

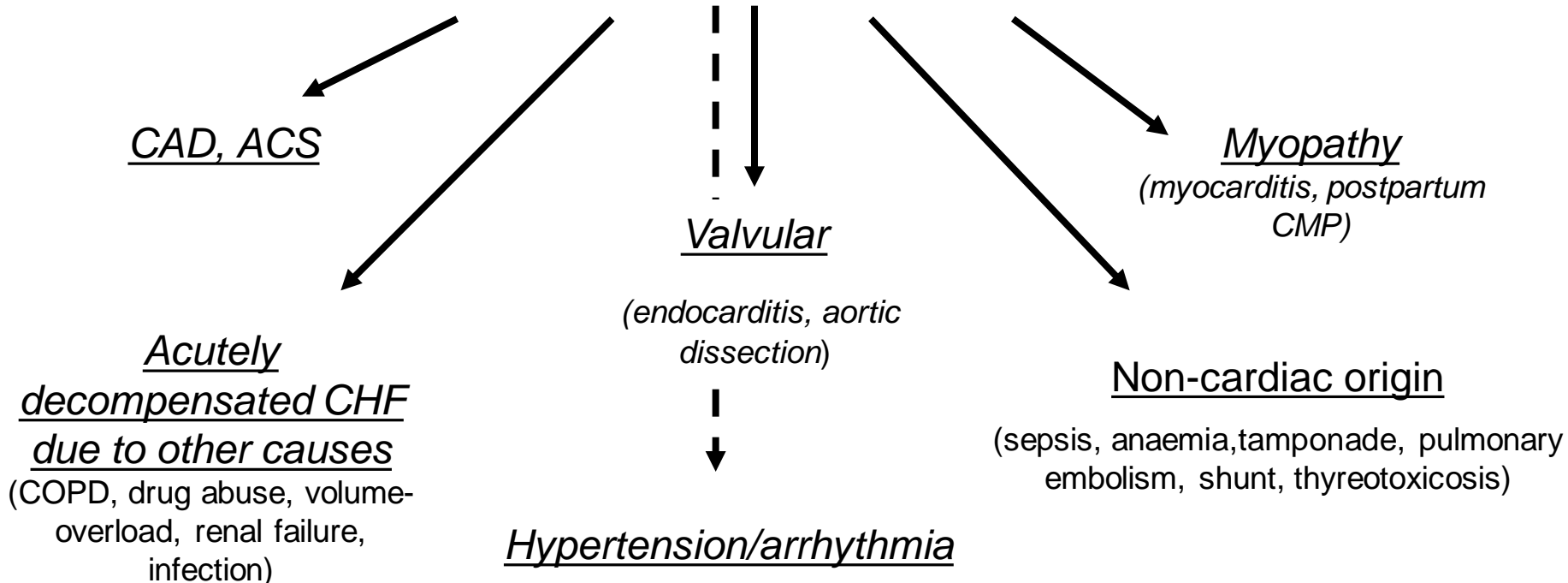
Acute heart failure

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What is acute heart failure (AHF)?

Fast appearance and progression of symptoms of circulatory failure that need immediate interventions

Causes of AHF



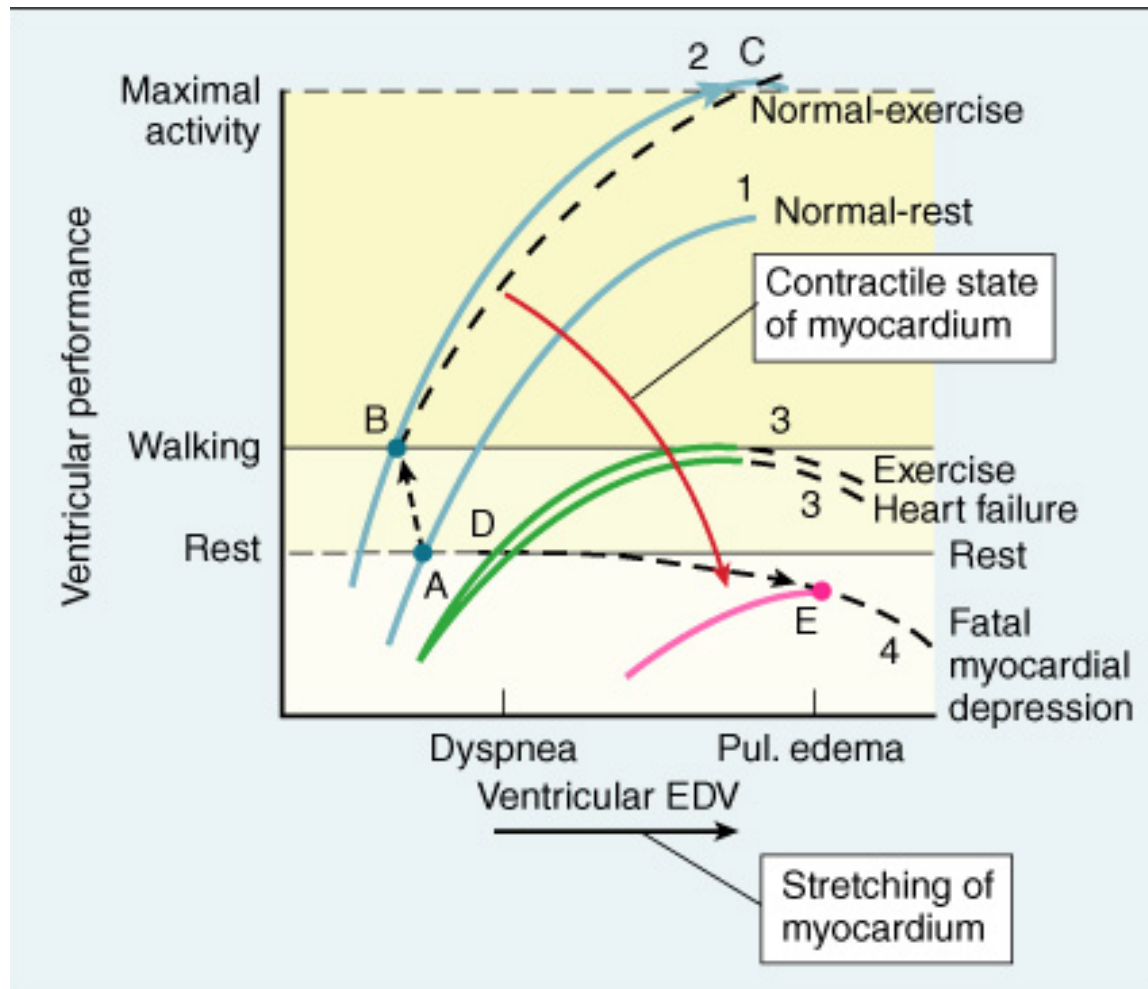
Different pathophysiology, no sharp border (dominant symptoms: clinical classification)

- **Acute decompensated CHF:** left and right heart congestion, pulmonary edema, hypotension
- **Pulmonary edema:** orthopnea, tachypnea, low O₂ saturation
- **Hypertensive attack:** increased afterload, congestion
- **Isolated right heart failure:** elevated jugular venous pressure, clear pulmonary sounds, hepatomegaly, anasarca
- **ACS:** during ischemia contractility worsens +/- arrhythmic events may occur (e.g. bradycardia, VT/VF)
- **Cardiogenic shock:** forward failure (low blood pressure, tissue hypoperfusion), backward failure (congestion), impaired contractility

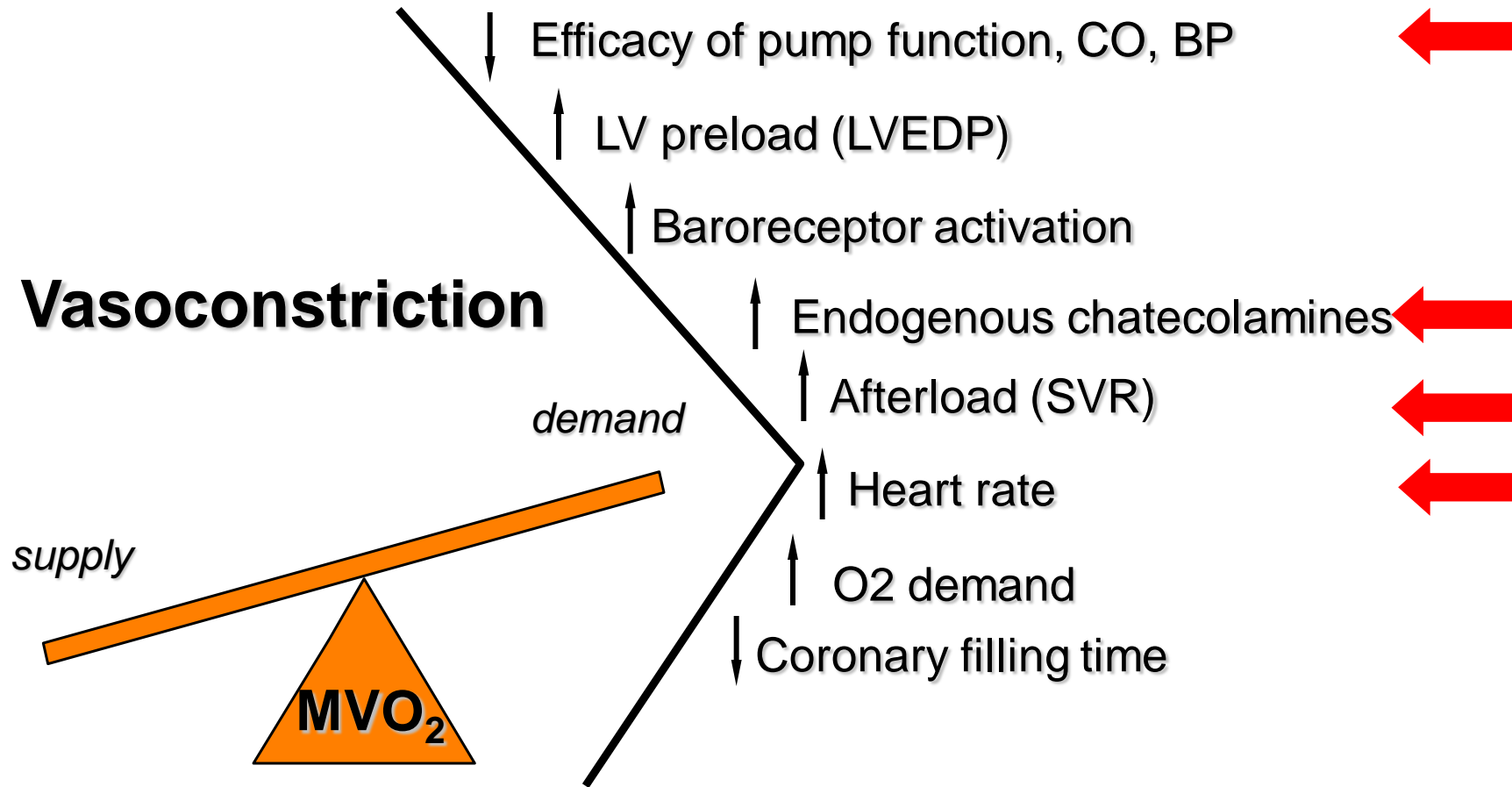
Acute decompensated left heart failure in ACS

- **Most often cause:** stable or unstable coronary artery disease
- Ischemia of left ventricle causes ***both systolic and diastolic dysfunction*** that leads to
 - Decreased cardiac output (CO)
 - Increased filling pressures (eg. pulmonary wedge pressure)
 - Pulmonary edema

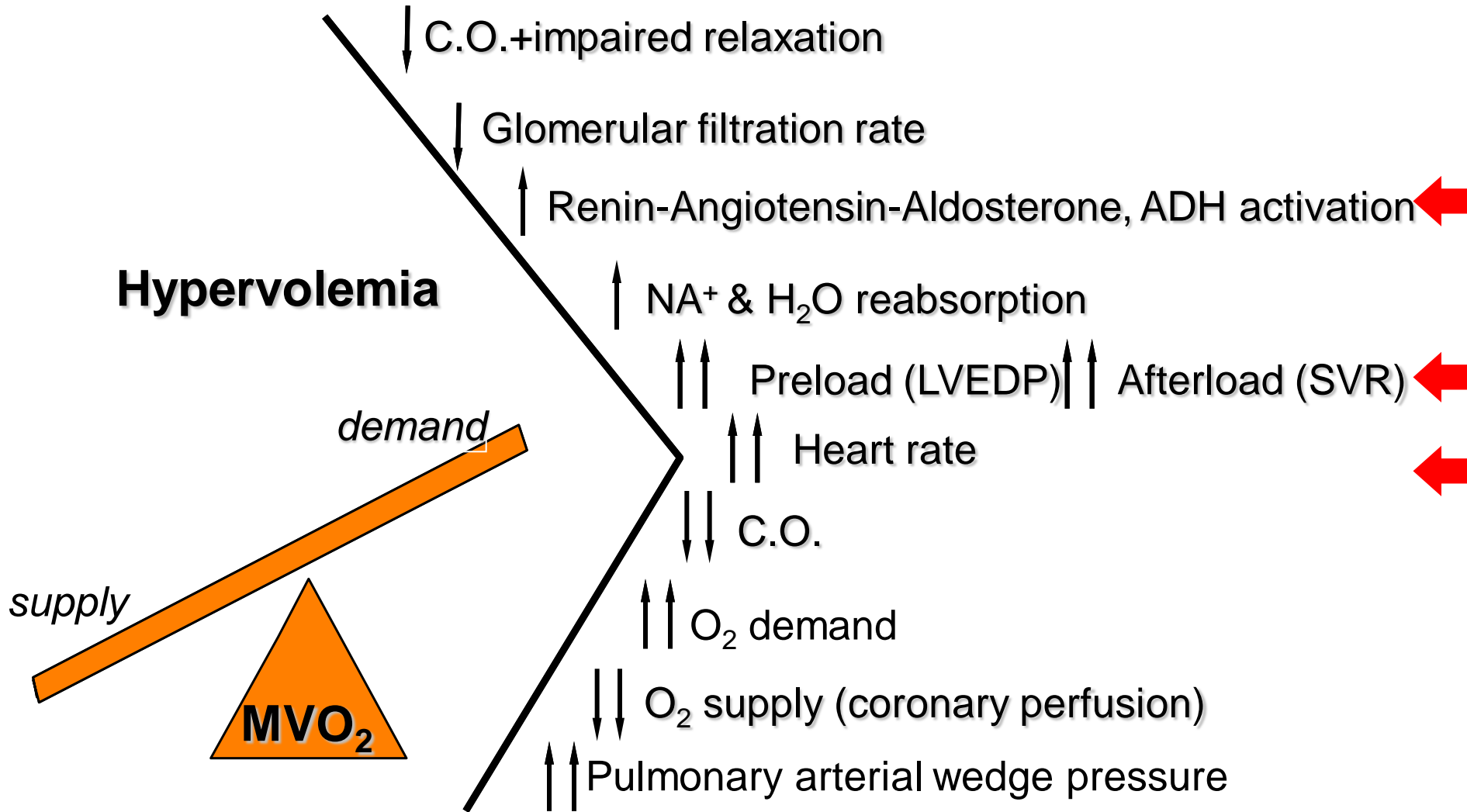
Pathophysiology



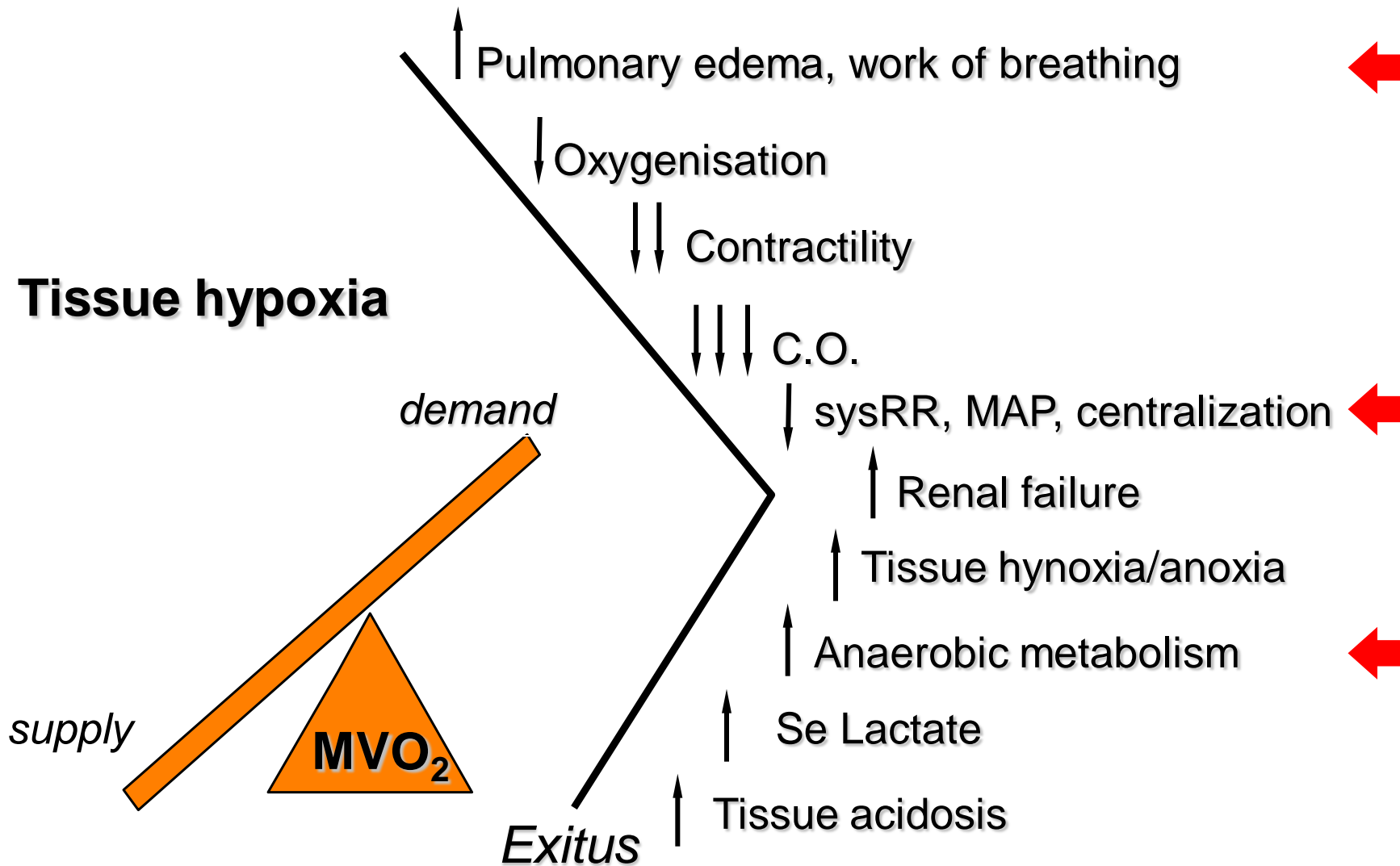
Pathomechanism



Pathomechanism



Pathomechanism



Classification of AHF

Stages of Heart Failure (NYHA)

NYHA class I.:

Symptoms present only at levels of exertion that would limit normal individuals.

NYHA class II.:

Symptoms present on ordinary exertion.

NYHA class III.:

Symptoms present on less-than-ordinary exertion.

NYHA class IV.:

Symptoms present at rest.

Stages of Heart Failure (AHA/ACC)

Stage A:

At high risk for heart failure but *without structural heart disease or symptoms* (hypertension, diabetes, obesity, metabolic syndrome, cardiotoxic drugs)

Stage B:

Structural heart disease but *without symptoms* of heart failure (previous MI, LV remodeling including LVH and low EF, asymptomatic valv. dis.)

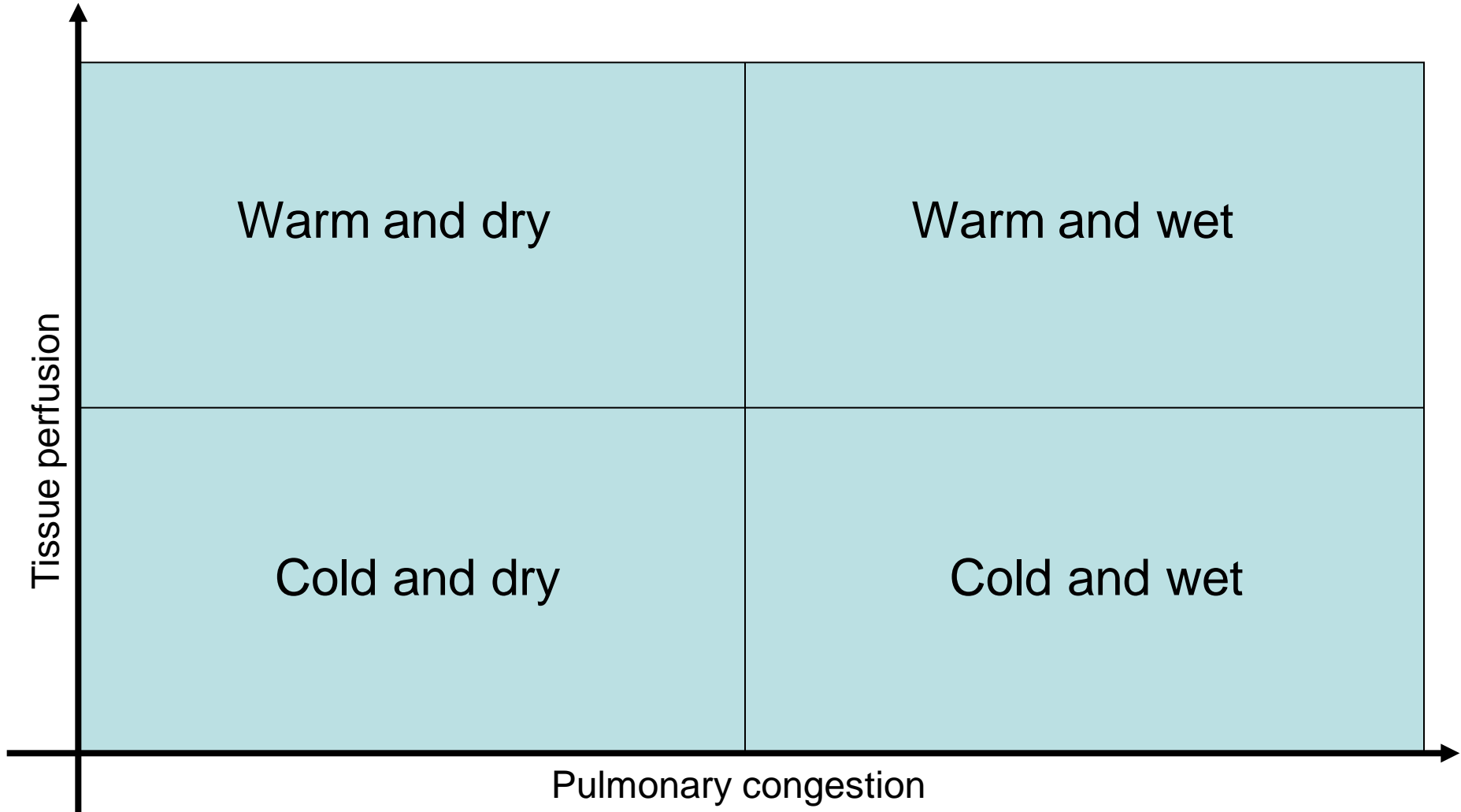
Stage C:

Structural heart disease with prior or current symptoms of heart failure.

Stage D:

Refractory heart failure requiring specialized interventions.

Clinical classification



Killip classification of AHF in acute MI

Killip Classification	% patients	Mortality (%)
I No CHF	30-50	5
II Rales, S3, Pulmonary venous hypertension	33	15-20
III Pulmonary edema	15	40
IV Cardiogenic shock	10	80-100

Forrester classification

Forrester Hemodynamic Classification		Mortality % (Clinical Dx)	Mortality % (Hemodynamic Dx)
I	PCW < 18 CI > 2.2	1	5
II	PCW > 18 CI > 2.2	11	9
III	PCW < 18 CI < 2.2	18	23
IV	PCW > 18 CI < 2.2	60	51

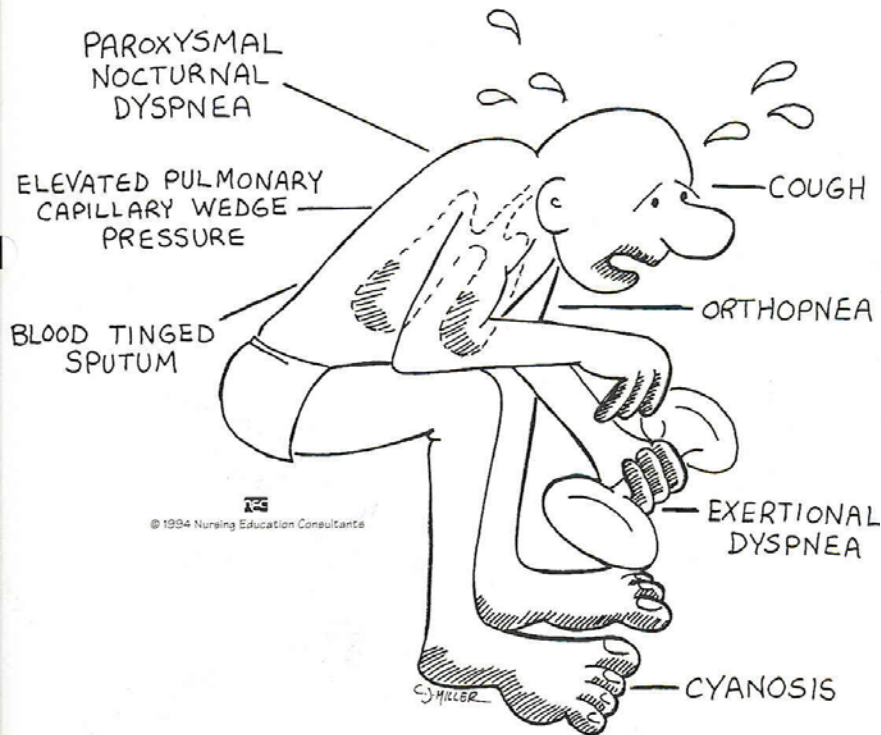
Diagnostic and therapeutic steps of AHF

Primary/emergency treatment of AHF

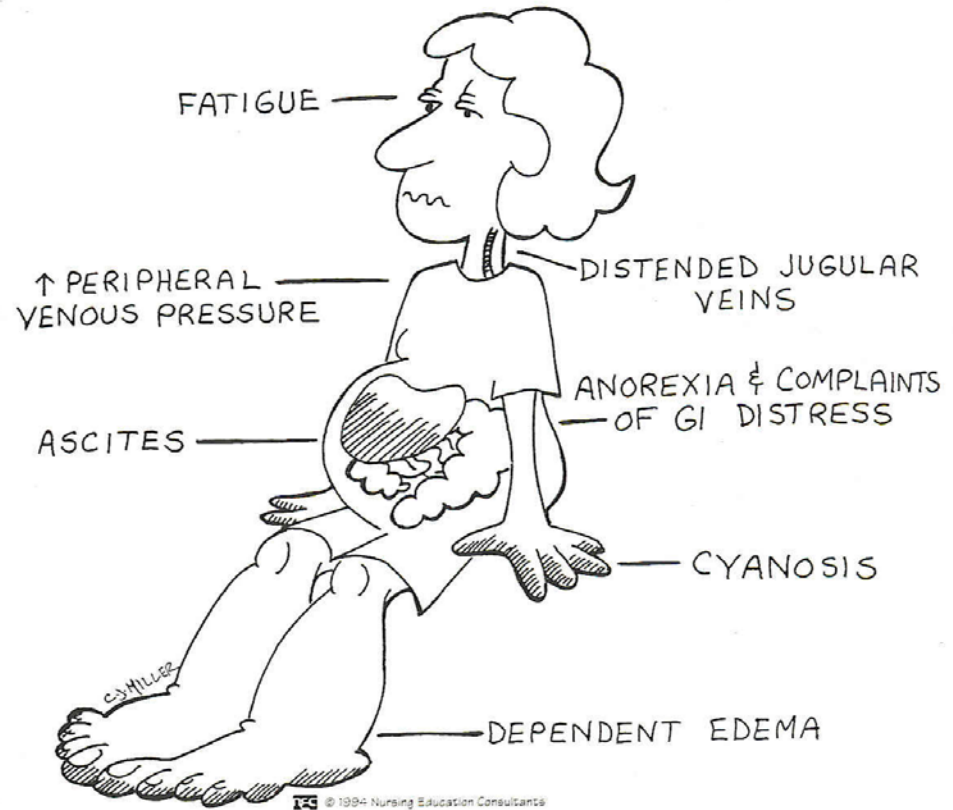
- Restoration of oxygenation and tissue perfusion
- Iv. access
- Monitoring – invasive if needed
- Positioning of the patient: sitting or half- sitting (45°)
- Symptom guided acute Rx
- Etiology evaluation and casual Rx
- Evaluation and control of fluid (volume) state of the patient (Stop of infusion in case of left heart failure)

Clinical signs and symptoms

LEFT SIDED FAILURE



RIGHT SIDED FAILURE



Evaluation: diagnostic options

ECG: ischemia (ST-elevation, Q-wave), arrhythmia, conduction disturbances (eg. LBBB)

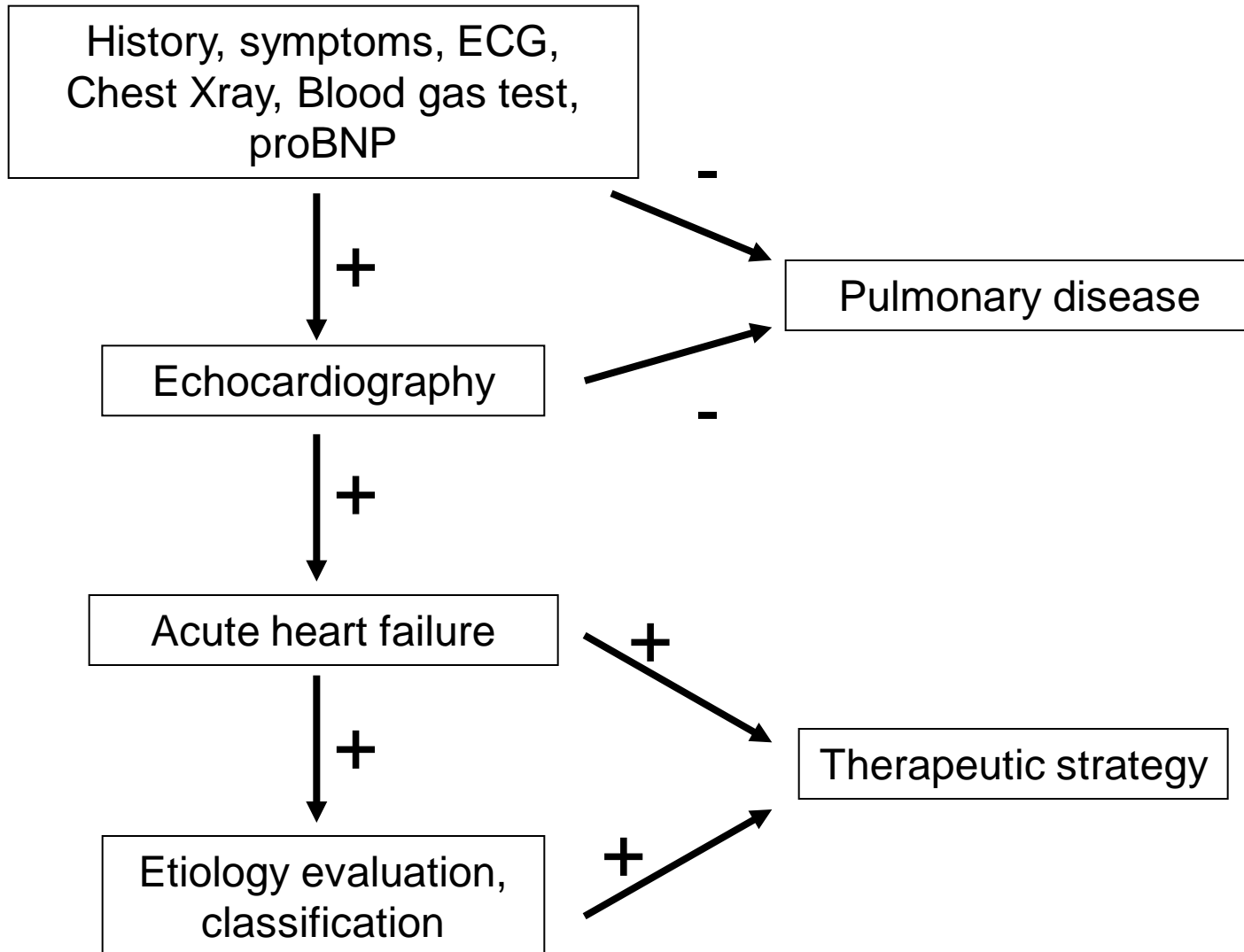
Chest X-ray: pulmonary congestion, pleural, pericardial fluids, cardiomegalia

Lab tests: electrolyte levels, renal&liver function, blood cell count, **ProBNP**

Blood gas analysis: pH, pO₂, PCO₂, BE, Lactate

Echocardiography: evaluation of systolic and diastolic function, dyssynchronia, valvular disease, mechanical complications

Algorithm: evaluation and diagnosis of AHF



Monitoring techniques

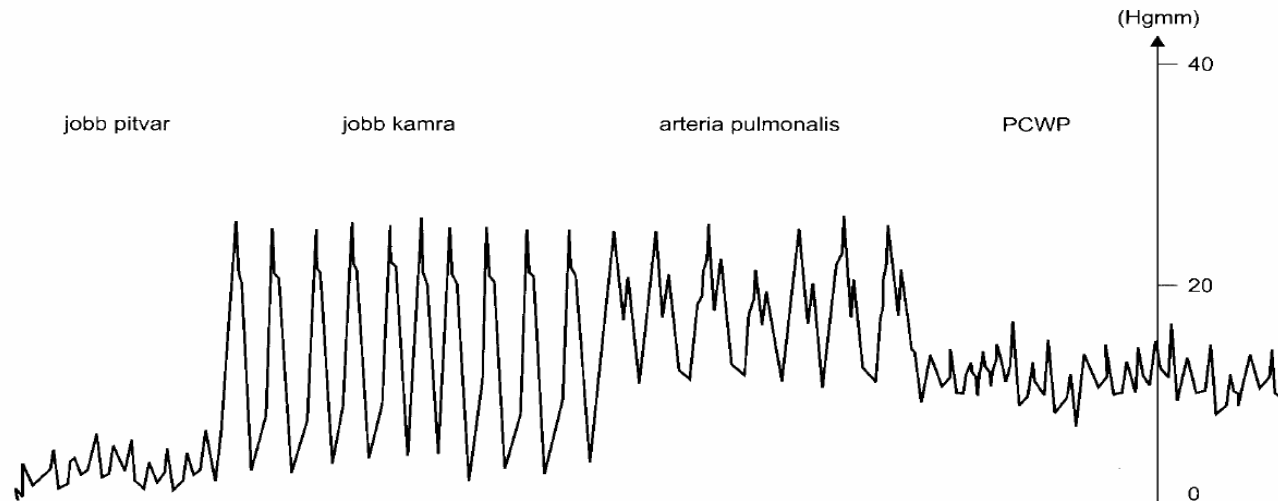
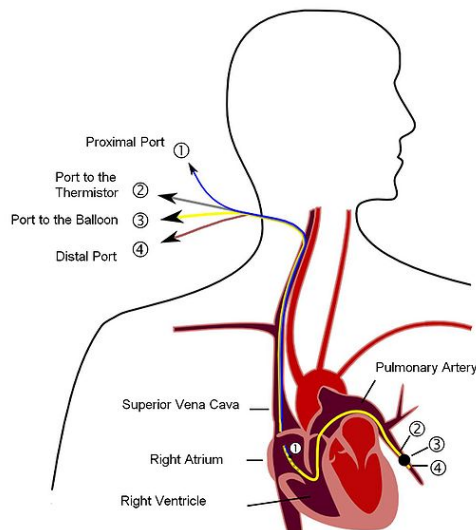
Non-invasive: obligatory at AHF patient (resp. rate, ECG, O2 saturation, NIBP)

Invasive: obligatory at unstable AHF patients (Noninvasive AND arterial line, central venous line – invasive pressures)

PAC (Swan Ganz) right heart catheterisation: unstable AHF and no reaction to conventional treatment

PAC right heart hemodynamics

Pulmonary capillary wedge pressure characterizes the end diastolic filling pressures (except mitral stenosis, aorta regurgitation, pulmonary vein occlusion, MV)



Cardiogenic shock

Cardiogenic shock (CS)

- is the most severe form of acute heart failure
- progressive circulatory insufficiency
- imbalance of oxygen supply-demand (inadequate tissue perfusion and hypoxia compared to oxygen demand)
- inadequate systolic and diastolic function of the heart *with* adequate intravascular volume and left ventricular filling pressures

Diagnosis of cardiogenic shock (international standards)

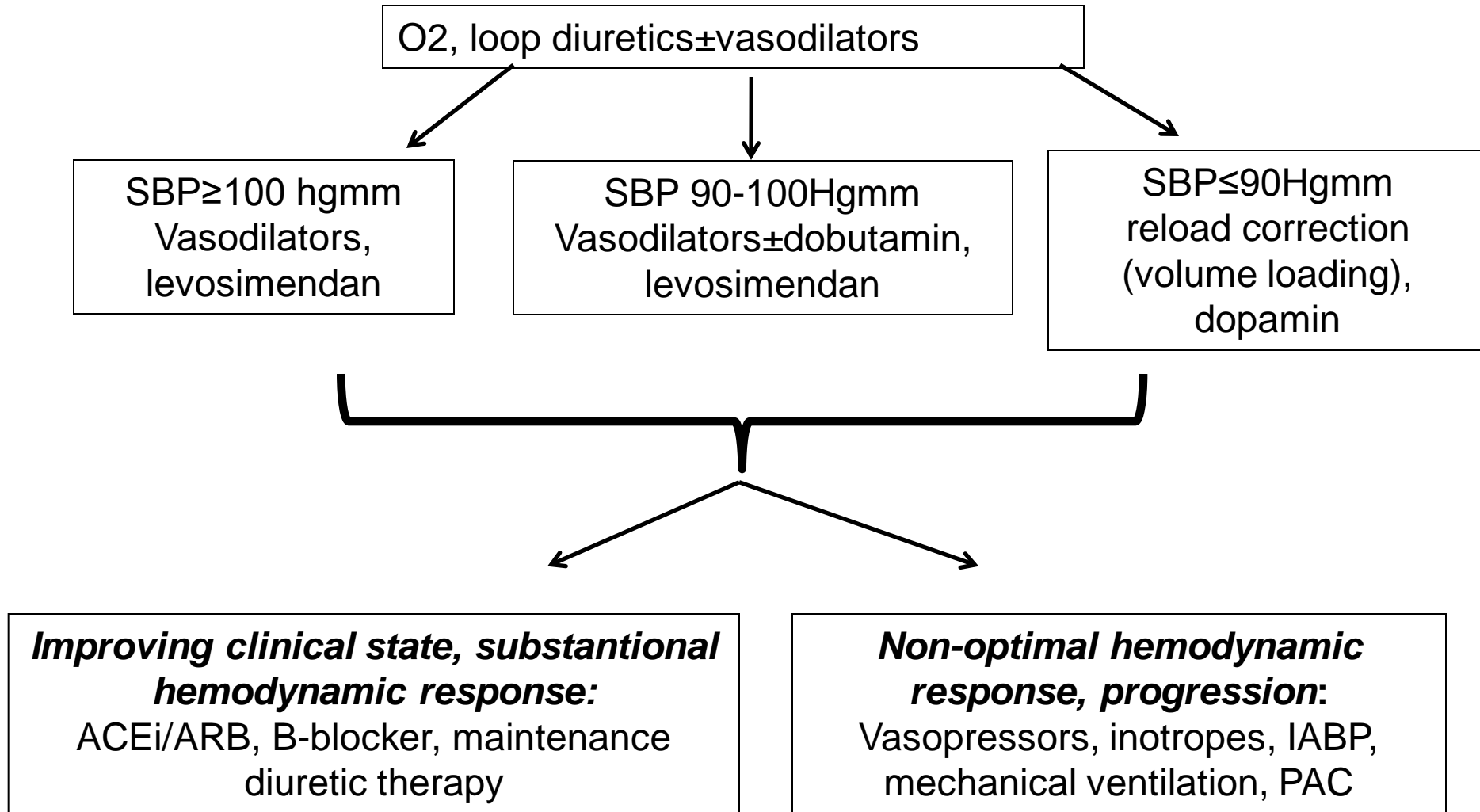
- Systolic blood pressure <90 Hgmm, or a decrease of >30 Hgmm in hypertensive patients for more than 30 minutes without intraaortic balloon pump treatment (IABP) and iv. catecholamin therapy
- reduced cardiac index (<2.2 L/min/m²)
- PCWP >18 Hgmm
- Heart rate >100 bpm
- Centralized circulation, cold extremities, prolonged capillary filling time, cyanosis (peripheral hypoxia)
- oligo-anuria (<30 ml/h)
- cerebral hypoperfusion: central nervous system impairment, altered mental state, coma
- signs usually persist after attempts to correct hypovolemia, arrhythmia, hypoxia, and acidosis.

Shock-differential diagnosis - PAC

Type	Heart rate	Stroke volume	CVP	PCWP	CO/CI	SVR
Hypovolemic	↑	↓	↓	↓	↓	↑
Distributive						
Spinal Shock	↑	n	↓	↓	↑	↓
Anaphylaxis	↑	n	↓	↓	↑	↓
Sepsis	↑	↓	↓	↓	↑	↓
Cardiogenic						
Conduction problem	↓	↑	↑	↑	↓	
Pump failure	↑	↓	low	Might be low	↓	↑
Volume load	↑	↓	↑	↑	↓	↑
Inflow obstruction	↑	↓	↑		↓	↑
Outflow obstruction	↑	↓	↑	↑	↓	↑

Therapy of AHF

Therapeutic algorithm based on systolic blood pressure (SBP)



Rx of acute decompensated HF

O2

- target O2 Sat: 95% (COPD:90%)
- aim: decrease of pulmonary vasoconstriction

Morphin

- aim: anxietas, dyspnea, chest pain relief
- dosage: initiating 2-4mg iv. , maitenance 2 mg in 5-15 mins (Massive dyspnea and fear takes over 40% of CO!)
- AE: antiemetic drug needed (eg. 1mg metoclopramid)
- monitoring is a must (resp. And heart rate, NIBP)

Loop diuretics (furosemid)

- effect reduced in acidosis, hyponatraemia
- CAVE: hypokalaemia!
- potentiating effect of ACEi/ARBs, hypotension!

Rx of acute decompensated HF

Vasodilators

if SBP > 90Hgmm

Nitroglycerine, isosorbide-dinitrate, nitroprussid-Na

Decreases hypertension (afterload)

Venodilator effect: pulmonary congestio decreases

Maximal dose: 8mg/h

Target BP 100-120 Hgmm

CAVE: significant aortic valvular stenosis! (i.v. hypotensio)

Ca-channel blockers are CONTRAINDICATED in AHF!

Beta-receptor blockers, ACEi, ARB

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CONTRAINDICATED in AHF!

Inotropes / Dopamin:

2-5 micg/kg/min: DA-R: renal perfusion, diuresis increase

5-10micg/kg/min: adjuvant B1-R.: + Inotrop, contractility increases

10+ micg/kg/min: adjuvant Alfa-1-R: vasoconstiction, SBP increases

Note: risk for arrhythmias

Rx of acute decompensated HF

Inotropes/ Dobutamin: dose dependent B1-R effect: +inotropic, +chronotropic
Note. Monitoring needed, risk for arrhythmia!

Phosphodiesterase inhibitors (PDEi)-milrinon, enoximone

i.c. cAMP increase: +Inotropic, peripheral vasodilation
increase in CO
decrease in SVR

Vasopressin antagonists (tolvaptan, conivaptan- not in Hungary)

renal „aqua channels,, outloading inhibition>>water reabsorption inhib.

Acute symptom relief, but 1 year mortality is not decreased
(EVEREST trial)

Rx of acute decompensated HF

Levosimendan

Ca-sensitiser (Troponin C), inodilator!

Cardiac contractility ↑ (stroke volume ↑) CO ↑,

Systemic and pulmonary vasc. resistance ↓,

Hemodynamic response in several days

Monitoring is necessary (hypotension, arrhythmia)

Vasopressor/ Norepinephrine: Not first choice of Rx!

Indication:

1., cardiogenic shock (when ineffective combination of inotrop agents and fluid challenge to restore 90Hgmm SBP)

2., sepsis and AHF

Vasopressors/Inotropes

DRUG	RECEPTOR	DOSE	ACTION
Dobutamine	Beta-1 >> Beta-2	2 – 10 mcg/kg/min	Inotrope
Norepinephrine	Alpha-1 >Beta-1 >> Beta-2	0.5 - 20 mcg/min; Max 30 mcg/min.	Increases SVR, +/- impact of CO
Dopamine	Dopaminergic Dopa + Beta-1 Beta-1 Alpha-1 , some Beta-1	1-2 mcg/kg/min 2-5 mcg/kg/min 5-10 mcg/kg/min > 10 mcg/kg/min	Renal + mesenVD/VC Above, inotrope Inotrope Vasoconstriction, inotrope
Epinephrine	Alpha-1 = Alpha-2 Beta-1 = Beta-2	1-2 mcg/min 2-10 mcg/min >10 mcg/min	Beta → incr. HR/SV Beta > alpha Alpha → vasoconstriction
Phenylephrine	Alpha-1 > Alpha-2 >>>>> Beta	2 – 200 mcg/min	Increases SVR
Vasopressin		0.03 units/minute	Vasoconstriction

Which drug Rx for which type of AHF?

Summary

Stable decompensated CHF

vasodilators, loop diuretics, hypoperfusion: positive inotropes

Pulmonary edema

morphin, loop diuretics, hypertension: vasodilators,
hypoperfusion: inotropes

Cardiogenic shock

fluid challenge, inotropes, vasopressors, intubation, mechanical ventilation, IABP

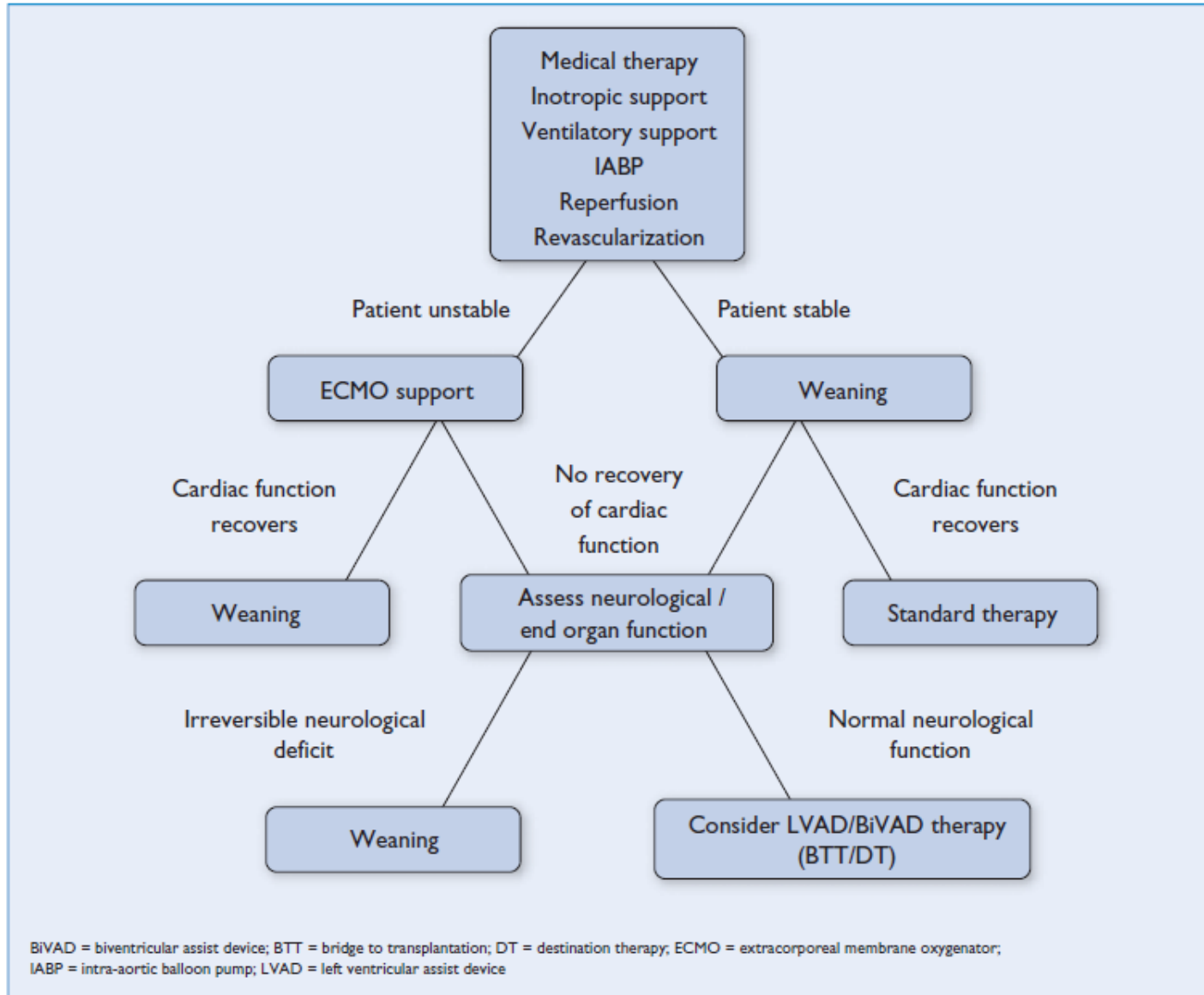
Isolated right heart failure

fluids, inotropes, rapid evaluation of etiology (pulmonary Embolism? AMI?)

Acute coronary syndrome

revascularisation (PCI, CABG, ...thrombolysis)

Device Rx algorithm of AHF ESC revascularisation guidelines 2010



Device Rx of AHF

Temporary mechanical circulatory support or acute „bridge” therapy even until heart transplantation

- **IABP**
- impella Recover
- extracorporeal membrane oxigenisation,
- continuous aortic-flow-augmentation Cancion-system
- TandemHeart
- **left-, right- or. biventricular assist device (VAD)**

Temporary pacemaker therapy

- antibradycardia
- antitachycardia
- resynchronisation

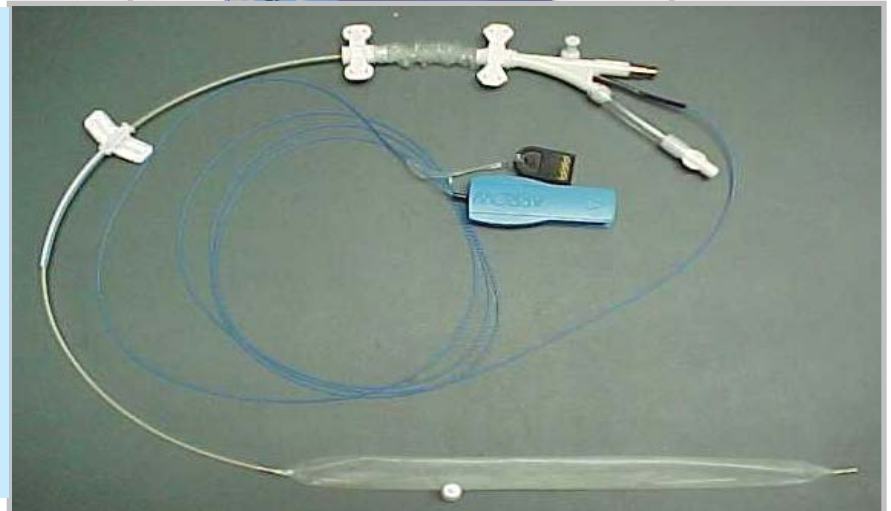
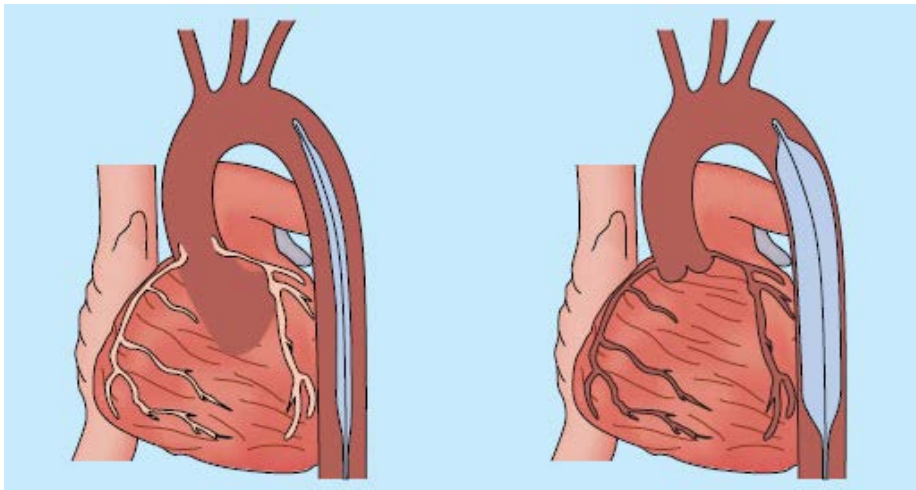
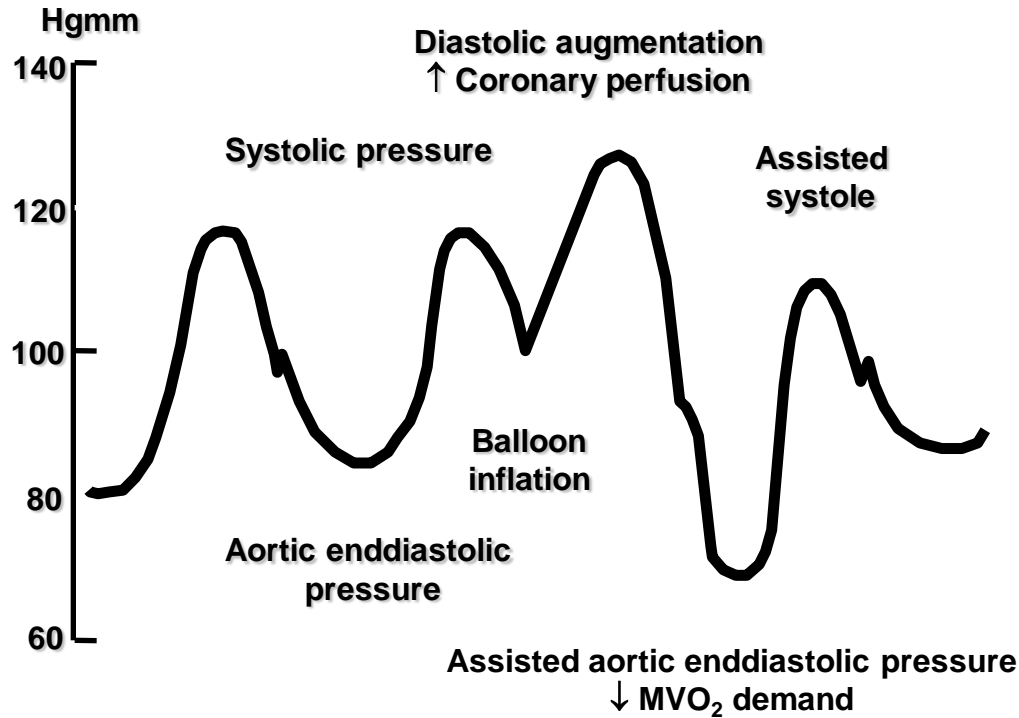
Mechanical ventilation – recovery of gas exchange, decrease work of breathing

- **invasive mechanical ventilation**
- **noninvasive respiratory support (CPAP, BiPAP)**
- PEEP 5-10 Hgmm (against edema)
- Pozitiv pressure ventilation (pre and afterload decrease)
- Overtake breathing work
- **Sedation**: analgesia, hipnotic effect, amnesia

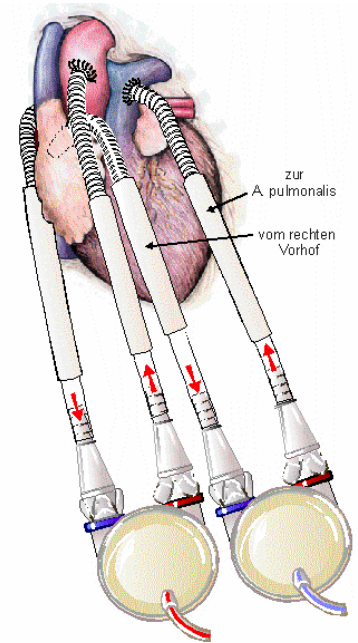
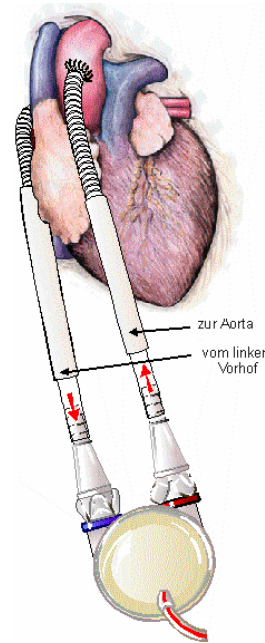
Renal replacement therapy: IHD / CVVH(DF)

Liver replacement therapy: MARS – not routinely used in Hungary

Intraaortic balloon counterpulsation, IABP



LVAD / BiVAD – Assist Device



Thank you for your attention!

