stenosis with overhanging edges and a narrow neck; are composed of a lipid-rich core with a thin fibrous cap. Patients with NSTE-ACS frequently have multiple such plaques that are
- Clin: chest discomfort is severe and has at least one of three features:
  - **occurrence at rest** (or with minimal exertion), lasting >10 min;
  - **relatively recent onset** (i.e., within the prior 2 weeks)
  - **crescendo pattern** (more severe, prolonged or frequent than previous episodes).
- Lab: ECG De novo **ST-segment depression** occurs in 1/3 of patients with. It may be transient but may persist I. **T-wave** changes are more common but are less specific signs of ischemia, unless they are new and deep T-wave inversions (≥0.3 mV).
- early risk of death (1-10%),recurrent ACS (5–15%) during the first year

Various ways of progression into MI



	MI	Pulmonary infarction	Pericarditis
Chest pain			
location	Retrosternal	Ant, post or lat	Retrosternal
onset	sudden	sudden	sudden
character	Pressure like heavy squeezing	Sharp, stabbing	Sharp, stabbing and sometimes dull
Change with respiration	no		**Worse with inspiration**
Change with position	no	no	**Worse in supine , improve c sitting up**
Radiation	Jaw, neck, shoulder or arms	shoulder	Jaw, neck, shoulder, arms, **trapezius**
Duration	Min to hours	Hours to days	Hours to days
Response to NTG	**improved**	No change	No change
others			
Pericardial rub	absent	rare	**present**
S3, pul cong	**present**	absent	absent

ACUTE CORONARY SYNDROME

- Dg: *A. ECG:* (ST seg. pathol. ; telemetry (Holter)

B. Imaging (chest X-ray)

C. Blood tests : *Biomarkers Early:*

h-FABP (heart specific Fatty Acid Binding Protein

(onset 30 min; peak 6 h; return 18 -24 h)

IMA (Ischemia-Modified Albumin) (in % of total) = conformational mode; low binding of Cu or Co

Myeloperoxidase (MPO)

Glycogen Phosphorylase Isoenzyme BB-(GPBB)

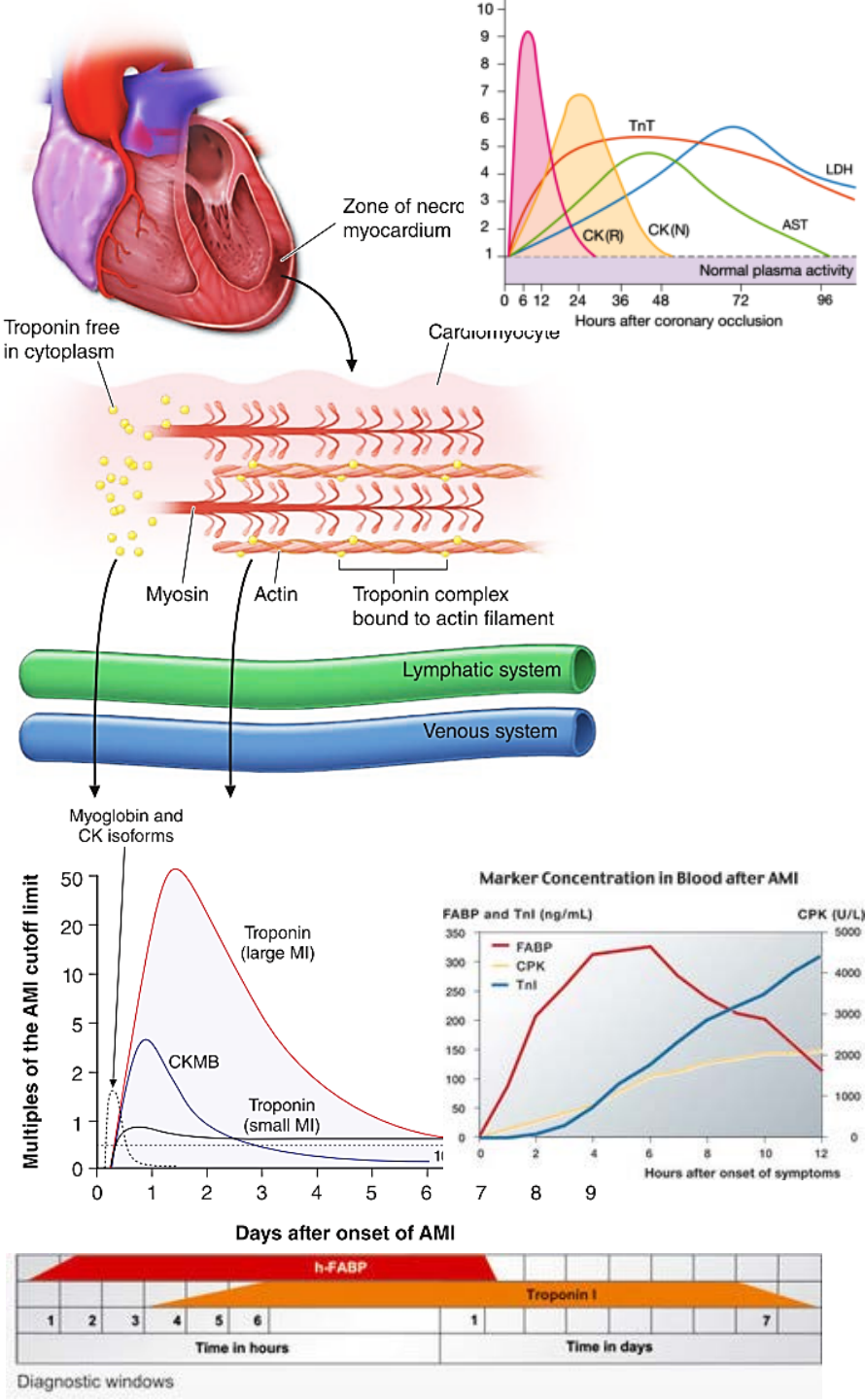
Biomarkers Late: troponin I or T (cTnI; cTnT), CK – MB, AST, LDH, ALT; D-dimer

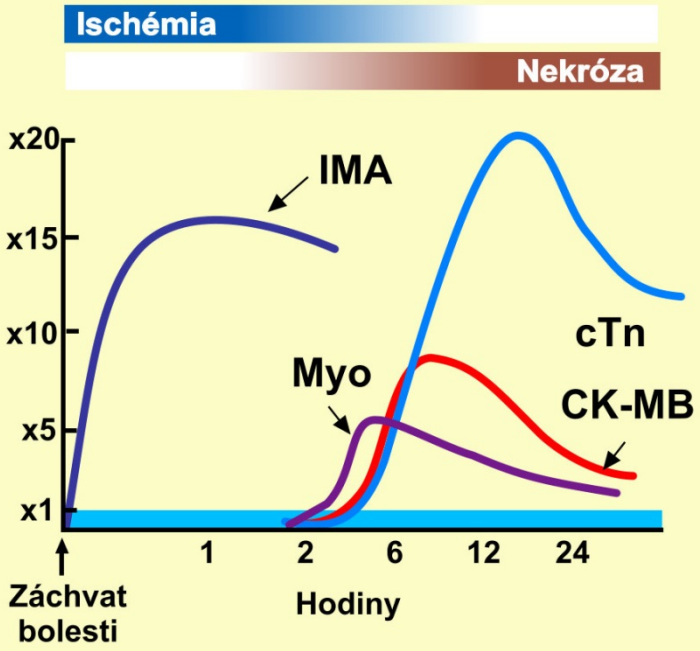
Prognostic markers:

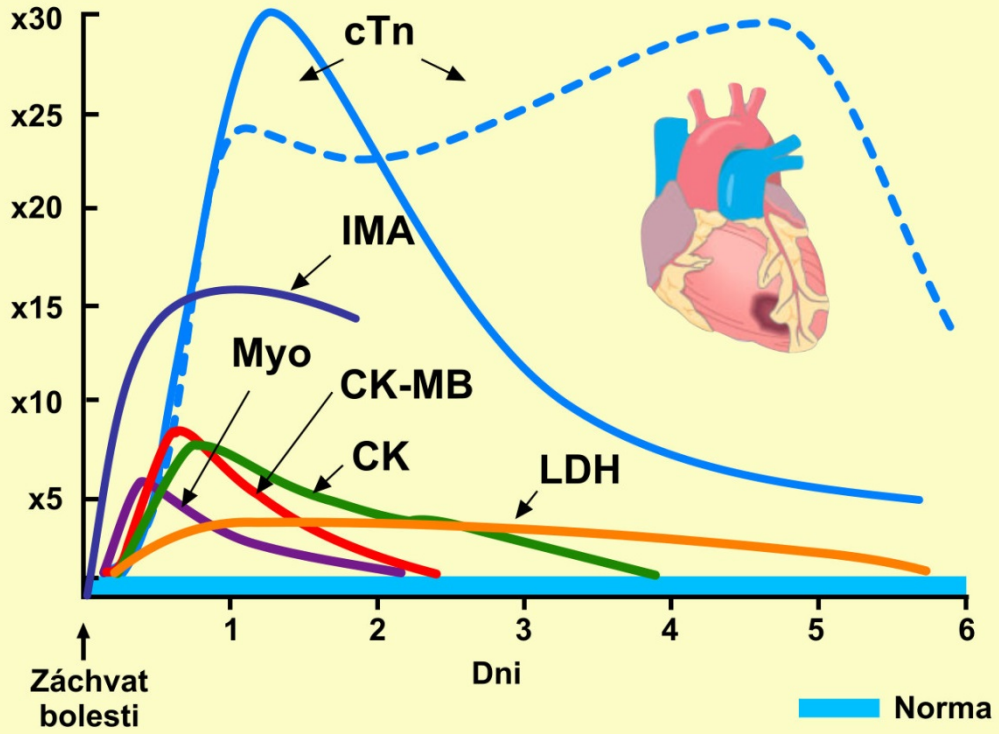
Natriuretic peptide - BNP and N-terminal Pro BNP (risk of death and heart failure following ACS)

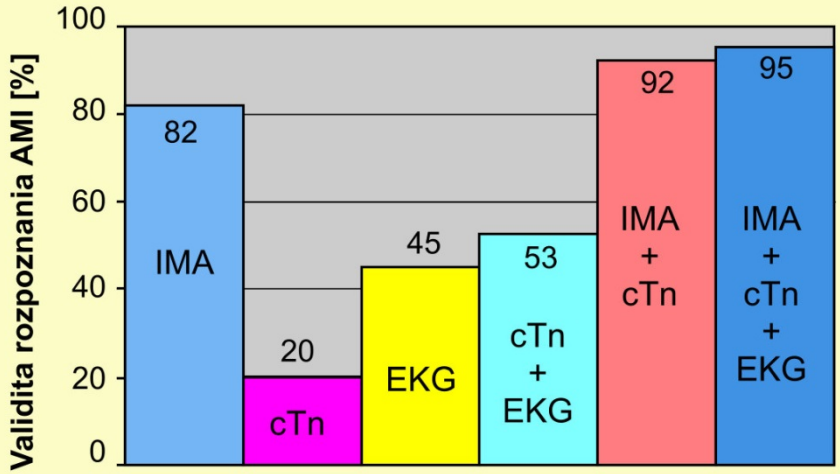
Monocyte chemoattractive protein (MCP)-1 - higher risk of adverse outcomes after ACS.

- Th: angioplasty or thrombolysis







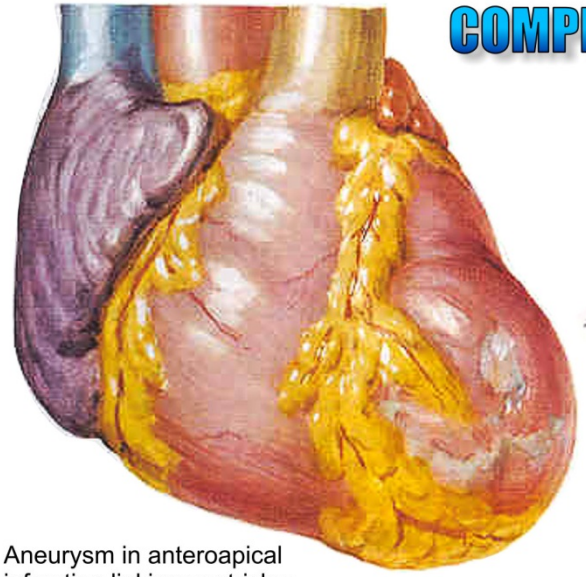


- Cardiac troponin I (cTnI) and T (cTnT) are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the heart
- cTnI and cTnT. high-sensitivity (hs)–cTn are the preferred biomarkers for the evaluation of myocardial injury
- CK-MB is less sensitive and less specific

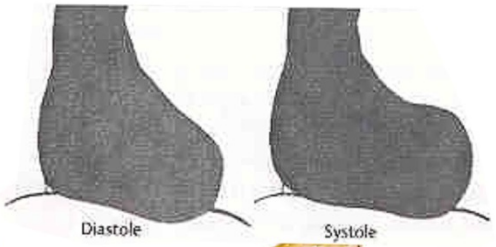
Elevation of Cardiac Troponin

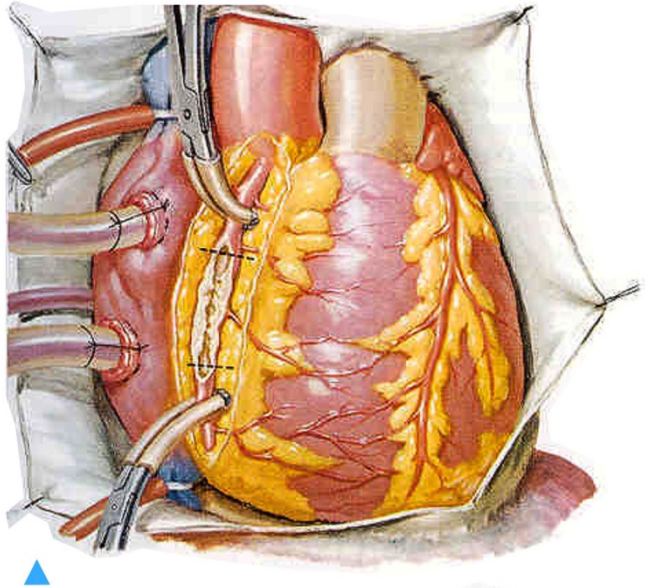
- Myocardial injury related to acute myocardial ischemia
  - Atherosclerotic plaque disruption with thrombosis
- Myocardial injury related to acute myocardial ischemia because of oxygen supply/demand imbalance
  - Reduced myocardial perfusion, eg, • Coronary artery spasm, microvascular dysfunction • Coronary embolism • Coronary artery dissection • Sustained bradyarrhythmia • Hypotension or shock • Respiratory failure • Severe anemia
  - Increased myocardial oxygen demand, eg, • Sustained tachyarrhythmia • Severe hypertension with or without left ventricular hypertrophy
  - Other causes of myocardial injury
    - Cardiac conditions, eg, • Heart failure • Myocarditis • Cardiomyopathy (any type) • Takotsubo syndrome • Coronary revascularization procedure • Cardiac procedure other than revascularization • Catheter ablation • Defibrillator shocks • Cardiac contusion
    - Systemic conditions, eg, • Sepsis, infectious disease • Chronic kidney disease • Stroke, subarachnoid hemorrhage • Pulmonary embolism, pulmonary hypertension • Infiltrative diseases, eg, amyloidosis, sarcoidosis • Chemotherapeutic agents • Critically ill patients

COMPLICATIONS OF INFARCTION

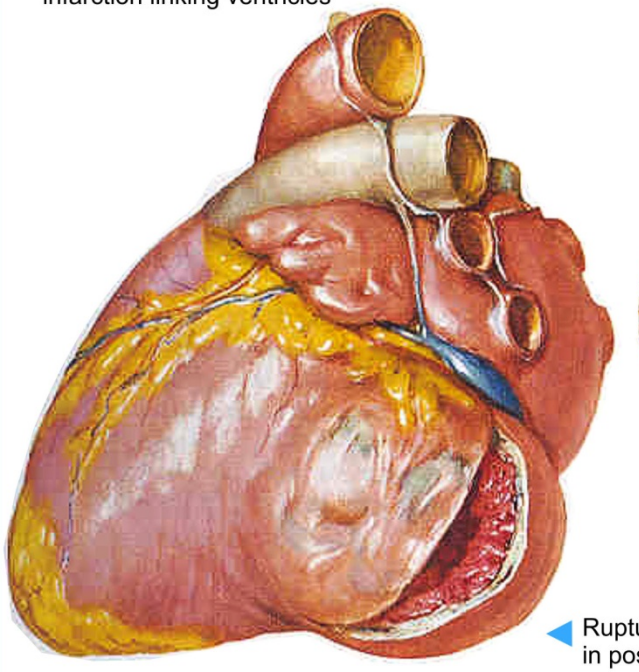


Aneurysm in anteroapical infarction linking ventricles

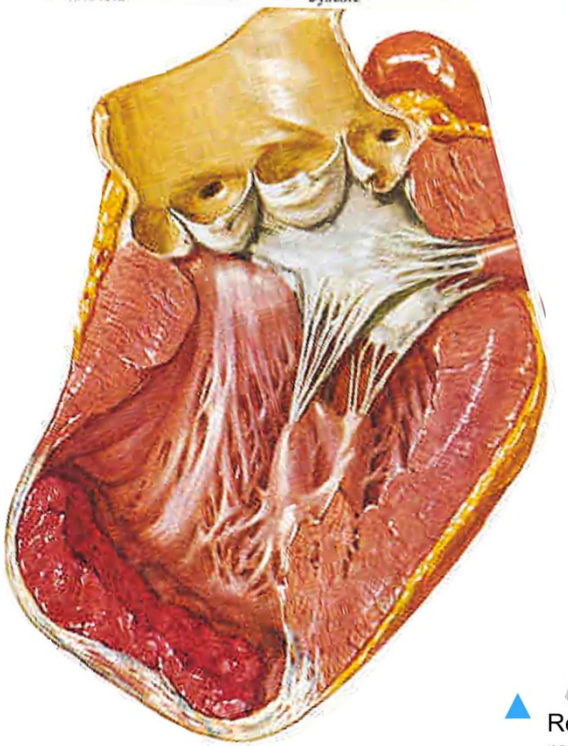




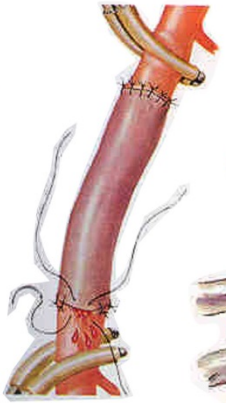
Obliterated segment of right coronary artery will be resected



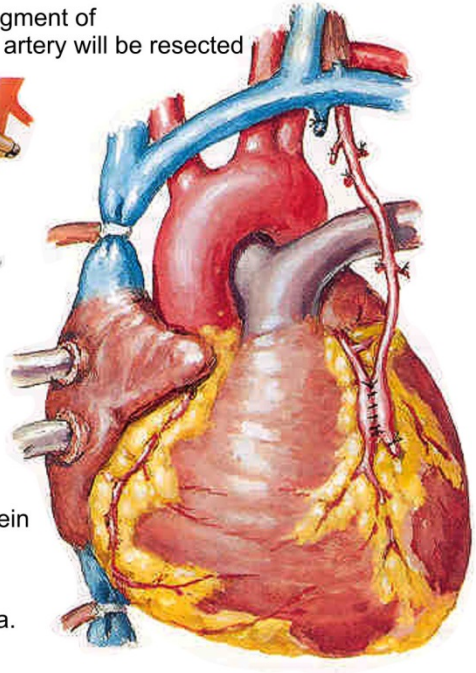
Rupture in aneurysm deleloped in posterior ventricular wall



Thrombus developing within aneurysm in ventricular septum



Replacement of resected part by vein stupm



IMA - bypass. Anastomosis between a. thoracica interna and r. interventricularis