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UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE "CAROL DAVILA" BUCUREŞTI

AD-COR Program inovativ de formare in domeniul cardiologiei pediatrice
POSDRU/179/3.2/S/152012

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MODUL TEORETIC

PAEDIATRIC CARDIAC CRITICAL CARE NURSING

Imputernicit: Prof. Dr. Tammam Youssef

Activitate prestata de I.R.C.C.S. POLICLINICO SAN DONATO – MILANO, ITALIA in baza contractului nr. 18/22144/29.07.2015

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Beneficiar: Universitatea de Medicină și Farmacie „Carol Davila” București

PAEDIATRIC CARDIAC CRITICAL CARE NURSING

Bucharest, September 24, 2015

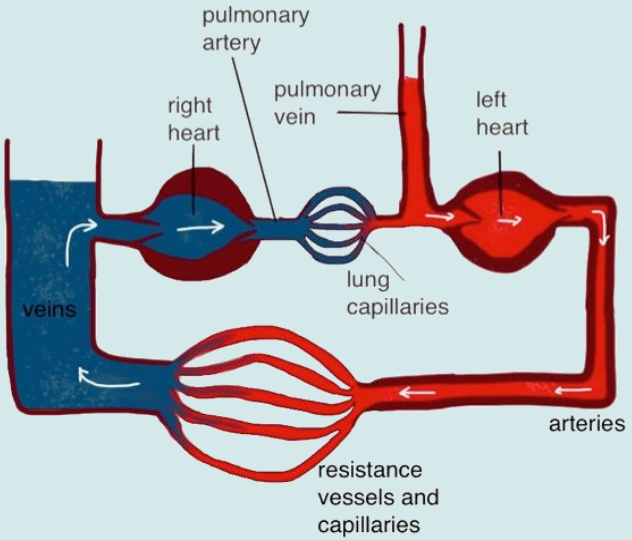
Welcome

14 lectures
during the next 4 months

1. **The importance of cardiac output and cardiovascular medications**
2. Human factors and crisis management in paediatric cardiac critical care
3. Receiving patient from OT: assessment, preparation and handing-over process
4. Invasive and non-invasive methods of monitoring in the PCICU
5. Respiratory assessment and management; respiratory complications
6. Strategies in sedation and pain management
7. Fluid management and treatment of metabolic disorders

Now let‘s start with the todays‘s topic

The importance of cardiac output and cardiovascular medications

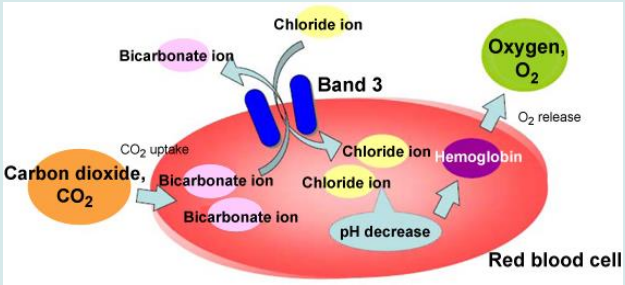


Cardiovascular system

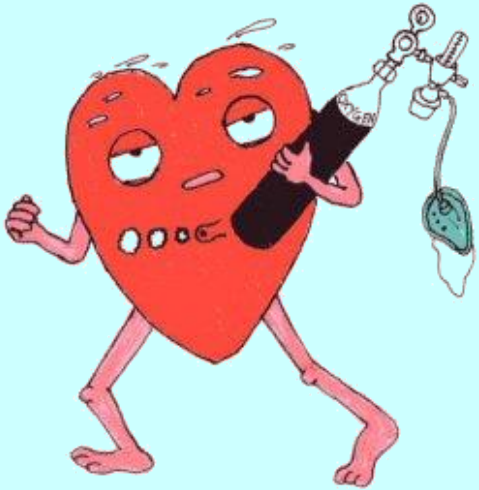
Important systems in postoperative paediatric cardiac management:

- **Cardiovascular**
- Pulmonary
- Renal
- CNS
- Pain Control
- Nutrition

The cardiovascular system is responsible for a continuous supply of oxygenated blood to every cell in the body (= **perfusion**).



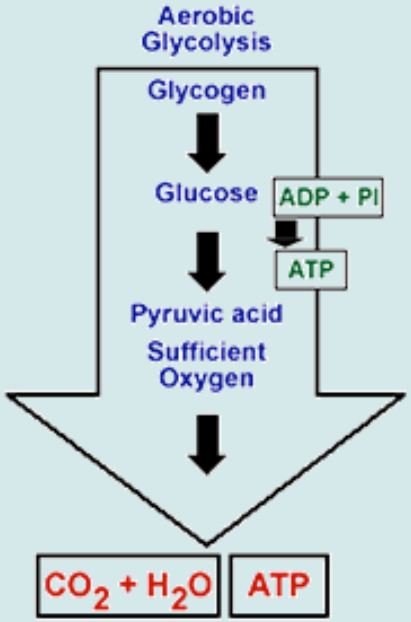
Basic understanding



An **understanding** of **oxygen delivery and demand** is central for the management of patients during critical illness.

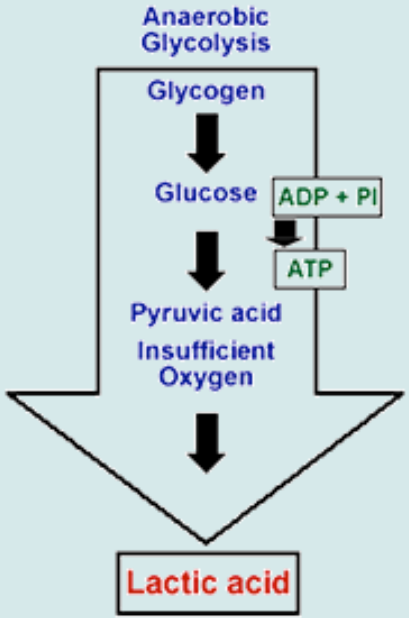
Why is oxygen so important

- Glycolysis is an **oxygen independent** metabolic pathway, that converts glucose into pyruvate under producing ATP.
- ATP transports chemical **energy within cells** for the entire metabolism.



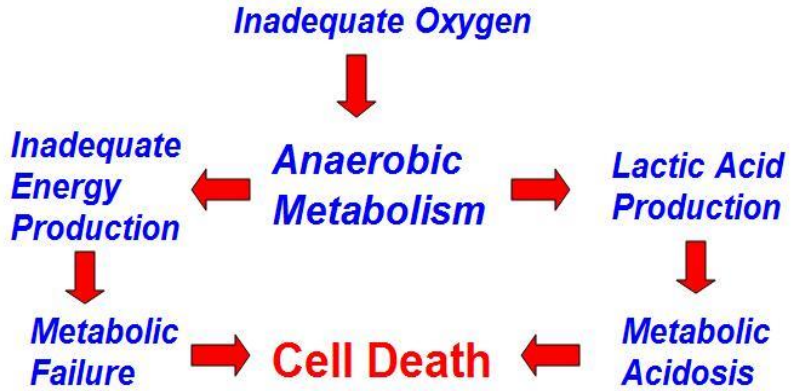
Why is oxygen so important

- When sufficient oxygen is not present for further oxidation pyruvate is converted to **lactic acid** by the enzyme lactate dehydrogenase as the **end point of anaerobic metabolism.**

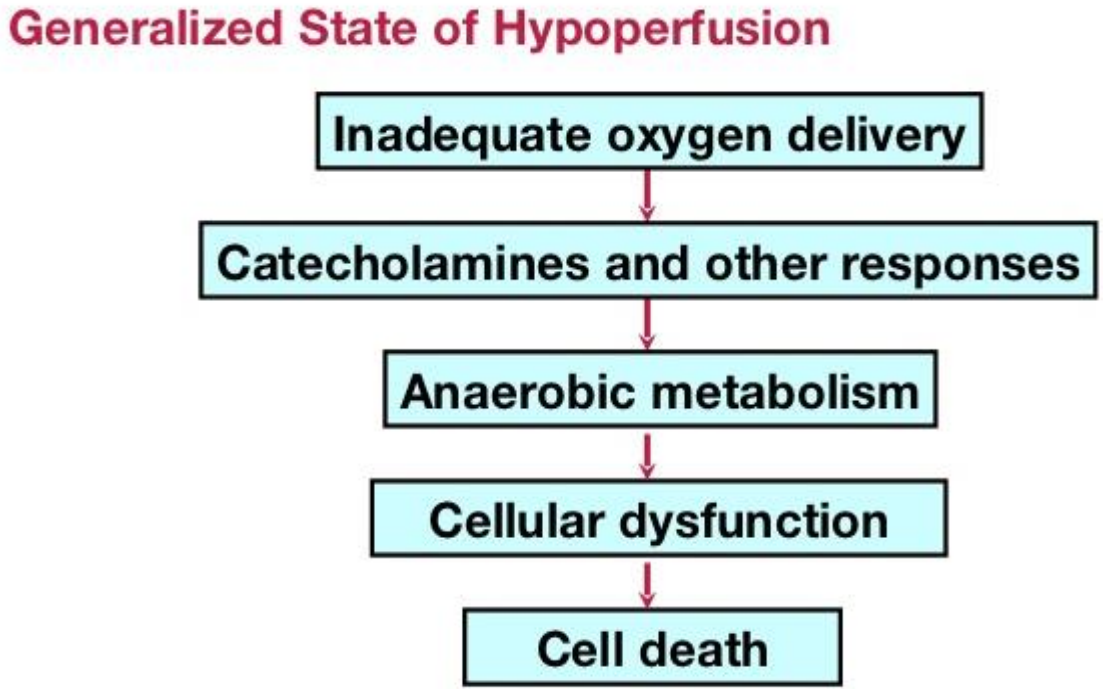


Why is oxygen so important

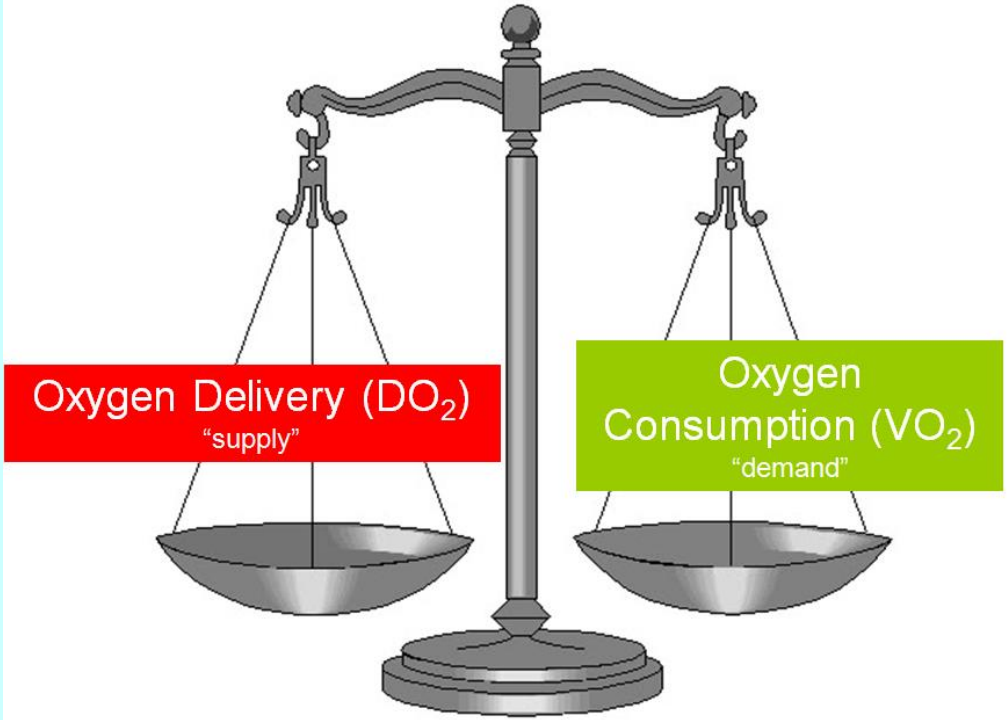
- An **imbalance** between tissue oxygen demand and delivery appears with the development of **cellular hypoxia** and leads lastly to **cell death**.



Why is perfusion so important



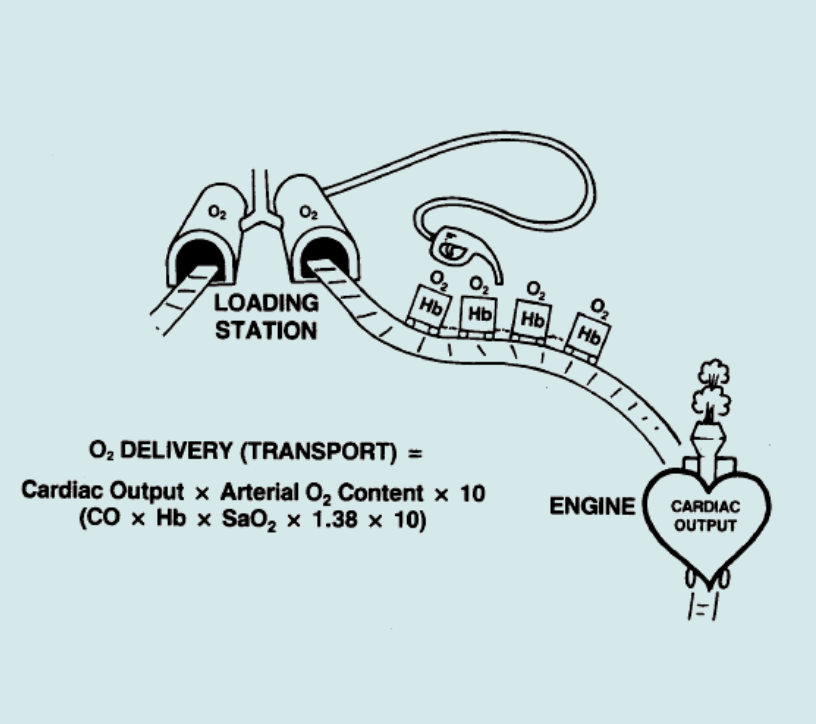
Oxygen Supply and Demand Balance



Oxygen Delivery

Amount of oxygen delivered (DO_2) to the body is the product of **systemic blood flow (Q)** and **oxygen content** (CaO_2) of systemic arterial blood:

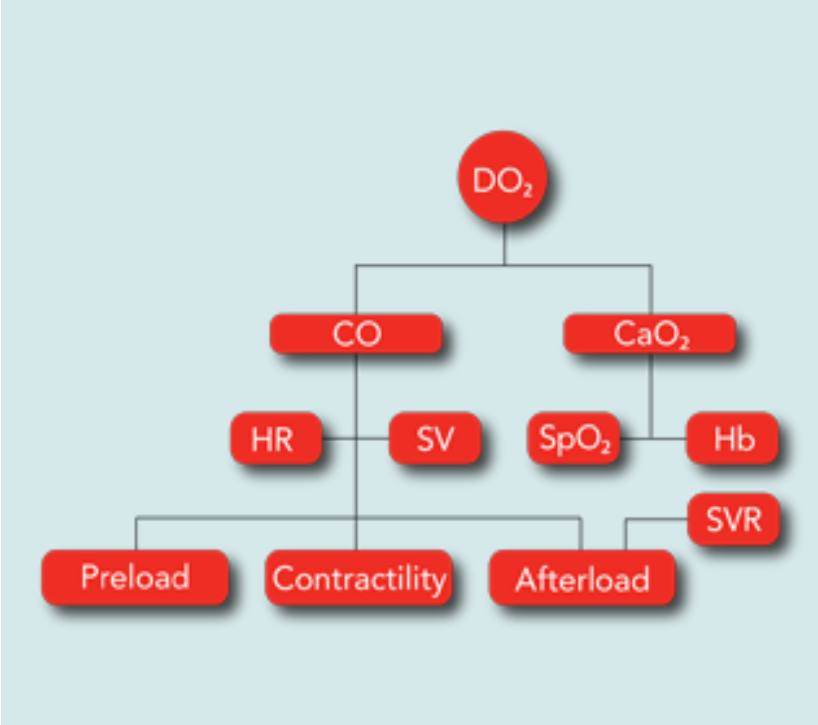
$DO_2 = Q \times CaO_2$ (Q = flow = cardiac output)



Poor Oxygen Delivery

There are **3 reasons for poor O_2 delivery**:

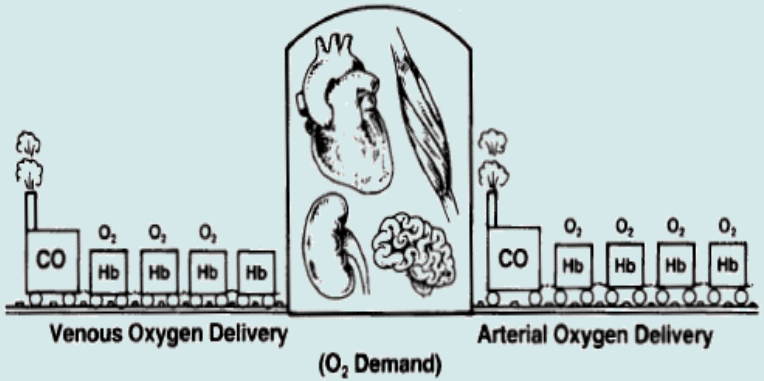
- Anoxic anoxemia - (**low SaO_2**)
- Anemic anoxemia - (**low Hgb**)
- Stagnant anoxemia - (**low CO**)



Oxygen Demand

Oxygen demand determined by metabolic activity:

- **High O_2 demand**:
  Heat production (maintain temperature)
  fever
  physical activity
- **Moderate O_2 demand**:
  Cardiac contraction
  normal respiration
- **Low O_2 demand**:
  Basic cellular function (ionic transport, electrical activity)



Oxygen Extraction

Oxygen extraction is the proportion of oxygen delivered that is consumed:

- **Normally about 30%**
- Oxygen extraction can be increased to ~**70% under** conditions of **stress**

O_2 Extraction Ratio (O_2 ER)

$$O_2 \text{ ER} = \frac{O_2 \text{ consumption (demand)}}{O_2 \text{ delivery (supply)}} = \frac{\dot{V}O_2}{\dot{D}O_2} \times 100 = \frac{Ca\text{-}\bar{v}O_2}{CaO_2}$$

$$\frac{([Hb] \times 1.34 \times Sao_2) + (Pao_2 \times 0.003) = Cao_2 \text{ (mL/dL)}}{98\% \text{ Bound} + 2\% \text{ Dissolved} = \% \text{ Oxygen content}}$$

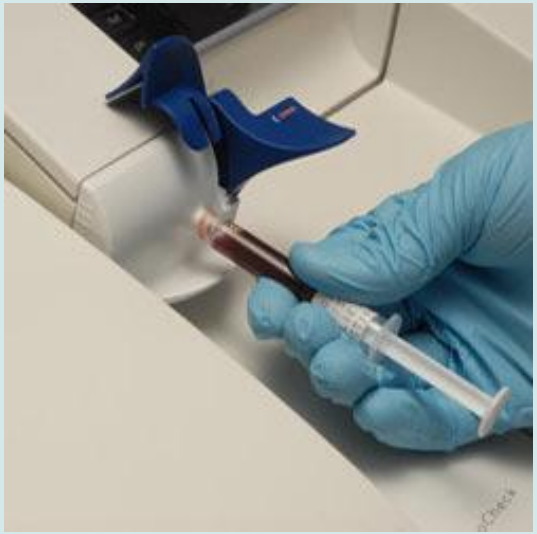
Low Cardiac Output

Low cardiac output syndrome (LCOS) is a clinical condition that is caused by a transient decrease in systemic perfusion secondary to myocardial dysfunction. The outcome is an **imbalance between oxygen delivery and oxygen consumption** at the cellular level which **leads to metabolic acidosis**.

Supply / demand imbalance

Ways to measure Oxygen supply / demand imbalance:

- Mixed venous saturation **SVO_2**
- **Lactate**



Mixed Venous Saturation

- Ideally measured where venous blood is completely mixed (ie. pulmonary artery)
- Reflects **balance of O_2 extraction by tissues and O_2 delivery**

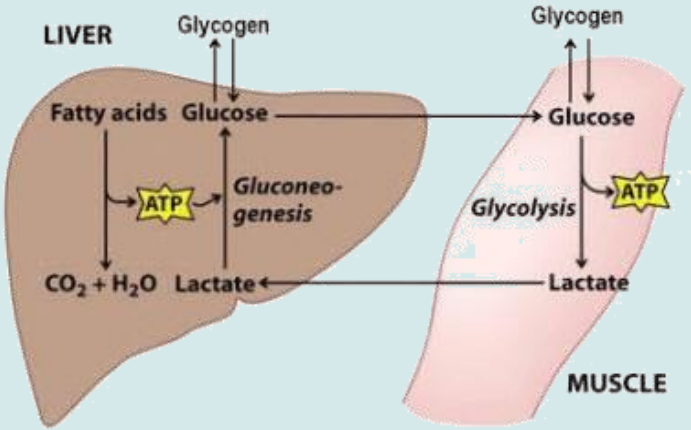
⇓

- SvO_2 ↓reflects O_2 extraction ↑or inadequate O_2 delivery or both

BLOOD GASES	
pH (a)	
pCO2 (a)	
pO2 (a)	
PH (v)	7.37
pCO2 (v)	50.4 H
pO2 (v)	38.2
HCO3	28.1 H
tCO2	29.6 H
Base Excess (vt)	3.1 H
Base Deficit (vt)	
tHB	14.0
Hct (Est)	43
ctO2 (a)	
O2-Saturation (a)	
ctO2 (v)	12.2
O2 Saturation (v)	61.9 L
Methemoglobin	1.0
CO-Hemoglobin	0.9

Lactate

- Product of anaerobic mitochondrial metabolism which occurs under conditions of **inadequate oxygen delivery**
- Can be measured by any blood sample
- Cleared by liver so level can be affected by liver failure
- Lactate can also be elevated by increase in pyruvate production or inhibition of pyruvate metabolism
- Serial measurements can determine lactate clearance which **correlates with mortality**



Supply / demand imbalance

When there is an imbalance you can:

- **Increase** oxygen **delivery**

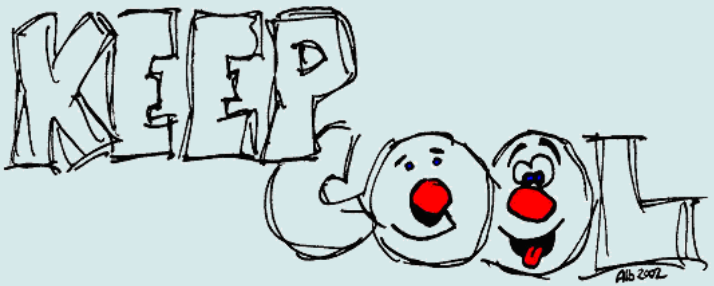
  and / or

- **Decrease** oxygen **demand**

↓ Oxygen Demand

Possible ways to ↓ oxygen demand:

- Regulating temperature – “**keep cool”**
- Decrease activity - **sedation** and/or paralysis
- Decrease work of breathing—pressure support or intubation with **mechanical ventilation**
- Hold enteral feeds



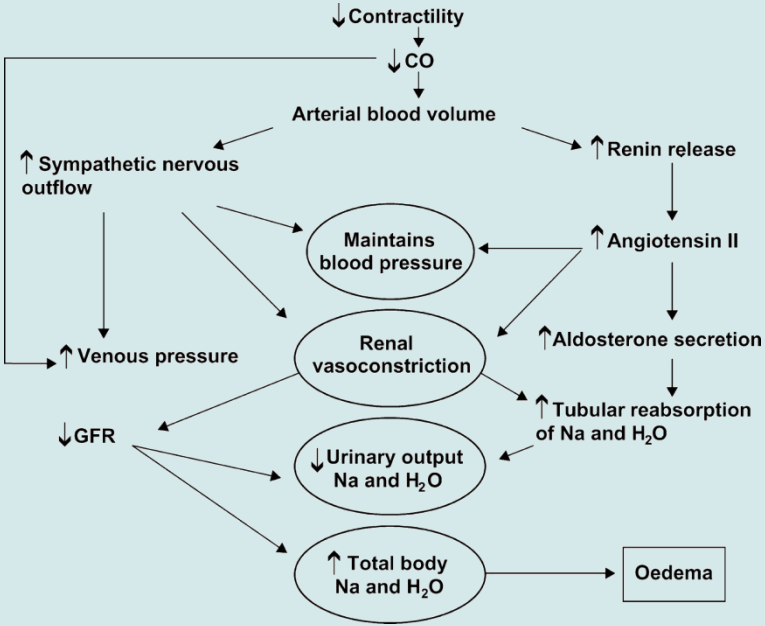
↑ Oxygen Delivery

Possible ways to ↑ oxygen delivery:

- Increase **Heart Rate** or **Stroke volume** => ↑ Cardiac Output
- Provide oxygen or establish airway to **improve saturation**
- Transfuse blood to **increase Hemoglobin**

Low Cardiac Output in paediatric cardiac surgery

- Low cardiac output syndrome (LCOS) is the most important **cause of morbidity and mortality** in the **early postoperative phase**.
- It is seen in **up to 25 %** of all patients undergoing pediatric cardiac surgery
- It is also occurring secondary to acute myocarditis and septic shock



Low Cardiac Output in paediatric cardiac surgery

- It occurs **typically 6 - 18 h after cardiopulmonary bypass**, which is usually **in the middle of the night**
- The resulting effects are

  shock and inadequate organ perfusion

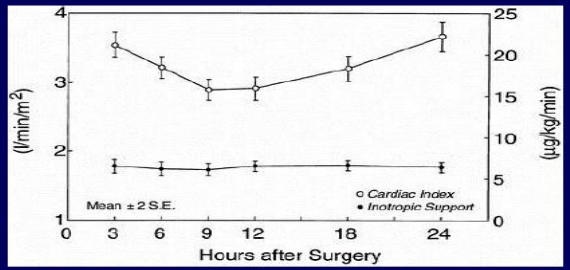
  ⇓

  organ dysfunction ⇓

  multi organ failure ⇓

  death

- Wernovsky et al reported that 25% of neonate with DTGA who underwent ASO had a decline in CI to <2L/Min/M2



Wessel, DL. Crit Care Med 2001;29(10):S220-S230

Low Cardiac Output in paediatric cardiac surgery

Etiology of Low Cardiac Output Syndrome following congenital heart surgery

- Inadequate myocardial protection
- Myocardial ischemia during aortic cross clamping and cardioplegia
- Reperfusion injuries
- Hypothermia
- Ventriculotomy
- **Post - bypass inflammatory injury:**
  systolic contractile dysfunction
  diastolic dysfunction (altered preload)
  altered vascular reactivity (altered afterload)

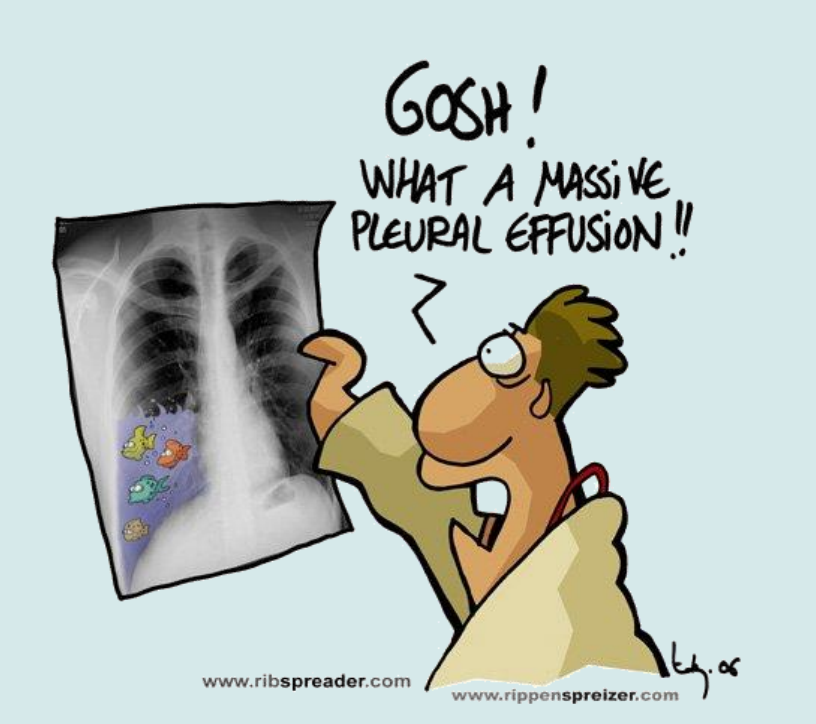
Low Cardiac Output in paediatric cardiac surgery

Etiology of Low Cardiac Output Syndrome following congenital heart surgery

- Loss of AV synchrony
- Residual cardiac lesion
- Post-op. bleeding
- Pulmonary hypertension
- **Stress by pain and / or fear**
- Endocrine derangement (Cortisol, Thyroid, Vasopressin)

Clinical presentation

- **Tachycardia** / Dysrhythmia
- Hypotonia, small amplitude of blood pressure
- **Mottled skin and delayed capillary refill**
- Centralization
- **Low urine output**
- Pulmonary edema
- Pericardial effusion
- Hepatomegaly
- Ascitis
- Generalized edema
- Multiorgan failure



Management

Catecholamine therapy

… **is not a curative intervention**, but a temporary support,

…effects and **side effects** have to be considered well,

…is **only a part** of medical treatment in LCOS,

…can be **necessary** to provide cellular oxygen supply + organ perfusion during / after CPB surgery.

Caution:

“perfusion is not the same as pressure!”

Management

First line :

Additional strategies

- **Reduction of oxygen demand**:
  sedation / analgesia / control fever / paralysis (?)
- **Normalization of acidosis** ($NaHCO_3$)
- **Optimizing of Oxygenation**
  ventilation
  FiO_2 high
  RBC - transfusion

Management

Before we use catecholamine therapy we have to …

- Perform **haemodynamic measurements**
- **Check** ventilation, x-ray…
- **Treat** metabolic acidosis
- **Optimize** fluid status
- **Rule out** arrhythmia

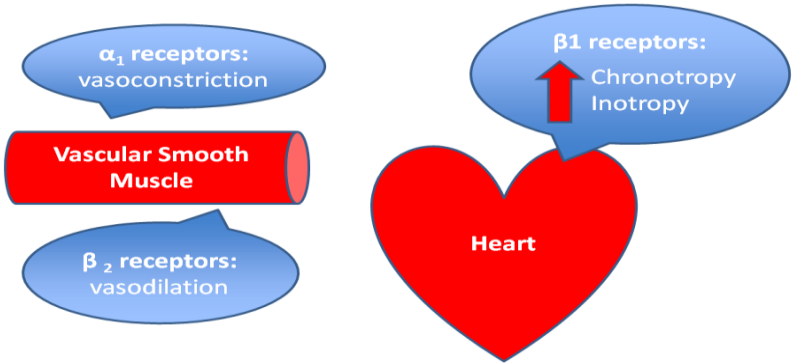
Monitoring

Invasive monitoring is necessary during catecholamine therapy

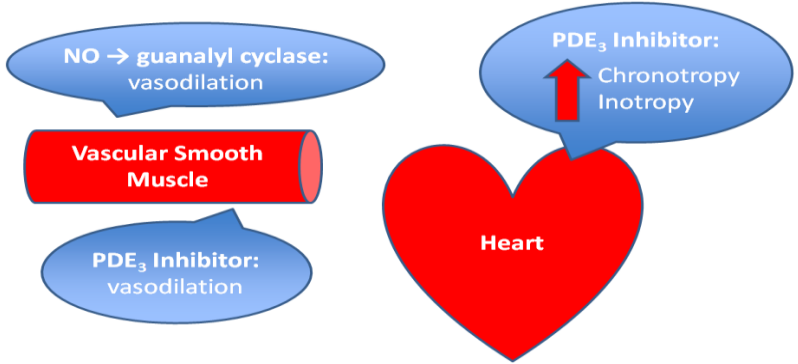
- NIBP-measurements are often misleading (too high)
- Central venous pressure (**CVP**): RV function and fluid status
- Mixed venous saturation (**SVO_2**): cardiac output
- **Arterial BP**: perfusion pressure (area under curve)
- Arterial saturation (**SaO_2**): organ perfusion
- **Lactate**: organ perfusion

Where do cardiovascular medications work?

Adrenoreceptors



NO & PDE Inhibitors



Definitions

- **Pressors**: increase systemic vascular resistance and increase blood pressure
- **Inotropes**: affect myocardial contractility and enhance stroke volume
- **Chronotropic** Agents: affect heart rate

- **Lusotropic** Agents: improve relaxation during diastole and decrease EDP in the ventricles
- **Dromotropic** Agents: Affects conduction speed through AV node; increases heart rate
- **Bathmotropic** Agents: affect degree of excitability

Dopamine

- Precursor of norepinephrine
- Releases norepinephrine in the heart
- Moderate α1, α2 and ß1- effect
- Direct effect on dopaminergic receptors (DA1, DA2)

- Effects are dose-dependent:
- **Low dose 1 - 5 mcg/kg/min:** increases renal and mesenteric blood flow (vasodilation)
- **Intermediate-dose 5 - 15 mcg/kg/min:** increases renal blood flow, heart rate, cardiac output (DA and ß1- effect)
- **High dose > 15 mcg/kg/min:** systemic and pulmonary vasoconstriction (α-effect)

Dopamine

BUT …. Severe adverse effects:

- **On heart rhythm:**
  tachycardia, induces VT´s
- ectopic beats
- AV-conduction abnormalities
- **pulmonary vasoconstriction**
- **Decrease of pituitary gland hormones:**
  prolactine (decrease of lymphocyte and macrophage activation)
  growth hormone (catabolism)
  Interaction with thyroid gland

Dopamine

There is no evidence-based data supporting the use of Dopamine as a renal protector in patients with heart failure !

We (and the most of the internantional centres all over the wolrd) **stopped giving Dopamine** in paediatric cardiac critical care.

Dobutamine

- Synthetic catecholamine with β1 inotropic effect (increases stroke volume) and β2 peripheral vasodilation (decreases afterload)
- Positive chronotropic effect (1β1, HR ↑)
- Some lusotropic effect
- No norepinephrine release

- Major metabolite is 3-O-methyldobutamine, a potent inhibitor of alpha-adrenoceptors
- Therefore, vasodilation is possible, secondary to this metabolite.
- Usual starting infusion rate is **5 mcg/kg/min**, with the dose being titrated to effect up **to 20 mcg/kg/min**.

Dobutamine

BUT …. Some adverse effects:

- **On heart rhythm:**
  tachycardia
  ectopic beats
- **Chest pain**
- **Contra indication:**
  LVOT obstruction in hypertrophic subaortic stenosis because increase of the outflow gradient

Norepinephrine

- Precursor of Epinephrine
- Acts primarily on α receptors
- **Increases Systemic vascular resistance** (SVR) without significantly increasing cardiac output (CO)
- Used in cases of low SVR and hypotension such as profound “warm shock” with a normal or high CO

- Dose:
  0,01 - 0,3 mcg/kg/min in LCOS
  0,4 - 2 mcg/kg/min in vasoplegia or under resuscitation by septic shock

Norepinephrine

BUT …. Severe adverse effects:

- Tachycardia and tachyarrhythmias
- Increased myocardial oxygen requirements and **potential to cause ischemia**
- **Decreased splanchnic and hepatic circulation** (elevation of AST and ALT)
- Dermal necrosis
- Anti-Insulin effects: **lactic acidosis, hyperglycemia**

Epinephrine

- Adrenergic agonist with multiple actions on various organs
- potent α1-, ß1- and ß2- effect
- **Effects are dose-dependent:**

- **Low dose < 0.02 mcg/kg/min:**
  ß2 - effect, but β1 predominantly
  HR ↑**,** Duration of Systole ↓
  Myocardial contractility ↑
  Peripher arteriolar dilatation
  Renal BF ↑**/** ↓
  Renin secretion ↑
  Splanchnic BF ↑**/** ↓
  Glucose ↑
  Hypokalemia

Epinephrine

- Adrenergic agonist with multiple actions on various organs
- potent α1-, ß1- and ß2- effect
- **Effects are dose-dependent:**

- **Reasonable inotropic dose**
  0,02 - 0,5 mcg/kg/min
  β1- effect still present, but also α1- effect
  HR ↑, Duration of Systole ↓
  Myocardial contractility ↑/ ↓
  Renal BF ↓
  Renin secretion ↑
  Splanchnic BF ↓
  Glucose ↑
  Lactat ↑

Epinephrine

- Adrenergic agonist with multiple actions on various organs
- potent α1-, ß1- and ß2- effect
- **Effects are dose-dependent:**

- **High dose > 0,5 - 2,0 mcg/kg/min**

  α1 effect predominantly

  Vasoconstriction

  Renal BF ↓

  Splanchnic BF ↓

  Glucose ↑

  Lactat ↑ ↑ ↑

Epinephrine

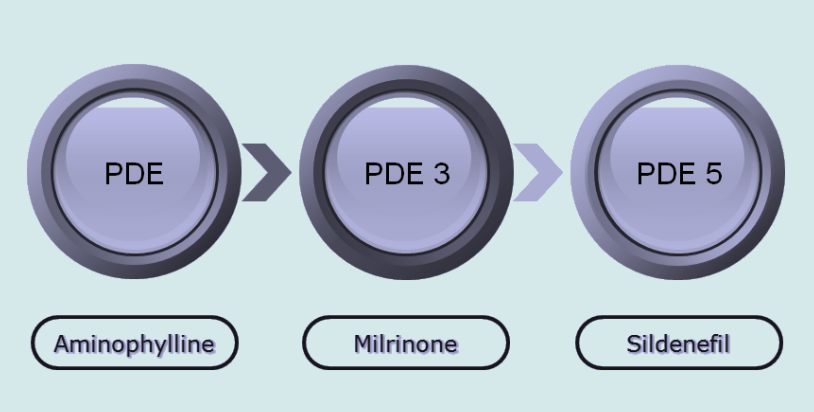
BUT …. Severe adverse effects:

- Marked metabolic effect (hyperglycaemia, leucocytes↑)
  Tachycardia, ectopic beats,
  Decreased renal blood flow
  Ischemia, abdominal pain, bladder retention
  High doses can induce apoptosis of myocardial cells

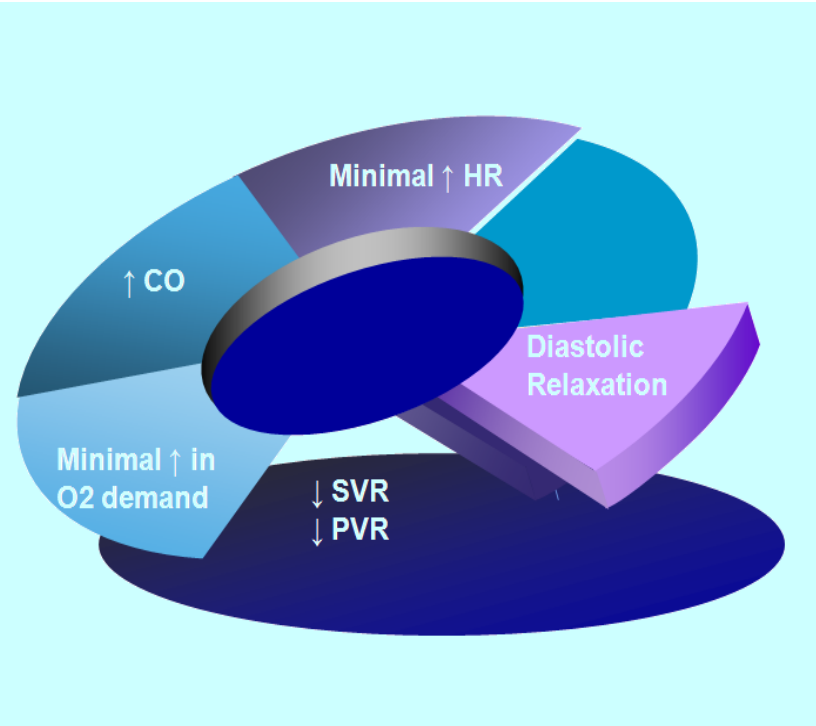
Milrinone

- Non-receptor mediated activity based on selective **inhibition of Phosphodiesterase Type III** enzyme resulting in cAMP accumulation in myocardium
- cAMP increases force of contraction and rate and extent of relaxation of myocardium
- **Inotropic, vasodilator and lusotropic effects**

- **Advantage over catecholamines:** Independent action from β - receptor activation, particularly when these receptors are down regulated (CHF and chronic catecholamine use)



Milrinone



- Increases CO by improving contractility
- Decreased SVR, PVR
- **Lusotropic effect**
- Decreased preload due to vasodilatation
- **Unique in beneficial effects on RV function**
- Half-life is 1-2 hours
- **Load with 50 mcg/kg** over 30 mins followed by **0.25 to 0.75 mcg/kg/min**
- No increase in myocardial O_2 requirement

Levosimendan

- Levosimendan is a calcium sensitiser
- It exerts its **positive inotropic effect** by increasing calcium sensitivity of myocytes by binding to cardiac troponin C in a calcium-dependent manner
- It also has a **vasodilatory effect** by opening adenosine triphosphate (ATP)-sensitive potassium channels in vascular smooth muscle to cause smooth muscle relaxation.

- Levosimendan is **indicated** for the **short-term treatment** of acutely decompensated severe chronic heart failure, and **in situations where conventional therapy is not considered adequate**.
- Preferred Dose: Load with **12 mcg/kg bolus** over 30 mins followed by **infusion with rates up to 0.2 mcg /kg /min /24h**

Inotropic agents

What is the ideal catecholamine?

- Dopamine
- Dobutamine
- Epinephrine / Adrenaline
- Norepinephrine / Noradrenaline
- **Milrinone**
- Levosimendan …..

- It increase the cardiac index + stabilize blood pressure

  WITHOUT

  increase in myocardial oxygen consumption

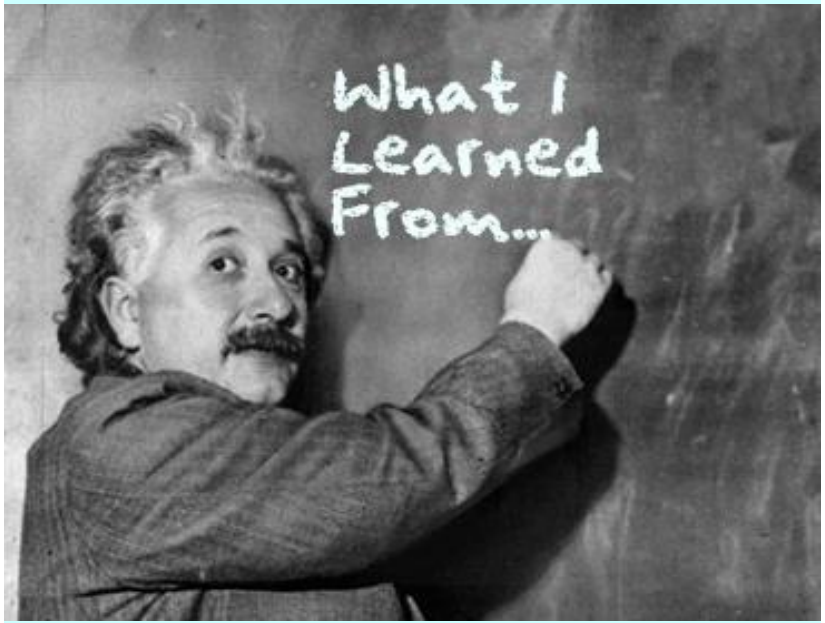
  disturbance of microcirculation due to vasoconstriction

  Inflammation, elevated cytokines (SIRS) + cardio toxicity

At a glance

	Dose: mcg/kg/min	Mechanism /Therapeutic Effects	Adverse Effects
Epinephrine	0.01- 2	β1 → ↑ HR, ↑ inotropy β2 → vasodilatation α1 → vasoconstriction → ↑ SVR	Arrhythmia ↑myocardial O_2 demand
Norepinephrine	0.01- 2	α1 → vasoconstriction → ↑ SVR β1 → ↑ HR, ↑ inotropy Min β 2 effects	Ischemic injury due to potent vasoconstriction ↑ afterload
Dopamine	< 5 5 -10 >10	D1→ diuresis, natriuresis, renal vasodilatation, (No proven benefit in preventing AKI or ↓ mortality) β1 → ↑ HR, ↑ inotropy α1 effects → vasoconstriction → ↑ SVR	Arrhythmia ↑myocardial O_2 demand Pulm. Vasoconstriction Decrease of pituitary gland hormones
Dobutamine	5-20	β1 → ↑ HR, ↑ inotropy Mild β2, α1 antagonist → vasodilation → ↓ PVR, SVR	Arrhythmia ↑myocardial O_2 demand Hypotension
Milrinone	0.25 -0,75	Phosphodiesterase Inhibitor (PDE_3 inhibitor): Myocardial : ↑ cAMP → ↑contractility + ↑lusiotropy Vasculature: ↑ cAMP → vasodilatation → ↓ SVR/PVR	Hypotension Arrhythmia
Nitroprusside	0.1-4	NO activates guanalyl cyclase (in vasc smooth muscle) → ↑cGMP → vasodilation	Cyanide toxicity ↑ V/Q mismatch

Take home messages



The cardiovascular system is responsible for a **continuous supply** of oxygenated blood to every cell in the body.

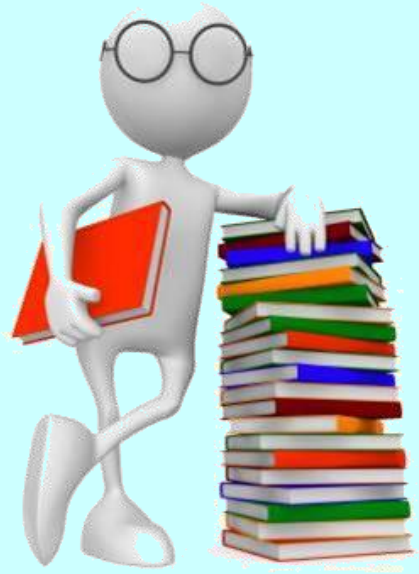
1.

Take home messages

An **imbalance** between tissue oxygen demand and delivery appears with the development of **cellular hypoxia** and leads lastly to **cell death**.

2.

Take home messages



Low cardiac output syndrome (LCOS) is the most important **cause of morbidity and mortality** in the **early postoperative phase.**

3.

Take home messages

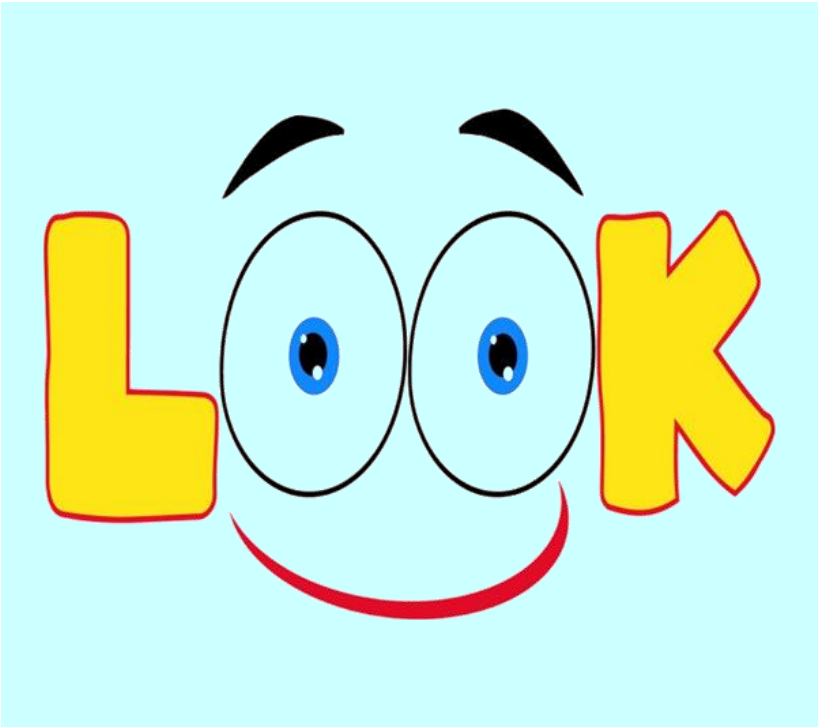
It occurs typically

6 - 18 h after cardiopulmonary bypass,

which is usually **in the middle of the night.**

4.

Take home messages



Tachycardia, centralisation and low urine output are most important clinical presentations and must be recognized as early as possible.

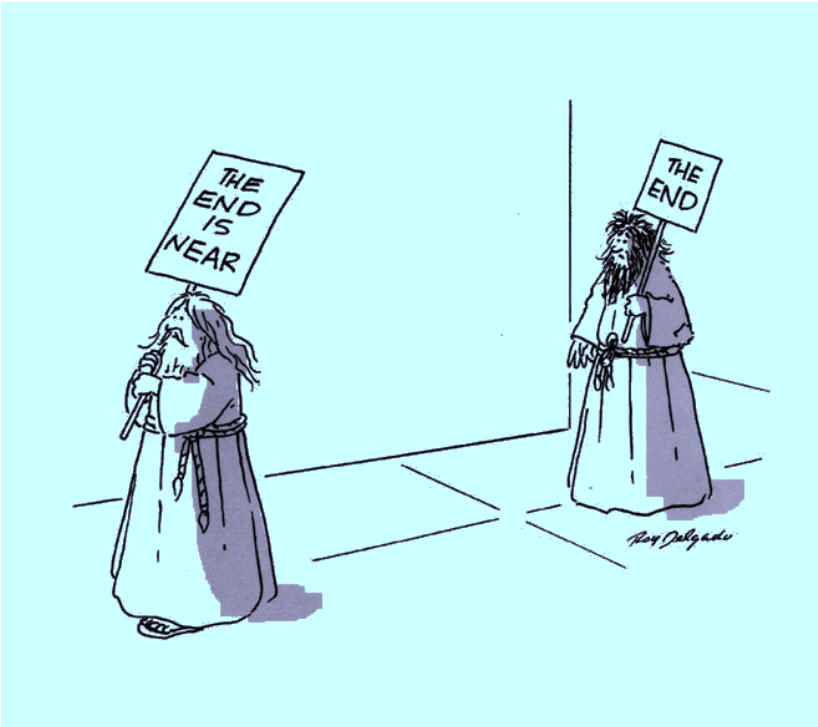
5.

Take home messages

Before we use catecholamine therapy we have to **perform** haemodynamic measurements, **check** ventilation, x-ray, treat metabolic acidosis, **optimize** fluid status and **rule out** arrhythmia.

6.

Take home messages



Invasive monitoring is necessary during catecholamine therapy and **SVO_2** as well as **lactate** are important parameters to control whether therapy is effective.

7.

Thank you for your attention