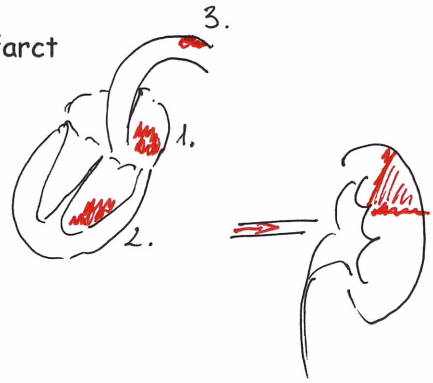
CLINICAL MANIFESTATION OF NECROSIS

	Coagulative	Liquefactive	Caseous	Fat necrosis
Anaemic	Renal infarct Splenic infarct Gangaena sicca AMI	Anaemic brain infarct Gangraena humida Decubitus	Tuberculosis	Pancreatitis
Haemorrhagic	Pulmonary infarct Intestine infarct	Haemorragic brain inf.		

1. **Renal infarct** Coagulative anaemic infarct

Pathogenesis: - Obstruction of renal arteries
- Most of them related to embolism (artial-, ventricular thrombus)
- Minority related to AS, vasculitis

Morphology: - Anaemic (white/yelow), sharply demarcated infarct
- Wedge shaped – apex pointing toward medulla
- Demarcated by a hyperaemic zone
- Healing with progressive fibrosis/scaring

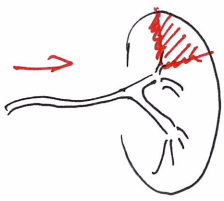


Clinical: - Most of them silent
- Some generate pain
- May generate hypertension

2. **Splenic infarct** - Coagulative anaemic infarct

Pathogenesis: - Occlusion of major splenic artery or branches
- Almost always related to embolism (artial-, ventricular thrombus)

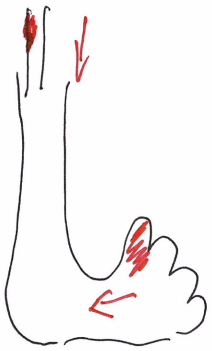
Morphology: - Pale, sharply demarcated infarct
- Wedge shaped, capsule is often covered by fibrin
- Healing with depressed scar



Clinical: - Most of them silent

3. **Gangraena sicca (dry gangraena)** Coagulative anaemic infarct

Pathogenesis: - Mostly related to thrombotic occlusion of a. femoralis or a. poplitea
- Rearly related to embolism



Morphology: - Starts at distal part of the leg
- Progression from distal to proximal

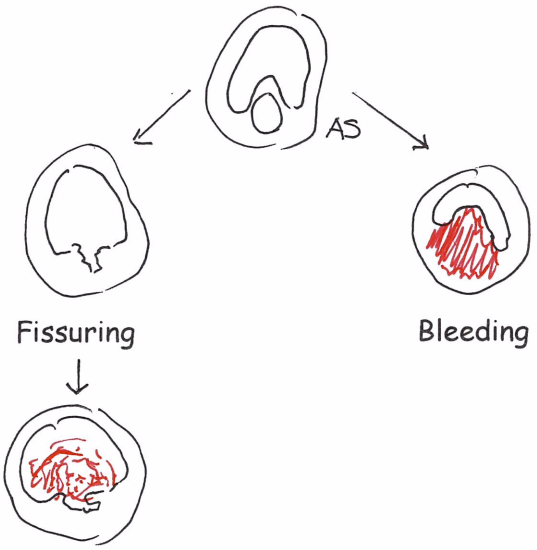
Clinical: - Previous sign – claudicatio intermittens
- Superimposed by infection – wet gangraena
- Can be source of sepsis

4. **Acute myocardial infarct (AMI)** - Coagulative anaemic infarct

- Necrosis of heart muscle resulting from ischaemia
- An imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand

Pathogenesis: - prexisting „fixed" AS plaqe + dinamic changes (thrombosis, vasospasm, bleeding)
- critical stenosis 70-75% - angina

Physical stress
(adrenergic stimuli)
↓
Spasmus
↓
Exposed to collagene
necrotic tissue
↓
Platelet aggregation
↓
Thromboxan A_2
Vasospasm



Fixed coronary

Dinamic changes

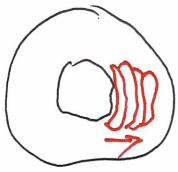
Myocardial response to ischaemia: biochemical + functional + morphologic alterations

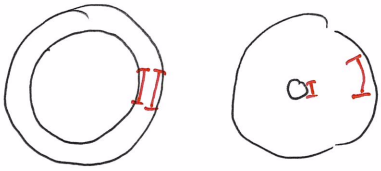
- Biochemical: - anaerobic glycolisis drops
- inadequate production of ATP
- accumulation of lactic acid

- Functional: - loss of contractility
- myofibrillar relaxation
- electric instability
- reversible 20-40 min (reperfusion)

3. LECTURE (2020.09.14.)

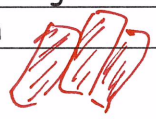
- Wavefront of cell death, wavefront theory:
  - All AMI starts subendocardial and progress transmural in 3-6 h
  - Some remains subendocardial

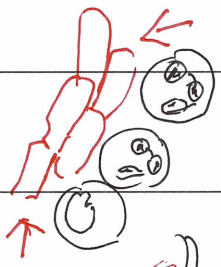


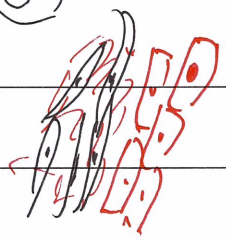


- Morphology:
  - Left anterior descending (LAD) (40-50%) – anterior left vent.
    - anterior septum
    - apex
  - Left circumflex (LCX) (30-40%)
    - posterior left vent.
    - posterios septum
  - Right coronar artery (RCA)
    - lateral left vent.

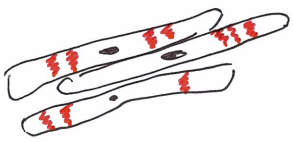
Time	Morphologic (macroscopic/microscopic) alterations
0,5-4 h	- no macroscopic, no microscopic alterations - detection with Diaphorase (triphenil tetrazolium Cl) substrate of dehydrogeneses
4-12 h	- pale, beginning of coagulative necrosis, oedema, sarcolemma disruption
12-24 h	- ongoing necrosis (hypereosinophilia, pycnosis) - beginning infiltration of neutrophils
1-3 days	- yellowish mottling - interstitial infiltration of neutrophils
3-7 days	- yellow center, hyperaemic border - beginnings of removal of death myofibers - massive infiltration of neutrophils, monocytes
7-10 days	- maximally yellow and soft - well developed phagocytosis - fibrovascular granulation
10-14 days	- well established granulatin - collagene deposition
2w-2mo	- scarring is complete - dense collagene scars





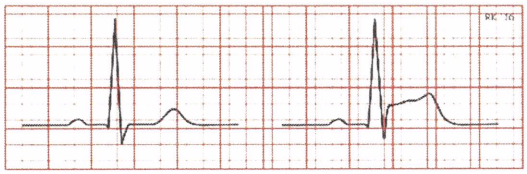


- Reperfusion:
  - Salvage of maximal amount of myocardium – main goal
  - Reperfusion – thrbolysis, balloon angioplastica, stenting, bypass
  - Reperfusion injurry – may iniciate grater damage
    - contraction bands – Ca2+ drive actin-myosis interaction
      - no ATP to relax
    - may generate haemorrhagic indarct



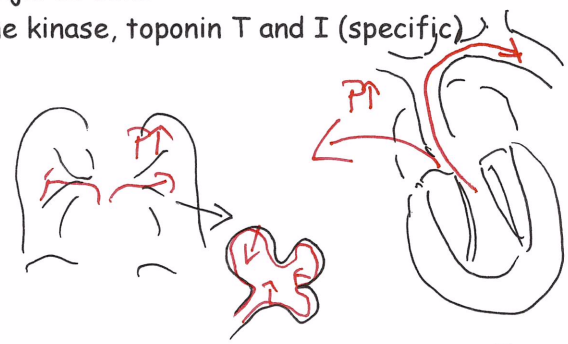
- Clinical features:
  1. Angina
     - substernal pain radiate to neck, jaw, epigastrium, left arm
     - no release of pain in 20 min. by rest or nitroglicetin
     - 10-15% asymptomatic (diabetic neuropathy)
     - posterior part of vertricule – abdominal pain

  2. EKG
     - ST abnormalities – ST elevation, T-en dome
     - Arrythmias

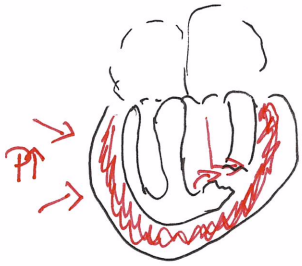


  3. Lab.tests
     - Macromolecules leak out of injured cells
     - Myocardial specific creatinine kinase, toponin T and I (specific)

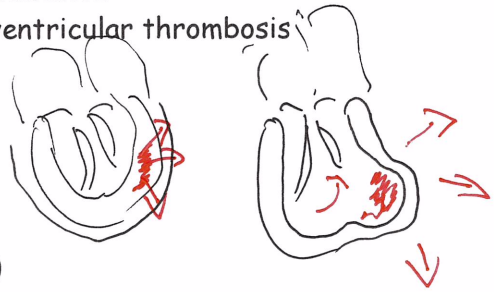
- Consequences/complications
  1. Contractile dysfunction „pump failure"
  2. Arrhytmias – bradycardia
     - fibrillation
     - tachycardia
     - heart block- AV block



  3. Myocardial rupture – high risk at day 3-7 (maximal myomalatia)
     - cardiac tamponade
     - left to right shunt
     - mitral insufficiency



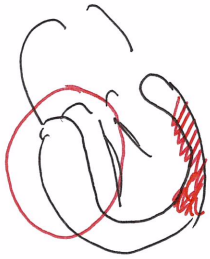
  4. Ventricular aneurysm – paradox pulsation
     - risk for ventricular thrombosis



  5. Mural thrombosis

  6. Pericarditis

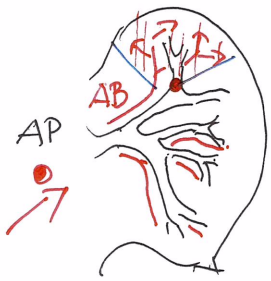
  7. Progressive heart failure (CIHD)



  8. Reinfarction

5. **Pulmonary infarct** - Coagulative haemorrhagic infarct

- Pathogenesis:
  - Blood clot occludes pulmonary arteries – thrombembolic origin
  - Deep vein origin (90%), minority from periprostatic- and parametric vein plexus
  - Predisposition – prolonged bed rest
    - surgery (mostly orthopedic)
    - severe trauma (multiplex fractures, burns)
    - congestive heart failure
    - anticoncipient drugs
    - disseminated cancer

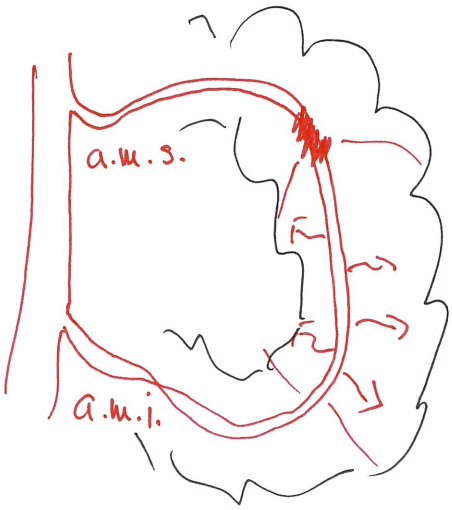


- Morphology:
  A/ - With adequate circulation and bronchial arterial flow the lung parenchyma is maintained
  B/ - With compromised cardiovasclular status (congestive heart failure) haemorrhagic pulmonary infarct will develope – coagulative necrosis with haemorrhage

- Clinical:
  - Clinically silent 60-80% (good systemic circulation)
  - Haemoptoe, pain
  - Right sided heart failure
  - Rapid removal by fibrolitic activity – recanalisation

6. **Intestinal infarct** – Coagulative haemorrhagic infarct

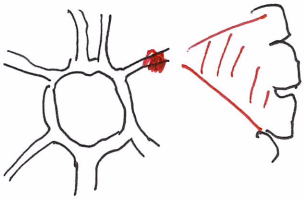
- Pathogenesis:
  - A/ Arterial thrombosis (AS, dissecting aneurysm)
  - B/ Arterial embolism (cardiac-, AS thrombosis)
  - C/ Venous (v. portae thrombosis) thrombosis
  - D/ Non occlusive ischeamia (cardiac failure, shock)



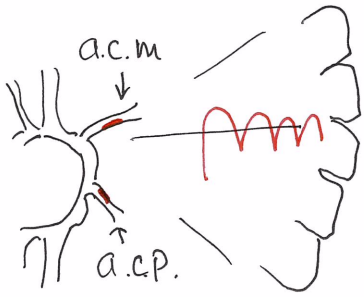
- Clinical:
  - Most common in old patients
  - Abdominal pain
  - Bloody diarrhea
  - Perforation – peritonitis
  - Mortality 90%

7. **Cerebral infarct** – Liquefactiv anaemic infarct

- Pathogenesis: - A/ AS occlusion of cerebral arteries
  - B/ Systemic embolism (cardiac mural thrombus, AS)
  - C/ Venous (sinus) thrombosis – Liquefactive haemorrhagic infarct



- Morphology: - A/ Encephalomalacia alba
  - First 36-48h
  - Necrotic area become swollen and soft
  - By 3dr day macrophages infiltrate the lesion
  - Increasing demarcatio
- B/ Encephalomalacia flava
  - Liquefaction of infarct
  - Irregular small cavities
- C/ Cysta post encephalomalaciam
  - Firm rim of gliosis around cyst
- D/ Encephalomalacia rubra
  - Reperfusion of anaemic cerebral infarct
  - Venous thrombus
  - Border zone infarct



8. **Acute pancreatitis** – Fat necrosis

Pathogenesis: - Implication of gallstone within the common bile duct
- Activation of pancreas enzymes by bile in the pancreas
- Autodigestion of pancreas by inappropriatly activated pancreatic enzymes
- Alcoholism – hypersecretion-obstruction theory

Morphology: - Necrosis of fat by lipase
- Proteolytic destruction of pancreatic parenchyma
- Destruction of blood vessels – haemorrhage

Clinical: - Abdominal pain – acute abdomen
- Elevated plasma levels of amylase and lipase
- Systemic relaese of digestive enzymes - DIC, ARDS