Acute coronary syndrome

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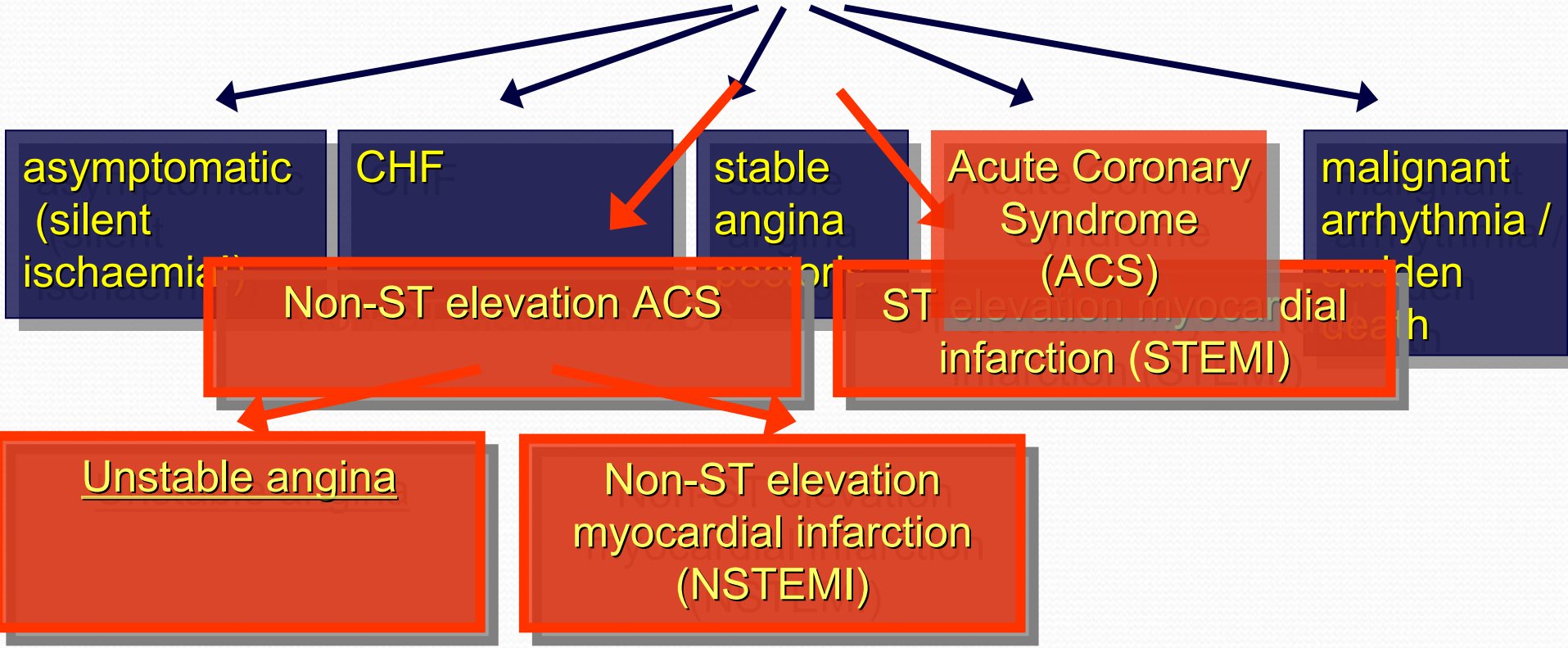
About the acute coronary syndrome

- Definition
- Pathophysiology
- Diagnosis
- Differential diagnosis
- Treatment

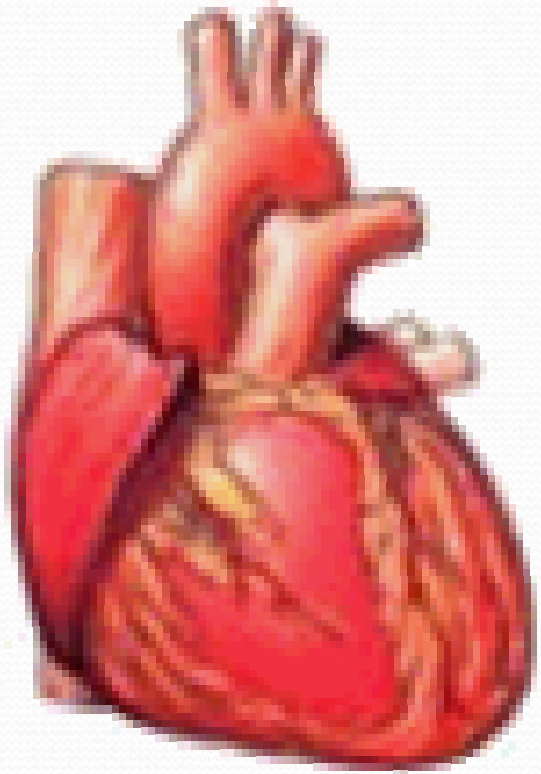
What is the acute coronary syndrome?

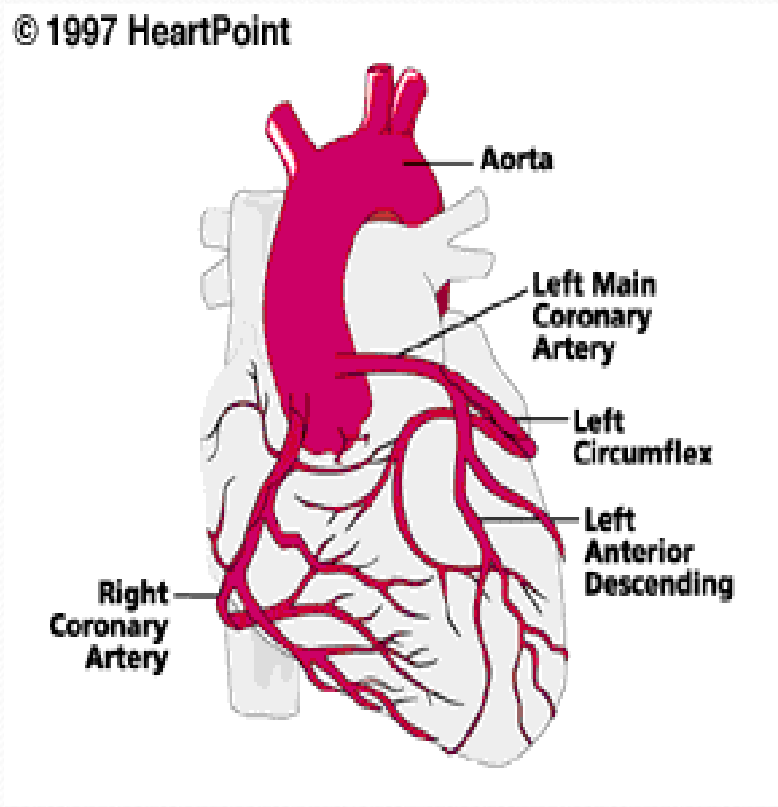
- Acute
- Left-threatening
- Coronary event

The clinical presentation of the ischaemic heart disease

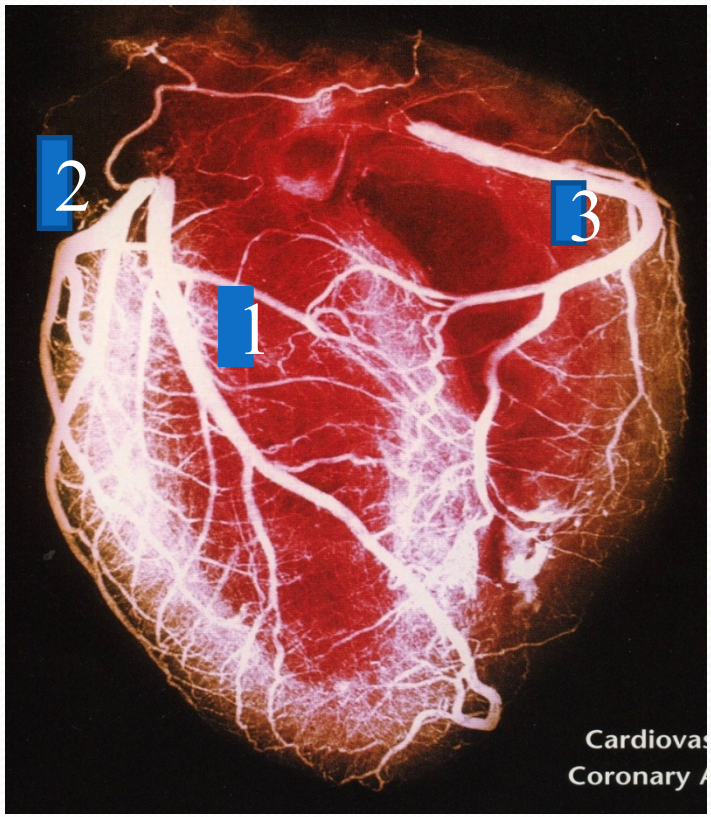


Coronary arteries





Coronary arteries (posterior view)



- 1. LAD (ด้านหน้า)
- 2. LCX (ด้านข้าง)
- 3. RCA(ด้านขวา)

World Health Organization

Diagnosis of Myocardial Infarction

requires ≥2 of the following

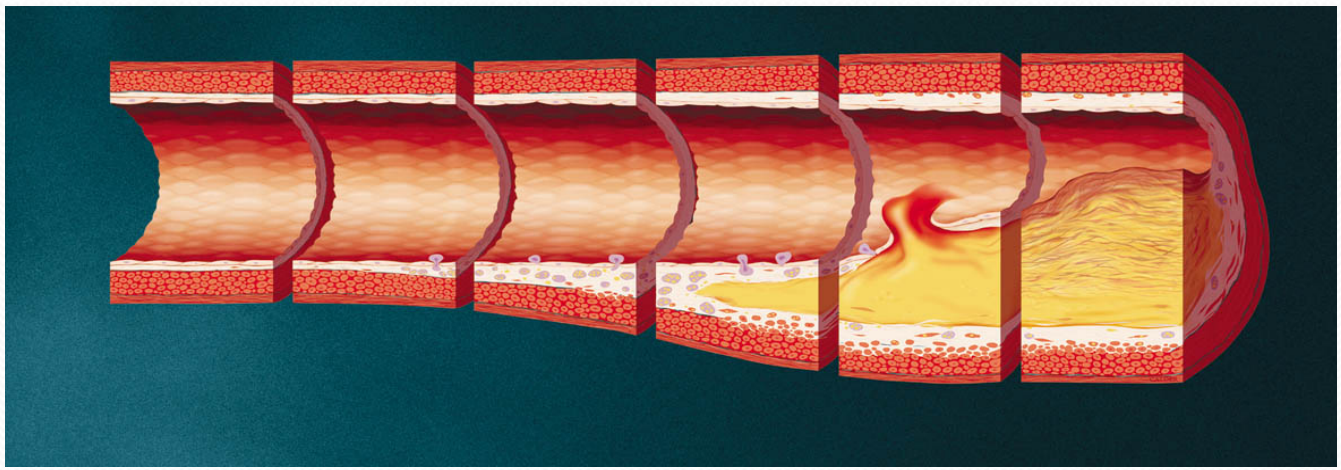
- 1. Prolonged ischemic-type chest discomfort
- 2.Serial ECG changes
- 3.Rise and fall of serum cardiac markers

Pathophysiology:

Acute coronary perfusion deficit

- Mechanism:
  - coronary plaque rupture (95%)→ lead to partial or total coronary occlusion
  - coronary spasm
    - Prinzmetal angina (transient ST elevation)
    - myocardial infarction (if the ischemic period is to long)
  - coronary embolisation

Degenerative change



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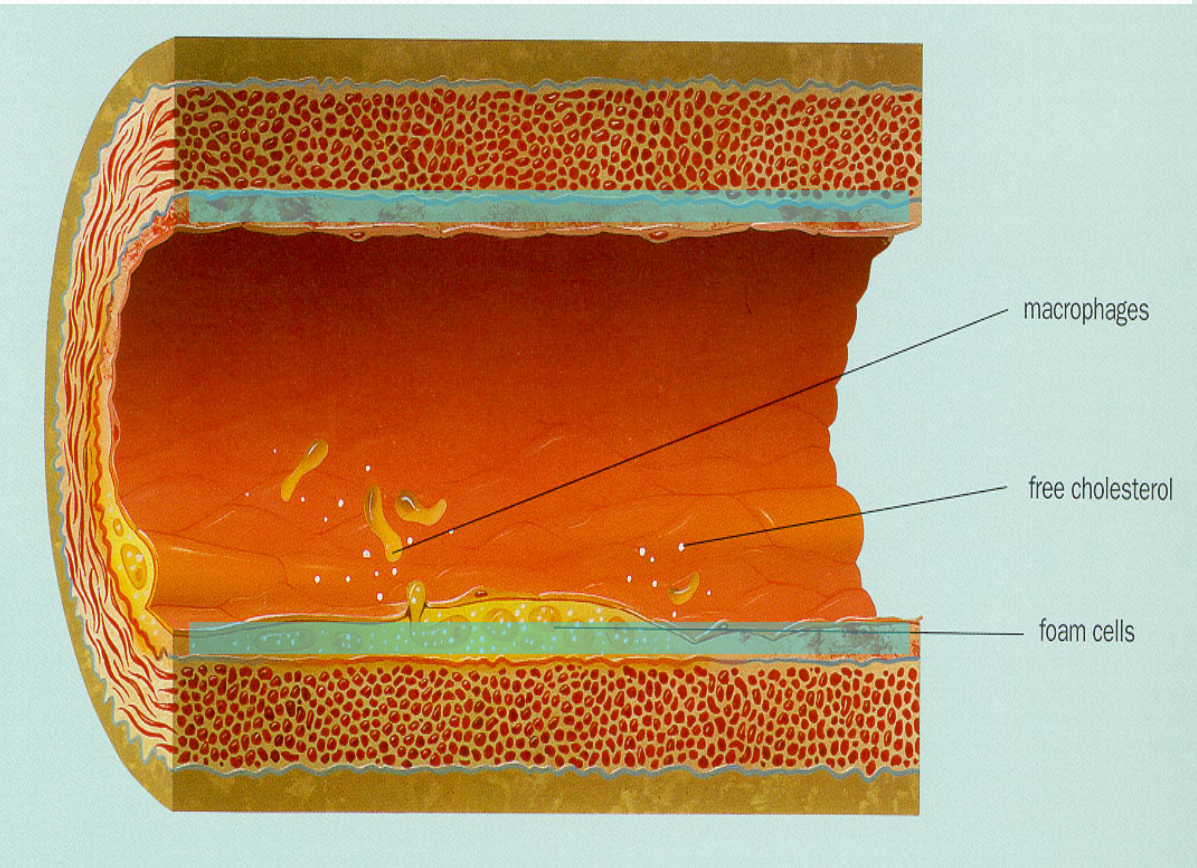

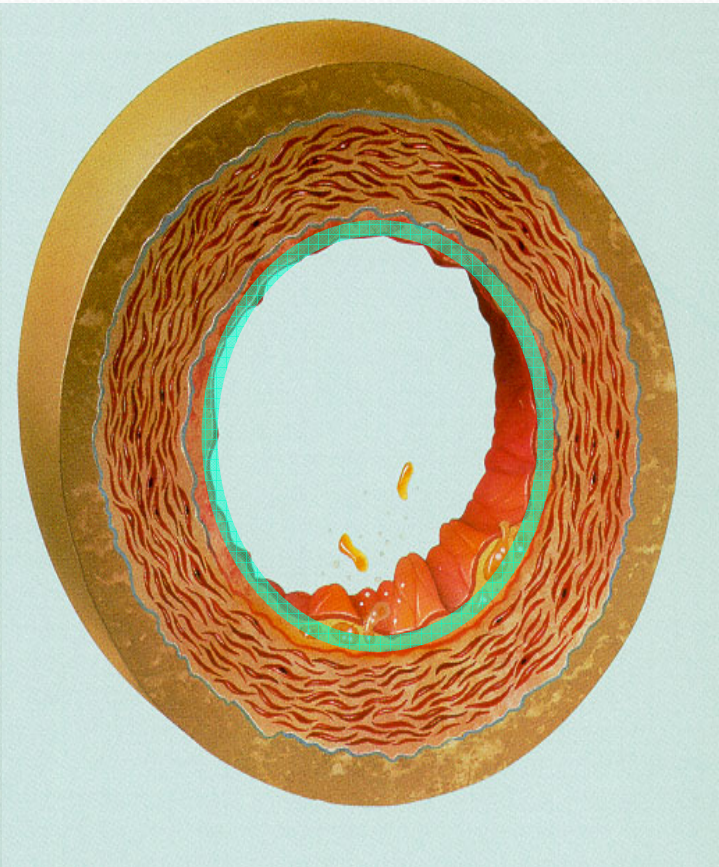

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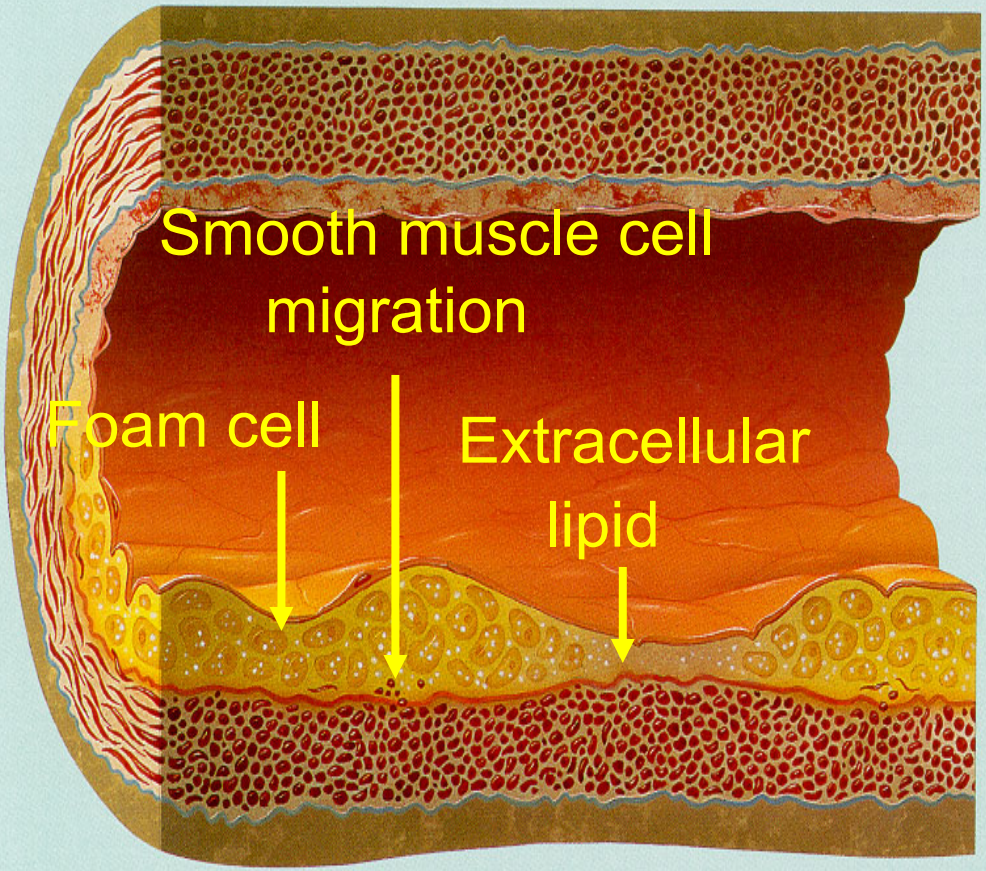

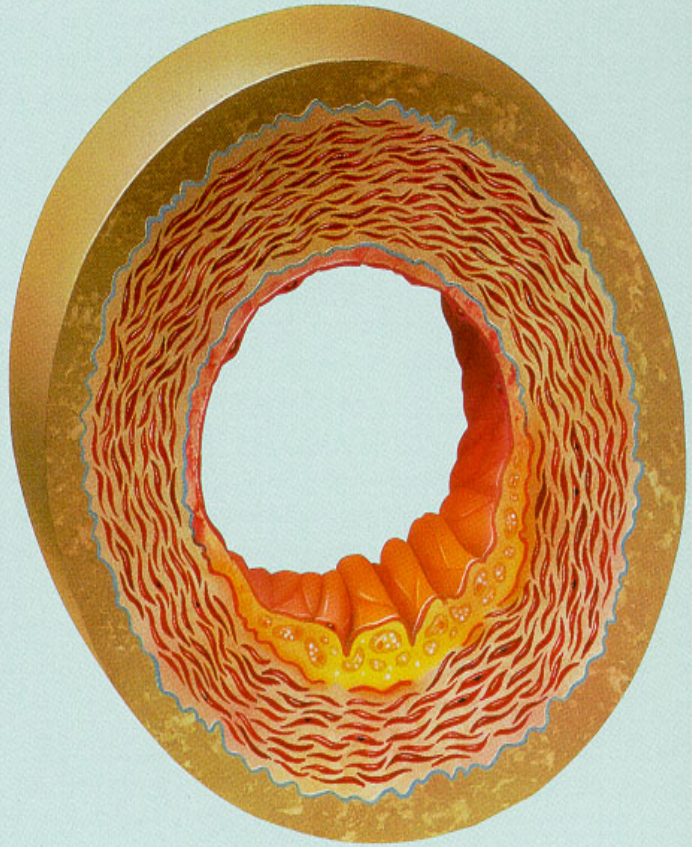

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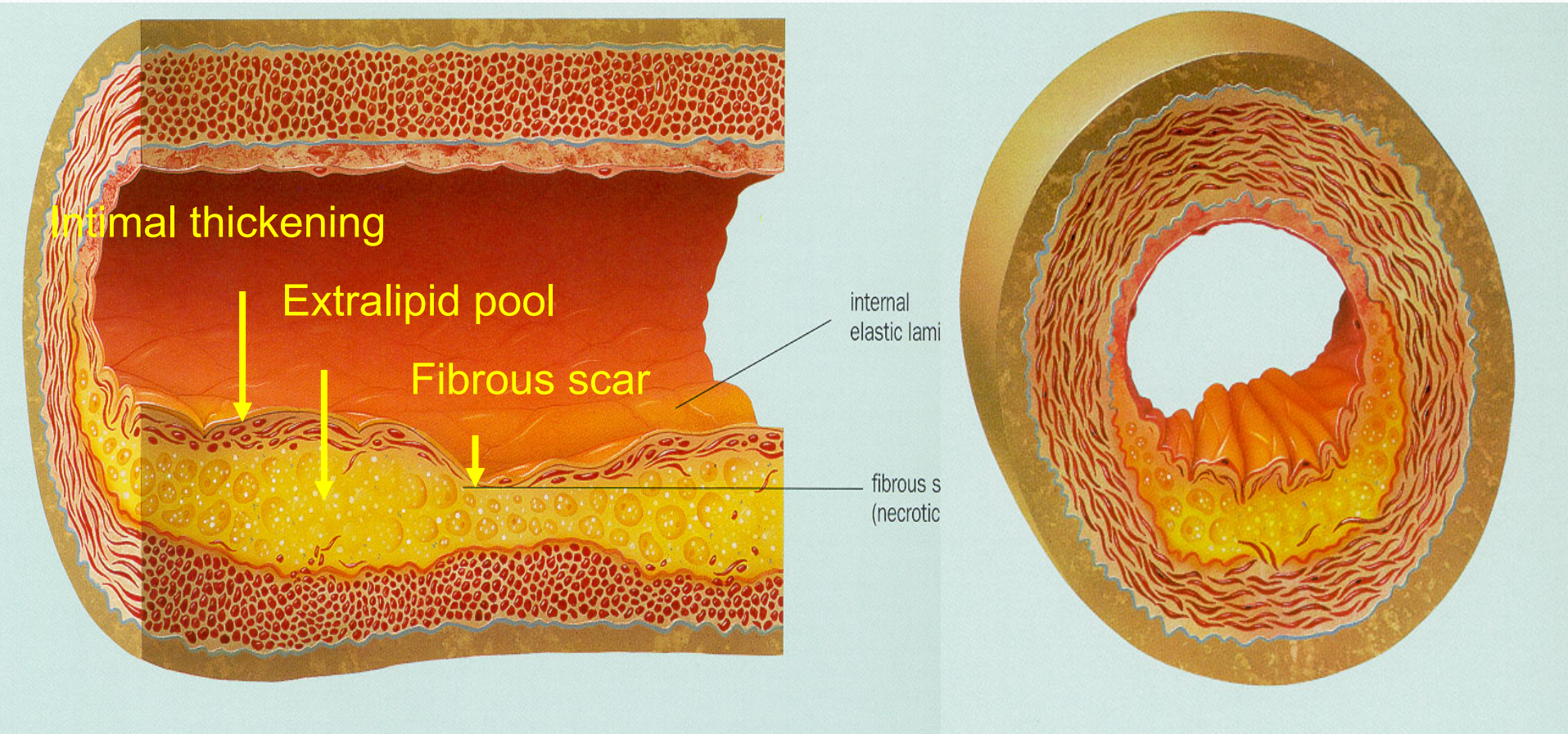

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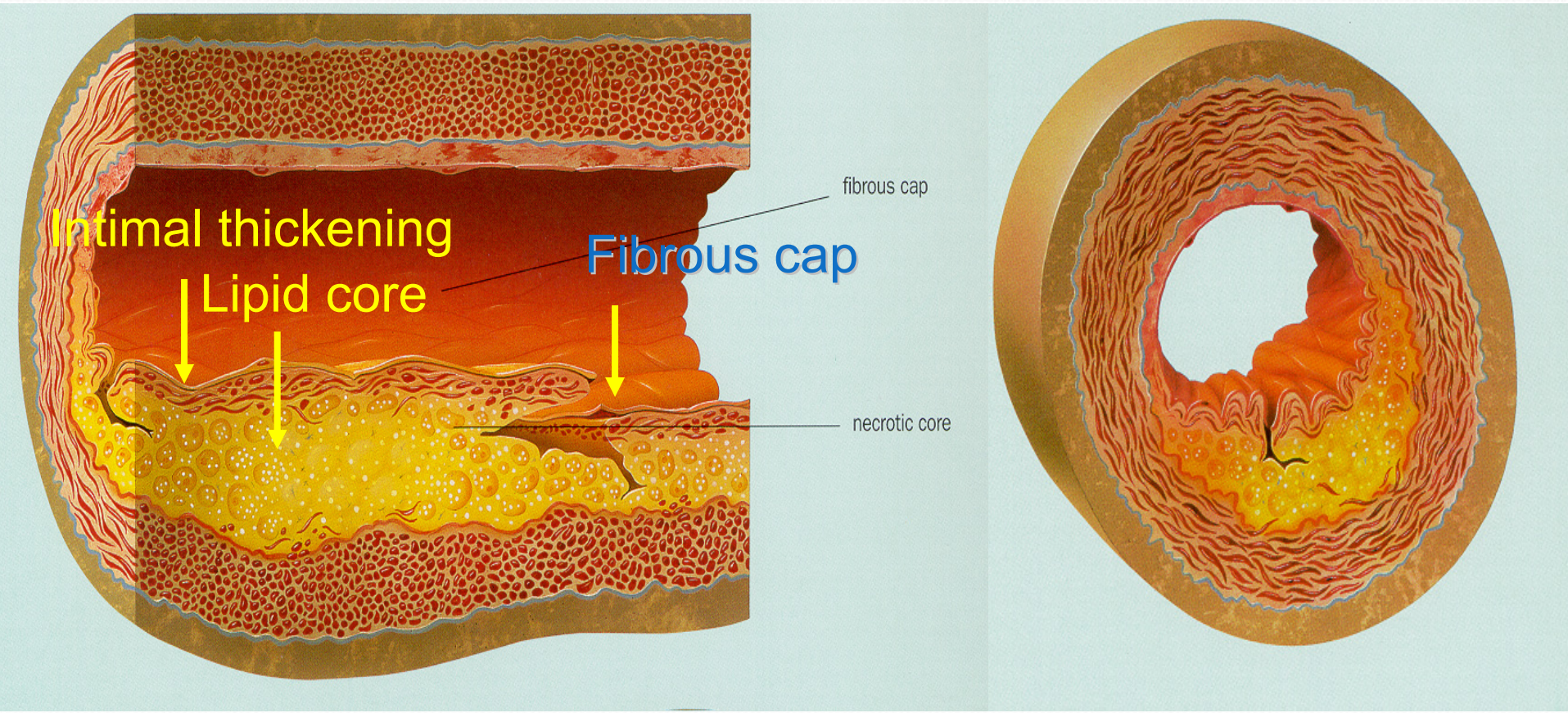

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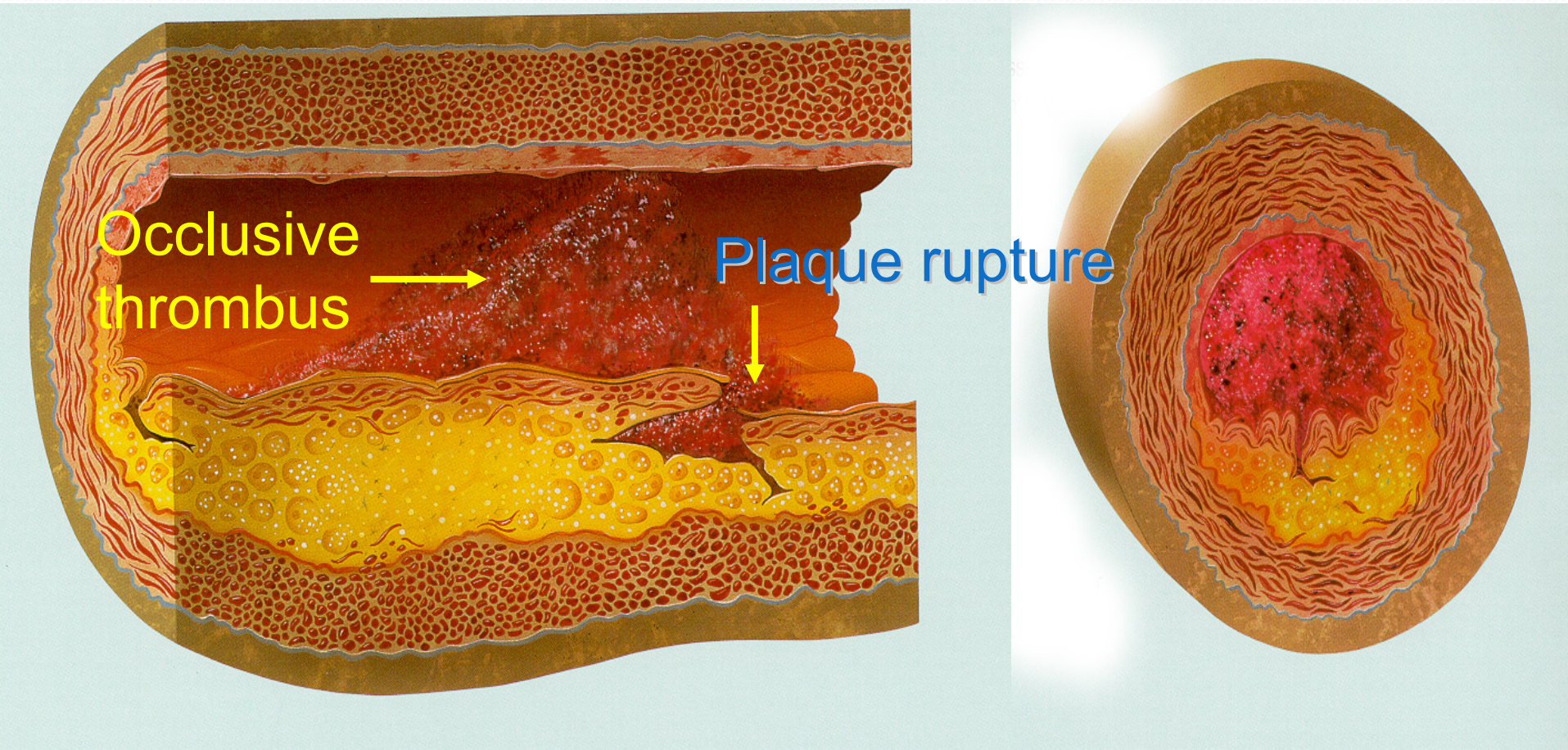
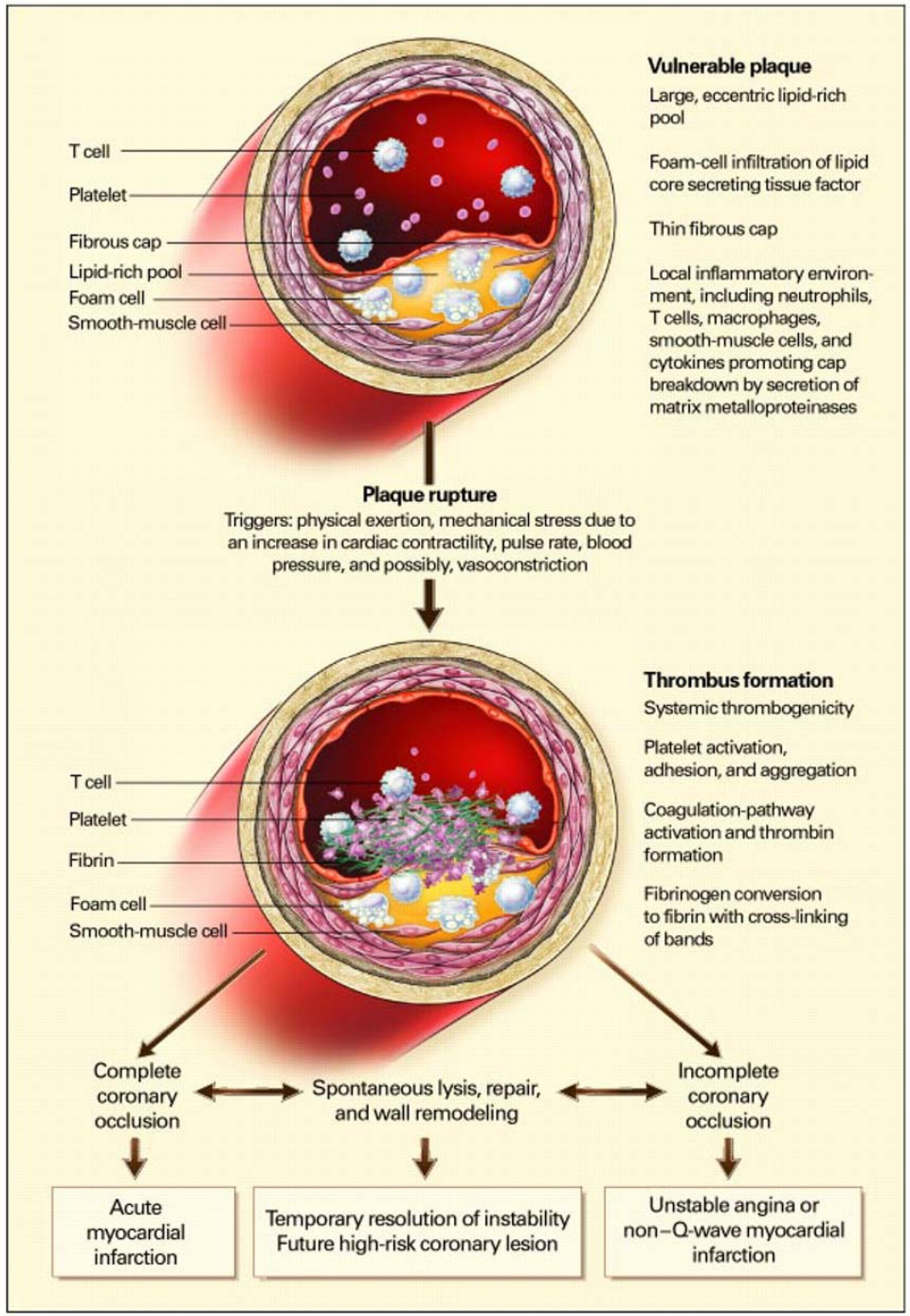
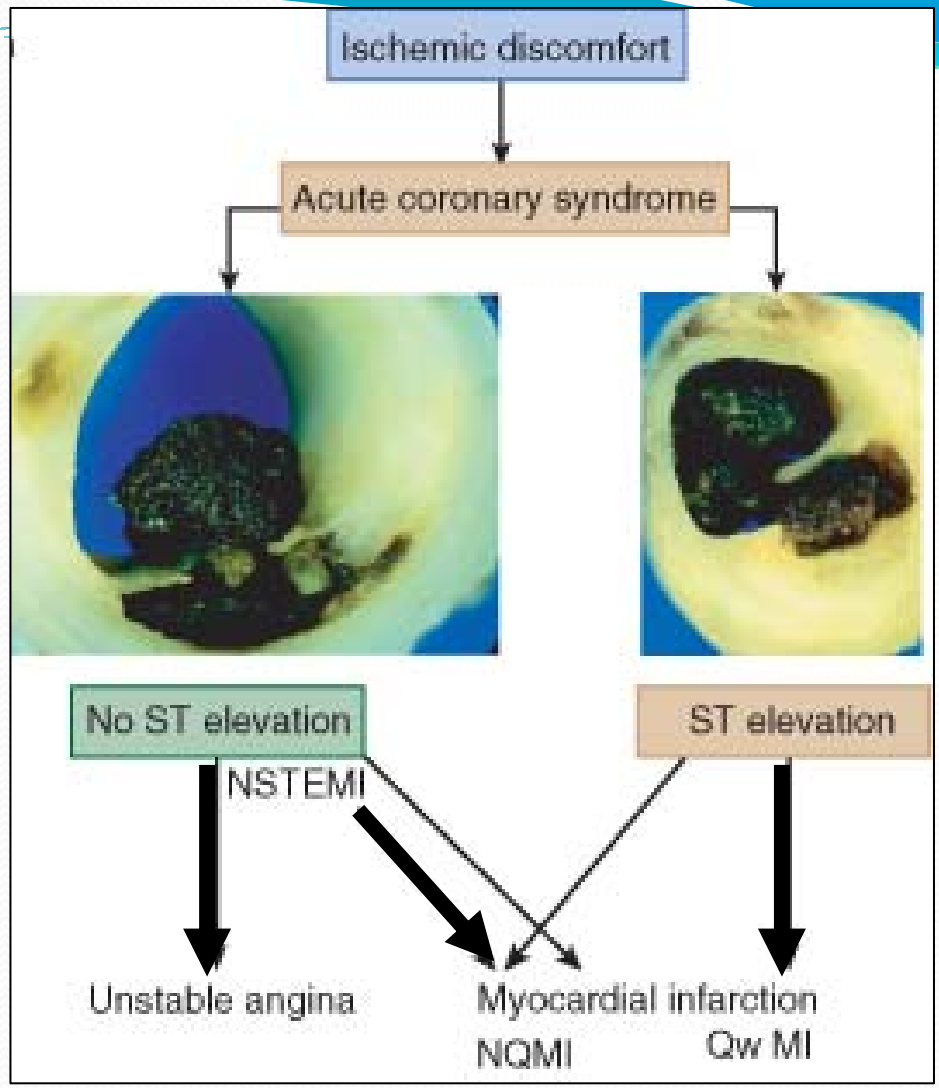



T cell
Platelet
Fibrous cap
Lipid-rich pool
Foam cell
Smooth-muscle cell
Vulnerable plaque
Large, eccentric lipid-rich pool
Foam-cell infiltration of lipid core secreting tissue factor
Thin fibrous cap
Local inflammatory environment, including neutrophils, T cells, macrophages, smooth-muscle cells, and cytokines promoting cap breakdown by secretion of matrix metalloproteinases
Plaque rupture
Triggers: physical exertion, mechanical stress due to an increase in cardiac contractility, pulse rate, blood pressure, and possibly, vasoconstriction
T cell
Platelet
Fibrin
Foam cell
Smooth-muscle cell
Thrombus formation
Systemic thrombogenicity
Platelet activation, adhesion, and aggregation
Coagulation-pathway activation and thrombin formation
Fibrinogen conversion to fibrin with cross-linking of bands
Complete coronary occlusion
Spontaneous lysis, repair, and wall remodeling
Incomplete coronary occlusion
Acute myocardial infarction
Temporary resolution of instability
Future high-risk coronary lesion
Unstable angina or non–Q-wave myocardial infarction



partial coronary occlusion

total coronary occlusion

By ECG (at admission)

biomarker

ECG (at discharge)

Pathophysiology:

Acute coronary perfusion deficit

- Mechanism:
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  - coronary spasm
    - Prinzmetal angina (transient ST elevation)
    - myocardial infarction (if the ischemic period is to long)
  - coronary embolisation

Prinzmetal angina

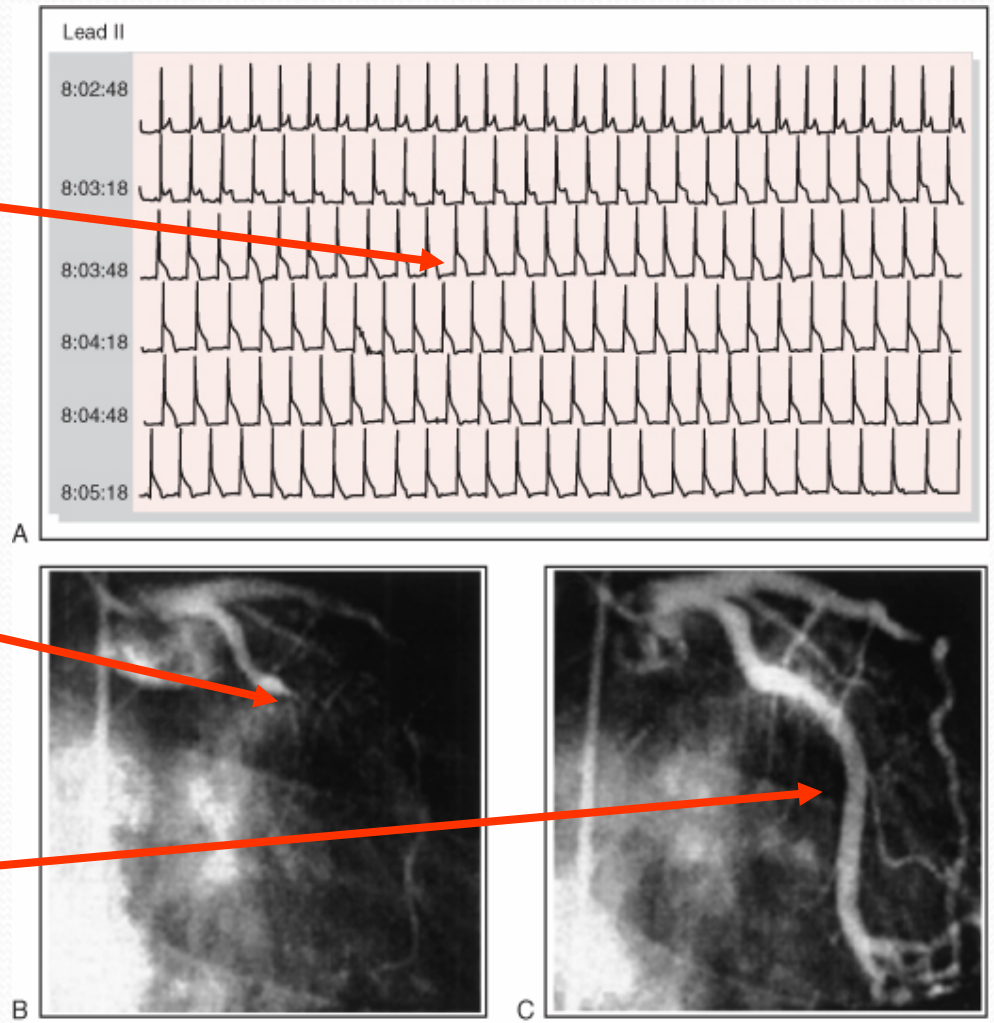
- Temporary partial or total coronary occlusion due to coronary spasm
- mainly at the middle adge women
- it can lead to malignant arrythmia (VF)
- Need an urgent coronary angiography
- prognosis is different:
  - with coronary stenosis w/o revascularisation poor, with revascularisation is good
  - w/o coronary stenosis with medical treatment is good

Prinzmetal angina

Transient ST elevation during pain on the 24 hour Tape Recorder

Transient coronary occlusion w/o stenosis due to coronary spasm

Normal coronary flow after intracoronary nitroglycerin administration



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    - myocardial infarction (if the ischemic period is to long)
  - coronary embolisation (rare)

Pathophysiology:

Acute coronary perfusion deficit

- Mechanism:

  other causes

  - with stable coronary stenosis without plaque rupture

    - extreme exercise

    - sudden dropped oxygen transport capacity (major bleeding)

Ischemic-Type Chest Pain

- Typically prolonged (>30 min) and at rest
- Pattern and accompanying symptoms (including “a sense of doom”)
- Can be mimicked by pericarditis, reflux, spontaneous pneumothorax, musculoskeletal disease (costochondritis)
- Clinical Pearl = 3 serious causes of severe chest pain: acute MI, aortic dissection, pulmonary embolus

Diagnosis

- Family history –close relative with acute MI in young age or sudden death
- Medical history for the risk factor
  - : smoking state
  - : hypertension
  - : hypercholesterorimia
  - : diabetis

Diagnosis

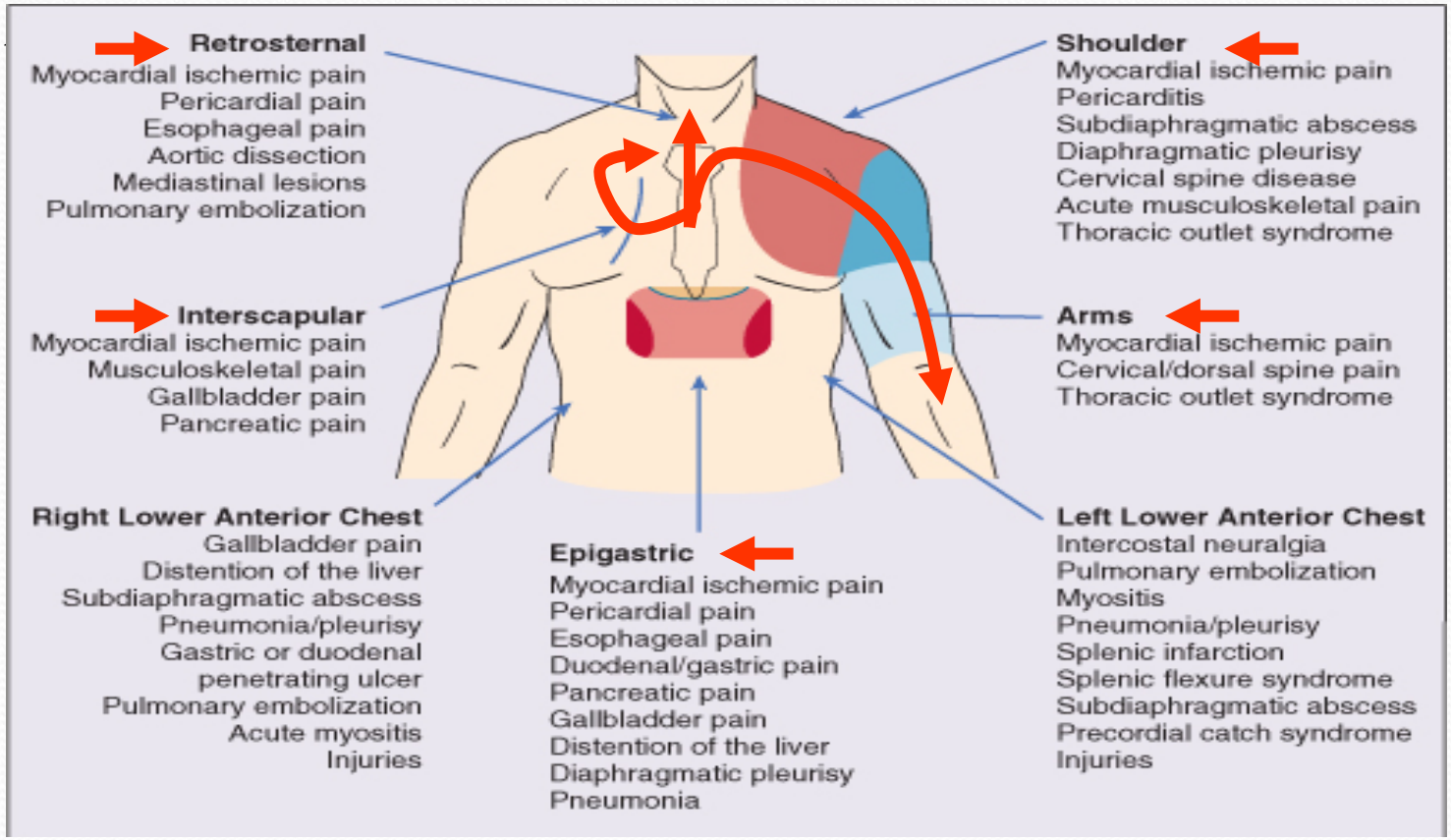
- Current complaint
  : pain
  : shortness of breath
  : sweating
  : dizzeness
  : fear of death

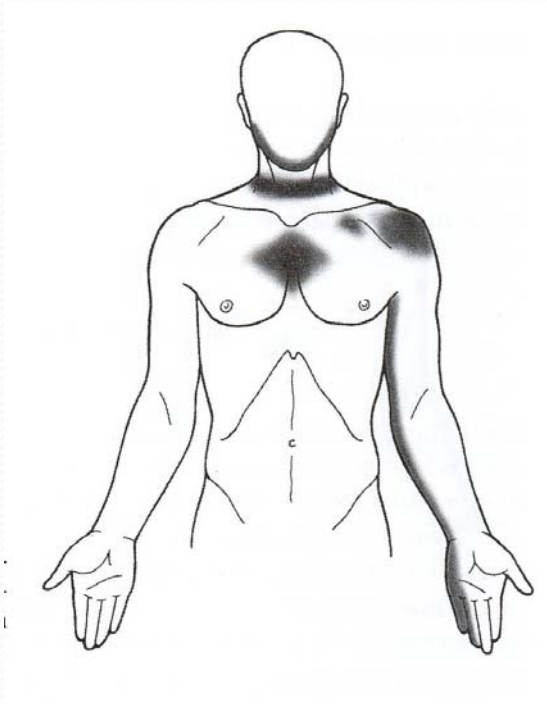
Diagnosis

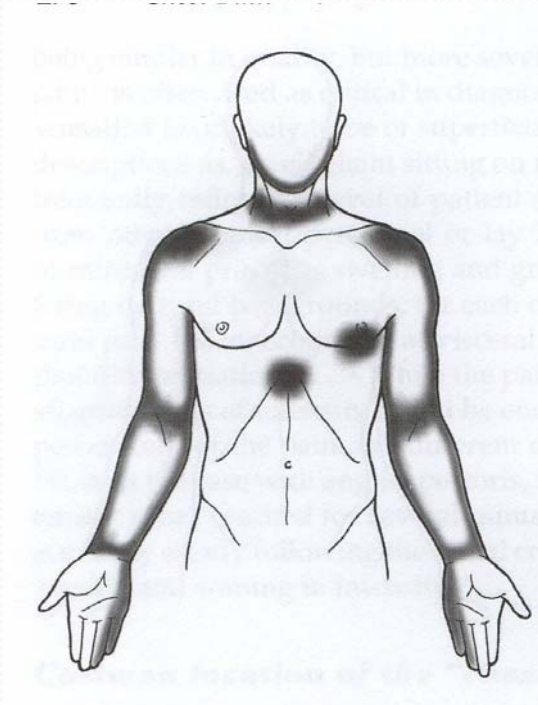
- current complaint:
  - pain
    - there are a lot of importment data of the pain:
      - localization
      - radiation
      - onset of the pain
      - the type (press, smart,cutting)
      - dinamic of the pain (continouosly, ongoing, undulaiting)
      - answer to the medical therapy
      - sometimes the MI patient dosn't has a pain – mostly from the diabetic patients through the autonomic neuropathy

Diagnosis

- current complaint:
  - pain
    -







อาการแน่นกลางหน้าอก หรือลิ้นปี่ ที่เป็นบริเวณกว้าง

๑ ใน ๓ จะมีเจ็บร้าวไปที่ คอ ขากรรไกรล่าง หรือ ด้านในของแขน

Diagnosis – physical examination

- presence of pallor – extracardiac cause – anemia!
- cyanosis
- shortness of breath, orthopnoe, periodic (Cheyne-Stokes) respiration
- distention of the neck veins

Diagnosis – physical examination

- Pulmonary congestion
- Blood pressure – cardiogenic shock?
- Murmur – complication?

Electrocardiography (ECG)

- ACC/AHA guidelines require ECG performed within 5 minutes of presentation to the ER with symptoms of chest pain

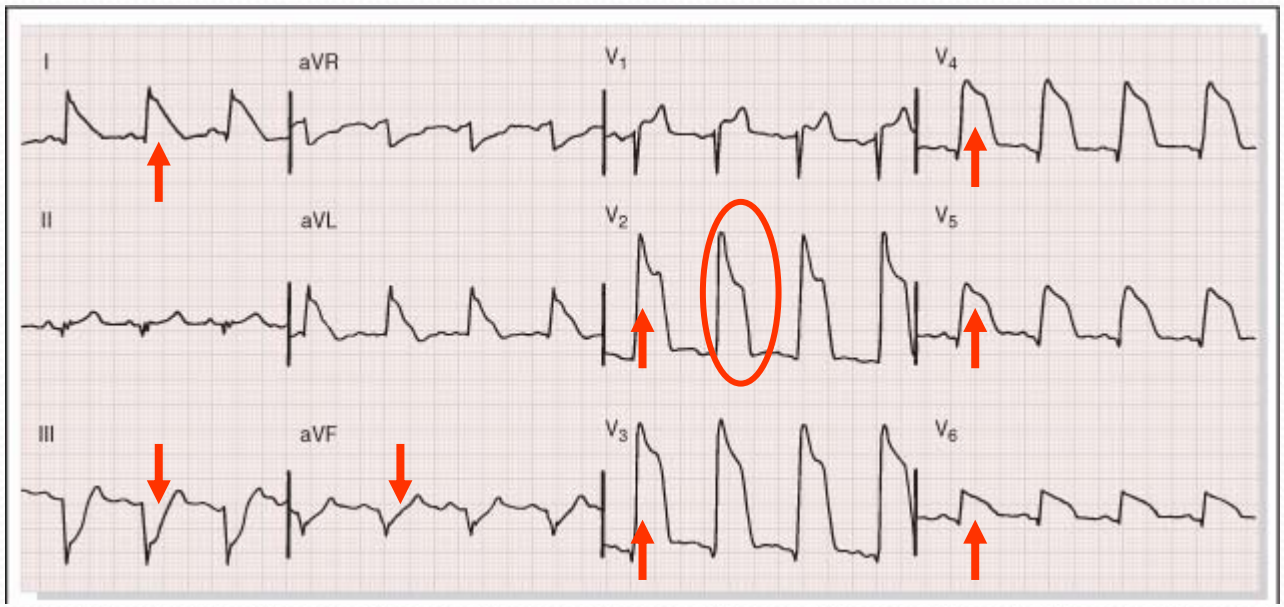
ECG with ST-segment elevation (STEMI)

- ST segment elevation (with compatible history) specificity=91%, sensitivity =46%
- The higher the elevation and the more the leads involved the large the infarction and the greater the mortality
- Watch at for other causes of ST- segment elevation , such as pericarditis, old MI (aneurysm) and normal varient (early repolarization)

ECG without ST-segment elevation(NSTEMI)

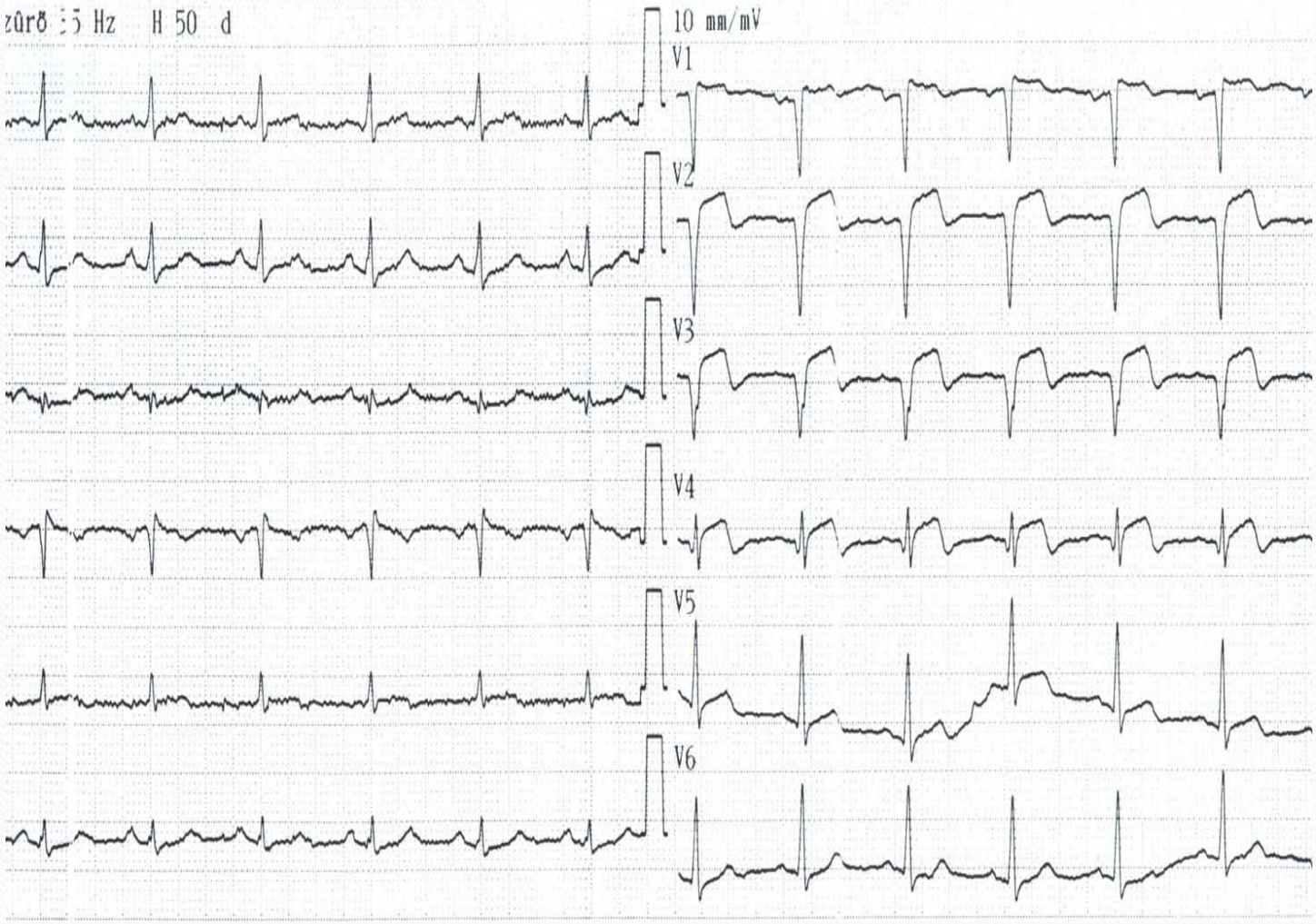
- Half of acute MI patients present without ST-segment elevation
- May see ST-segment depression , T wave inversion , non specifit ST-T wave changes or rarely entirely normal ECG
- Left bundle branch block

Diagnosis – ECG - STEMI

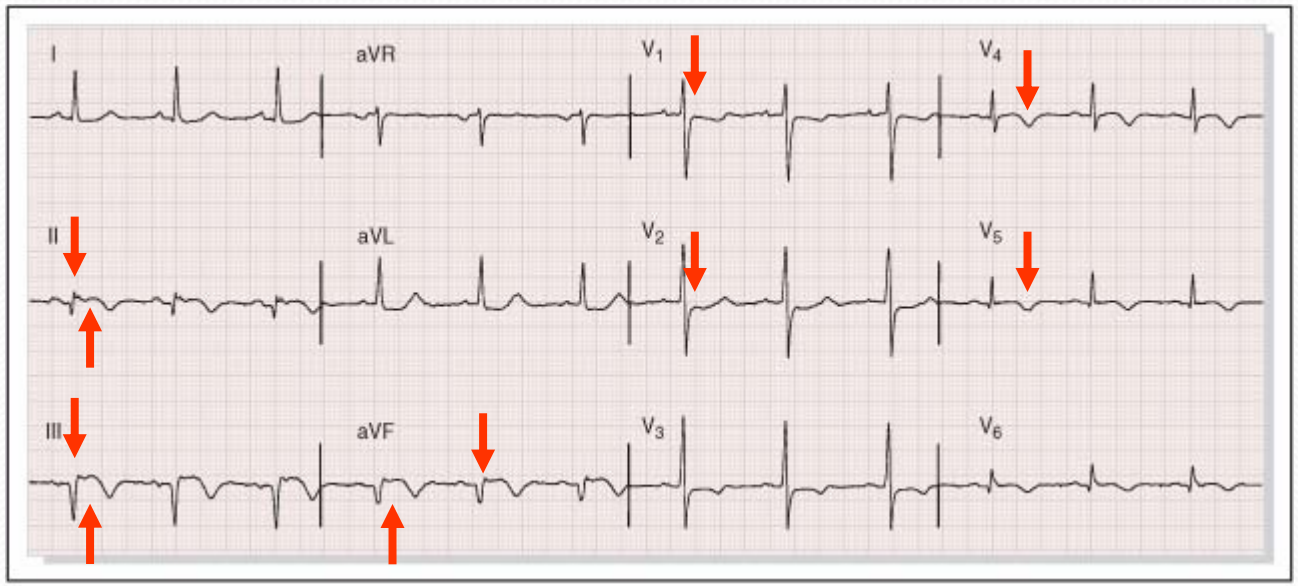


Hyperacute phase of extensive antero-lateral myocardial infarction.

Diagnosis – ECG - STEMI



Diagnosis – ECG - STEMI



Acute inferior
myocardial infarction.

In case of STEMI…

Very important the *quick* diagnosis!!!

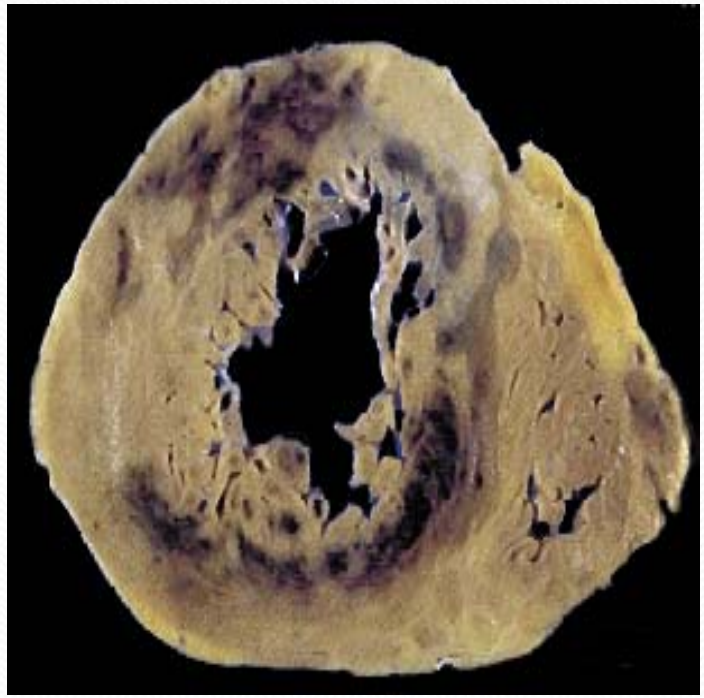
TIME = MYOCARDIUM = LIFE!

If the time window ≤ 12 h

open artery theory =

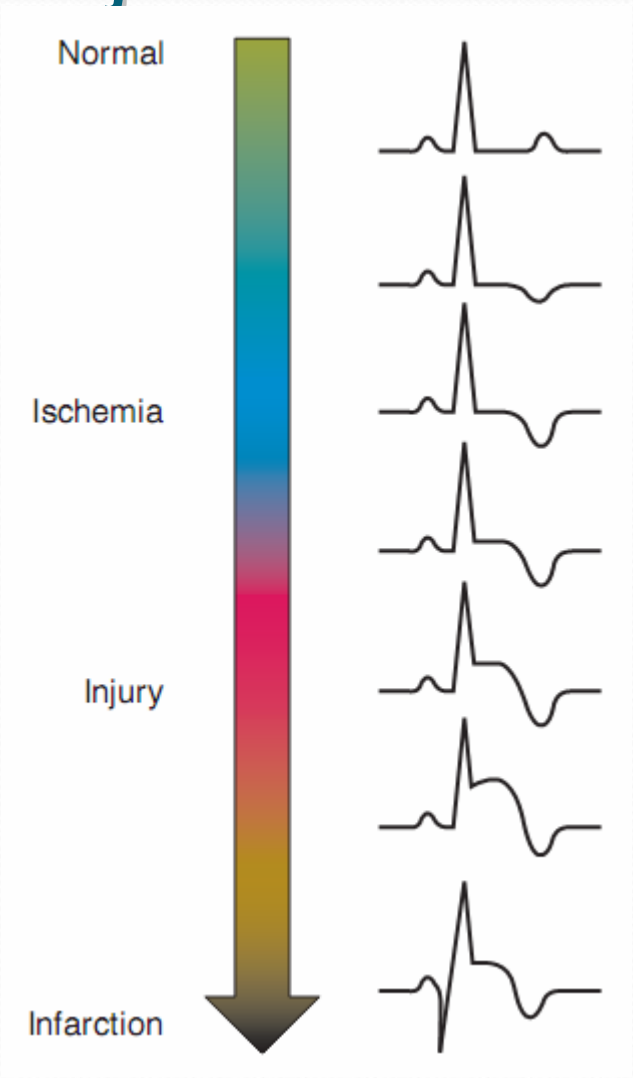
open the occluded coronary artery – save the myocardium!

If the patient has typical chest pain + typical ECG with acut STEMI – *it is enough to diagnosis!*

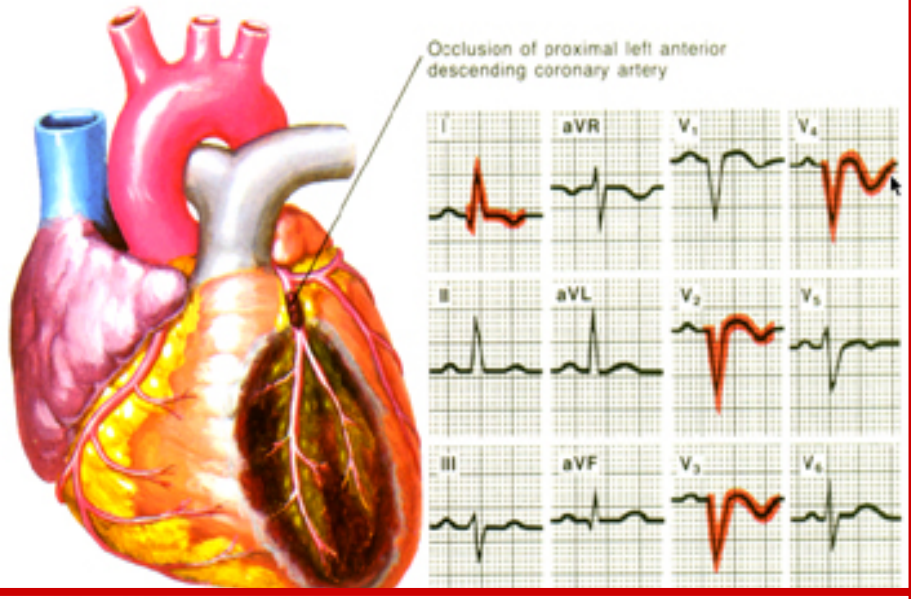


Progression of myocardial injuries

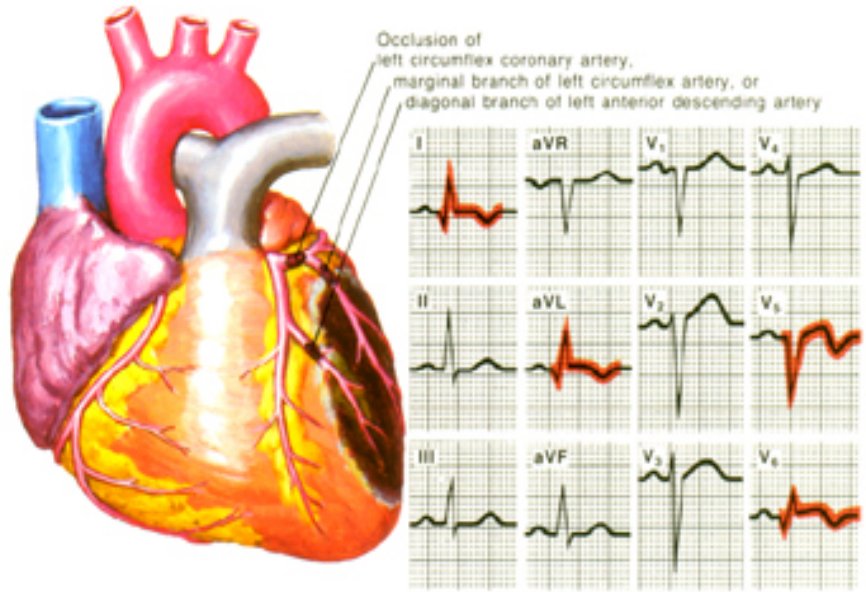
- Ischemia—Lack of oxygen to the cardiac tissue, represented by ST segment depression, T wave inversion, or both
- Injury—Arterial occlusion with ischemia, represented by ST segment elevation
- Infarction—Death of tissue, represented by a pathological Q wave



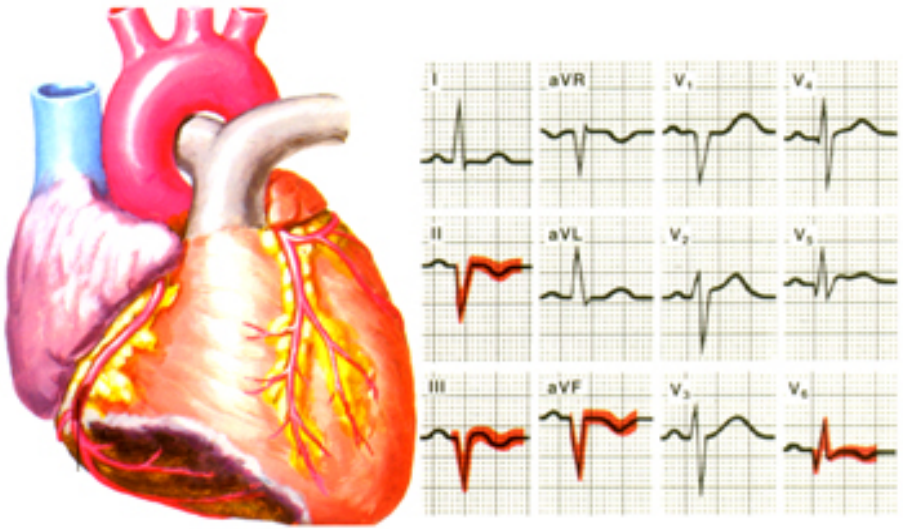
Anterior MI



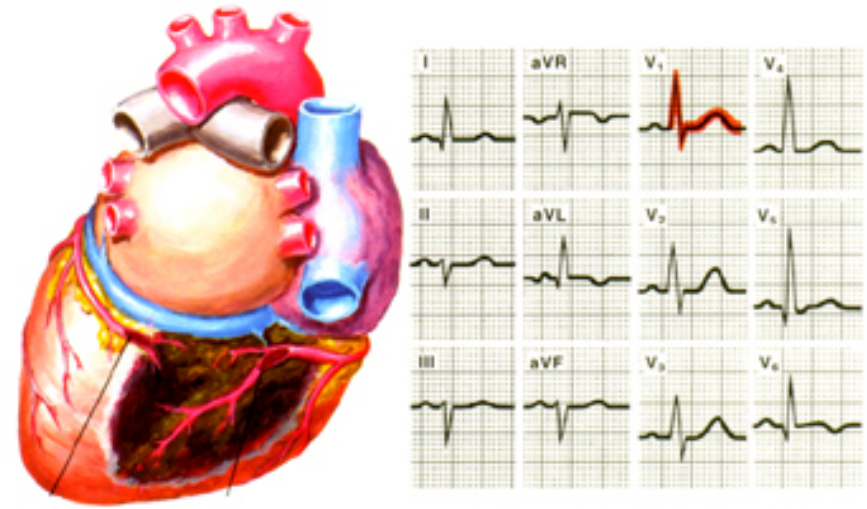
Lateral MI



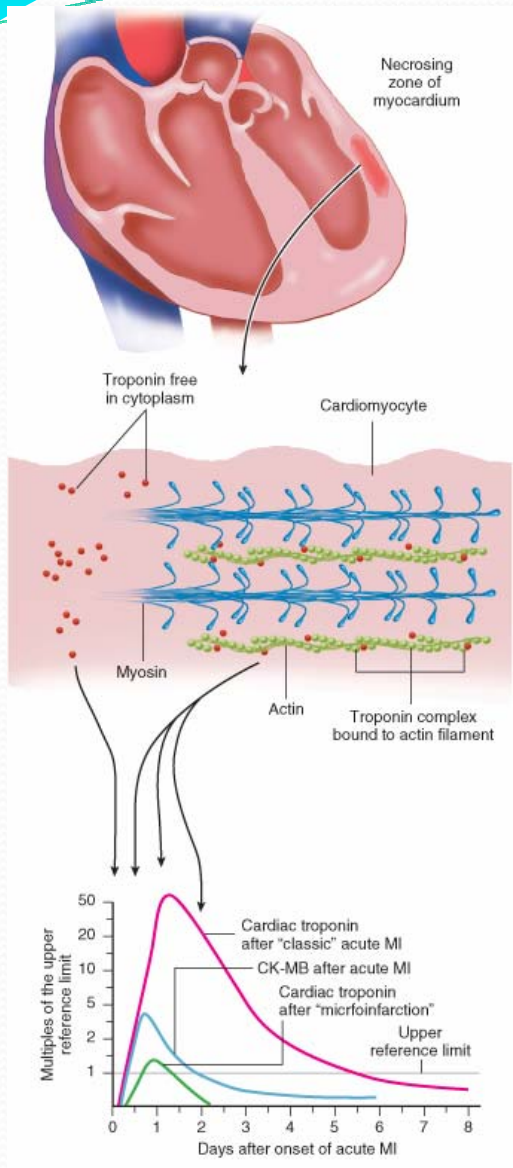
Inferior MI



Posterior MI



Laboratory Findings



myocardium injury

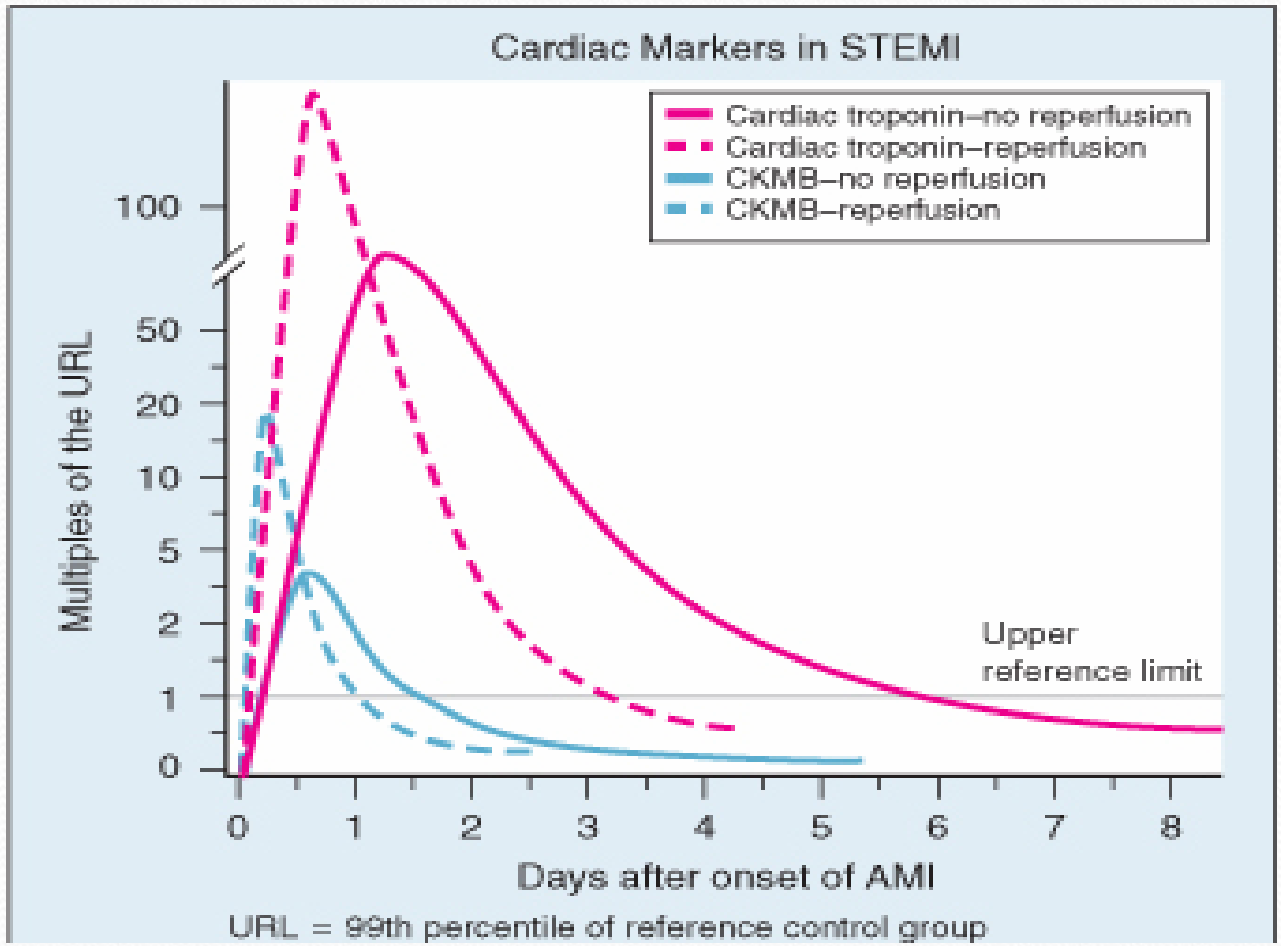
releasing biomarkers

Troponin T,I,C and
MB fraction of creaitine kinase (CK-MB)

If the patient has only the Troponin elevation: microinfarction

Laboratory Findings

rapidly and higher peak of cardiac marker after successful reperfuse therapy: **„WASH-OUT PHENOMENON”**



Different type of enzyme kinetics

Differential diagnosis

- From other life-threatening acute chest-disease:
  - pulmonary embolism
  - aortic dissection
- From other disease:
  - pleuritis
  - gastro-oesophageal disease
  - myositis
  - gastric or duodenal ulcer
  - gallbladder pain

Differential diagnosis

- From other life-threatening acute chest-disease:
  - pulmonary embolism
    - pain related to breathing, sharp
    - blood-gas report (hypoxaemia and hypocapnia)
    - ECG sign S_I-Q_{III}
    - echocardiography (transthoracal and transoeosophageal)
    - Ultrasound image from the lower limbs (search for deep vein thrombi)
    - CT – direct image of the pulmonary emboli
    - SCAN (ventilation and perfusion scintigraphy together

Differential diagnosis

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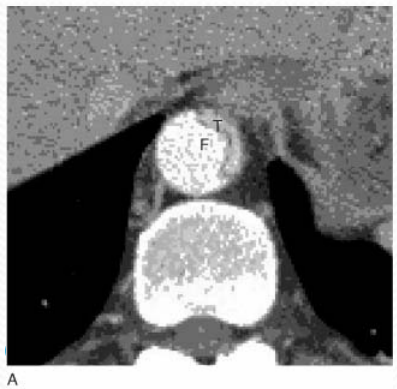
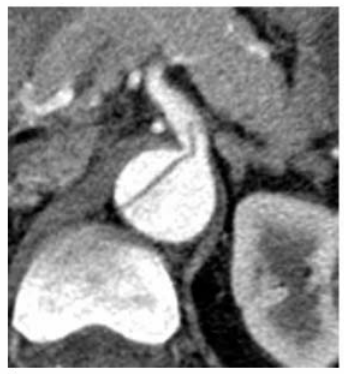
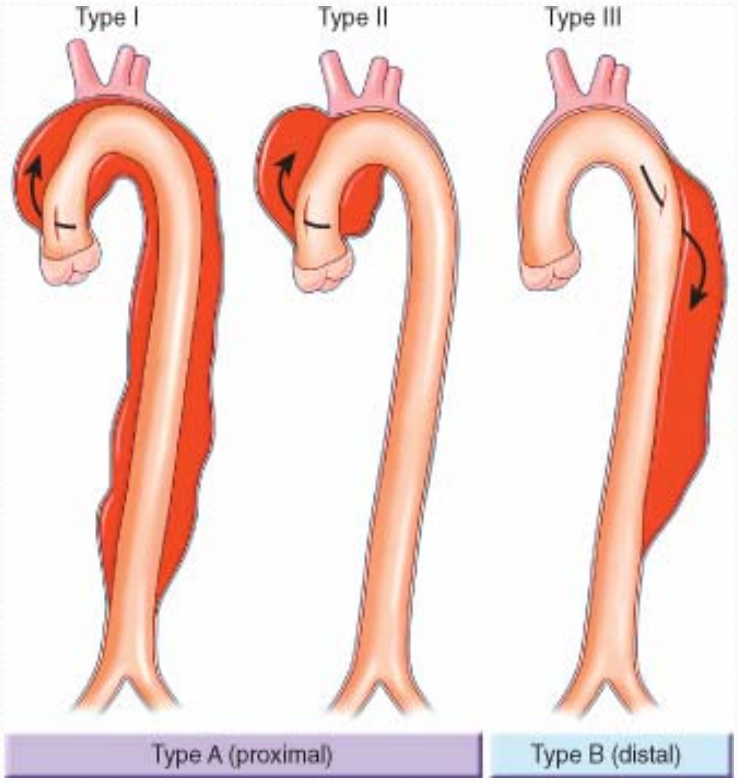
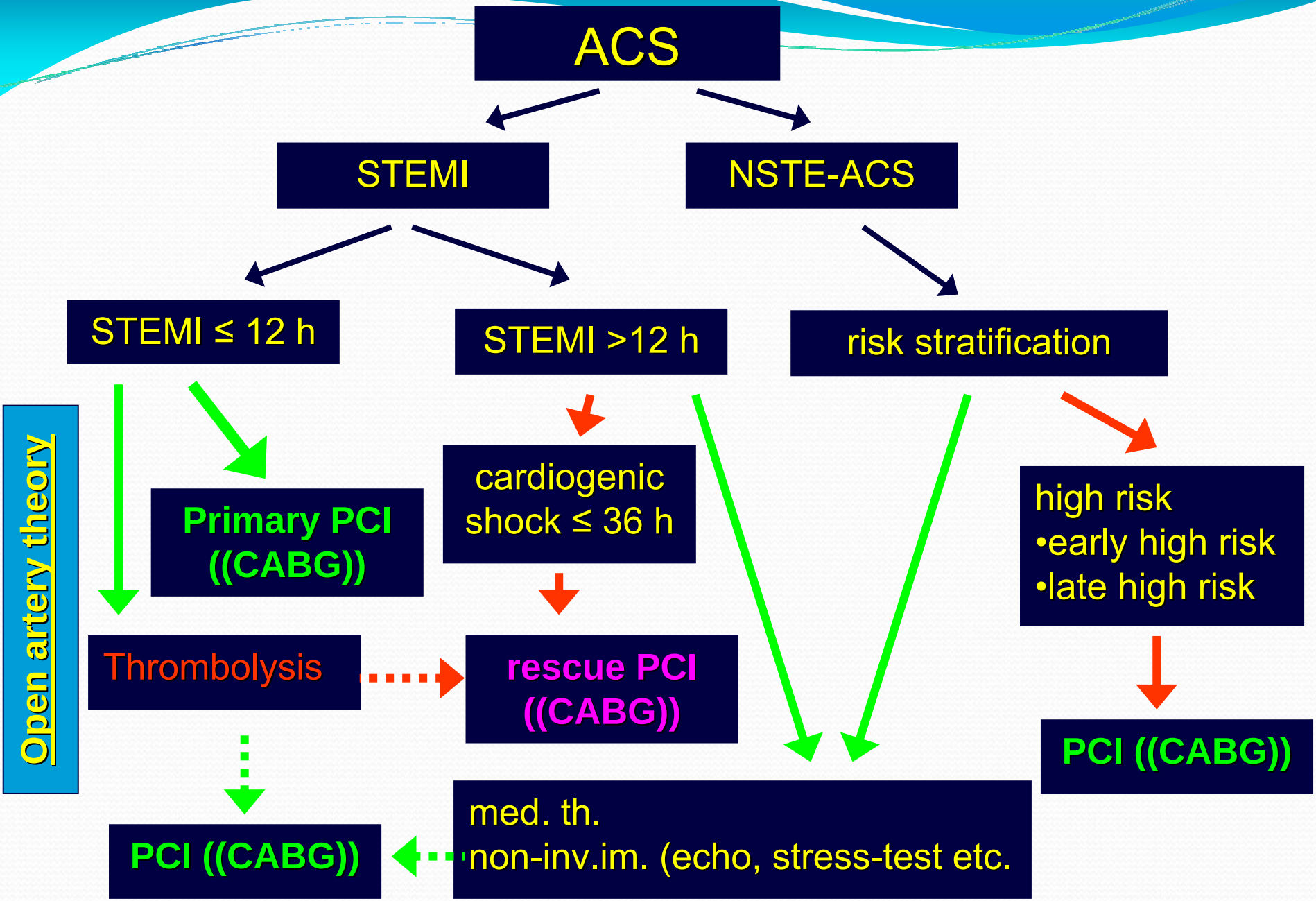


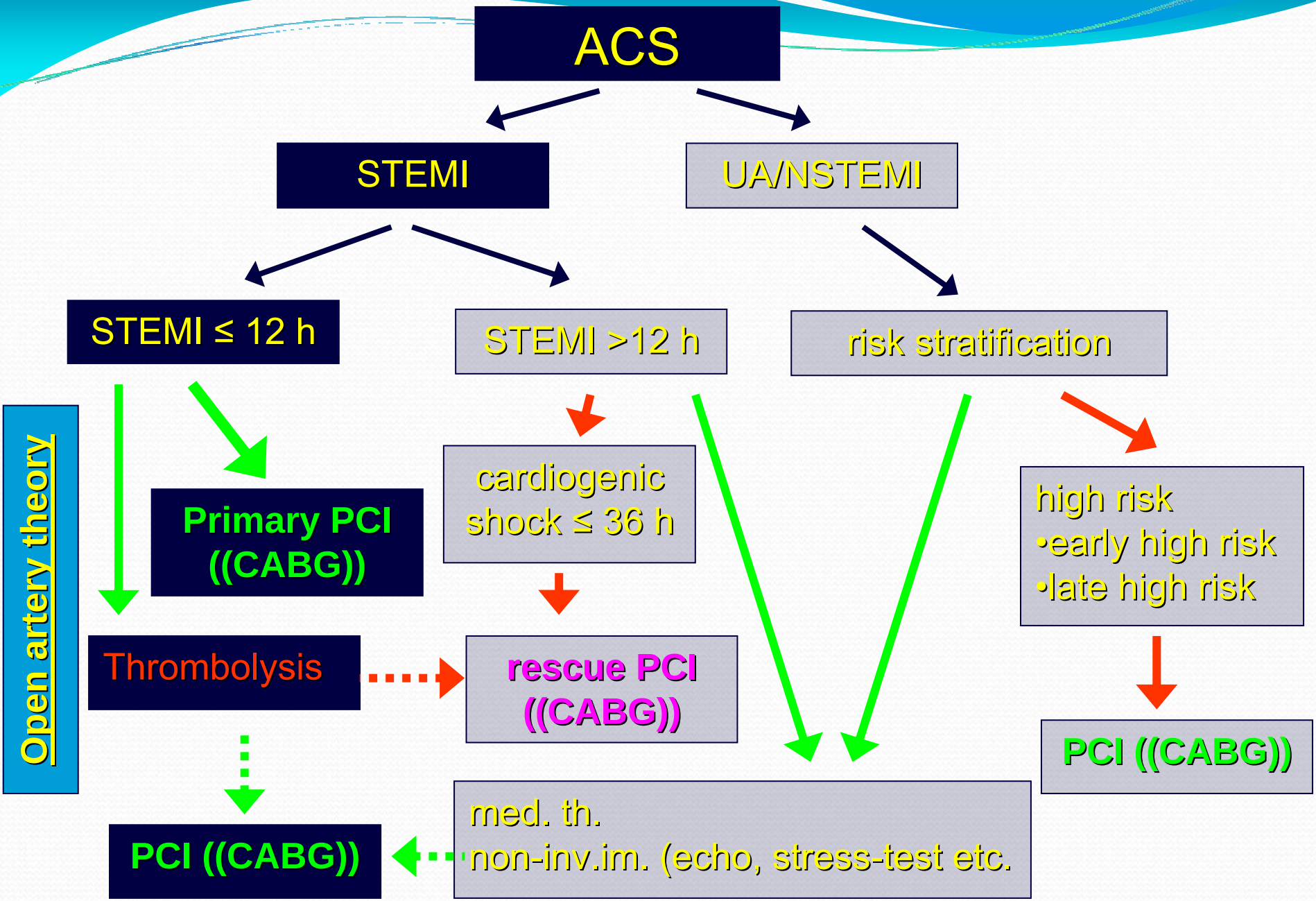





ACS
STEMI
NSTE-ACS
STEMI ≤ 12 h
STEMI >12 h
risk stratification
Open artery theory
Primary PCI ((CABG))
cardiogenic shock ≤ 36 h
high risk
•early high risk
•late high risk
Thrombolysis
rescue PCI ((CABG))
PCI ((CABG))
med. th.
non-inv.im. (echo, stress-test etc.
PCI ((CABG))

STEMI

- Quick diagnosis (Typical chest pain and ECG)
- Time window?
- Prehospital therapy
  - aspirin
  - morphine
  - nitroglycerin
    - again the pain, hypertensive state, left ventricular failure
    - Attention! Right ventricular infarction can cause sever hypotension!
  - O2
- Send the patient to the hospital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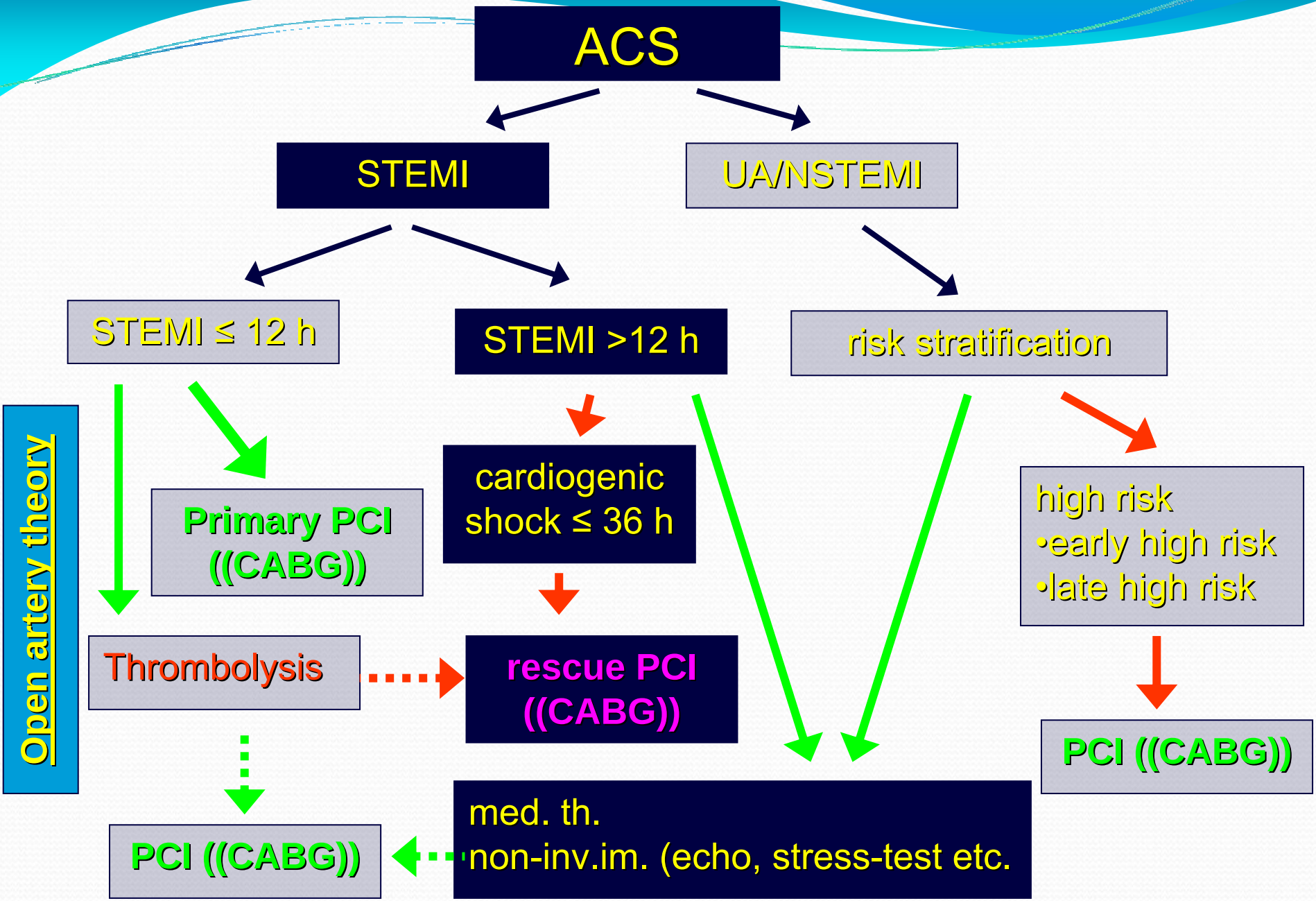
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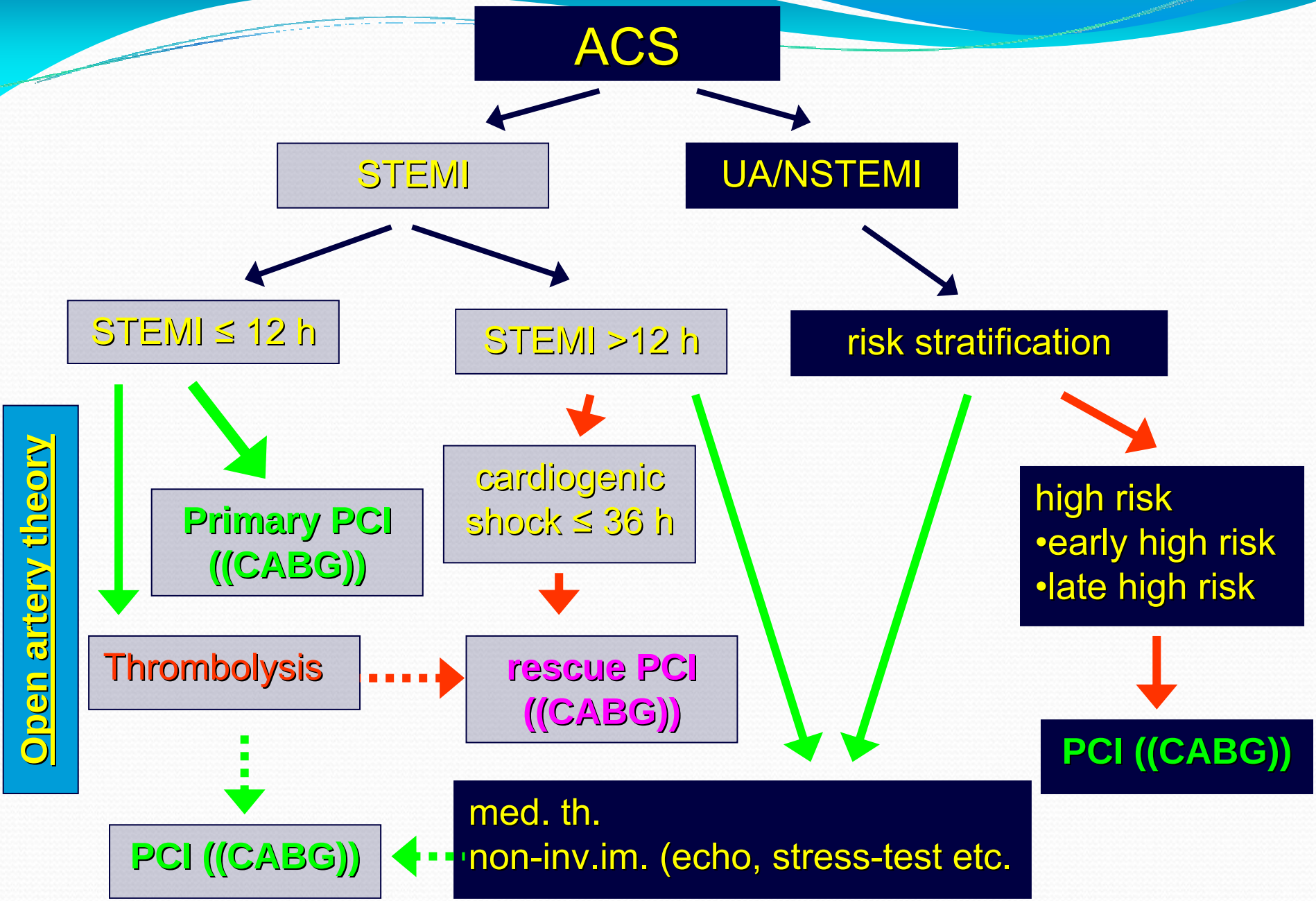
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Acute Management of MI
General Measures

- Oxygen: modest hypoxemia (common V/Q mismatch)
- Bed rest
- ECG monitoring: 48-72 hours for acute MI, 12-36 hours to rule out MI
- Analgesics: commonly underdosed (pain, catecholamines, myocardial oxygen demand)

Analgesic - Morphine Sulfate

- Good dose response, easily reversible; 2-5mg every 5-30 minutes
- Peripheral venous and arterial dilation; blocks sympathetic efferent discharge at CNS level; reduces preload and afterload - good with CHF
- Side effects: hypotension and bradycardia occur rarely; respiratory depression with severe COPD

Acute Management of MI: Pharmacotherapy - Aspirin

- Acute Aspirin - ASA 325mg chewed immediately on presentation

- ISIS-2 results (Lancet 2:349, 1988) based on 17,187 patients; reduced one month mortality 19%
- Additive effect to streptokinase - reduced one month mortality 23%
- Give immediately to anyone with suspected MI unless STRONG contraindication

Acute Management of MI: Pharmacotherapy - NTG

- Sublingual NTG given to all patients initially if systolic BP >90
- Avoid long acting nitrates initally
- Data from trials show acute MI pain due to ongoing ischemia rather than completed myocardial necrosis so NTG may be rational choice for ongoing ischemic pain
- Helpful in pulmonary edema

Acute Management of MI: NTG (continued)

- Dosage: 5-10 μg/min, increase 5-10 μg/min every 5 to 10 minutes
- Nitrate tolerance after > 24 hours
- Recommend routinely for most MI's for 24-48 hours (particularly with CHF), hypertension or recurrent ischemia, and regularly for unstable angina

Acute Management of MI: NTG Side Effects

- Headache - quite common, decreases with time
- Hypotension - particular care needed with right ventricular infarction
- Hypoxemia from V/Q mismatch - need to be alert for this phenomenon
- Bradycardia with hypotension - under appreciated

Acute Management of MI: Pharmacotherapy - Heparin

- Potential Uses
  - To aid in recannalization or reduce reocclusion of coronary artery
  - To reduce systemic embolism and stroke from left ventricle mural thrombus
  - To reduce deep venous thrombosis and pulmonary embolus

Acute Management of MI: Heparin (continued)

- Definite indication for IV heparin (for 48 hours)
  - Unstable angina
  - As adjunctive therapy for thrombolysis with tissue plasminogen activator (tPA)
  - As adjunctive therapy for primary angioplasty
  - Large anterior MI or known mural thrombus (to reduce stroke)

Acute Management of MI: Heparin (continued)

- Ideal target dose: aPTT = 50-75 sec; higher doses lead to intracranial hemorrhage
- Be aware of hypercoagulable state with abrupt termination of heparin
- Give to large majority of patients with acute coronary syndromes

Heparin-Induced Thrombocytopenia

- 3% incidence
- Most often occurs after day 4
- Check platelets daily
- Associated with prothrombotic events, particularly deep venous thrombosis

Acute Management of MI: Pharmacotherapy - Lidocaine

- Treatment of choice for sustained ventricular tachycardia (VT) and fibrillation (VF) and shock if necessary
- More benign ventricular arrhythmias (including nonsustained VT) generally not treated
- Prophylactic use no longer advised - meta-analysis of 14 randomized trials showed ↓VF by 33% but slight ↑mortality possibly due to asystole and electromechanical dissociation

Acute Management of MI: Lidocaine (continued)

- Dose: 1mg/kg (100mg max) followed by 0.5mg/kg every 10 minutes to 4mg/kg max
- Maintenance 20-50µg/kg/min IV
- $t_{1/2}$ = 1-2 hours in normal individuals, >20 hours with bad CHF secondary to ↓liver metabolism

Acute Management of MI: Lidocaine Side Effects

- Frequent
- CNS : dizziness, confusion, drowsiness, nausea, slurred speech, perioral numbness, tremor, respiratory depression, double vision
- Cardiovascular: bradycardia, hypotension, sinus arrest
- Consider IV Amiodarone and procainamide as alternatives

Acute Management of MI: Pharmacotherapy-Beta Blockers

- Beta-blockers sigfnicantly ↓ MI size by enzymes, ST segments, etc.
  - MIAMI trail (Eur H J, 6:199,1985) 5600 patients, MI smaller with metoprolol if treated within 7 hours, 15-day mortality reduced
  - TIMI II (NEJM 320:618,1989) B-blocker + thromolytics ↓ ischemia and reinfarction but not mortality

Acute Management of MI: Beta-blockers (continued)

- Mortality evident by day 1 and sustained
- Quickly reversed by isoproterenol
- Surprisingly safe
- Good candidate patients - early presentation, ↑HR, ↑BP, anterior MI
- Contraindications - HR<60, BP<100, moderate/severe CHF, AV block, bad COPD
- Typical dose metoprolol 5mg IV every 5 minutes x 3

Acute Management of MI: Pharmacotherapy - Ace Inhibitor

- Definite indication - within 24 hours of moderate or large anterior MI's or MI's associated with CHF of LVEF <40%
- Controversial indication - all MI's within 24 hours, stopped in 4-6 weeks if no CHF or significant left ventricular dysfunction (LVEF<40%) evident

All Early ACE Inhibitor Trails Have Shown Mortality Benefit

- SAVE Study - 2231 patients 3-13 days post-MI, half received 50mg captopril TID: ↓4 year mortality 19%, ↓severe CHF 35%, ↓recurrent MI 25% (NEJM 327:669,1992)
- GISSI-3 - lisinopril in >19,000 patients ↓mortality at 6 weeks 12% (Lancet 343:1115,1994)

All ACE Inhibitor Trails Show Mortality Benefit (continued)

- ISIS-4 - 58,000 patients showed 7% ↓ 5 week mortality with captopril (Lancet 345:8951,1995)
- Meta-analysis: 4.6 fewer deaths per 1000 patients treated
- Contraindication: SBP<100, significant renal failure
- Give ACE inhibitors in the first few hours to all MI's or at least large MI's or MI's associated with CHF or ↓ejection fraction

Acute Management of MI: Pharmacotherapy-Calcium Channel Antagonists

- Generally best avoided unless patient experiences continued ischemia unresponsive to nitrates or beta-blocker

Acute Management of MI: Pharmacotherapy - Magnesium

- Meta-analysis - showed 50% ↓mortality (BMJ 303:1499,1991)
- LIMIT-2 trial - 24% ↓mortality with 8mmol $MgSO_4$ for 5 min then 3 mmol/hour (Lancet 339:8809,1992)
- ISIS-4 - no difference in mortality with Mg^{++} but given late (Lancet 345:8951,1995)
- Mg^{++} best used in high risk (elderly) and non-thrombotic candidates

Acute Management of MI: Referfusion by Thrombolysis

- Rationale:
  - ST-segment elevation MI nearly always due to acute coronary thrombosis
  - All thrombolytic agents work by converting plasminogen to plasmin
- Clearly saves lives:
  - Meta-analysis - 35 day mortality ↓ by 21%
  - 18 lives per 10000 treated

Acute Management of MI: Reperfusion by Thombolysis (continued)

- GISSI - 11,700 patients using streptokinase ↓mortality 18% with difference persisting at one year (Lancet 2:871,1987)
- ISIS-2 - 17,200 patients using streptokinase (+ASA) ↓one year mortality 23% with significant improvement noted even when treatment started 12-24 hours after the onset of symptoms

Acute Management of MI: Reperfusion by Thrombolysis (continued)

- Underused - Use in good candidates 50-70%; in patients >65 years = 20%
- Indication:
  - ST elevation
  - LBBB
  - MI <12 hours since onset

Acute Management of MI: Reperfusion by Thrombolysis (continued)

- Controversial potential contraindications:
  - Patients > 75 years old
  - Late presentations (12-24 hours)
  - Hypertension (>180/100 mmHg)
- Clear contraindications:
  - CVA/TIA within one year
  - Hemmorrhagic CVA at any time
  - Intracranial neoplasm
  - Active internal bleeding (not including menses)
  - Suspected aortic dissection

Acute Management of MI: Reperfusion by Thrombolysis (continued)

- Time to delivery is critical:
  - <1 hour - 35 lives saved per 1000; 7-12 hours - 16 lives saved per 1000
  - Community education programs
  - Educate your own patients with coronary artery disease
  - Hospital goal - “door to needle” time of <30 minutes
  - Thrombolytic “code” team

Acute Management of MI: Choice of Thrombolytic Agent

- tPA
  - Less allergic reactions
  - Less fibrinogen depletion (“clot selective”)
  - Faster thrombolysis
  - Slightly lower overall mortality
- Streptokinase (SK)
  - Less expensive
  - Lower stroke rate (0.3% vs 0.8%)
  - Can’t use again secondary to antibody formation

Acute Management of MI: Choice of Thrombolytic Agent (continued)

- 90 minute patency better with tPA than SK (70% vs 55% in Euro Coop Study and 70% vs 43% in TIMI-1)
- Patency at 24 hours roughly equal between tPA and SK
- ISIS-3 - mortality identical in head to head comparison of tPA and SK

Thrombolytics: Bottom Line

- Generally choose tPA for large MI's presenting early or in patients who have previously received streptokinase, otherwise choose streptokinase because of cost.

Acute Management of MI: Reperfusion by Primary PTCA

- Theoretic advantages - higher early vessel patency (90% vs 50-75%) and less strokes
- Only 10% US hospitals capable of emergent PTCA
- “Door-to-balloon-inflation” time should be <90 minutes

Reperfusion by Primary PTCA: Comparative Data

- Meta-analysis of 7 trials: 6-week mortality and reinfarction reduced
- PTCA + thrombolytics vs thrombolytics alone much less favorable
- PAMI trial: 395 patients randomized to tPA vs primary angioplasty (12 hours)
  - 97% success rate of PTCA
  - In-hospital mortality PTCA 2.6% and tPA 6.5%
  - Stroke PTCA 0% and tPA 2%
  - Results persisted 6 months

Reperfusion by Primary PTCA: Indications

- Reperfusion candidates (ST-segment elevation <12 hours, etc.) with contraindications to thrombolysis (such as recent CVA)
- Reperfusion candidates as an alternative to thrombolysis in an experienced high volume center
- Suitable candidates in cardiogenic shock

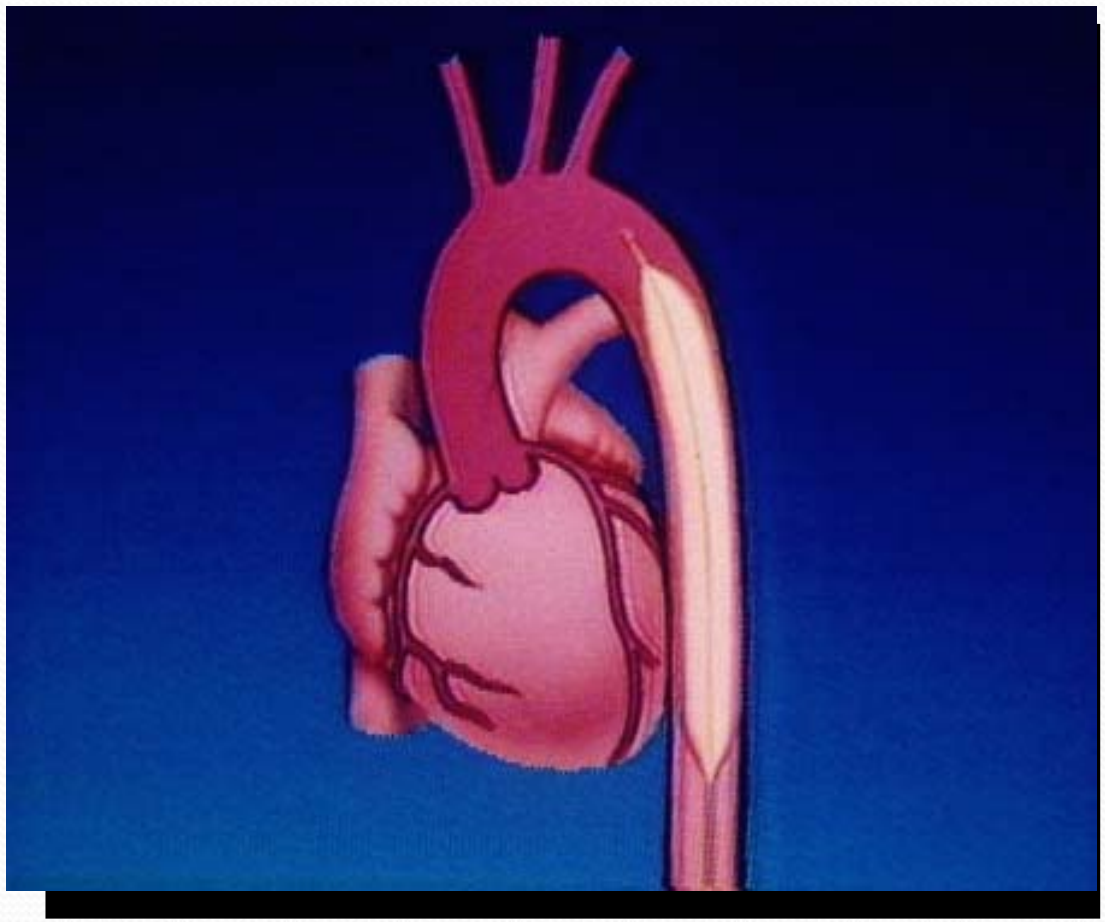
Reperfusion by Primary PTCA Conclusion

- If quickly available in a good quality center, PTCA is a reasonable alternative to thrombolysis, especially in high-risk patients presenting early, or in patients likely to bleed with thrombolytics

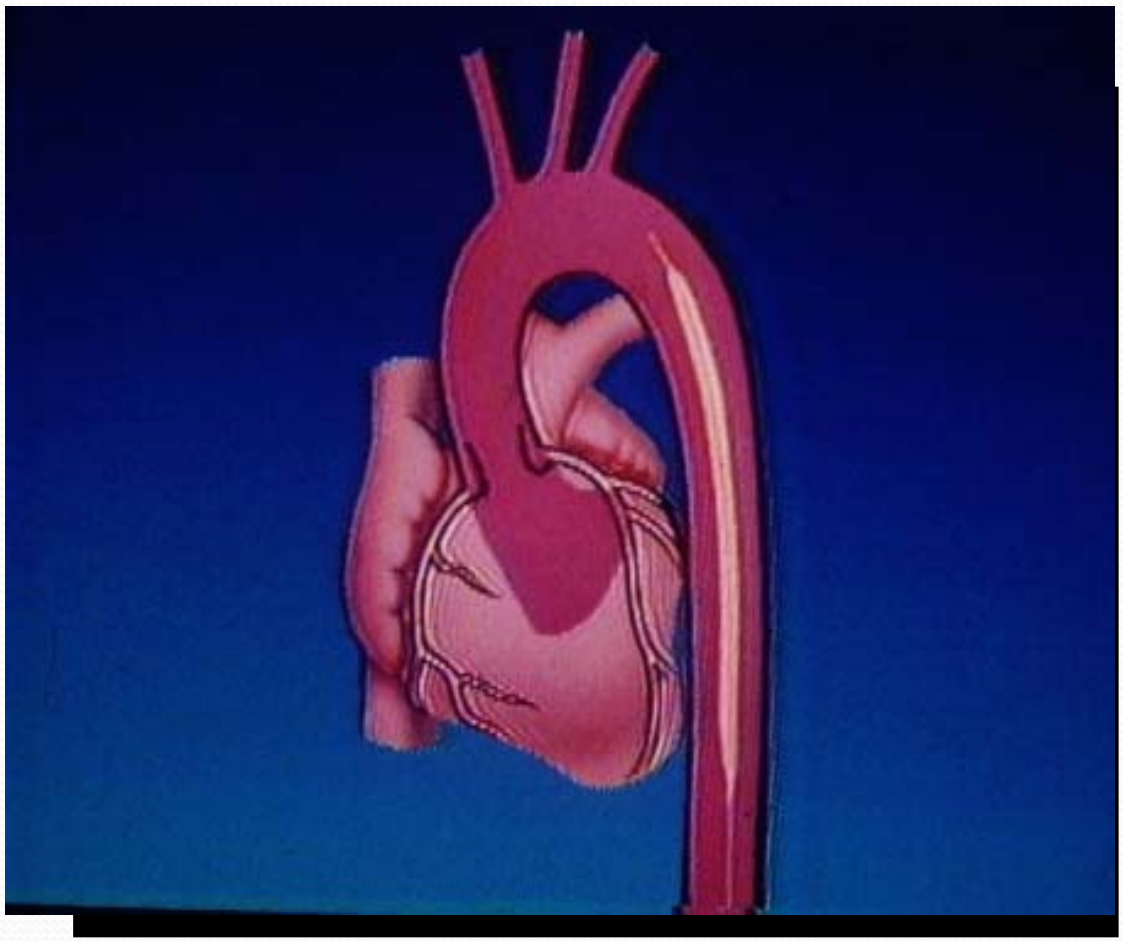
Acute Management of MI: Intra-Arterial Pressure Monitoring

- Indications:
  - Severe hypotension (<90mmHg) or cardiogenic shock
  - Vasopressor agents
  - Potent vasodilators
- Do not leave in for more than 72 hours
  - Thrombosis, infection

IAB Inflation



IAB Deflation



Acute Management of MI: Right Heart Catheter (Swan-Ganz Catheter)

- Indications:
  - Severe or progressive CHF/pulmonary edema
  - Progressive hypotension or cardiogenic shock
  - Suspected mechanical complication of MI (VSD,papillary muscle rupture, pericardial tamponade)
  - Hypotension without pulmonary congestion unresponsive to fluid challenge (uncertain fluid status)

Acute Management of MI: Intra-Aortic Balloon Counterpulsation ("Balloon Pump")

- Improves coronary flow and ↓myocardial O_2 demand
- Indications:
  - Unresponsive cardiogenic shock (as a "bridge" to revascularization)
  - Refractory post-MI angina (as a "bridge" to revascularization)
  - Acute MR or VSD
  - Almost always used to stabilize the patient until more definitive treatment is performed (PCI or CABG)

Long-Term Management After MI

- Aspirin
  - 13% ↓mortality, 31% ↓nonfatal MI
  - Give to nearly everyone lifelong
- Beta-blocker
  - Metoprolol, timolol, propranolol all shown to reduce mortality 1 to 6 years in more than 35,000 patients
  - ↓Mortality 30%
  - Give to nearly everyone indefinitely

Long-Term Management After MI

- Ace Inhibitor
  - Best if started early (25% ↓ mortality)
  - Probably should be stopped in 4-6 weeks for patients with preserved LV function and no CHF symptoms
  - Continue indefinitely if LV dysfunction/CHF is present

Long-Term Management After MI

- Lipid Lowering Agents
  - Prognosis improved even in post-MI with “normal” cholesterol level
  - CARE trial - mean cholesterol 209, LDL 139 at entry showed 24% ↓mortality/nonfatal MI at 5 years with pravastatin
  - Aggressive approach to lipid control (goal LDL<100) mandatory for all patients with CAD

Long-Term Management After MI

- Warfarin (coumadin)
  - Definitely indicated for: post-MI patients with large anterior MI's with/without thrombus or patients with atrial fibrillation (to prevent systemic embolism from LV thrombus)
  - Use for 3 months for LV thrombus or large anterior MI
  - Use indefinitely for atrial fibrillation

Long-Term Management After MI

- Lifestyle modification / Therapeutic Lifestyle Changes (TLC)
  - Diet
  - Exercise
  - Smoking

Long-Term Management After MI

- Exercise testing and stress testing
- Three goals post-MI
  - Assess functional capacity
  - Evaluate efficacy of patient's current medical regimen
  - Risk stratification
- For post-MI patients lacking spontaneous angina who are potential revascularization candidates, an exercise/stress test can be used to select appropriate candidates for coronary angiography

Complication of myocardial infarction

- Arrhythmias
  - Life-threatening:
    - Ventricular tachycardia / ventricular fibrillation – sudden death (I. symptom?)
    - II-III degree AV block – asystolia

- Ventricular failure (LV mass loss >40%)
  - pulmonary congestion
  - cardiogenic shock
  - right ventricular failure – impared filling pressure (CAVE: NITRO!)

- Mechanical complication
  - mitral papillar rupture – acute mitral regurgitation
  - ventricular septal rupture
  - free wall rupture – pericardial Tamponade

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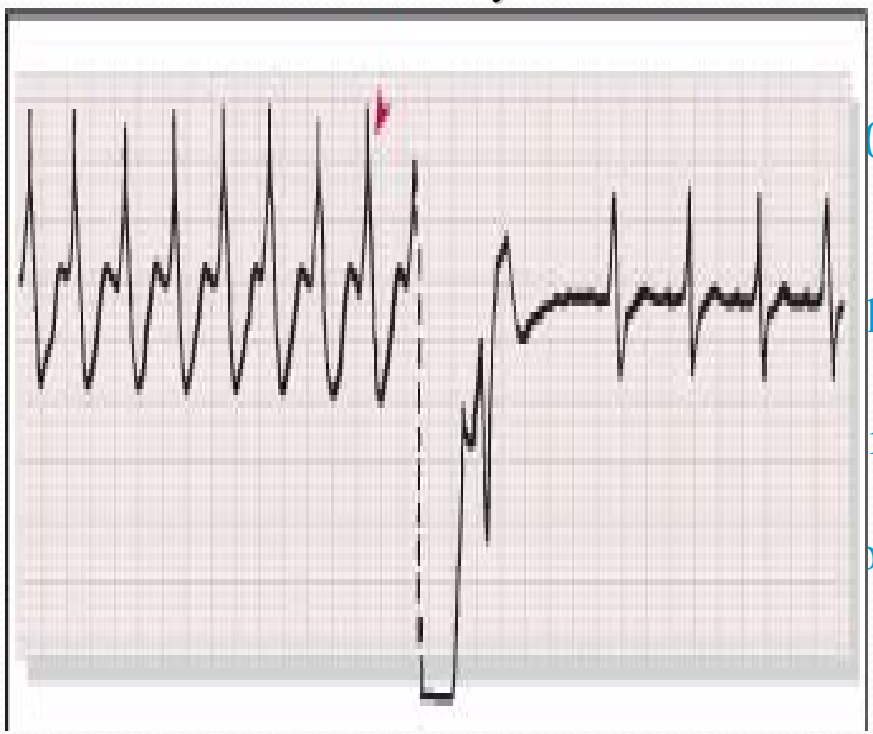

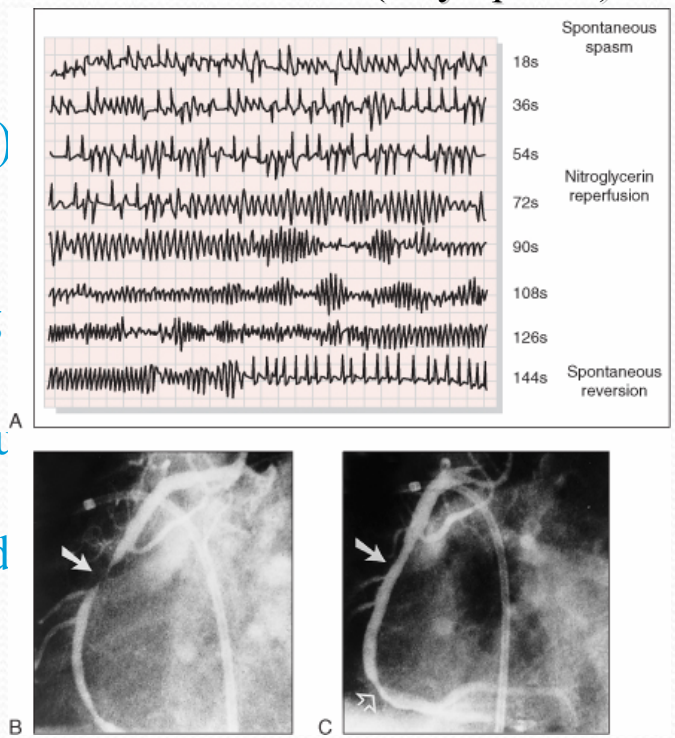

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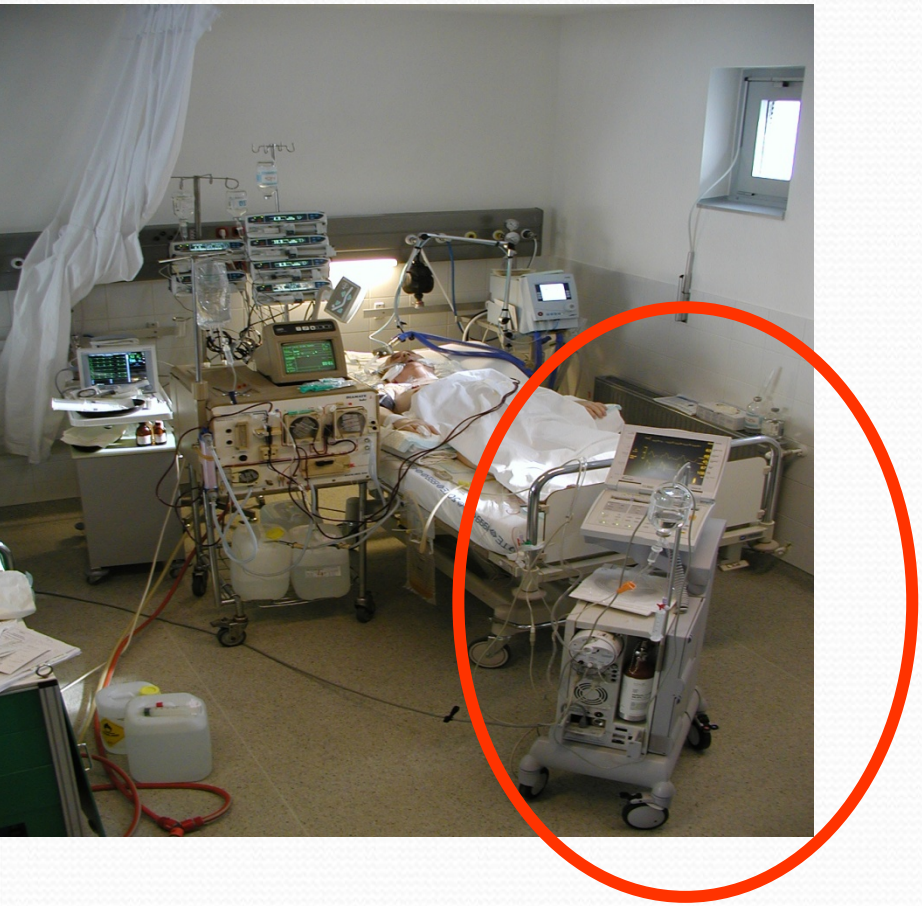
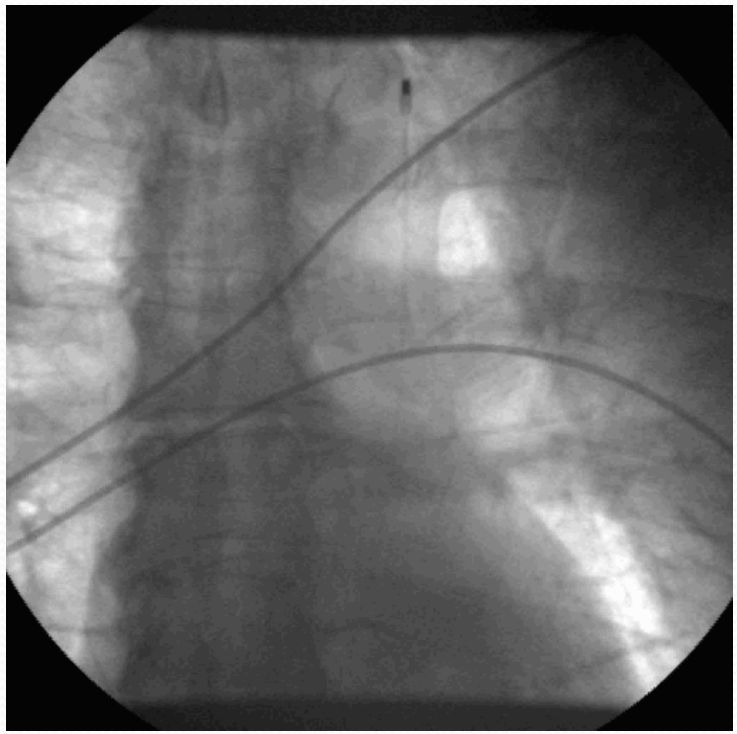
In the CCU a lot of technical devices (IABP,respirator, dialysator) are necessary





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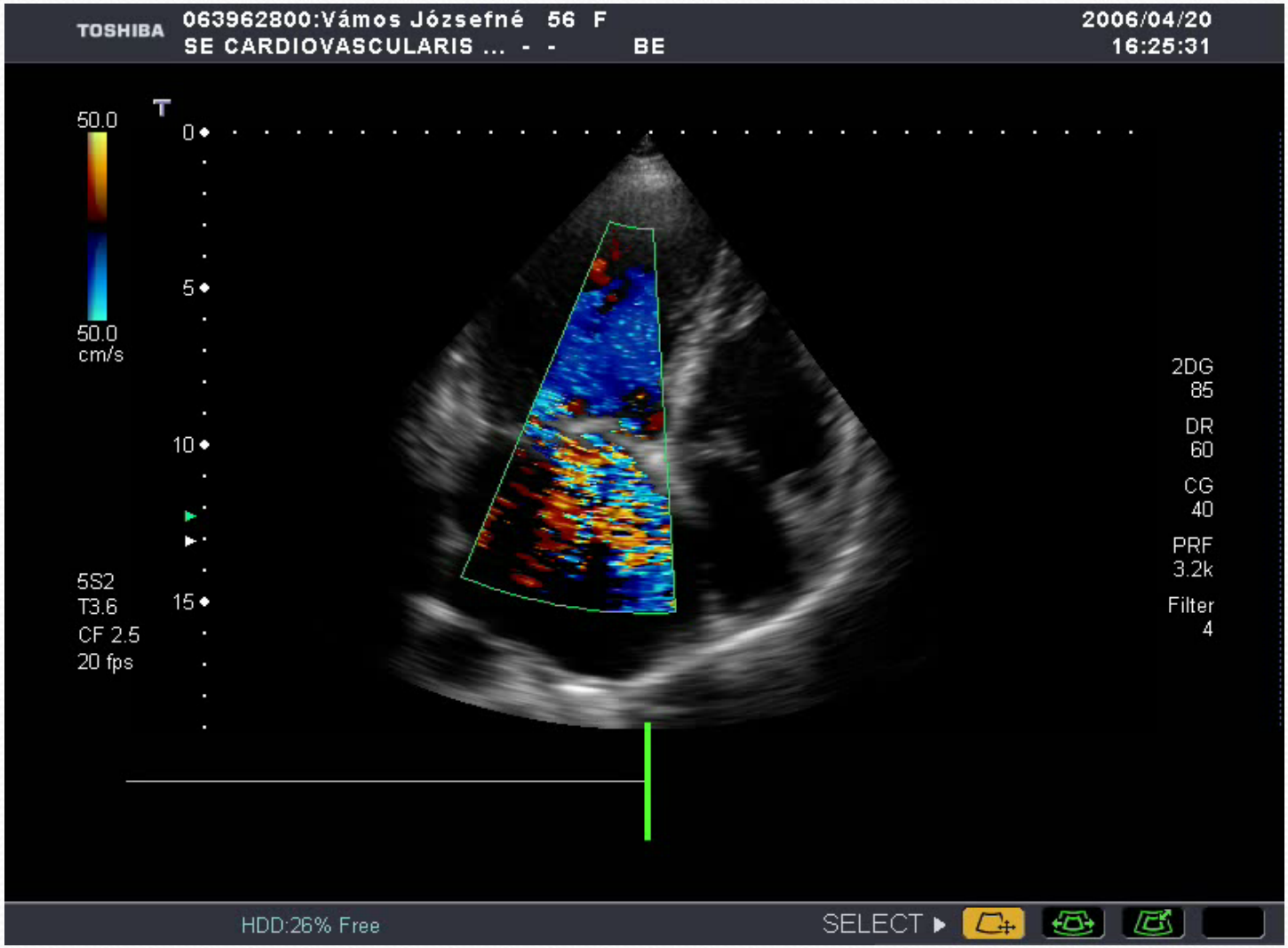
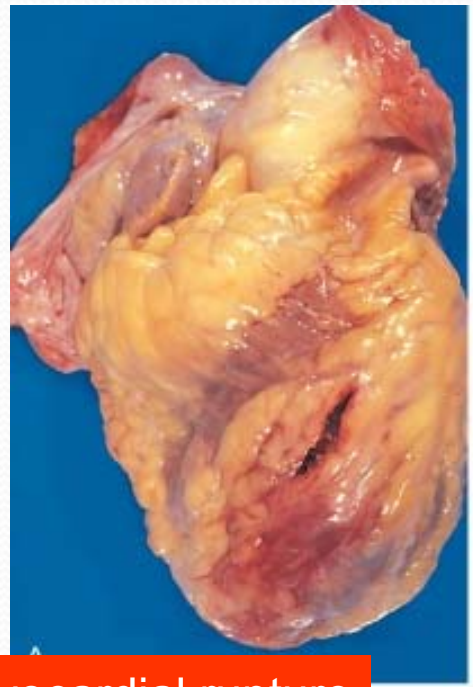
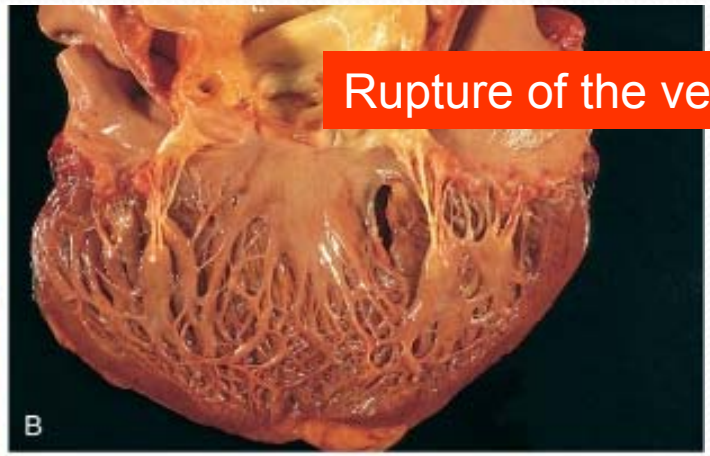
Acute mitral flail, chordal rupture



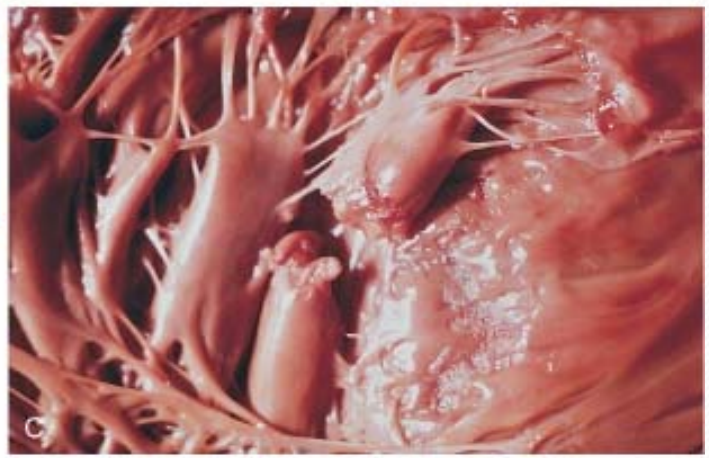
Cardiac rupture syndromes complicating STEMI



Anterior myocardial rupture

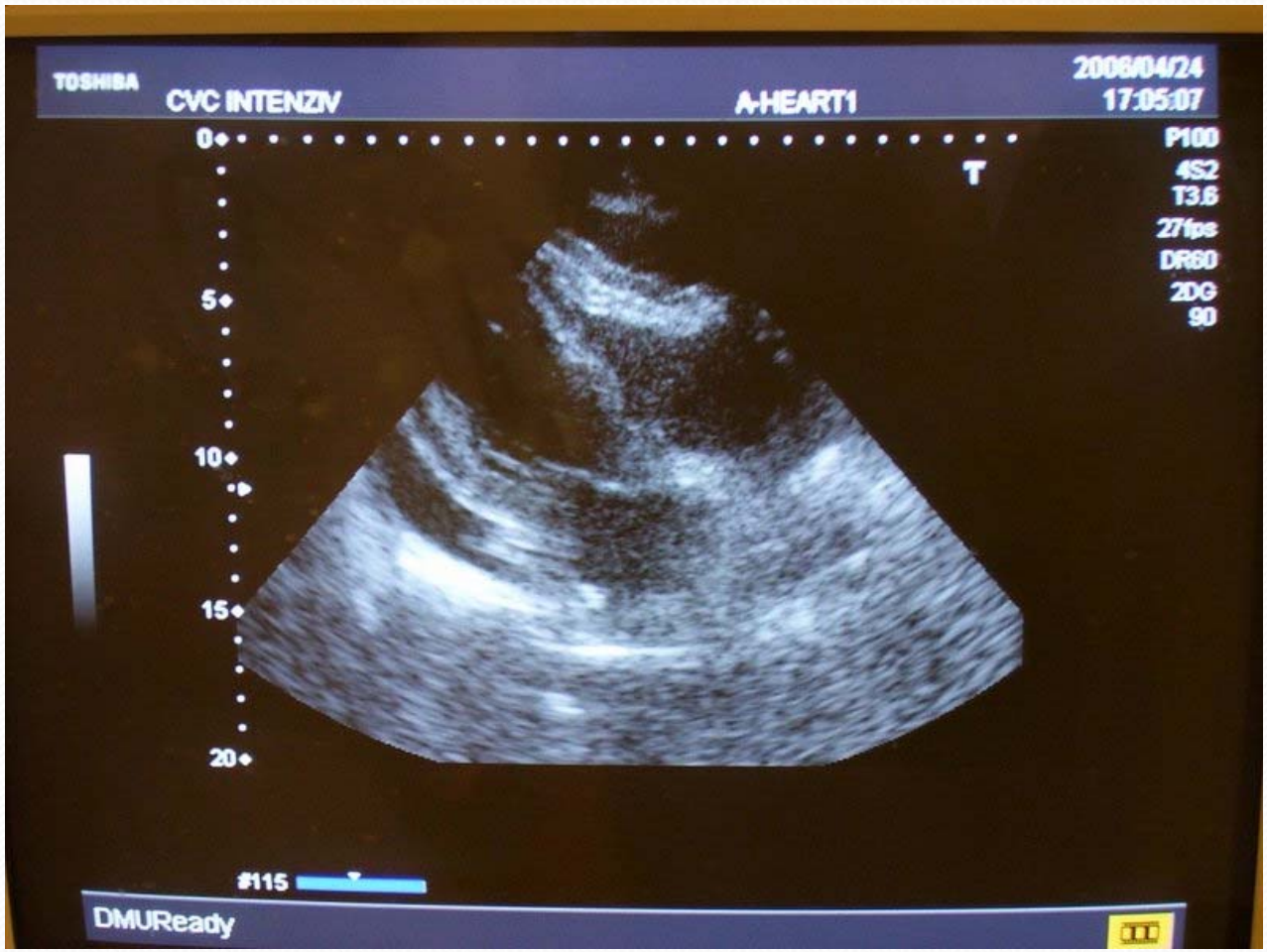


Rupture of the ventricular septum

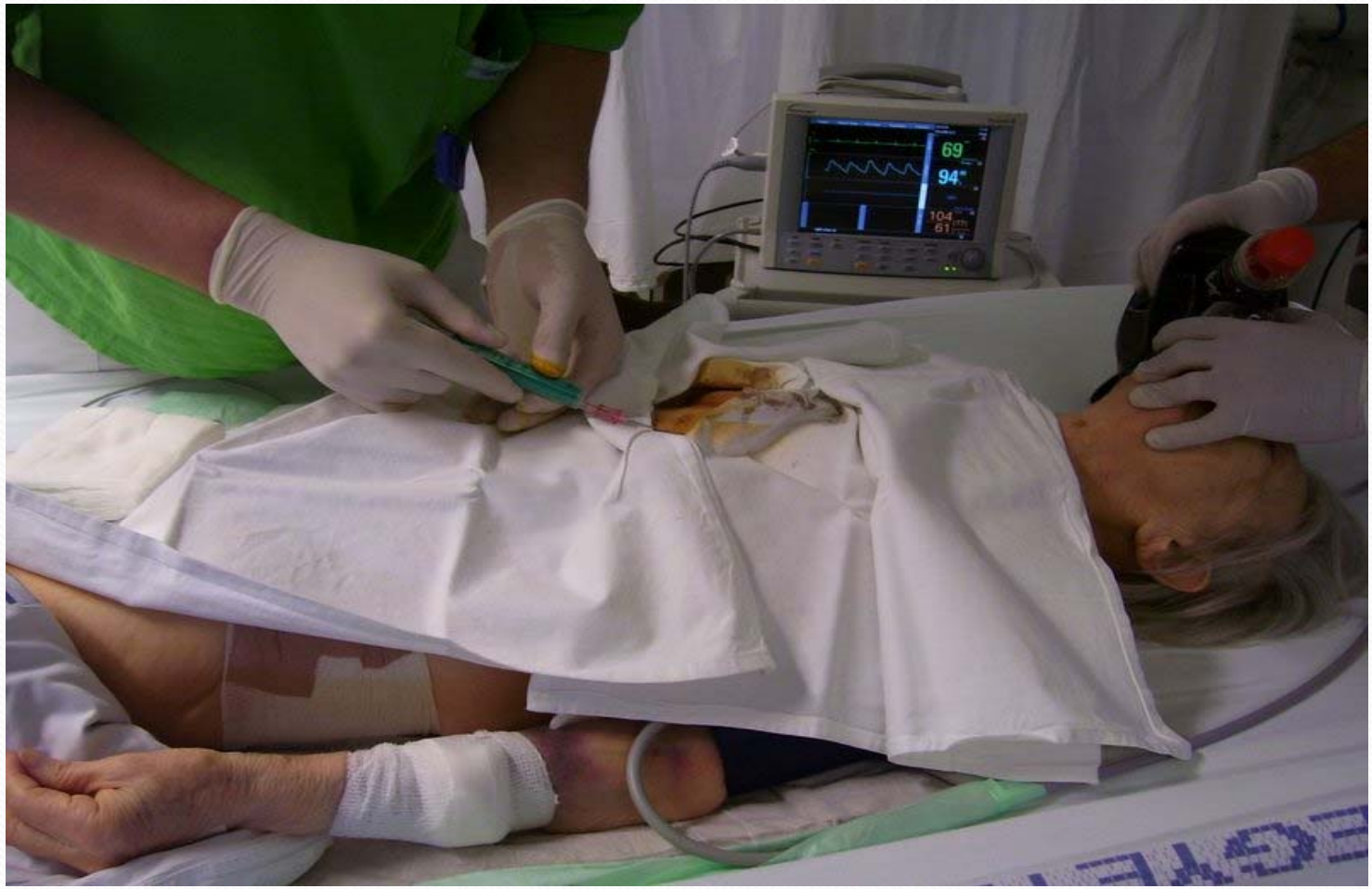


Complete rupture of a necrotic papillary muscle

Pericardial Tamponade



Pericardial Tamponade



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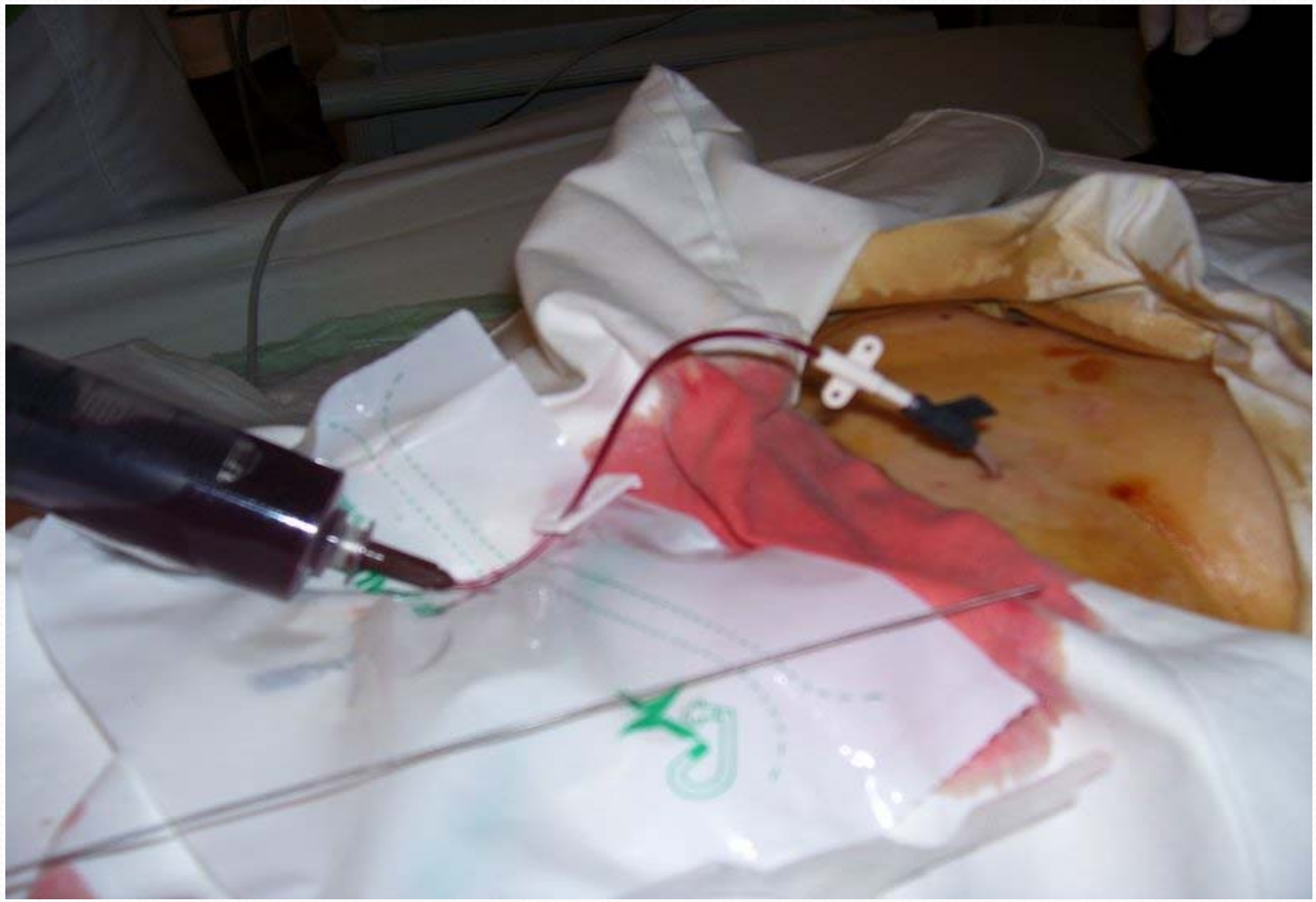

Conclusion

- The acute coronary syncrome is an acute, life-threating coronary event
- Need an urgent hospitalisation
- Short anamnesis (mostly the pain!!), physical examination
- rapidly perfom an ECG

  according to the ECG: NSTE-ACS or STEMI
- In case of NSTE-ACS: risk stratification
- In case of STEMI:
  - If the patient has typical chest pain + typical ECG with acut STEMI – *it is enough to diagnose!*
  - If the time-window is <12 hours: reperfusion therapy (primary PCI or if pPCI is not feasible thrombolytic therapy)