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

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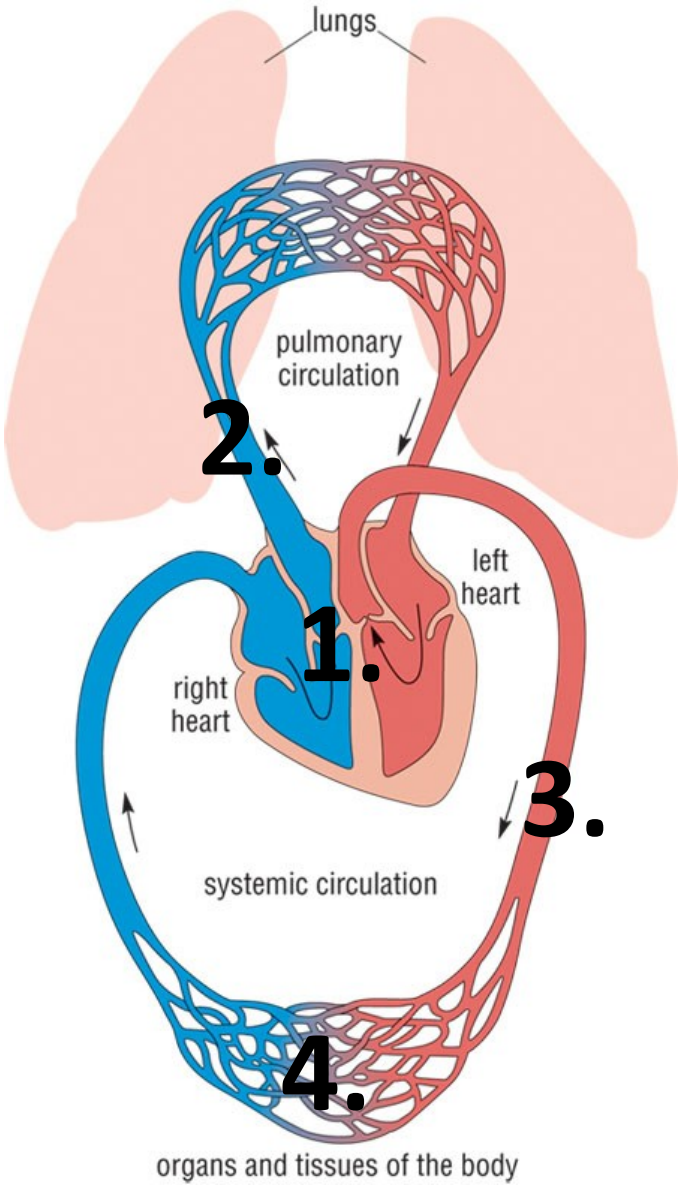
2016

Shock in general

Shock

- Circulatory failure – supply ≠ 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**
  - Imunity, inflamatory mediators
  - Localy OK, but generalized response is harmful

Pathophysiology

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
- Iritation
- 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

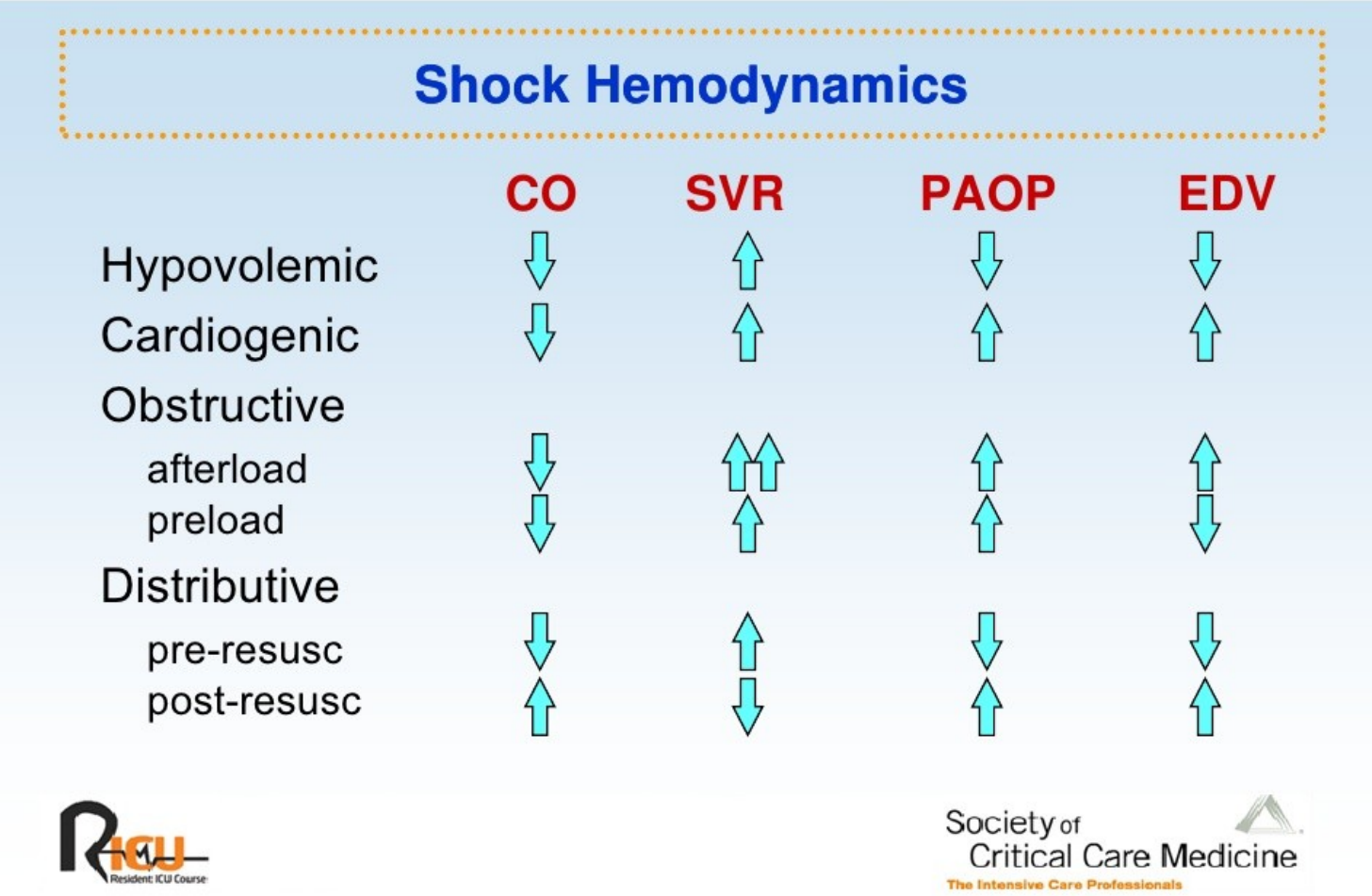
Lac

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

ScvO2

- $O_2ER = (SaO_2 – SvO_2) / SaO_2$, normaly 25%
- normal SvO_2 is 75%
- **SvO_2 < 70% = O_2supply impairment**

Extended Hemodynamics



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 (**relativ hypovolemia**)

Treatment

1. **Initial resuscitation**
2. **Causal treatment**

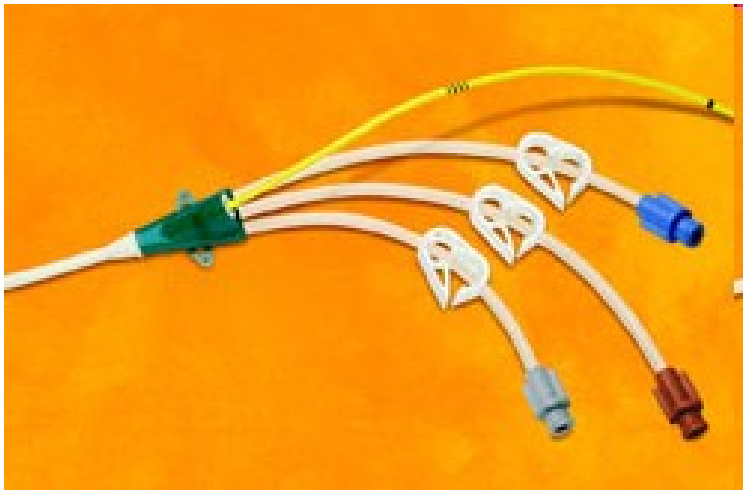
- Goal is to restore organ perfusion, O2 supply
- Early initiation

- 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 (Edwars AVA 9F)

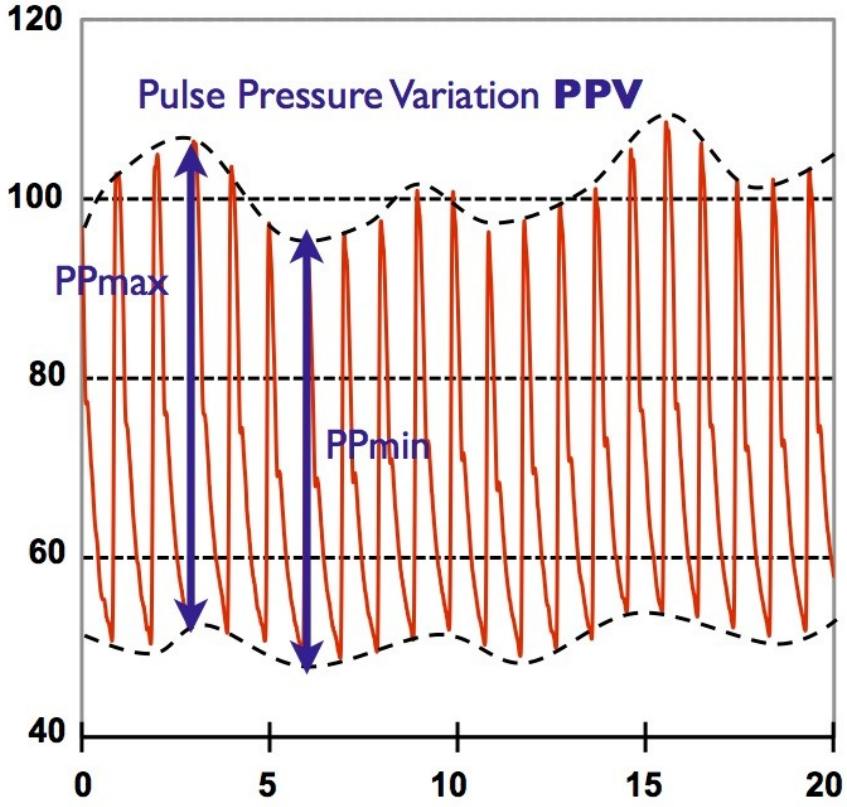
$$\frac{\Delta V}{\Delta t}(Q) = \Delta P \cdot \frac{\pi}{8} \cdot \frac{1}{\eta} \cdot \frac{R^4}{L}$$



Arterial Catheter

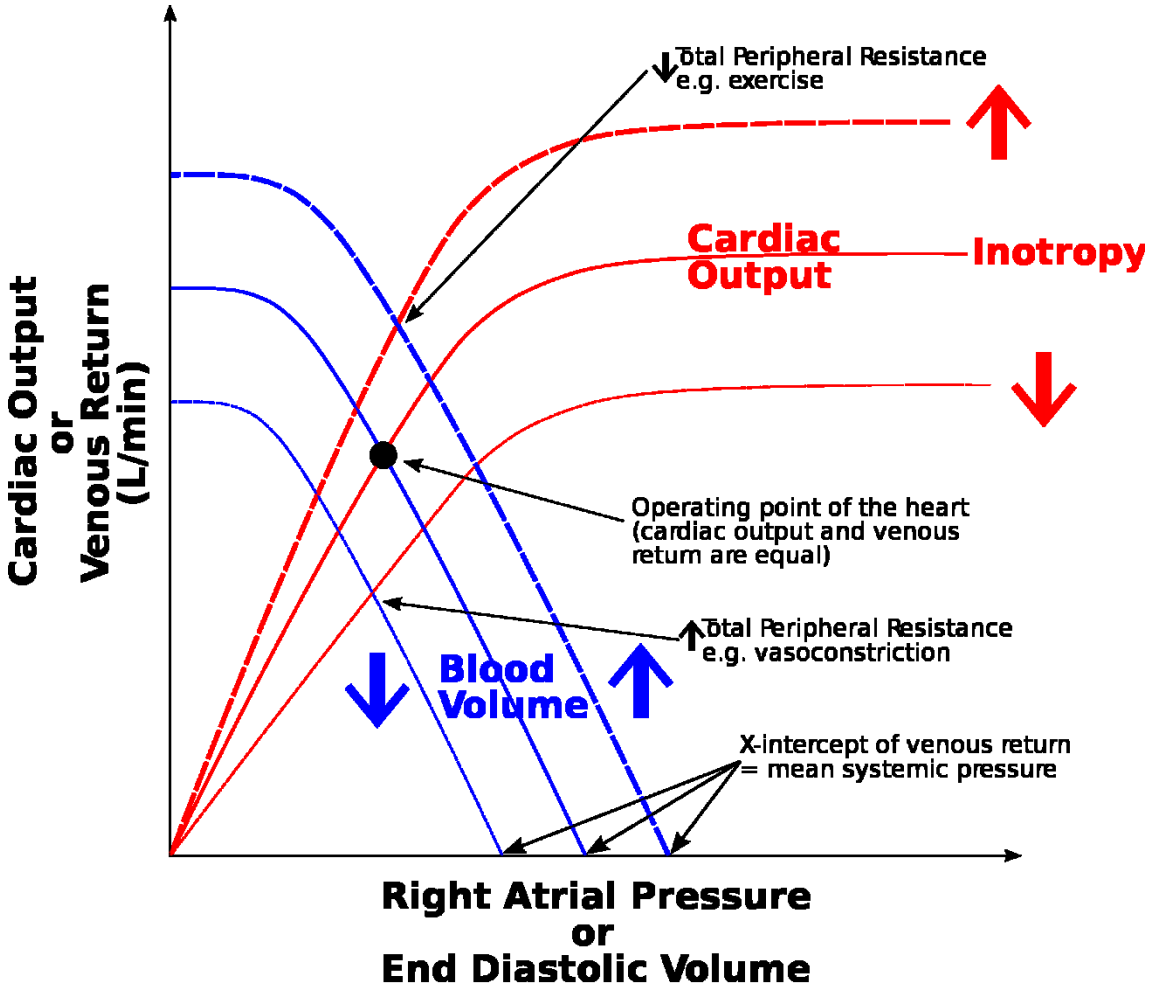
- Continuous blood pressure monitoration
- accurate
- PPV
- Repeated blood draws

SPV / PPV



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

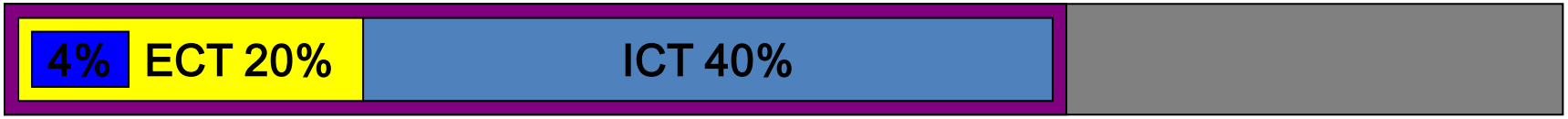
SPV / PPV


Total Peripheral Resistance
e.g. exercise
Cardiac Output
Inotropy
Operating point of the heart
(cardiac output and venous return are equal)
Total Peripheral Resistance
e.g. vasoconstriction
Blood Volume
X-intercept of venous return
= mean systemic pressure
Cardiac Output
or
Venous Return
(L/min)
Right Atrial Pressure
or
End Diastolic Volume

Witch fluid to use?

- Ions Na^+ and K^+ - ICT/ECT distribution

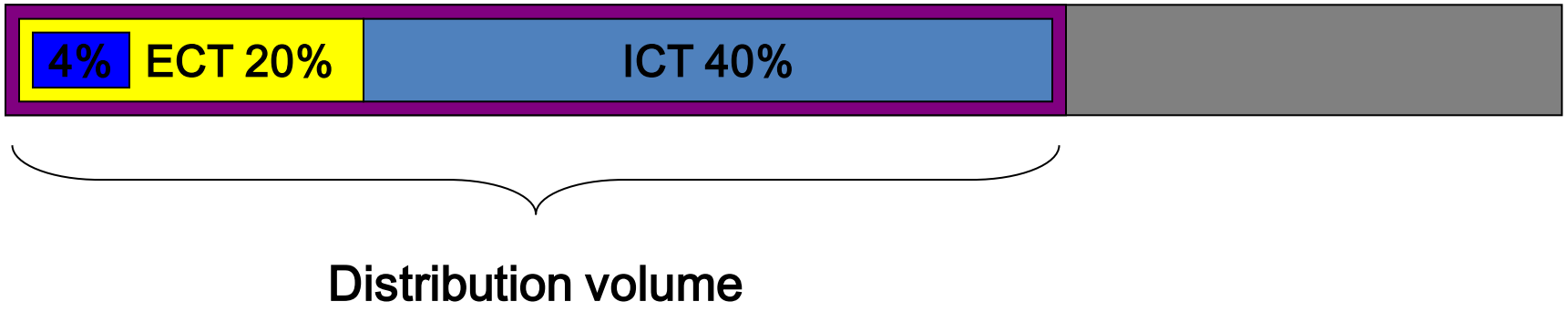
- Oncotic pressure plasma/ECT distribution



Glucose

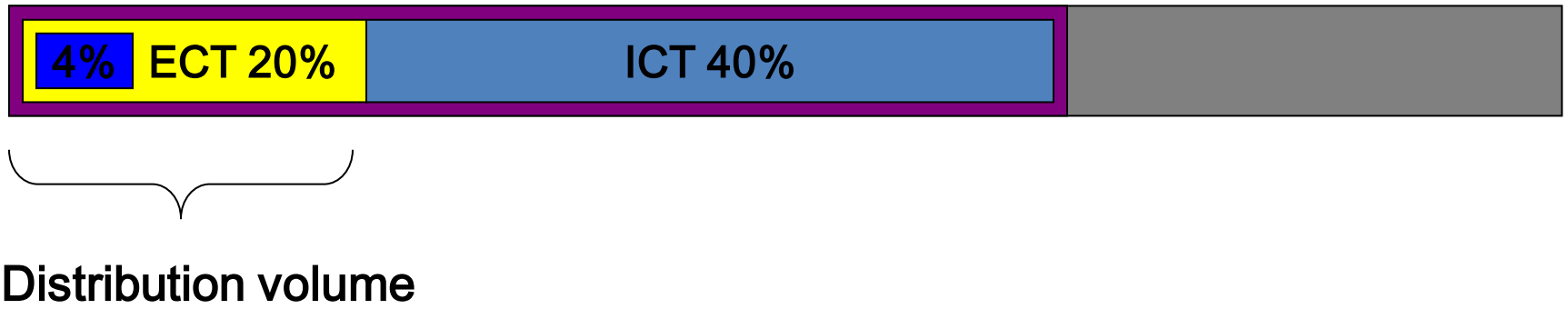
- **Inadequate**

- Absolute water deficit
- Hypernatremia correction



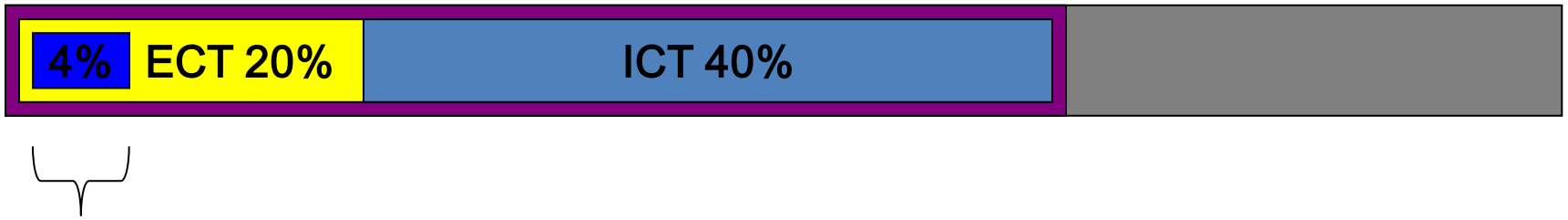
Crystaloids

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



Coloids

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



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)

Diagnostics

1. Patinet 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
Analgosedation - 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 consuption by myocardial muscle

- Preload optimalization: diuretics/fluids
- Afterload optimalization: vasodilatation / cave coronary arthery perfussion
- 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 cloth, fat, tumor, air/gas, foreign body, ...)

Etiology:

- 85% low extremity/pelvic DVT

Risc Factors

- **Virchov 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

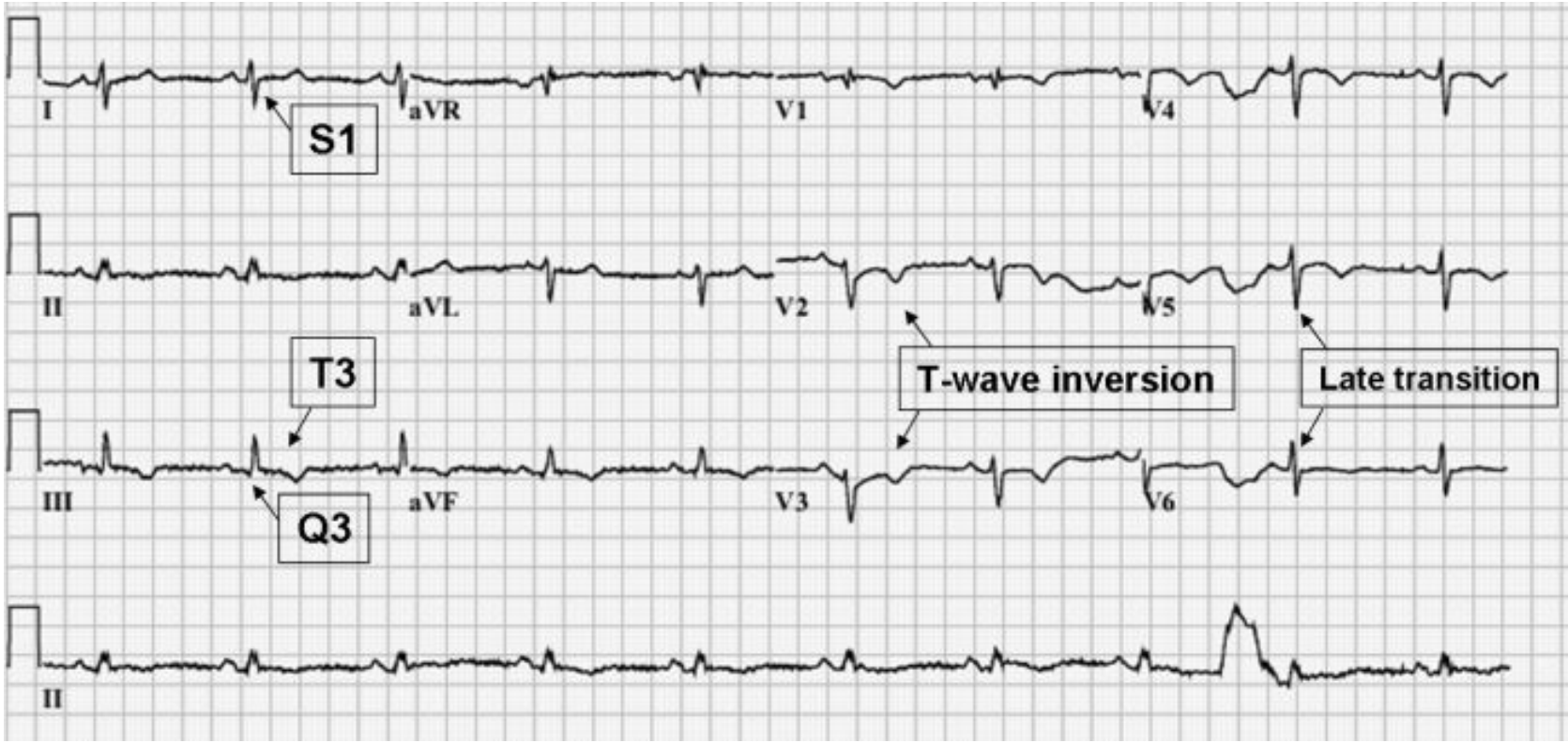
Clinical evaluation

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

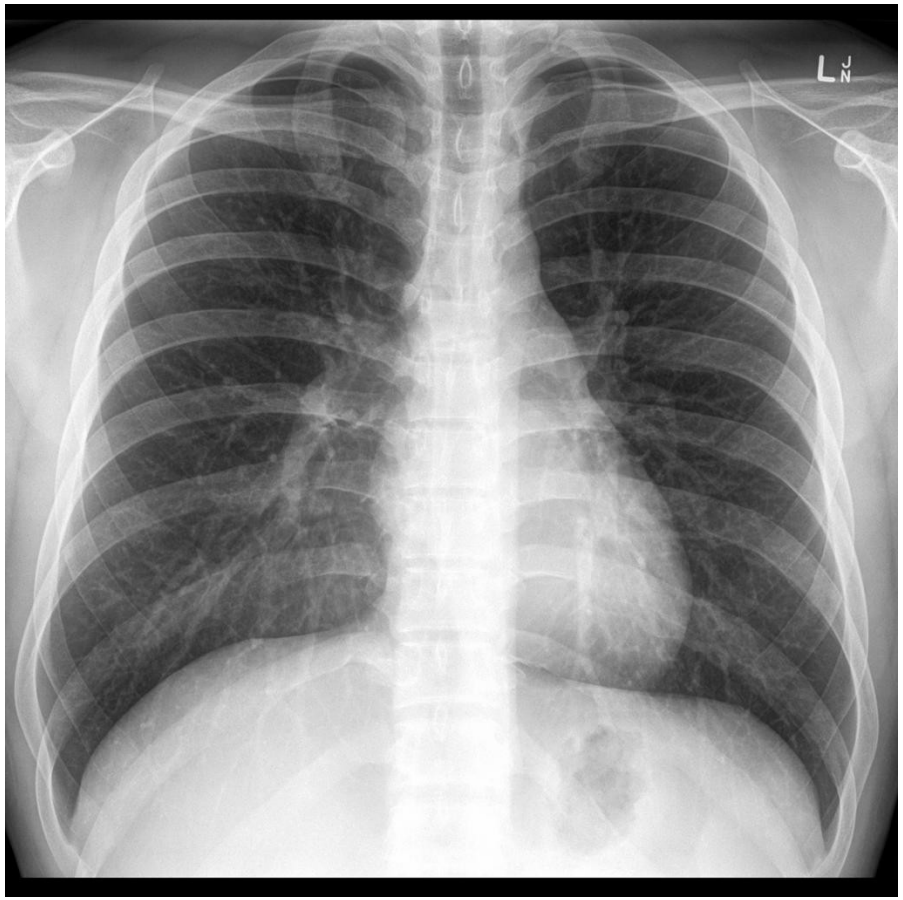
Lab

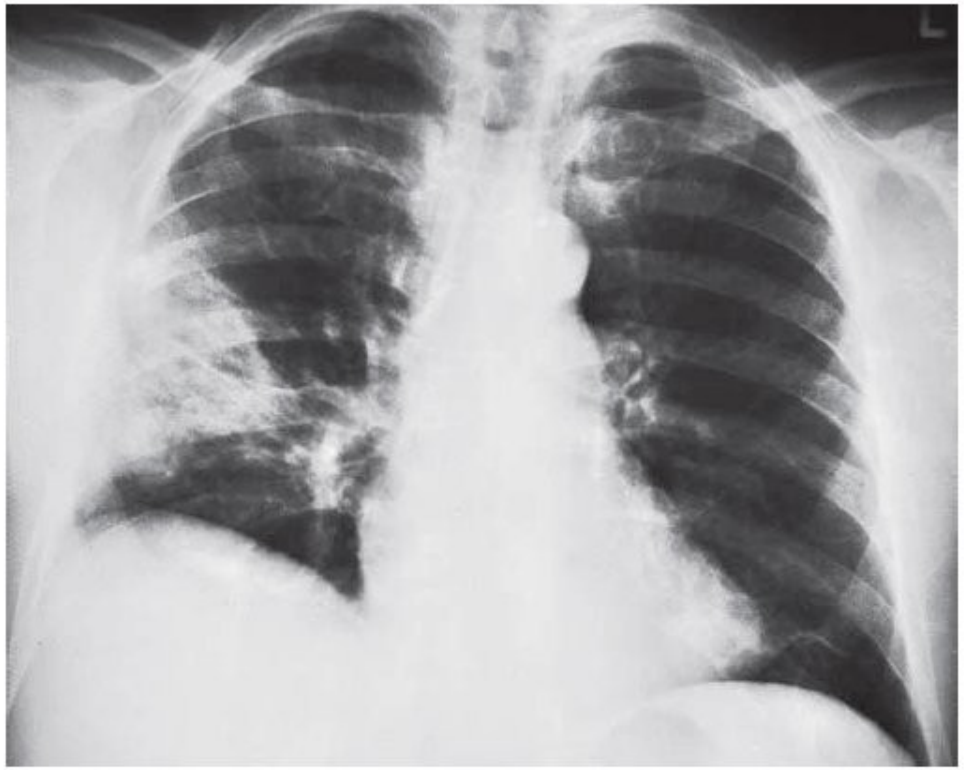
- ABG - hypoxia, hypocapnea, Ralc
- DD- negative – practically excludes PE
- DD- positive – tumors, inflammation, post-surgery, sepsis ...

EKG


I
aVR
V1
V4
S1
II
aVL
V2
V5
T3
T-wave inversion
Late transition
III
aVF
V3
V6
Q3
II

Chest X-ray

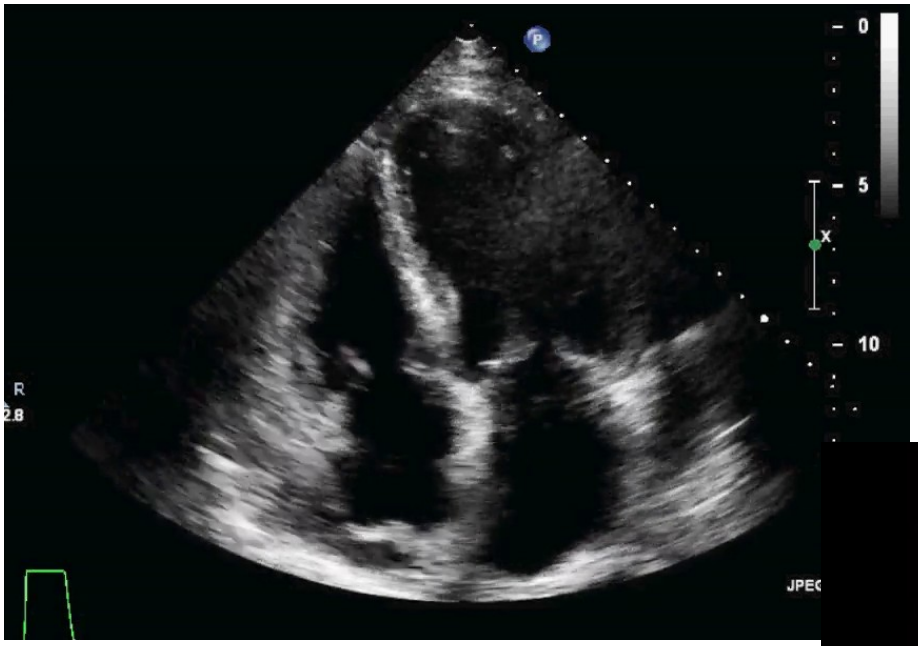




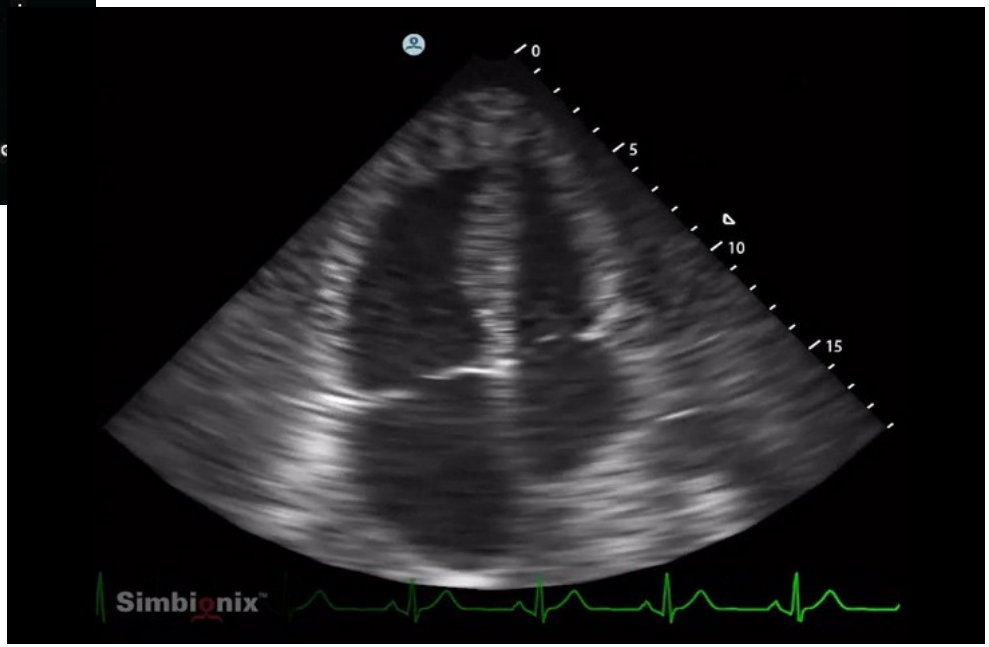
Excludes other reasons for dyspnea

Fleischman sign- atelectasis
Westerman sign – decreased pulmonary vascularization

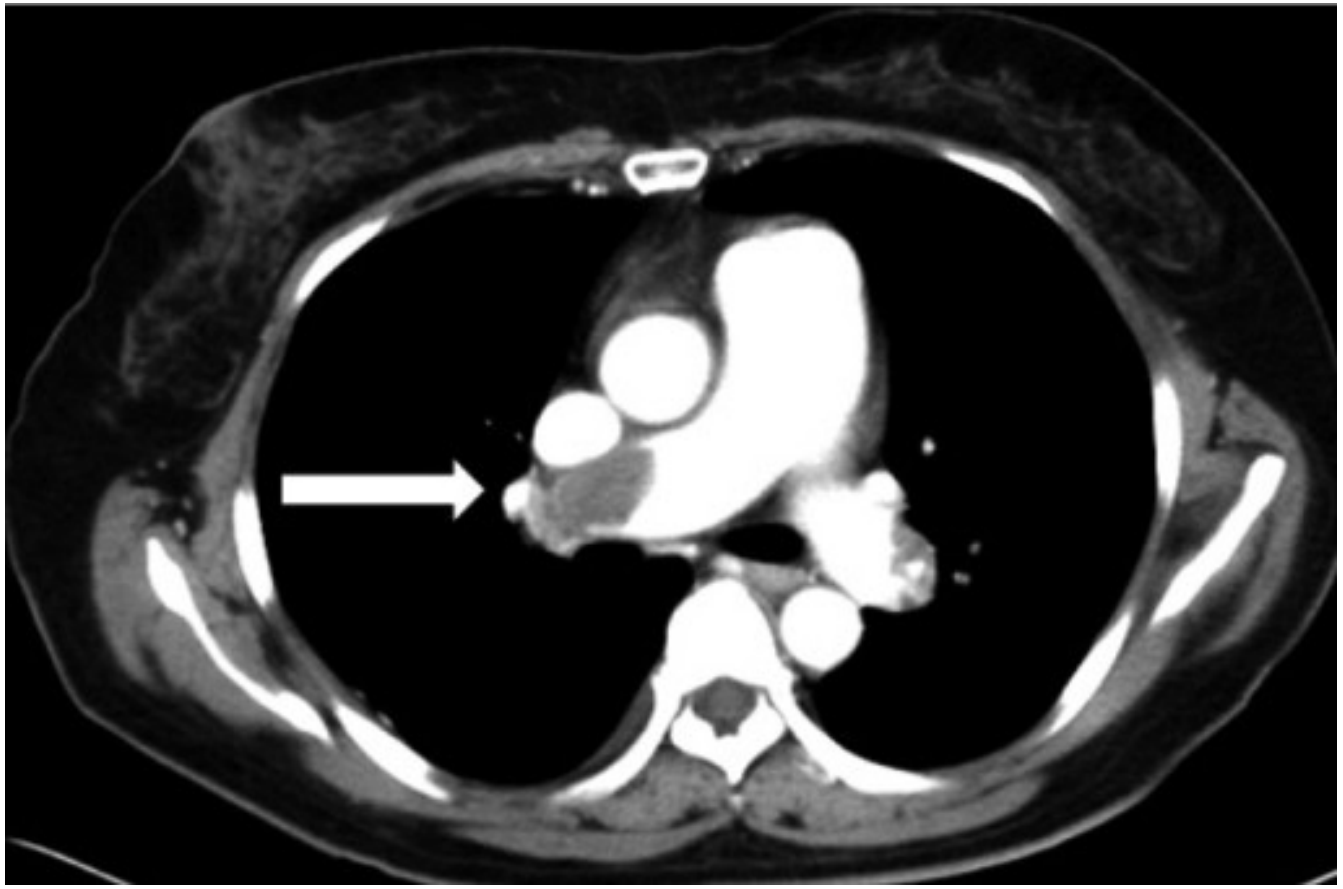
ECHO



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

Managment

- Clinical probability, DD, echo and CT angio

Signs of DVT	3	Clinical probability:
Other dg improbable	1,5	- low 0-1 (3,4%) - moderate 2-6 (20%)
Tachycardia 100	1,5	- high 7 (63%)
Immobilization more than 3 days, surgery within 4 weeks	1,5	- 0-4 PE less probable
DVT, PE in history	1,5	- More than 4 - PE highly probable
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 fibrinolysis[46]

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. ateriovenous 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