

1. hypovolemia/shock
2. pulmonary embolism/shock
3. acute myocardial infarction/shock

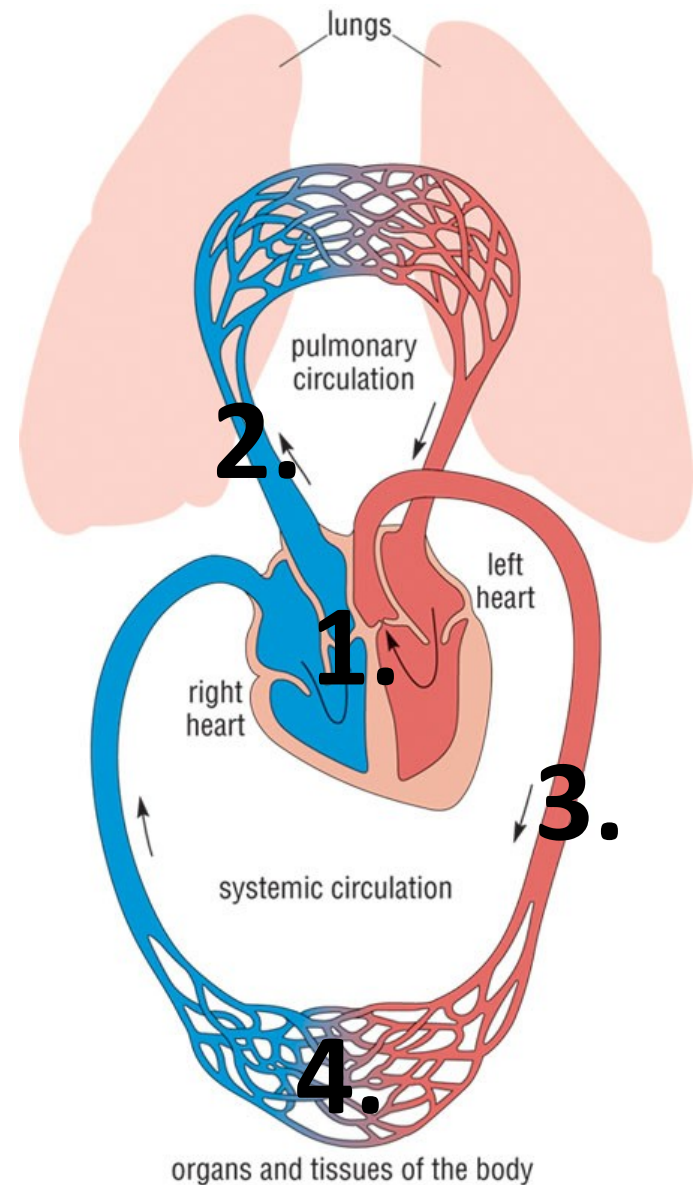
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Shock in general

Shock

- Circulatory failure – supply \neq demand
- **1. cardiogenic – pump**
- **2. obstructive – obstruction**
- **3. hypovolemic – filling**
- 4. distributive - shunts



Phases of Shock

1. Compensation

2. Decompensation

3. Refractory

- Inflammatory cascade induction and organ damage - „secondary-hit model“
- Organ damage further increases inflammatory cascade induction – vicious circle
- Each type of shock differs at the beginning, however during the late phase all types of shock look similar (like distributive shock)

Pathophysiology

- The main problem is cell **hypoxia**
- **Stress response**
 - catecholamines, RAAS, cortisol, glucagon
- **Systemic inflammatory response**
 - Immunity, inflammatory mediators
 - Locally OK, but generalized response is harmful

Changes are in ...

1. **Macrocirculation**

- „blood flow centralization“
- rarely „warm shock“

2. **Microcirculation**

- Endothelium damage
- Increased vascular leakage, leucocytes adherence
- Main role in shock

3. **Coagulation**

- Intravascular coagulation

4. **Metabolism**

- Increased gluconeogenesis, proteolysis
- Lactate acidosis

MODS

1. Circulation

- Vasoplegia, cardiomyopathy

2. Lungs

- ARDS

3. Kidney

- AKI

4. Coagulation

- DIC

5. CNS

- Altered consciousness

6. GIT

- Loss of barrier function

Signs/Symptoms

- Nonspecific
- Variable
- Unreliable

Hypotension, tachycardia:

- SBP < 90 mmHg
- MAP < 60 mm Hg
- Tf > 100/min
- Cave compensatory shock/BB

Oliguria:

- diuresis < 0,5 ml/kg/hr for 1 – 6hrs

Tachypnea

- > 30 breaths/min, dyspnea

Skin:

- Wet, cold
- CRT(> 2 s)

Mental state:

- Confusion
- Irritation
- coma

Diagnostics

1. Basic Lab

- BC, coagulation (Q/INR, aPTT, fib)
- ions, gly
- urea, kreatin
- CRP (sepsis?)

2. ABG

- Ventilation/oxygenation
- Lac, SvO₂, ScvO₂

ABG

Lac

- Product of anaerobic glycolysis
- Non-toxic, serves also as a fuel
- normal < 2 mmol/l
- Mortality predictor
- Early sign of shock

ScvO₂

- $O_2ER = (SaO_2 - SvO_2) / SaO_2$, normally 25%
- normal SvO₂ is 75%
- **SvO₂ < 70% = O₂ supply impairment**

Extended Hemodynamics

Shock Hemodynamics

| | CO | SVR | PAOP | EDV |
|--------------|----|-----|------|-----|
| Hypovolemic | ↓ | ↑ | ↓ | ↓ |
| Cardiogenic | ↓ | ↑ | ↑ | ↑ |
| Obstructive | | | | |
| afterload | ↓ | ↑↑ | ↑ | ↑ |
| preload | ↓ | ↑ | ↑ | ↓ |
| Distributive | | | | |
| pre-resusc | ↓ | ↑ | ↓ | ↓ |
| post-resusc | ↑ | ↓ | ↑ | ↑ |

Initial resuscitation

- **Preload optimization** – increasing CO, fluids „volume challenge“, PLR
- Persistent hypotension – catechols (**norepinephrine**)
- If CO does not rise with fluids, add **inotropes** (dobutamin)
- Lowering of inadequately high **afterload** (hypertension crisis)

Causal treatment

1. Cardiogenic shock:

- SCG - PCI
- Arrhythmia treatment (AV block III., VT)

2. Hypovolemic shock:

- fluids
- hemotherapy
- damage control surgery/damage control resuscitation

3. Obstructive shock:

- thrombolysis
- Pericardial effusion evacuation

1. Hypovolemic Shock

Most common Causes of Hypovolemia

- **Bleeding**
- **Loss of fluids** (sweating, vomiting, diarrhea,)
inadequate intake
- **Burns**
- **3rd space losses**
 - Ileus
- anafylaxis, sepsis (**relative hypovolemia**)

Treatment

1. Initial resuscitation

2. Causal treatment

- Goal is to restore organ perfusion, O₂ supply
- Early initiation

- Secondary goal: restoration of O₂ transportation capacity (ERY...)

Venous access

- 2-3 thick peripheral cannulas
- Central venous access is secondary (good for catecholamine, not fluids)
- Exception: thick central lines (Edwards AVA 9F)

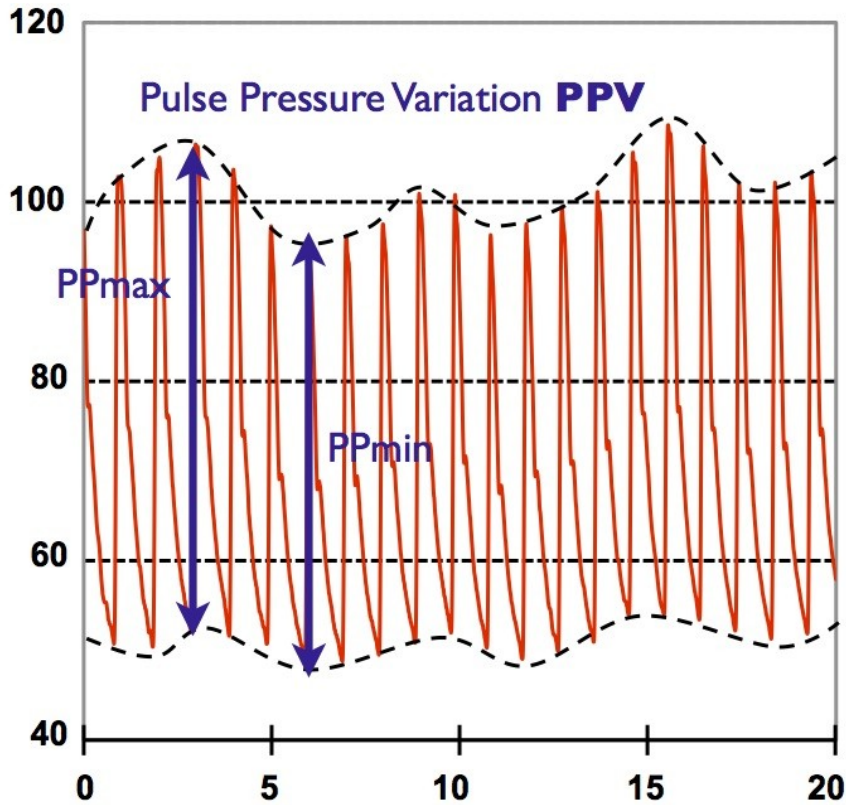
$$\frac{\Delta V}{\Delta t} (Q) = \Delta P \cdot \frac{\pi}{8} \cdot \frac{1}{\eta} \cdot \frac{R^4}{L}$$



Arterial Catheter

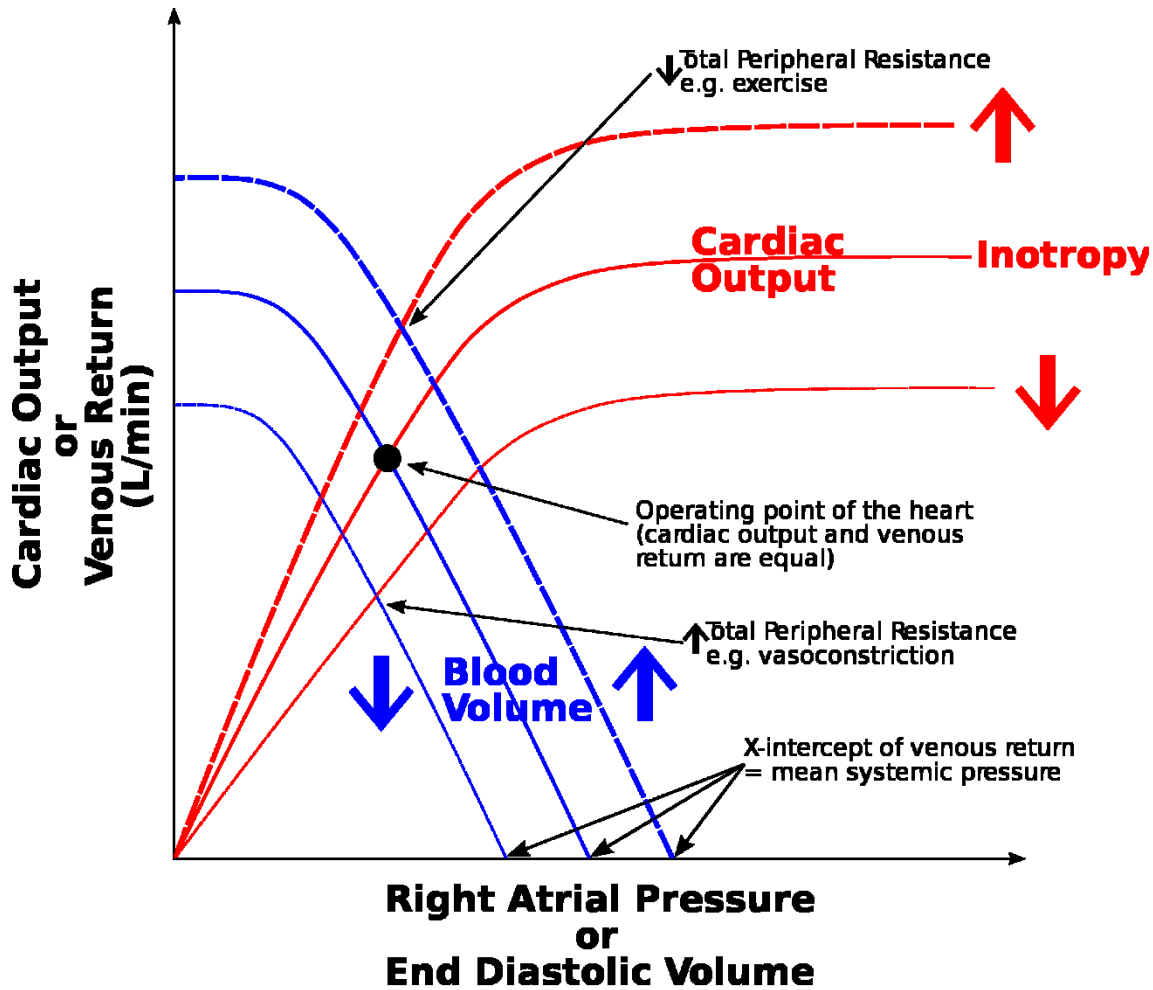
- Continuous blood pressure monitoring
- accurate
- PPV – only sinus and positive pressure vent.
- Repeated blood draws

SPV / PPV



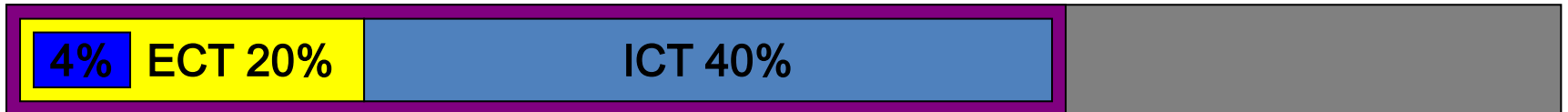
$$: 100 \times \left\{ \frac{(PP_{\max} - PP_{\min})}{\left[\frac{1}{2} (PP_{\max} + PP_{\min}) \right]} \right\}$$

SPV / PPV



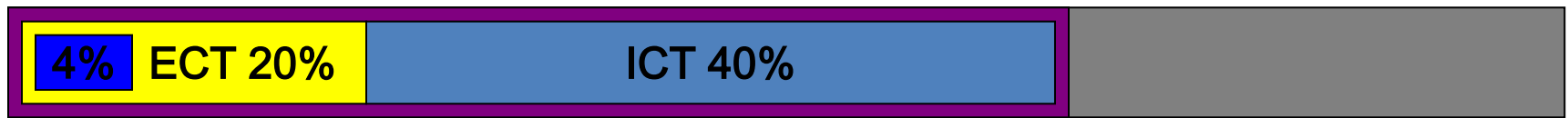
Witch fluid to use?

- ⦿ Ions Na^+ and K^+ - ICT/ECT distribution
- ⦿ Oncotic pressure plasma/ECT distribution



Glucose

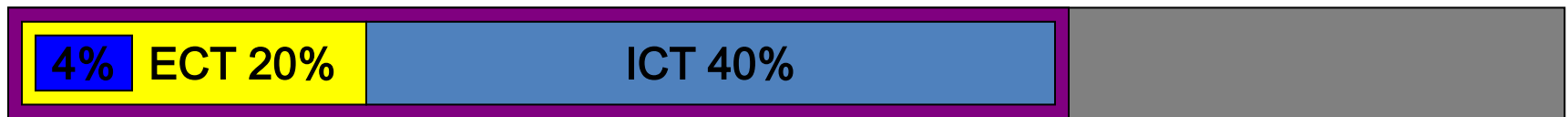
- **Inadequate**
- Absolute water deficit
- Hyponatremia correction



Distribution volume

Crystalloids

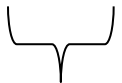
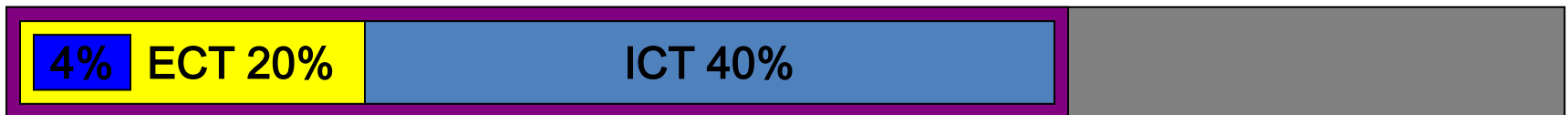
- Fast leak into the ECT compartment
- Substitution has to be 4x higher than the deficit (...recently questioned) → **swellings**



Distribution volume

Colloids

- Do not leave the intravascular compartment
- Equal the deficit
- Adverse reactions, contraindication – sepsis – renal damage
- Good for acute blood loss



Distribution volume

Blood products

- Only for blood loss corrections
- 5% albumin – natural colloid
 - expensive

Fluid resuscitation goals

- Blood pressure, heart rate
- Centralization reversal
- diuresis
- Decrease of the PPV / SVV
- Sc(v)O₂ a lactate normalization

- Filling pressures(CVP, PAOP) not a good target

Acute bleeding

Blood loss

- **15%** (750 ml) well compensated
- **30%** (1,5 l) – tachycardia, oliguria, normotension – **however ↓ organ perfussion!**
- More than **30%**: hypotension, tachycardia, oligo-anuria, ...

fractures

- pelvis (5000ml)
- femur (2000ml)
- tibia (1000 ml)
- humerus (800 ml)
- radius (400ml)

Treatment

- Basic approach ... ABCD
- Stop the bleeding
- Give i.v. Fluids + catecholamine
- Blood: type O- (4 immediately available), after 30 minutes type matched
- Fresh frozen plasma 1:1 with erythrocytes
- Target Hb 70-90 g/l, CNS trauma 100g/l
- Thrombocytes 50 – 100 tis/ul
- Fibrinogen 1,5 g/l
- Prevent hypothermia, hypotension and acidosis

2. Cardiogenic shock/ AIM

AIM

- **Myocardial ischemia**

Causes

1. Increased demand – tachycardia
 2. Low oxygen content – anemia, CO poisoning, hypotension, pulmonary disease
 3. Low coronary artery blood flow
- 90 % low coronary artery flow – coronary atherosclerosis
 - Transmural ischemia – 3/4 of the myocardial wall (complete closure)
 - Laminar/subendomyocardial – 1/3 of the myocardial wall (partial closure + increased demand)

Diagnosics

1. Patient history/clinical evaluation
2. ECG a Lab
3. ECHO, SCG

| | STEMI | NSTEMI | AP |
|---------|---|--|--|
| History | Chest pain | Chest pain | Chest pain |
| ECG | ST elevation at least 2 mm in leads V1–V3 or at least 1 mm in V4–V6, I, aVL, II, III, aVF. ST elevation in at least two adjacent leads. New LBBB or (RBBB + LAH, RBBB + LPH). | ST depression at least 1 mm and /or T wave inversion | ST depression at least 1 mm and /or T wave inversion |
| Lab | Positive TNT | Positive TNT | Negative TNT |

Localization

| | |
|--------------------|----------------|
| Anteroseptal | V1-V4 |
| Anterolateral | V1-V6 |
| Lateral | I, aVL, V5, V6 |
| Lower/diafragmatic | II, III, aVF |

Treatment

Continuous vital signs / ECG

IV access

Oxygen 4–8 l/min

12 lead ECG

Blood draw – Lab /TNT

Analgesedation - morphine

ASA 500 mg i.v./200–400 mg p.o.

heparin 5000 j i.v./enoxaparin 1 mg/kg s.c./i.v.

clopidogrel 300 nebo 600 mg p.o.

metoprolol i.v. If tachycardia

Cardiogenic Shock

- Severe, long-lasting arterial hypotension
- Low CO
- Increased filling pressure CVP/PAOP
- Alteration of consciousness, oliguria, cold periphery, sweat, cyanosis

Treatment

- Most important is to **increase oxygen supply** and **lower oxygen consumption** by myocardial muscle
- **Preload** optimization: diuretics/fluids
- **Afterload** optimization: vasodilatation / cave coronary artery perfusion
- **Inotropy** – dobutamin
- Treatment of the cause – PCI/thrombolysis

Avoid

- **Tachycardia** – short diastolic phase, increased work load (however, sometimes only chance how to increase CO)
- **Severe hypotension, hypovolemia, vasodilatation** – low coronary artery perfusion pressure (Ao pressure – EDP LV)
- **Increased preload/afterload** – increase of wall tension, work

Treatment

- **Oxygen** – increase O₂ supply
- **NIV, invasive ventilation** – oxygenation, decreases preload/afterload
- **Diuretics/fluids** – decrease preload, in later phase optimization of preload (fluid challenge/PLR)
- **Catecholamine** – norepinephrine for blood pressure, dobutamin (milrinon, levosimendan) for inotropy
- **Vasodilatancia** – nitrates, coronary artery, but also systemic vasculature (increased blood pooling, preload lowering; arterial – afterload lowering)
- **Morphine** – improves dyspnea

3. Obstructive shock/ PE

Pulmonary Embolism

- Sudden obstruction of pulmonary vasculature with emboli (blood clot, fat, tumor, air/gas, foreign body, ...)

Etiology:

- 85% low extremity/pelvic DVT

Risc Factors

- **Virchow trias** - venostasis, hypercoagulation, vessel wall damage
- Major surgery
- Lower extremity fractures
- Hypercoagulation (Leiden ...)
- Heart Failure (blood stasis)
- Sepsis (coagulation activation)
- High age (70 years)
- Immobilization
- Obesity
- Pregnancy
- Economy class syndrome
- corticoids, diuretics, HAC

Diagnosis

History

- Sudden dyspnea, chest pain, tachypnea, cough, syncope, hemoptysis

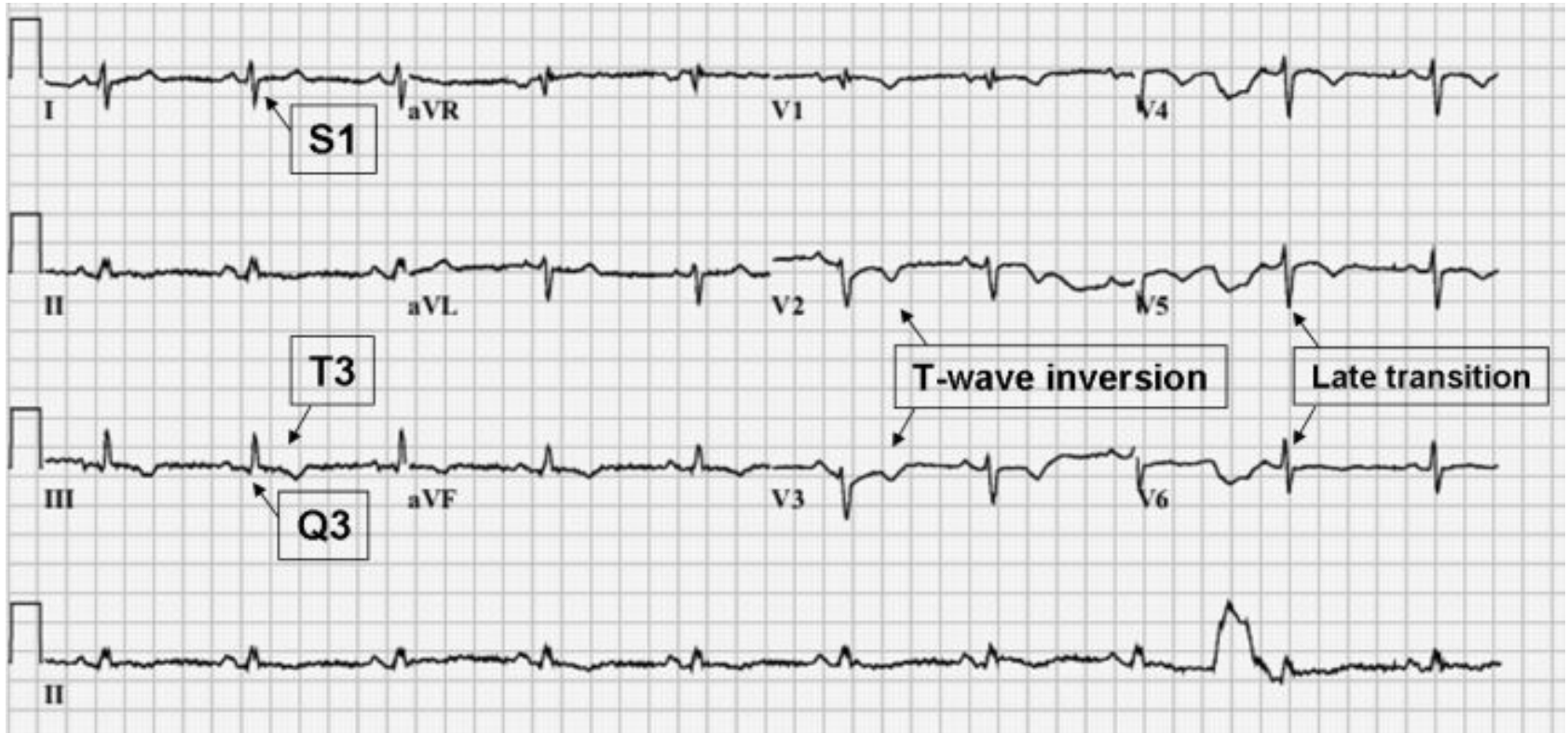
Clinical evaluation

- tachypnea, cyanosis, hypotension, shock, tachycardia, neck veins distension

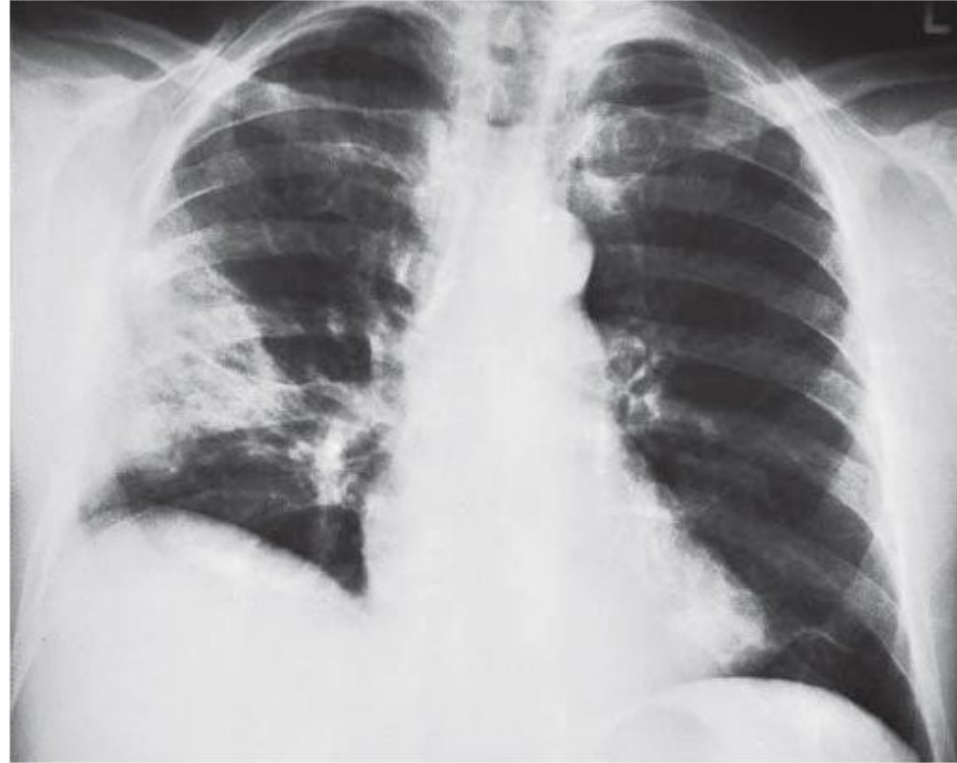
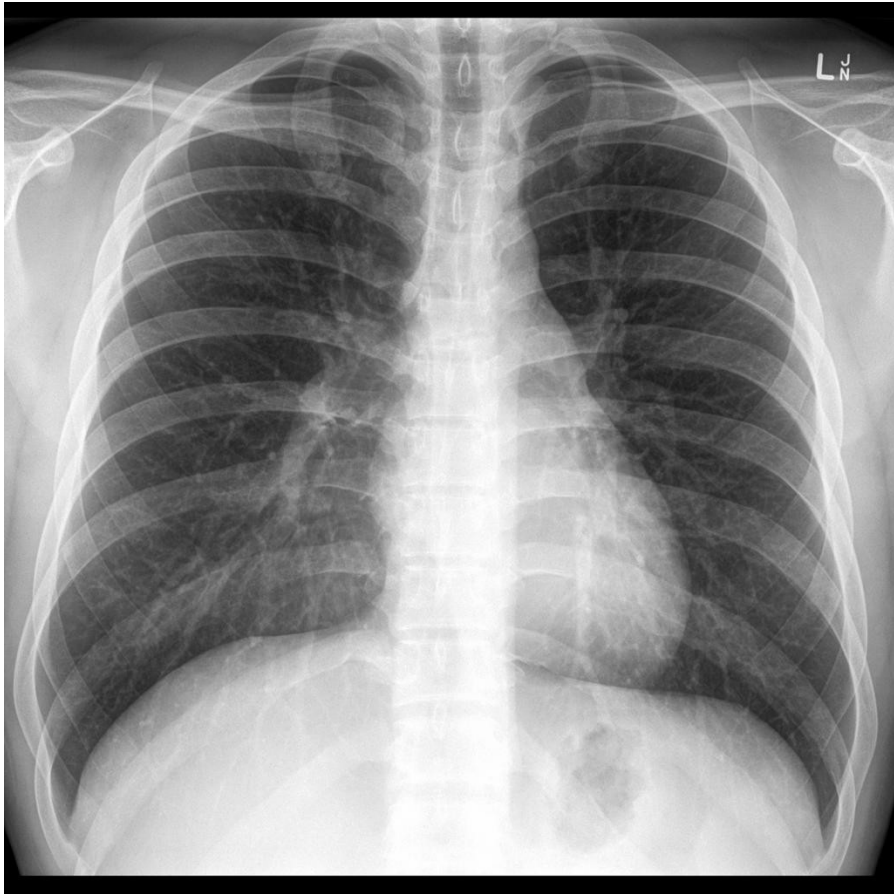
Lab

- ABG - hypoxia, hypocapnea, Ralc
- DD- negative – practically excludes PE
- DD- positive – tumors, inflammation, post-surgery, sepsis ...

EKG



Chest X-ray

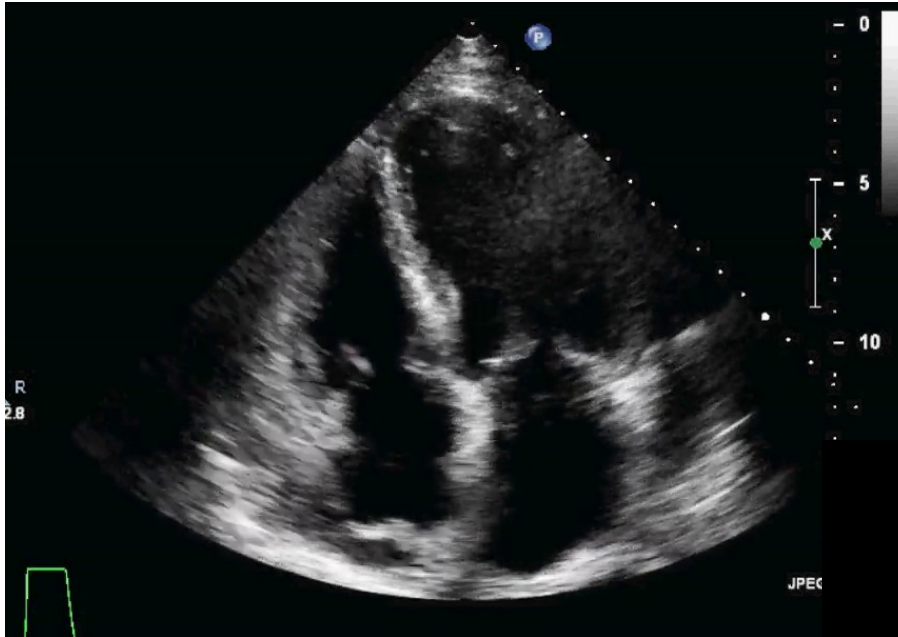


Excludes other reasons for dyspnea

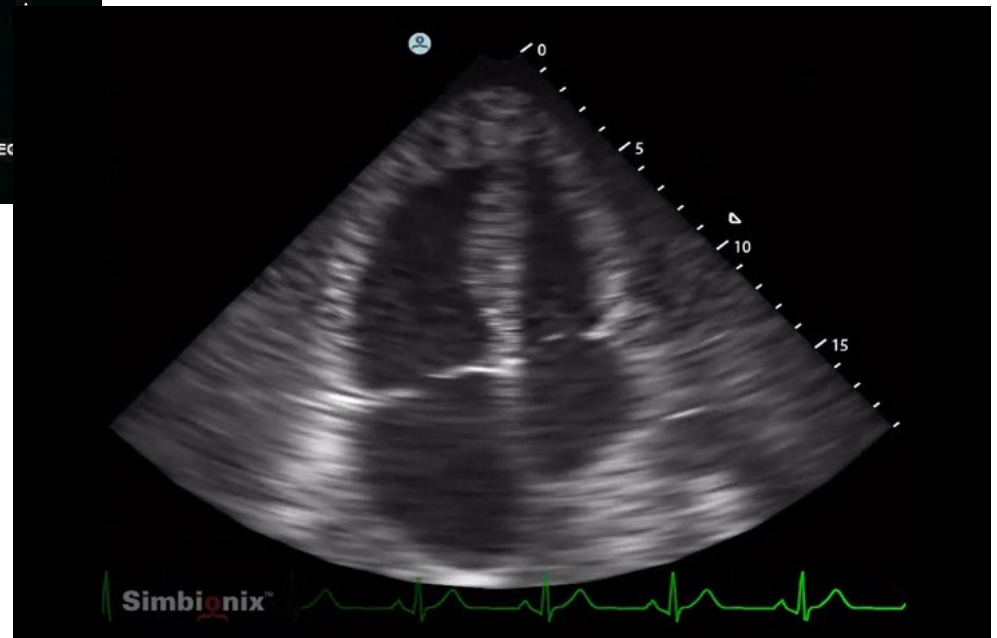
Fleischman sign- atelectasis

Westerman sign – decreased pulmonary vascularization

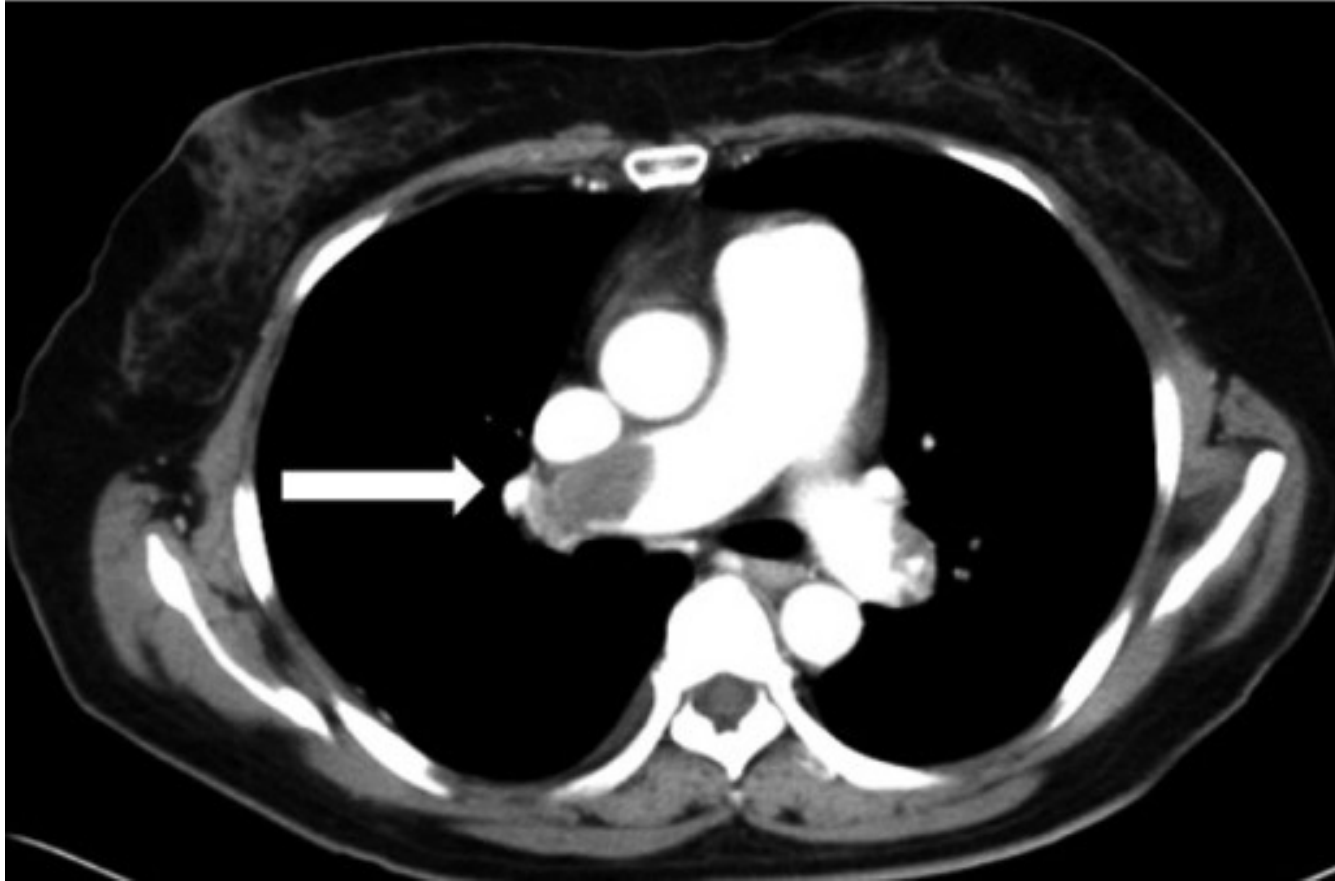
ECHO



RV dilatation, paradoxical septum movements, pulmonary hypertension, Tri regurgitation



CT - AG



Other

- **Vein US** – femoral, popliteal
- **TEE** – thrombus in pulmonary artery
- **Swan-Ganz** - precapillary PH, high CVP, high RV pressure, increase PAP,
- **Ventilation/perfusion scan** – low specificity

Management

- Clinical probability, DD, echo and CT angio

| | | |
|---|-----|---|
| Signs of DVT | 3 | Clinical probability: <ul style="list-style-type: none">- low 0-1 (3,4%)- moderate 2-6 (20%)- high 7 (63%) - 0-4 PE less probable- More than 4 - PE highly probable |
| Other dg improbable | 1,5 | |
| Tachycardia 100 | 1,5 | |
| Immobilization more than 3 days, surgery within 4 weeks | 1,5 | |
| DVT, PE in history | 1,5 | |
| hemoptysis | 1 | |
| malignancy | 1 | |
| | | |

1. High risk PE (shock, hypotension)

- CT angio or ECHO, if CT unavailable/impermissible for the patient
- CT/ECHO positive - trombolysis

2. Low risk PE (without shock/hypotension)

- High clinical suspicion – CT angio
- Low clinical suspicion – DD
- Negative DD nearly completely exclude PE
- TNT, NT pro BNP, RV dysfunction – thrombolysis/heparinization

Massive PE – unstable or RV dysfunction, TNT, NTproBNP

- **Thrombolysis** – optimally within 48 hrs
alteplasis (0,9mg/kg)—10 mg bolus iv. + 90 mg cont. iv. for 2 hrs
- + heparin for min 72 hrs - UHF 80 IU/kg bolus + 18 IU/kg/hr

Thrombolysis contraindications

Table 1

Contraindications for fibrinolysis⁴⁶

Absolute contraindications

Haemorrhagic stroke or stroke of unknown origin at any time

Ischaemic stroke in the preceding 6 months

Central nervous system damage, neoplasms or structural vascular lesions (e.g. arteriovenous malformation)

Recent major trauma/surgery/head injury (within the preceding 3 weeks)

Gastro-intestinal bleeding within the last month

Known bleeding disorder (excluding menses)

Aortic dissection

Relative contraindications

Transient ischaemic attack in preceding 6 months, dementia

Oral anticoagulant therapy

Pregnancy within 1-week post-partum

Non-compressible punctures

Traumatic resuscitation

Refractory hypertension (systole. blood pressure >180mmHg)

Advanced liver disease

Infective endocarditis

Active peptic ulcer

Small PE

- **UF heparin** – bolus 80IU/kg + 18IU/kg/hr—
aPTT 1,5-2,5 times norm
- At least 6-10 days, than warfarin
- **LMWH**- as effective as UHF, s.c. every 12 hrs
- At least 6-10 days, than warfarin
- Cave – renal dysfunction, antiXa (terap. 0,6-1,0 U/ml) 3 hrs after administration