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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**

***Data:*** **17-09-2015**

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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PAEDIATRIC CARDIAC CRITICAL CARE NURSING

Bucharest, September 17, 2015

# Welcome

Matthias Angrés, MD, PhD
RobinAid Foundation
Hamburg / Germany

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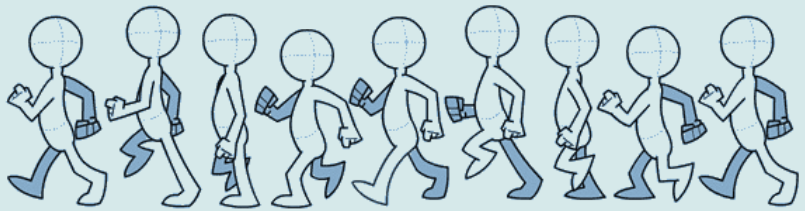
# Let‘s start a walk


**Important topics**

**in paediatric cardiac critical care nursing**



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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

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14 lectures
during the next 4 months

8. Fast-track extubation in pediatric cardiac surgery
9. Resuscitation: management of cardiac arrest
10. Medistinal bleeding and cardiac tamponade blood transfusion, coagulation disorders, and postoperative anticoagulation
11. Infections in the cardiac intensive care unit
12. Cardiac arrhythmias; care of patients with temporary pacemaker;
13. Renal failure: prevention, identification, management, and replacement therapy
14. Neurological complications, gastrointestinal complications, and nutrition

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# About me

- Training in anaesthesia and intensive care
- Specialization in cardiac anaesthesia and cardiac intensive care (adults as well as paediatrics)
- Deputy Head of Department of Anaesthesiology and Intensive Care at Bad Rothenfelde Cardiac Centre
- Medical Director and Head of Department of Anaesthesiology and Intensive Care at Cottbus Cardiac Centre
- Medical and Managing Director at Friedrichshafen Medikor WUND Group of Companies
- Medical Director and Chairman Department of Intensive and Emergency Care at Hamburg Albertinen Hospital

- Starting full-time humanitarian profession in 2008 and founding of Hamburg based RobinAid
- Regularly medical missions as paediatric cardiac intensivist in many different projects worldwide
- Extensive teaching activities on subjects of cardiac anaesthesia, intensive care, emergency care, hospital management and medical ethics

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# About RobinAid

RobinAid is an independent and non-profit humanitarian, medical organization.

Chaine de l'Espoir Europe
international medical network

La chaîne de l'espoir ▪ France
De Keten van Hoop ▪ Belgium
Bambini Cardiopatici nel Mondo ▪ Italy
RobinAid Foundation ▪ Germany
Cadeia da Esperança ▪ Portugal

United as **Chaîne de l'Espoir Europe** we work as an international medical network in order to give disadvantage children around the world access to the best healthcare.

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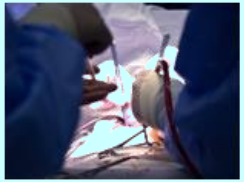
# What we do

Paediatric Critical Care

Urgent Medical Care



Centres of Expertise

RobinAid stands for highly specialized medical care. We provide urgent medical assistance, support the set-up of local centres of expertise and accompany them as partners on their own way to autonomy.

We focus on the treatment of lifethreatening disorders by neonates, infants and elderly children, particularly congenital and acquired heart disease.

Paediatric Cardiac Critical Care

Paediatric General Critical Care

Paediatric Critical Care

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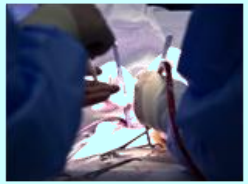
# How we work

We are always working in cooperation with local partners. By sending medical teams as well as equipment we assist them to take medical responsibility for their little patients.

- Qualified Medical Experts
- Medical Equipment
- Urgent Medical Care



- Centres of Expertise

- Certified Training & Fellowship Programs
- Access to International Medical Networks

We share our knowledge with the local colleagues. Training and access to international medical networks are of primary importance to encourage them in their own abilities.

# Fellowship Programs

On the way to independence access to international knowledge and experience are of great importance.


RobinAid performs the following fellowship programs:

Paediatric Cardiac Critical Care
Paediatric Cardiac Critical Care Nursing
Paediatric Cardiac Anaesthesia

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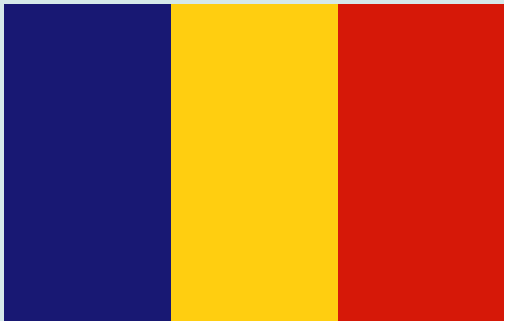
# Paediatric Cardiac Surgery in Romania

Annually more than 2,000 Romanian children with congenital heart disease need surgeries but currently less than 200 can be performed in the country.

The health system in Romania is affected by two major problems: An insufficient funding and a lack of qualified medical staff.

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# Paediatric Cardiac Surgery at Bucharest Marie Curie Children‘s Hospital

Because of the urgent need in 2011 the local NGO Inima Copiilor realized the construction of a brand new paediatric cardiac unit which was financed by a big fundraising campaign throughout Romania and the Ministry of Health.

The unit includes two operating theaters, 6 beds in the intensive care unit as well as a cath lab and is very well equipped. In September 2013 Bambini Cardiopatici nel Mondo started the first mission and in beginning of 2014 RobinAid joined the project.

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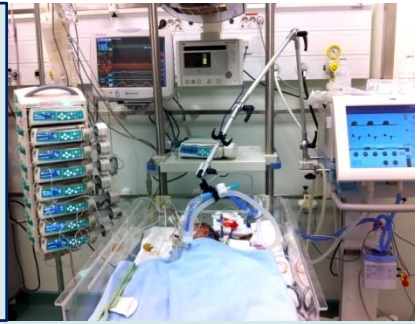
# Involved partners

Bambini Cardiopatici nel Mondo:

Surgical teams

RobinAid Foundation:

ICU teams



Local Department of Paediatric Cardiology:

Preparation and follow up of the patients

Local Neonatal Intensive Care Unit:

Follow up by need of prolongued intensive care

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# Frequency of missions

| | January | February | March | April | May | June | July | August | September | October | November | December |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 2013 | | | | | | | | | | | | |
| 2014 | | | | | | | | | | | | |
| 2015 | | | | | | | | | | | | |

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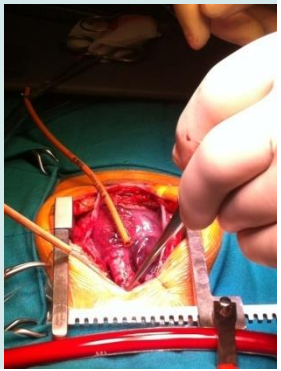
# Procedures 9/13 - 6/15

| DATE | DIAGNOSIS | INTERVENTION |
|---|---|---|
| 23-Sep-13 | ASD OS | ASD closure |
| 23-Sep-13 | ASD OS | ASD closure |
| 24-Sep-13 | ASD OS | ASD closure |
| 24-Sep-13 | ASD OS | ASD closure |
| 25-Sep-13 | ASD OS | ASD closure |
| 2-Dec-13 | ASD OS | ASD closure |
| 3-Dec-13 | ASD OS | ASD closure |
| 4-Dec-13 | PDA | PDA closure |
| 4-Dec-13 | ASD OS | ASD closure |
| 9-Dec-13 | Co Ao | Co Ao repair (Crafoord technique) |
| 9-Dec-13 | VSD | VSD closure |
| 10-Dec-13 | Co Ao | Co Ao repair (patch enlargement) |
| 10-Dec-13 | VSD | VSD closure |
| 11-Dec-13 | AVSD partial | AVSD repair |
| 12-Dec-13 | ASD + VSD | ASD + VSD closure |
| 12-Dec-13 | ASD OS | ASD closure |

Data collected by Dr. Cristian Bulescu

| DATE | DIAGNOSIS | INTERVENTION |
|---|---|---|
| 3-Feb-14 | Infundibular stenosis | Infundibular stenosis repair |
| 3-Feb-14 | VSD (Pezzi-Laubry) | VSD closure |
| 4-Feb-14 | TOF | TOF repair |
| 4-Feb-14 | ASD + VSD | ASD + VSD closure |
| 5-Feb-14 | VSD | VSD closure |
| 5-Feb-14 | VSD (Pezzi-Laubry) | VSD closure |
| 6-Feb-14 | VSD | VSD closure |
| 6-Feb-14 | Ao regurgitation S/P VSD repair | Ao valve replacement |
| 7-Feb-14 | ASD | ASD closure |
| 7-Feb-14 | VSD | VSD closure |
| 7-Apr-14 | VSD + mid-ventricular stenosis | VSD closure + resection |
| 7-Apr-14 | VSD | VSD closure |
| 8-Apr-14 | Co Ao | Co Ao repair (patch enlargement) |
| 8-Apr-14 | ASD + PAPVR | ASD closure + PAPVR repair |
| 5-May-14 | DORV type Fallot | Total repair |
| 5-May-14 | Subvalvular Ao stenosis | Subvalvular stenosis resection |
| 6-May-14 | TOF | TOF repair |
| 6-May-14 | Tricuspid atresia | Modified B-T shunt |
| 7-May-14 | AVSD partial | AVSD repair |
| 7-May-14 | VSD | VSD closure |
| 8-May-14 | ASD + PAPVR | ASD closure + PAPVR repair |
| 8-May-14 | Vascular ring | Vascular ring resection |
| 9-May-14 | ASD OS | ASD closure |
| 9-May-14 | ASD + PAPVR | ASD closure + PAPVR repair |
| 8-Jul-14 | PDA | PDA closure |
| 8-Jul-14 | ASD OS | ASD closure |
| 9-Jul-14 | PDA | PDA closure |
| 9-Jul-14 | PDA | PDA closure |
| 9-Jul-14 | PDA | PDA closure |
| 25-Sep-14 | PDA | PDA closure |
| 25-Sep-14 | DSA OS | ASD closure |
| 26-Sep-14 | PDA | PDA closure |
| 26-Sep-14 | ASD OS | ASD closure |
| 26-Sep-14 | VSD | VSD closure |
| 29-Sep-14 | TOF | TOF repair |
| 29-Sep-14 | VSD | VSD closure |
| 30-Sep-14 | DORV type Fallot | Total repair |
| 1-Oct-14 | TOF | TOF repair |
| 1-Oct-14 | ASD OS | ASD closure |
| 2-Oct-14 | VSD + infundibular stenosis | VSD closure + infundibular patch repair |
| 2-Oct-14 | TOF | Modified B-T shunt |
| 3-Oct-14 | ASD + VSD | ASD + VSD closure |
| 13-Nov-14 | Recurrent pericardial effusion | Pleuro-pericardial window |
| 13-Nov-14 | VSD | VSD closure |
| 13-Nov-14 | VSD | VSD closure |
| 14-Nov-14 | ASD OS | ASD closure |
| 14-Nov-14 | VSD | VSD closure |
| 8-Dec-14 | DORV type Fallot | Total repair |
| 8-Dec-14 | VSD | VSD closure |
| 9-Dec-14 | TOF | Total repair |
| 9-Dec-14 | VSD | VSD closure |
| 10-Dec-14 | TOF | Total repair |
| 10-Dec-14 | VSD | VSD closure |
| 11-Dec-14 | Co Ao | Co Ao repair (Crafoord technique) |
| 11-Dec-14 | Supravalvular pulmonary stenosis | Main pulmonary artery enlargement |
| 12-Dec-14 | ASD OS | ASD closure |

| DATE | DIAGNOSIS | INTERVENTION |
|---|---|---|
| 12-Jan-15 | VSD | VSD closure |
| 12-Jan-15 | TGA | Arterial switch operation |
| 13-Jan-15 | LCOS S/P ASO | Mediastinal inspection |
| 14-Jan-15 | Co Ao | Co Ao repair (patch enlargement) |
| 14-Jan-15 | VSD | VSD closure |
| 15-Jan-15 | S/P ASO | Delayed sternal closure |
| 9-Feb-15 | TOF | Total repair |
| 9-Feb-15 | VSD | VSD closure |
| 10-Feb-15 | VSD + mid-ventricular stenosis | VSD closure + resection |
| 10-Feb-15 | VSD + mid-ventricular stenosis | VSD closure + resection |
| 11-Feb-15 | TGA | Arterial switch operation |
| 12-Feb-15 | DORV type Fallot | Total repair |
| 12-Feb-15 | Subvalvular Ao stenosis + PDA | Ao valve repair + Ao valve replacement + PDA closure |
| 13-Feb-15 | Supravalvular aortic stenosis | Doty |
| 23-Mar-15 | TOF | Total repair |
| 23-Mar-15 | VSD | VSD closure |
| 24-Mar-15 | VSD + mid-ventricular stenosis | VSD closure + resection |
| 24-Mar-15 | VSD | VSD closure |
| 25-Mar-15 | VSD | VSD closure |
| 25-Mar-15 | PDA | PDA closure |
| 26-Mar-15 | Fibroadenoma of the right thigh with vascular involvement | Tumor resection |
| 27-Mar-15 | PDA | PDA closure |
| 11-May-15 | TOF + absent pulmonary valve | TOF repair |
| 11-May-15 | TOF | TOF repair |
| 12-May-15 | AVSD intermediate | AVSD repair |
| 13-May-15 | VSD + mid-ventricular stenosis | VSD closure + resection |
| 13-May-15 | DORV type Fallot | Total repair |
| 14-May-15 | ASD + VSD | ASD + VSD closure |
| 15-Jun-15 | AVSD complete | AVSD repair |
| 16-Jun-15 | AVSD complete | AVSD repair |
| 16-Jun-15 | TAPVR cardiac type | Total repair |
| 17-Jun-15 | Pulmonary pseudoatresia & intact septum | Pulmonary commissurotomy + infundibular patch |
| 18-Jun-15 | DORV type Fallot | Total repair |
| 18-Jun-15 | Pulmonary atresia & intact septum | Pulmonary commissurotomy |
| 19-Jun-15 | AVSD partial + pectus excavatum | AVSD repair + pectus repair |
| 22-Jun-15 | Pu veins stenosis + DORV + Pu stenosis + ASD S/P TAPVR (cardiac) repair | Pulmonary veins enlargement |
| 23-Jun-15 | PDA | PDA closure |
| 24-Jun-15 | End-stage kidney disease | Brachio-basilic fistula creation |



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# Results

| Year | 2013 | | | | 2014 | | | | 2015 (by June 30) | | | | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Age | 0 - 28 days | 29 days - 1 year | > 1 - 5 years | > 5 years | 0 - 28 days | 29 days - 1 year | > 1 - 5 years | > 5 years | 0 - 28 days | 29 days - 1 year | > 1 - 5 years | > 5 years | |
| No of patients | 0 | 0 | 8 | 8 | 0 | 14 | 19 | 23 | 7 | 15 | 5 | 11 | 110 |
| Aristotle Basic Complexity Score | 0 | 0 | 3,5 | 4,13 | 0 | 5,27 | 5,06 | 5,79 | 8,04 | 6,7 | 6,5 | 6,8 | 5,59 |
| Discharge Mortality | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 (0,9%) |

Data collected by Dr. Cristian Bulescu

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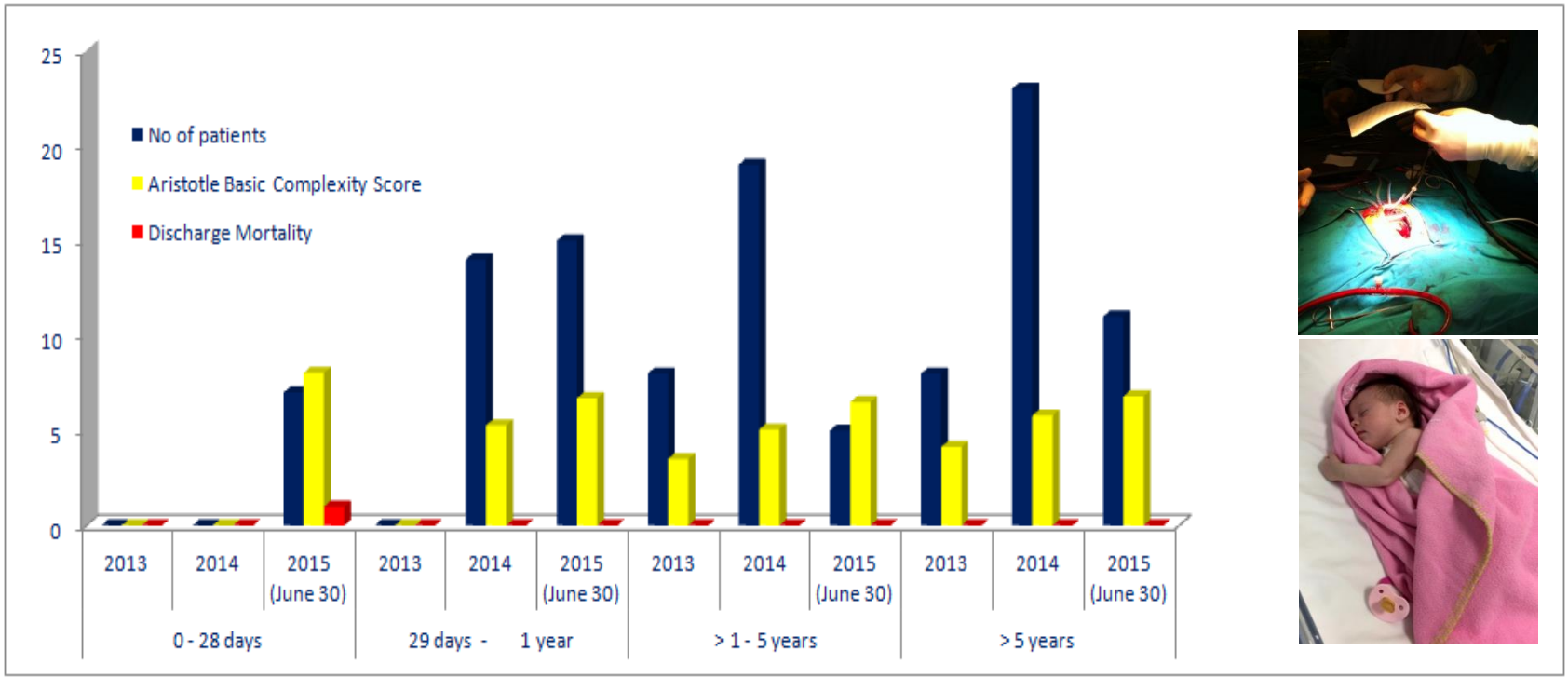
# Results




Data collected by Dr. Cristian Bulescu





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# Further common projects with Bambini Cardiopatici nel Mondo

Dohuk Azadi Heart Centre

Cairo
Giza El Agouza Hospital

Dakar Paeditric Cardiac Centre
Fan University Hospital

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# Main project with La Chaine de l‘Espoir

## French Medical Institute for Children
## Kabul / Afghanistan

This children hospital was founded in 2006 by our French parent organization La Chaîne de l‘Espoir and is managed by the Aga Khan Development Network. The PICU is the only mechanical ventilation unit throughout Afghanistan. More than 1,200 children are treated and more than 250 paediatric cardiac surgeries are performed annually.

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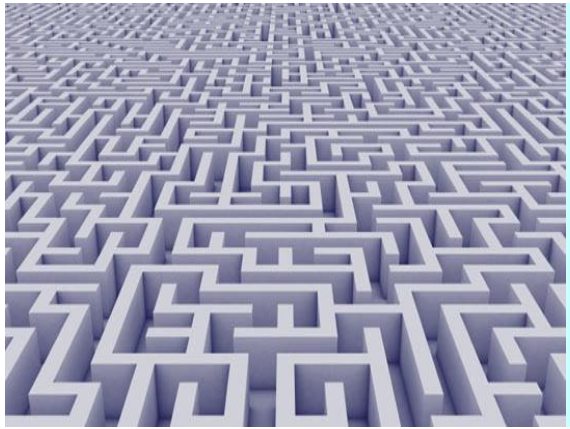
# First important message

The highly complex paediatric patients with congenital or acquired heart disease require **interprofessional teamwork and collaboration** to ensure high quality outcomes with low mortality and morbidity.

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# Paediatric Cardiac TEAM


- Intensivist
- Cardiologist
- Neonatologist
- Anaesthetist
- Surgeon
- Nurses
- Physiotherapist
- Technicians
- Perfusionist
- Radiologist


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# Benefit

To achieve more means to **decrease** the number of risks as well as **complications** and to **increase patients' safety**.



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# How to reach the goal


Hard Skills ● Soft Skills = value to the patient

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# Second important message

The care of critically ill children after cardiac surgery is challenging and best performed with the emphasis on an **anticipatory** (rather than reactive) **approach**.


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# In other words

**Anticipation and prevention** are the most important **keys for recognition, diagnosis, and successfull management** of all potential risks and complications in the postoperative critical care of paediatric cardiac patients.

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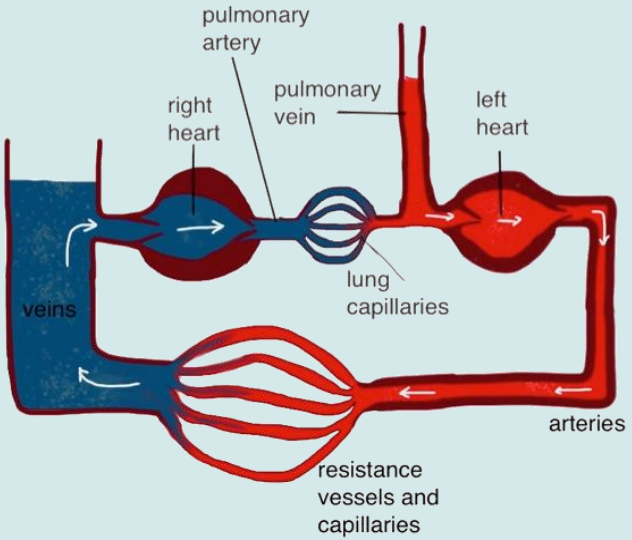
Now let‘s start with the todays‘s topic

# The importance of cardiac output and cardiovascular medications

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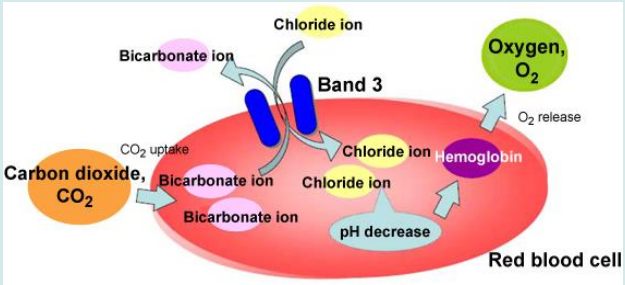
# Cardiovascular system

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**).

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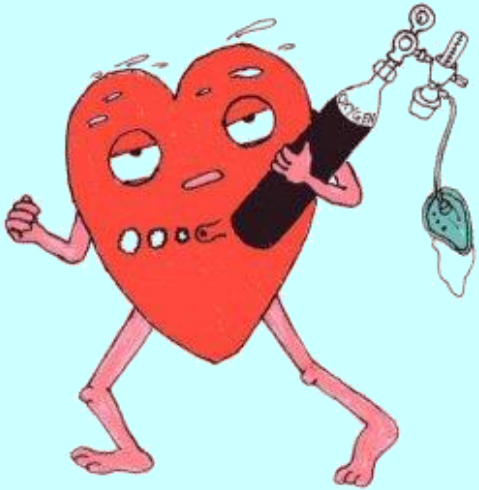
# Basic understanding



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

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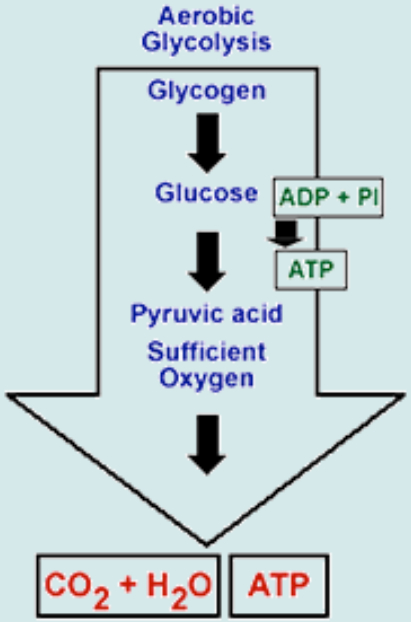
# Why is oxygen so important

- 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.

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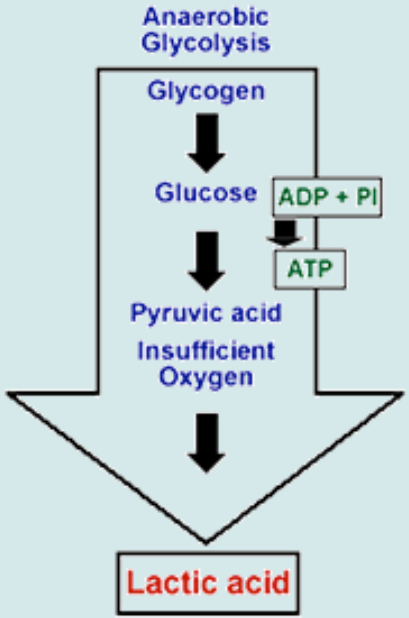
# Why is oxygen so important

- 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.**

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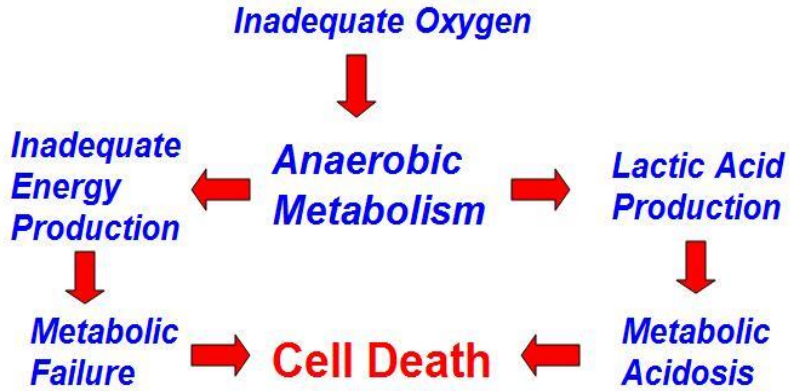
# Why is oxygen so important

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

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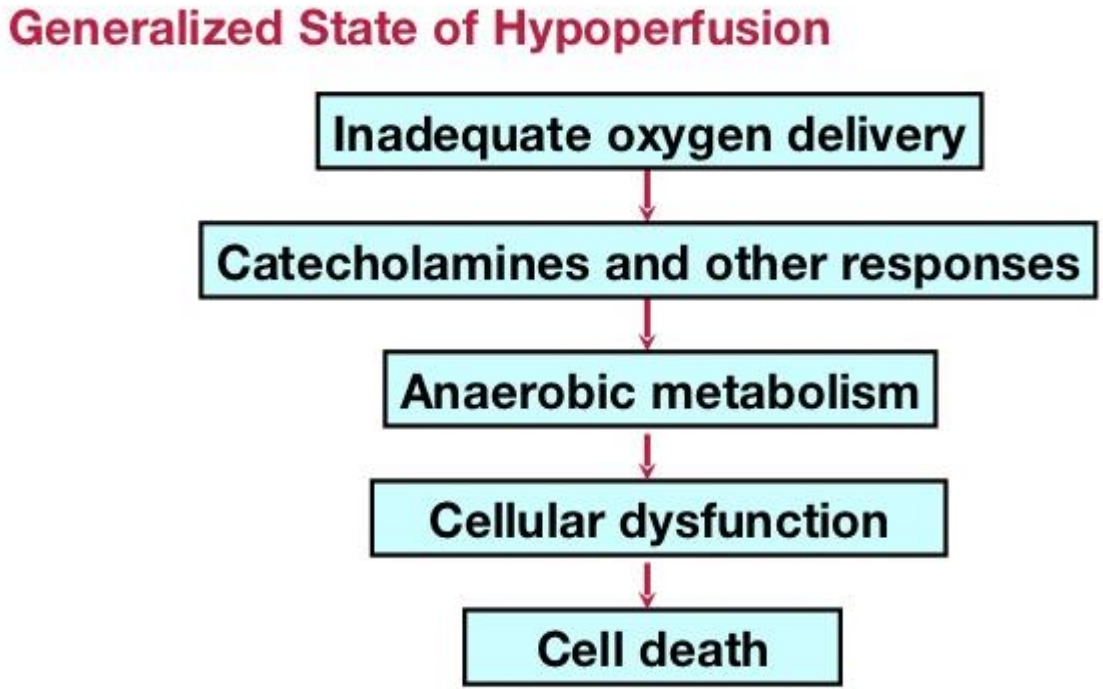
# Why is perfusion so important

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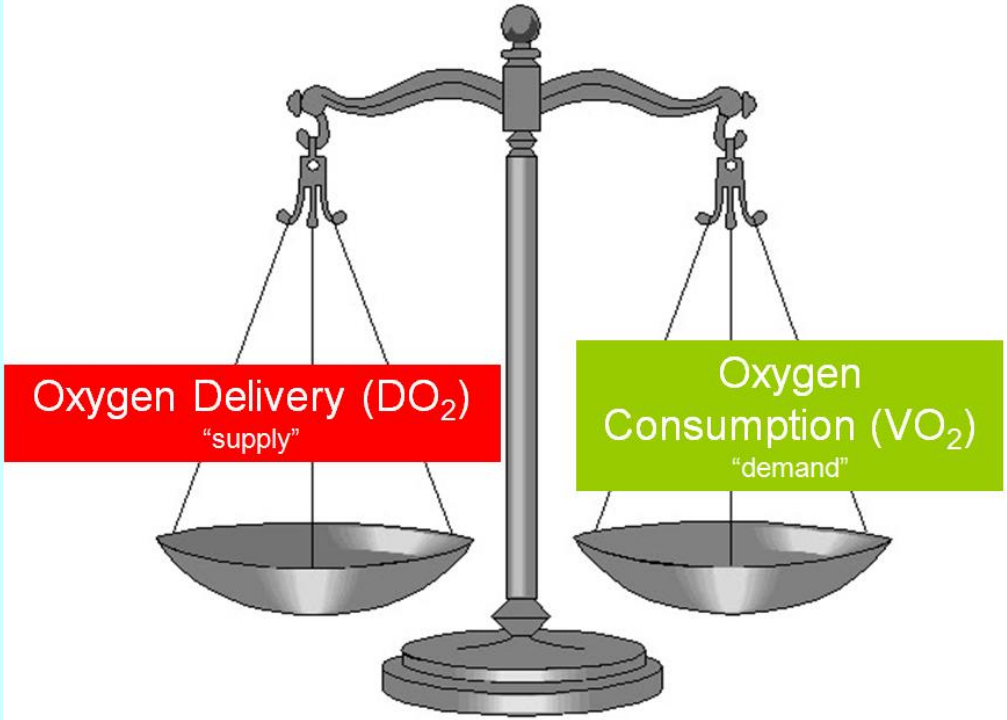
# Oxygen Supply and Demand Balance

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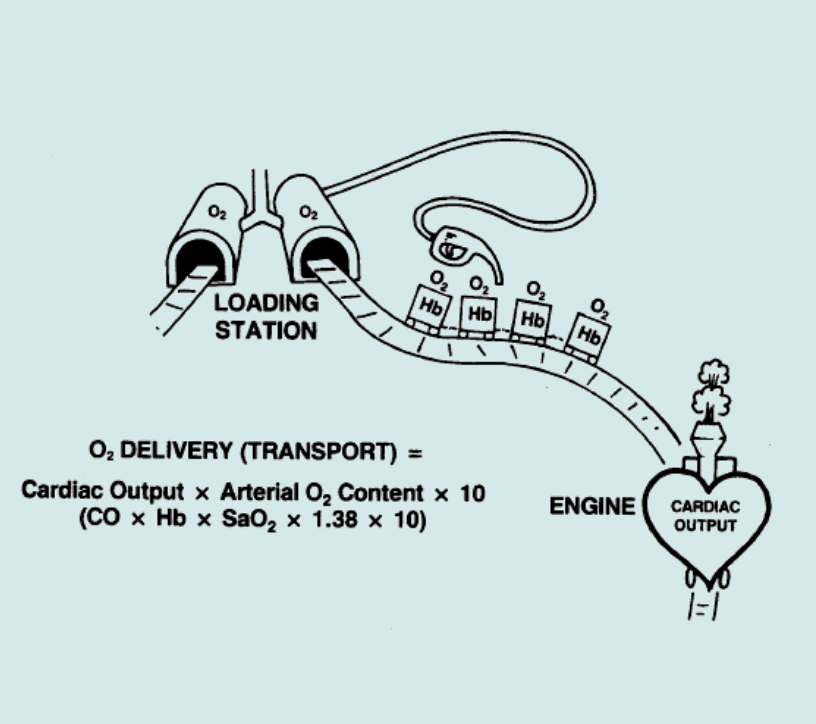
# Oxygen Delivery

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)

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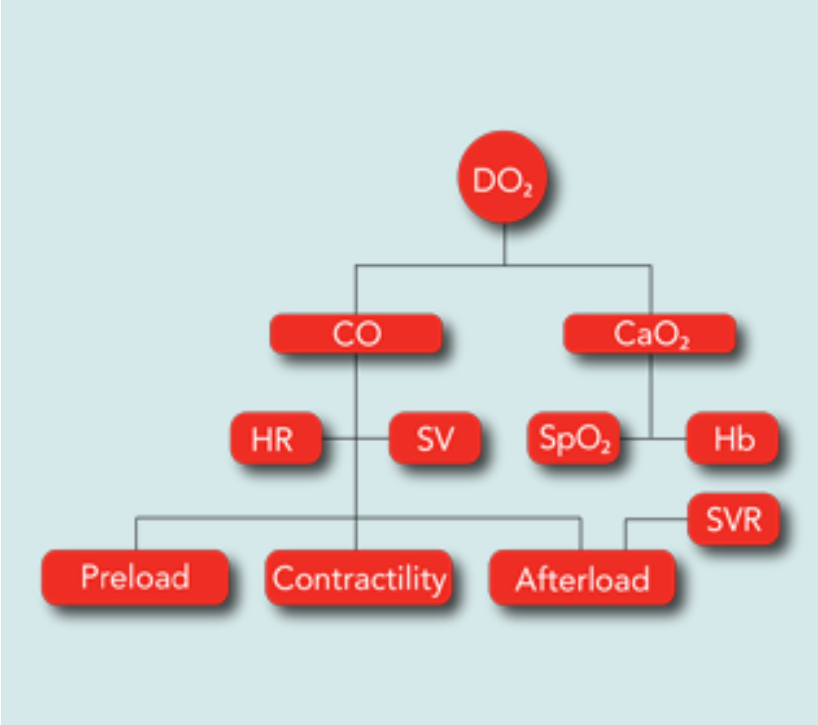
# Poor Oxygen Delivery

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

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

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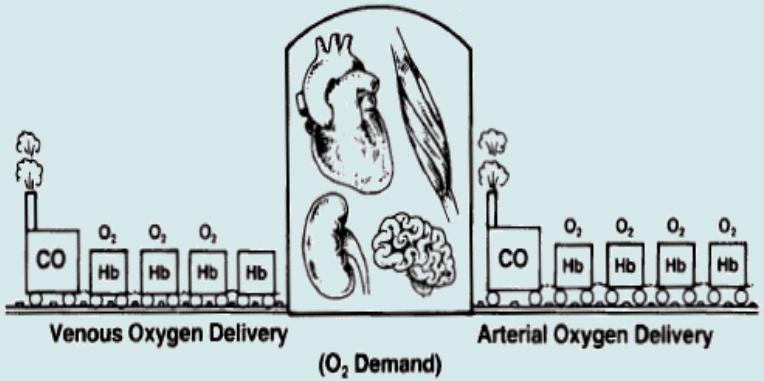
# Oxygen Demand

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)

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# 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)

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

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

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# 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**.

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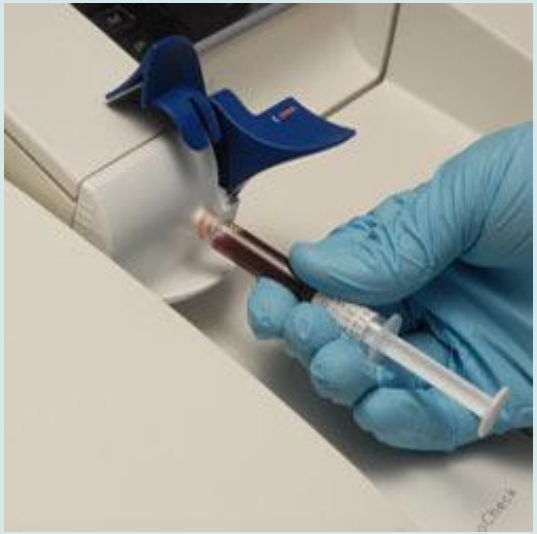
# Supply / demand imbalance

Ways to measure Oxygen supply / demand imbalance:

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



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# 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 |

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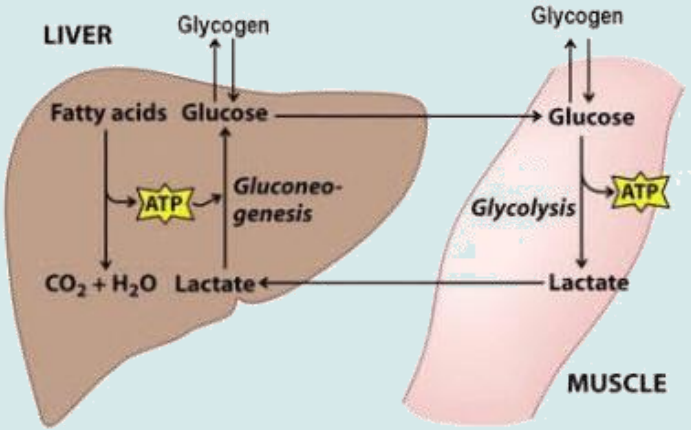
# Lactate

- 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**

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# Supply / demand imbalance

When there is an imbalance you can:

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

  and / or

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


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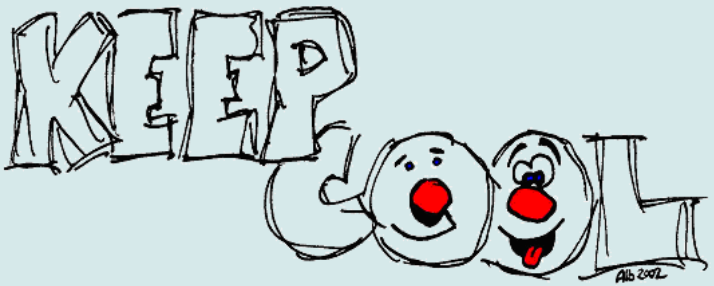
# ↓ Oxygen Demand

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

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# ↑ 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**

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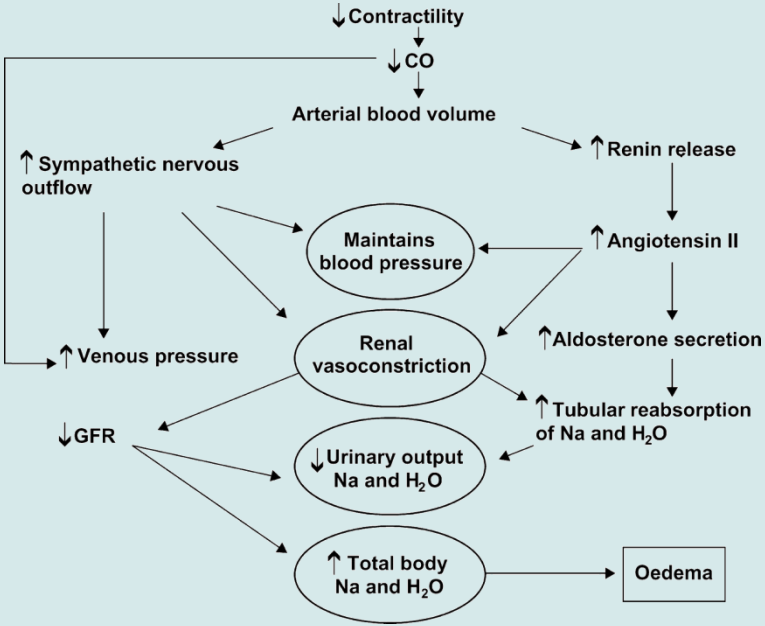
# Low Cardiac Output in paediatric cardiac surgery

- 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

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# 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

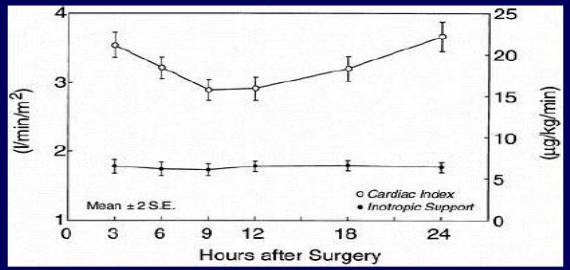
  ⇓

  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

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# 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)

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# 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)

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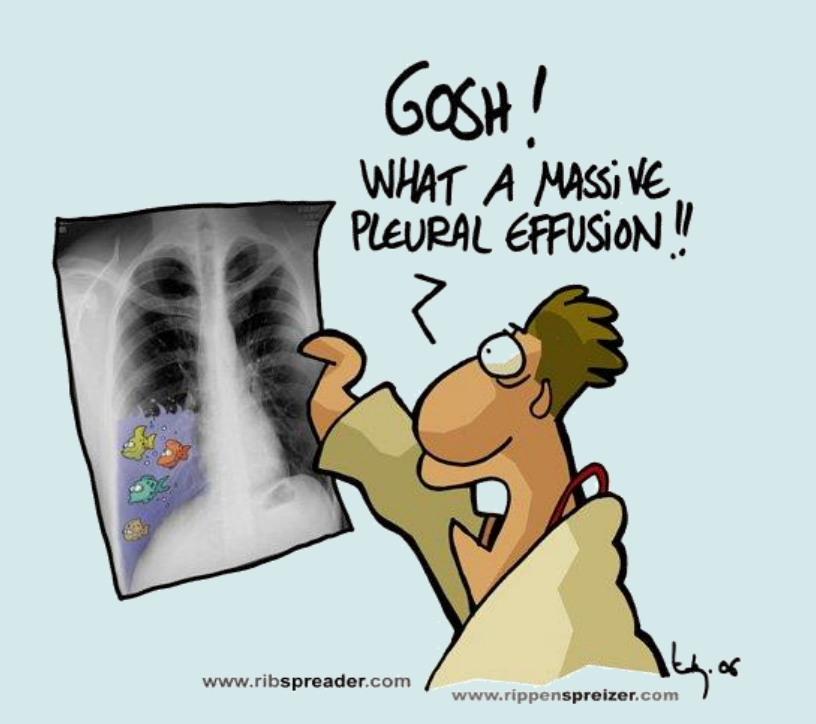
# Clinical presentation

- **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

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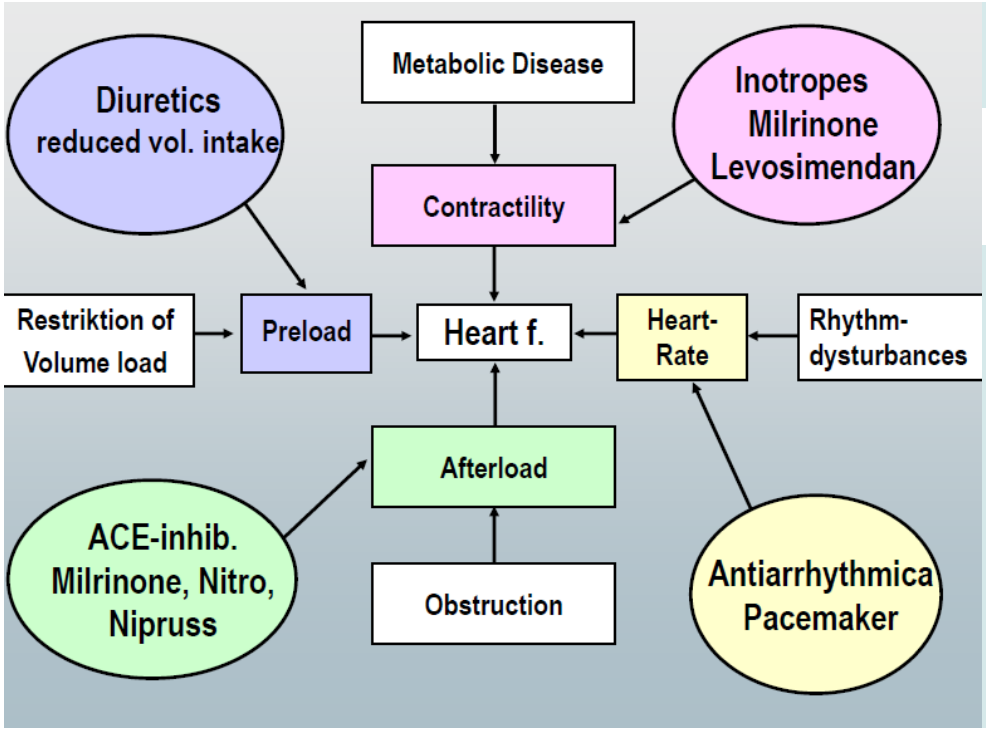
# Management




**Heart failure treatment is more than inotropes!**

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# 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!”***

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# 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

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# Let‘s have a look to a case

## Clinical findings

- 8 years, VSD, 24 kg, ICU for 20 min.
- RR 70/40 mmHg
- ECG: SR 155 bpm
- Lactate elevated
- Normal oxygenation and ventilation
- CVP 2 mmHg
- Mixed venous saturation: 55 %

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# Let‘s have a look to a case

**What is the therapy of choice?**

- Adrenalin iv 0.25 mcg bolus
- Adrenalin infusion (0.1 mcg/kg/min)
- Crystalloide volume (300 ml in 30 min)
- Noradrenalin iv 0.4 mcg bolus
- Milrinone (0.6 mcg/kg/min)
- Levosimendan (0.2 mcg/kg/min)

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# The right answer

RR 72/40 mmHg → ▶ 95/40 mmHg
ECG: SR 135 bpm → ▶ SR 95 bpm
Lactate elevated → ▶ normalized

Normal oxygenation and ventilation
CVP 2 mmHg → ▶ 5 mmHg
Mixed venous saturation: 55 % → ▶ 65 %

- Adrenalin iv 0.25 mcg bolus
- Adrenalin infusion (0.1 mcg/kg/min)
- **Crystalloide volume (300 ml in 30 min)**
- Noradrenalin iv 0.4 mcg bolus
- Milrinone (0.6 mcg/kg/min)
- Levosimendan (0.2 mcg/kg/min)

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# 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

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# 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

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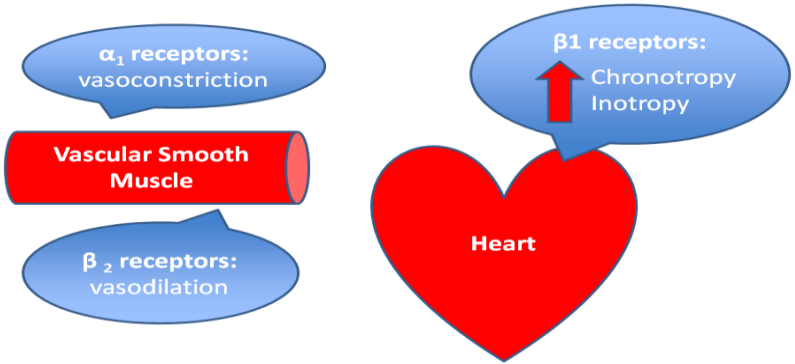
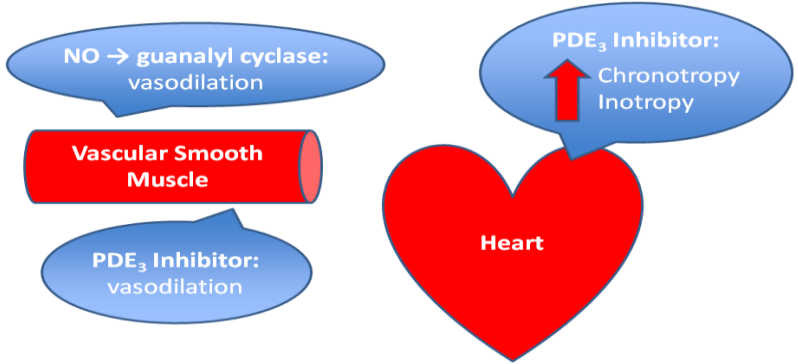
## Where do cardiovascular medications work?

### Adrenoreceptors




### NO & PDE Inhibitors

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# 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

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# Catecholamine

**What is the ideal catecholamine?**

- It should **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

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# 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)

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# 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

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# 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.


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# 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**.

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# 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

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# 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

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# 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**

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# 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

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# 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 ↑

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# 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 ↑ ↑ ↑

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# 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**

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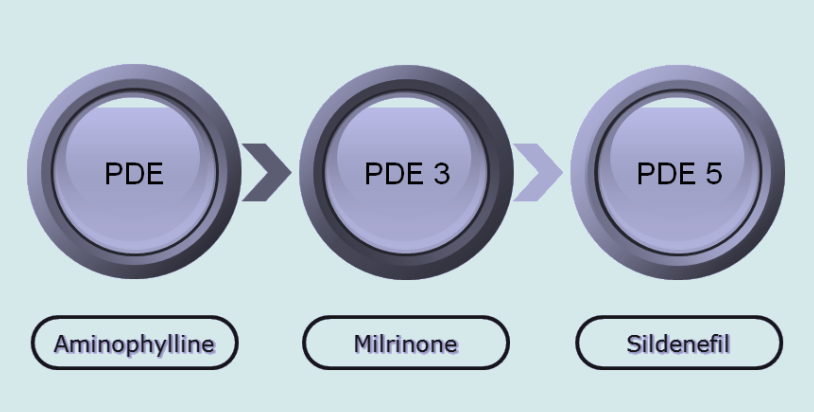
# Milrinone

- 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)

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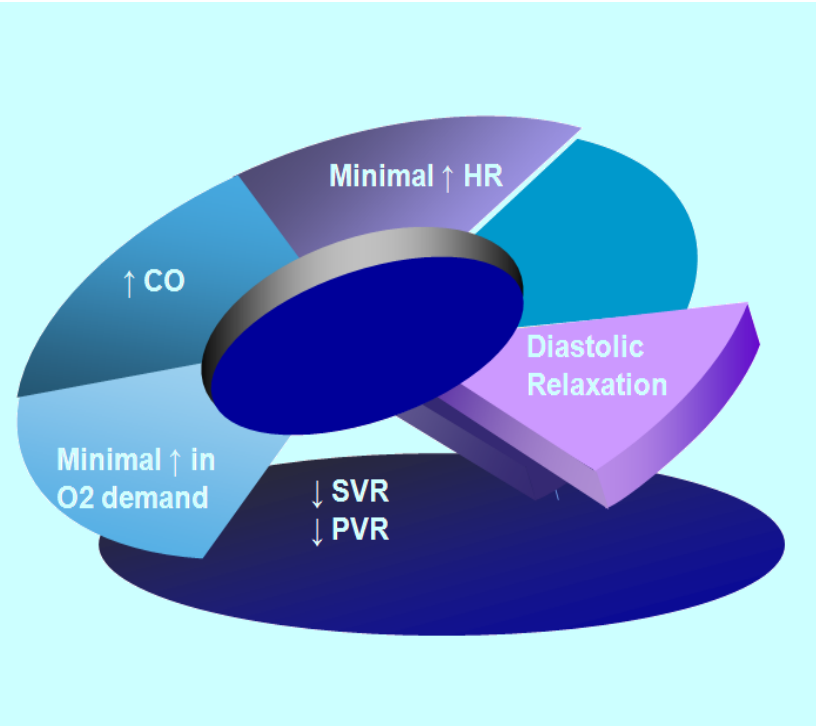
# Milrinone




- 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

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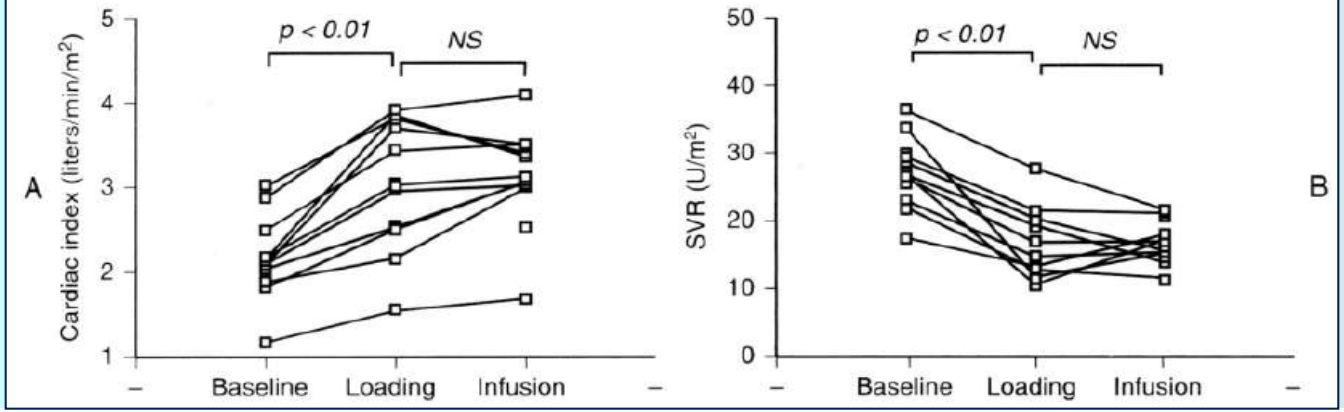
## PRIMACORP study — Milrinone vs Placebo

**Prophylactic iv Use of Milrinone after Cardiac Operation in Pediatrics (n=220)**

Crit Care Med 1995:33:1907

### 46% reduced risk to develop LCOS after cardiac surgery

**Cardiac Index improves** — **Systemic resistant reduced**

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# 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**

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# 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

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# At a glance

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


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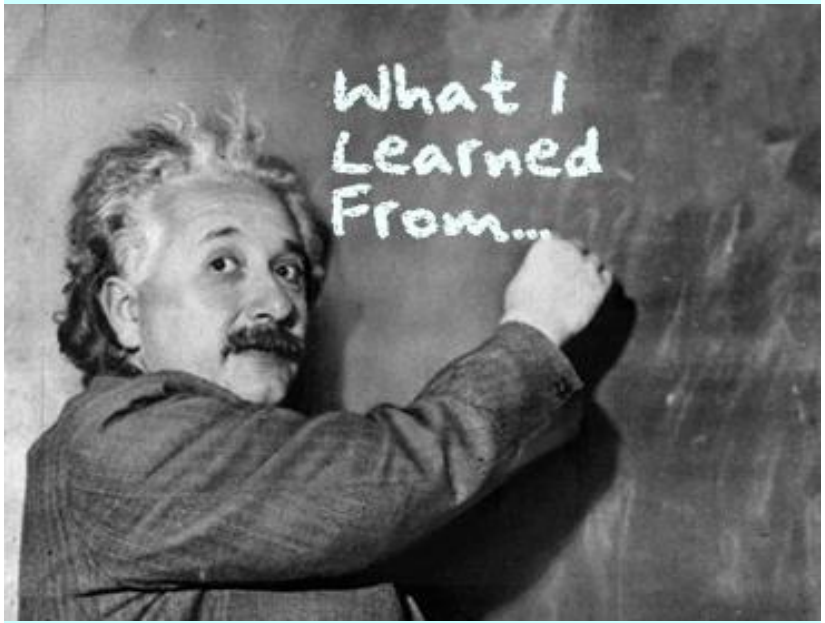
# 8 take home messages




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

**1.**

# 8 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.**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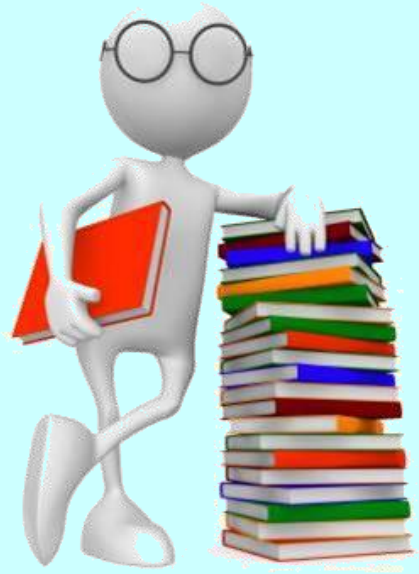
# 8 take home messages



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

**3.**

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# 8 take home messages

It occurs typically

**6 - 18 h after cardiopulmonary bypass**,

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

**4.**


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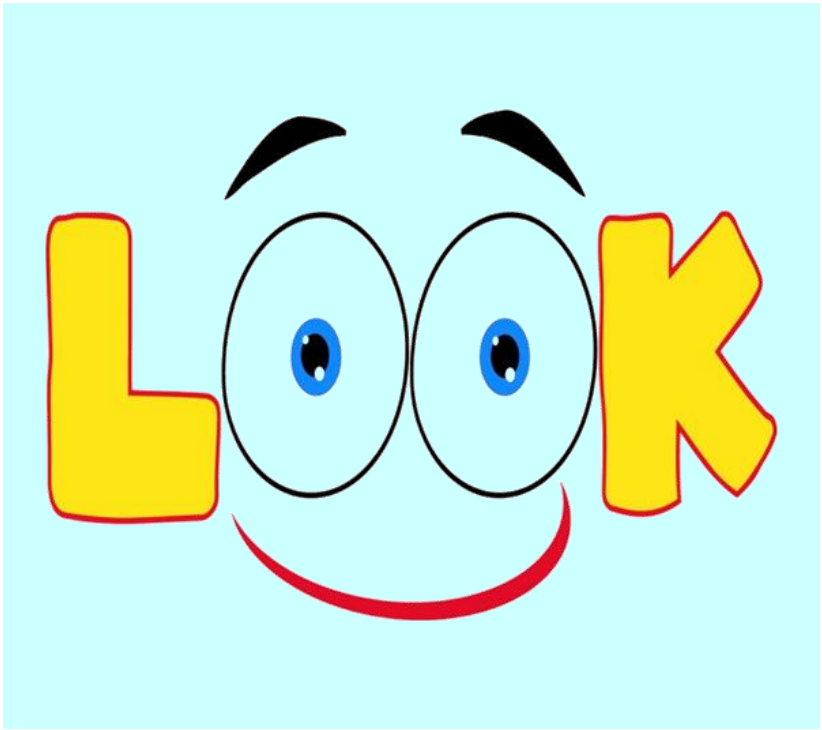
# 8 take home messages




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

**5.**

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# 8 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.**

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# 8 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.**

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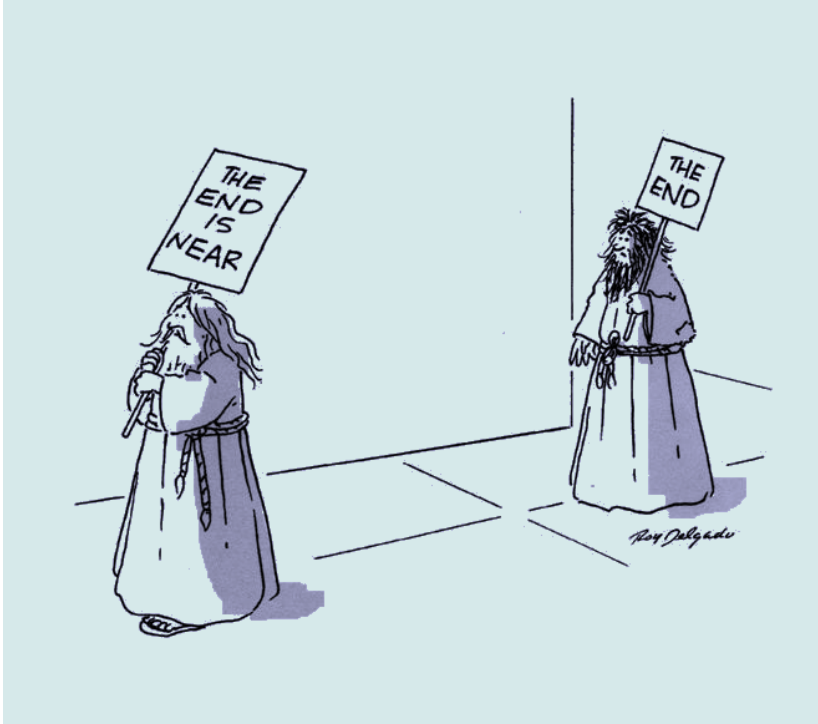
# 8 take home messages

**Anticipation and prevention** are the most important keys **for recognition, diagnosis, and successfull management** of all potential risks and complications in the postoperative critical care of paediatric cardiac patients.

**8.**

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Thank you for your attention


Yes, you can!

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