# Cardiopulmonary monitoring

## Non-invasive cardiopulmonary monitoring

Non-invasive cardiopulmonary monitoring includes **data on heart rate, respiratory rate, blood pressure** (non-invasive NIBP measurement), **pulse oximetry** and **ECG**.

### Heart rate and respiratory rate (HR, RR)

Continuous monitoring with adjustable alarm values is best. **Approximately**, HR can be determined **by counting the heartbeats over 6 seconds and multiplying the result by 10**. When **assessing RR**, it is necessary to determine the value by **counting over a 1-minute period**, as the respiratory rate is slower and more variable than the heart rate and calculating over a shorter period of time would risk miscalculation. **We always set upper and lower limits for the alarm**, and the values depend on the age of the child and the underlying disease. In terms of RR, the most important thing is an **alarm to catch apnoea**, which is a superurgent condition in medicine. We usually set the threshold for apnoea at 15 seconds.

| **Normal values of heart rate and respiratory rate** | | |
|---|---|---|
| | **normal respiratory rate (per minute)** | **normal heart rate (per minute)** |
| **newborns** | 40–60 | 100–180 |
| **infants** | 30–50 | 80–150 |
| **toddlers** | 25–40 | 80–130 |
| **pre-school** | 25–35 | 80–120 |
| **younger school kids** | 20–30 | 70–100 |
| **older school kids** | 12–20 | 60–100 |
| **grown-ups** | 12–16 | 60–90 |

### Non-invasive blood pressure measurement (NIBP)

In general, BP measurements should be a **routine for children older than 3 years**; it is clearly **preferred to use a mercury manometer with auscultatory measurement**. When measuring BP, simultaneous measurement of the child's height and weight should become the rule. If BP values exceed 90-95 during the measurement, BP is automatically measured on the lower limb as well. **BP on the lower limbs tends to be 10-20 mmHg higher**. In no case **should the upper limb BP exceed the lower limb BP**. In this case, we strongly suspect aortic coarctation.

| **Physiological values of respiration** | | | | |
|---|---|---|---|---|
| | **newborn** | **infant** | **toddler** | **older kid** |
| **Tidal volume** | 8 | 8 | 8 | 8 |
| **Resistance** | 40 | 20–30 | 20 | 1–2 |
| **Compliance** | 3–5 | 10–20 | 20–40 | 70–100 |

In some children, echoes are audible when measuring BP, possibly down to 0 mmHg. In these cases, it is recommended to repeat the measurement with less pressure on the head of the stethoscope. If the echoes are still audible up to 0 mmHg, the diastolic BP value should be recorded at the first obvious weakening of the echoes. The **width of the cuff of the tonometer** should be **approximately 40% of the circumference of the arm**; a narrow cuff is the source of falsely high BP values, while a cuff that is too wide is the cause of falsely lower BP values (in this case, however, the significance of the error is small). The width of the cuff of the tonometer means the **inner, i.e. rubber, part** of the cuff.

The arm on which the BP is measured should be completely free (the child should be at least halfway undressed), the cuff should be placed in the middle of the arm between the olecranon and the acromion. If borderline BP values are detected, the measurement should be repeated.

**Non-invasive BP monitoring:**

- ***oscilometric*** (most common)
- ***sphygmomanometric*** (mercury tonometer) – determine Korotkov phenomena (the accuracy of the measurement affects the correct choice of cuff)
- ***doppler principle***

In children in the ICU, the mercury tonometer is disadvantageous in the youngest children, in uncooperative children and when frequent measurements are necessary.

**Doppler technique** is suitable for young children and conditions with impaired perfusion. A small Doppler probe is placed over the radial or brachial artery. The blood movement is well detected by sensitive ultrasound. A cuff placed on the upper arm is inflated until the Doppler signal has completely disappeared. It is then slowly deflated. Systolic pressure is read when the first Doppler signal appears, diastolic pressure is read when the length and quality of the signal decreases. Correlation with pressure measured directly intra-arterially is good, but **the method is not suitable for continuous measurement**.

**The oscillometric method** is easy to implement. When the cuff is inflated, the blood flow in the artery causes oscillations. If the pressure in the cuff begins to drop, the device registers the sBP, dBP and MAP. However, all techniques have limitations in conditions with a significant decrease in cardiac output, severe hypotension or systemic vasoconstriction, conditions with generalized edema, and extreme obesity.

Recommended sizes of tonometer cuffs

| Age group | Size of cuff |
|---|---|
| Premature babies | 3,75 |
| Newborns | 4,0 |
| Young kids < 5 let | 7,0 |
| School-age kids | 11,0 |

In addition to systolic BP and diastolic BP, the determination of **mean arterial pressure (MAP)** is very important. MAP represents the organ perfusion pressure and is useful to assess circulatory failure and to define hypotension. It is not the arithmetic mean of systolic sBP and diastolic dBP.

**MAP = (sBP + 2x dBP) / 3**

Indirect methods of BP measurement have limited accuracy, therefore, **intra-arterial monitoring is necessary in severe conditions** such as shock, rhythm disturbances, and administration of vasoactive agents.

MAP values indicative of circulatory failure in mmHg

| Age | MAP |
|---|---|
| Newborns | < 40 |
| 3.–6. months | < 40 |
| 6.–12. months | < 45 |
| 1–4 years | < 50 |
| 4–10 years | < 55 |
| 10–14 years | < 60 |
| 14–18 years | < 65 |
| >18 years | < 70 |

**Perfusion pressure** is of great significance in shock conditions. Arithmetically, it is the **difference between MAP and CVP:** $PP = MAP - CVP$

Lower limit of the norm for perfusion pressure values in cm H2O

| Age | Perfusion Pressure |
|---|---|
| Newborn | 55 |
| Infants | 60 |
| Toddler | 65 |
| Preschool | 65 |
| School Age | 65 |

## Pulse oxymetry

Pulse oximetry **non-invasively measures the oxygen saturation of haemoglobin in the arterial part of the bloodstream** (pulsatile flow).

## Astrup – blood gas testing

The aim of the blood gas examination is to obtain data to assess the oxygenation function of the lungs, the adequacy of alveolar ventilation and, together with other biochemical parameters, to detect the possible existence of an ABR disorder and to determine the degree of its compensation.

## Capnometry, capnography

The measurement of the CO2 concentration (**capnometry**) and the graphical representation of this value (**capnography**) in exhaled air is **based on the measurement of the absorption of infrared light**.

Under normal circumstances, the gradient between arterial tension $paCO_2$ and end-expiratory $CO_2$ tension (end-tidal $CO_2$ = $etCO_2$) is 2-5 torr (0.25-0.66 kPa) and reflects the size of the ventilatory dead space and the ratio of the size of the tidal volume to the dead space. An increase in anatomical or alveolar dead space under pathological conditions in which pulmonary perfusion is reduced leads to an increase in the gradient between $paCO_2$ and $etCO_2$. In practice, this change is usually manifested by a decrease in $etCO_2$.

**Clinical causes of the increase in the gradient between $paCO_2$ and $etCO_2$:**

- enlargement of the anatomical dead space;
- enlargement of the alveolar dead space;
    - hypotention;
    - low cardiac output;
    - high PIP and/or PEEP;
    - pulmonary embolism;
    - bronchospasm

## The most commonly used indices assessing the respiratory component

### PF index

In patients with severe forms of pulmonary dysfunction, the PF index = **hypoxemic index = Horowitz index** is often used **to assess the oxygenation function of the lungs**. Its determination requires blood gas testing with knowledge of $FiO_2$. The actual value is strongly dependent on the $FiO_2$ parameter used and the level of airway pressures at the time of the blood gas examination. In patients with hypercapnia, the effect of changes in partial pressure of $CO_2$ may also need to be taken into account, as a significant rise in alveolar partial pressure of $CO_2$ results in a decrease in $pAO_2$ and subsequently in $paO_2$.

$$\mathbf{PFi = paO_2 : FiO_2}$$

- *$FiO_2$ is given as a decimal number*
- *$paO_2$ is given in torr*

**Normal values** are > **500**, values < **300** represent **acute lung injury**, and values < **200** are one of the criteria defining **ARDS**. PFi < 200 corresponds to a lung shunt value > 20%.

### Alveolo-arterial oxygen gradient A-a$DO_2$

The alveolar-arterial oxygen gradient A-a$DO_2$, sometimes referred to as the **alveolar-arterial difference**, is a parameter used **to assess the degree of impaired oxygenation of the lungs**. It primarily **indicates the quality of alveocapillary diffusion.**

$$\mathbf{A\text{–}aDO_2 = pAO_2 - paO_2}$$

$$\mathbf{A\text{–}aDO_2 = (760 \times FiO_2) - \{(paO_2 + paCO_2) + 47\}}$$

- *$FiO_2$ is given as a decimal number*
- *$paO_2$ and $paCO_2$ are given in torr*
- *760 = barometric pressure at sea level*
- *47 = partial pressure of water vapour in the inhaled air*

This formula can be used if data on inhaled $O_2$ concentration and arterial gas values are available. Values >350 are indicative of **respiratory insufficiency**, values >**550** are one of the criteria for **ECMO** (extracorporeal membrane oxygenation).

### Oxygenation index

The oxygenation index **OI** is widely used in paediatrics, unlike the PFi it also **reflects pressure changes**.

$$\mathbf{OI = (FiO_2 \times Pmaw) : paO_2}$$

- *$FiO_2$ is given in percentage!*
- *Pmaw is given in $cmH_2O$.*
- *$paO_2$ is given in torr.*

**Normal** values are < **5**.

**Dead space ventilation**

**For an indicative assessment of the relationship between the size of the functional dead space and the size of the tidal volume** (Vd/Vt), the difference between the arterial tension of $CO_2$ and the tension of $CO_2$ in the exhaled mixture at the end of expiration ($etCO_2$) is used. Under normal circumstances, this difference is minimal (2-5 torr), but under pathological circumstances it increases significantly.

$$\mathbf{Vd/Vt = (paCO_2 - etCO_2) : paCO_2}$$

If an increase in airway pressure (e.g. after PEEP adjustment) results in an increase in this parameter without a concomitant beneficial effect of the increase in pressure on oxygenation, this may be considered a sign of an exceedance of the optimal airway pressure. Similarly, changes in the magnitude of the Vd/Vt ratio may occur when cardiac output or pulmonary pressures decrease.

**In normal subjects**, the **Vd/Vt** value is in the range of **0.2-0.3**. A rise in Vd/Vt is associated with the development of both hypoxemia and hypercapnia. Hypercapnia usually occurs when **Vd/Vt is greater than 0.5**.

### Gastric tonometry

The principle of the method is the **regional measurement of the partial pressure of $CO_2$ ($PtCO_2$) of the gastric mucosa**. Using this method, we can **detect very early perfusion disturbances of the splanchnic region**, **which will be manifested by a very early rise in mucosal $PtCO_2$.**

## Invasive cardiopulmonary monitoring

### Invasive BP measurement

Measuring arterial blood pressure is an essential part of monitoring any acute condition. **The mean arterial pressure MAP depends on cardiac CO output and systemic SVR resistance:**

$$\mathbf{MAP = CO \times SVR}$$

**For children, it is necessary to use the indexed values of these parameters,** i.e. the values relative to the body surface. Then the equation will look like this:

$$\mathbf{MAP = CI \times SVRI}$$

The equation itself shows the limits in the measurement of arterial blood pressure. **Blood pressure does not inform about blood flow.** It can therefore be normal even with increasing peripheral resistance and simultaneously decreasing cardiac output, and therefore with reduced organ blood flow. Thus, we consider **MAP** as only a **crude indicator of organ perfusion**, especially since many organs have the ability to autoregulate, i.e., their perfusion is kept constant over a wide range of perfusion pressures through changes in vascular resistance.

**Arterial BP is measured directly or indirectly.** Indirect methods are simple and non-invasive. Direct methods are more accurate. The differences between indirect and direct blood pressure measurement are especially obvious in shock, hypertension, hypothermia, and obesity.

**Advantages of direct BP measurement:**

- continuous monitoring;
- consistent measurement accuracy;
- rapid recognition of circulatory disorders;
- direct monitoring of the hemodynamic effects of heart rhythm disturbances;
- indirect assessment of myocardial contractility from the rate of rise of the arterial pressure curve;
- estimation of pulse volume from the systolic part of the pressure curve;
- access to the artery to take blood samples: the Astrup and other laboratory.

***Indication***:

- hemodynamically unstable patient: shock states, hypertensive crisis, hypotension;
- intracranial hypertension;
- the need to administer vasoactive substances: catecholamines, sodium nitroprusside;
- ventilationally unstable patient (need for repeated and frequent blood gas testing);
- the need for repeated blood draws;
- regular blood sampling;
- angiographic examination;
- hemofiltration/hemoperfusion.

## Central venous pressure, CVP

A central venous catheter is a catheter whose distal end lies in a hollow vein. **Normal values** of central venous pressure CVP are **2-12 cm H2O** (ideal 3-10 cm $H_2O$).

***Transfer relations:***

- 1 cm $H_2O$ = 0,74 mmHg
- 1 mmHg = 1,36 cm $H_2O$
- 1 kPa = 7,5 mmHg = 10,2 cm $H_2O$

**Reduced CVP values** are found in **hypovolemia**.

**Increased CVP values** in **hypervolemia, right heart insufficiency, pulmonary embolism, superior vena cava obstruction, cardiac tamponade**.

**Catheters** for long-term insertion are **coated with an antibacterial surface**. Currently, all catheters are **radiocontrast**. To eliminate risks, the latest catheters are fitted **with a one-way valve to prevent air embolism.**

When choosing an access to the superior vena cava, the following factors in particular should be taken into account: the experience of the physician with a particular method, the accessibility of the veins suitable for puncture, the risks of each approach for a particular patient and the expected time of catheter insertion.

For long-term cannulation, we prefer a central approach (v. jugularis interna, v. subclavia) because catheters inserted in this way have a lower risk of infectious and thrombotic complications than catheters inserted from the periphery (**swimming catheters**). Never introduce catheters through an infected puncture site.

Central venous cannulation is a very common procedure in intensive care. The availability of quality sets has expanded its use and safety. In this context, it is appropriate to emphasize the need for correct indication and professional humility of the physician in the decision-making process, including strict adherence to the methodology for each approach.

We need to check the position of each CVK and adjust if necessary to avoid severe complications. **The optimal position is immediately in front of the orifice of the superior vena cava into the right atrium**. There are no longer any venous valves in this area. Two procedures are appropriate: chest X-ray and ECG check with a catheter tip lead. On the chest radiograph, the carina tracheae is an important landmark for the position of the tip of the central venous catheter. The carina always lies cranial to the pericardium. For safety reasons, the end of the catheter must lie just above the carina. ECG diagnosis is simple, without great expense, and should therefore be preferred to the less prompt and more expensive radiographic imaging. **The position of the catheter tip in the right atrium is indicated by a clearly elevated P wave in the ECG image** on the monitor. The catheter is then advanced until the normal P wave reappears on the monitor.

## Saturation of hemoglobin in the central venous system - $SvO_2$

In the critically ill patient, it is essential to determine whether the oxygen supply to the tissues is adequate in relation to the tissue oxygen demand. **$SvO_2$ monitoring allows** a fairly accurate determination of the ability **to determine whether tissue oxygen demand is in balance with oxygen delivery**. $SvO_2$ represents **the average percentage of oxygen bound in mixed venous blood.**

The delivery of oxygen to the tissues is a fundamental task of the cardiovascular system and directly depends on cardiac output, arterial blood $O_2$ saturation $SaO_2$ and haemoglobin concentration. **Adequate oxygen delivery** to the tissues is ensured by a **$SaO_2$ > 92%** and an optimal hemoglobin value (depending on the age of the child). Cardiac output is an equally important component of maintaining good tissue oxygenation. In critical conditions, the effort is always to maximize cardiac output by manipulating preload, afterload, contractility and heart rate.

The **physiological $SvO_2$ value** represents a range of **60-80%** and means that the tissue oxygen demand is covered by a sufficient supply. In case of significant deviations, the values of $SaO_2$, Hb, CO/CI and $O_2$ consumption should always be reassessed.

***Changes in $SvO_2$ values that require reassessment of the patient's condition:***

- change of plus/minus 10 % persisting for at least 5 minutes
- decrease < 60 % or increase > 80 %

- trend showing a gradual but steady decline

**Drop in $SvO_2$** indicates that the patient needs to use up $O_2$ stores to meet their needs. This occurs when the $O_2$ supply decreases despite an equal or increased $O_2$ requirement, or when the $O_2$ requirement increases despite an equal or decreased $O_2$ supply.

**An increased $SvO_2$ value** is observed in the case of increased $O_2$ supply despite equal or decreased demand, or decreased $O_2$ demand despite equal or increased supply.

***Reduced value of $SvO_2$***:

- reduced supply of $O_2$:
  - reduced cardiac output,
  - high PEEP,
  - cardiogenic shock,
  - hypovolemia,
  - hypotension,
  - arrhythmias.
- reduced $SaO_2$:
  - hypoxia,
  - respiratory failure,
  - dyspnoea,
  - decrease in hemoglobin (anemia, bleeding).
- increased need for $O_2$:
  - hyperthermia,
  - pain,
  - increased physical activity,
  - cramps,
  - increased respiratory work.

***Conditions with increased value of $SvO_2$***:

- increased supply of $O_2$:
  - increased cardiac output (inotropics, sepsis),
  - increase in $SaO_2$ (high $FiO_2$, hyperoxia),
  - increased Hb (transfusions).
- reduced need for $O_2$:
  - hypothermia,
  - anesthesia.
- other causes of the increase in $SvO_2$:
  - VVV heart with L-R shunt,
  - tissue necrosis,
  - nitroprusside toxicity,
  - septic shock.

## Basic physiological calculations of ventilation

### Oxygen delivery ($DO_2$)

## Oxygen delivery:

Oxygen delivery (oxygen delivery, $DO_2$) is **directly proportional to cardiac output and arterial blood oxygen content** (arterial oxygen content, $CaO_2$). For paediatrics, we always choose indexed values, i.e. values relative to body surface area.

$$DO_2 \text{ (index)} = CI \times CaO_2 \times 10$$

$$CI = HR \times SV$$

$$CaO_2 = (Hb \times 1{,}34 \times SaO_2) + (0{,}003 \times PaO_2)$$

$$CvO_2 = (Hb \times 1{,}34 \times SvO_2) + (0{,}003 \times PvO_2)$$

$$a\text{–}v\ DO_2 = CaO_2 - CvO_2$$

***$DO_2$*** = oxygen delivery, represents oxygen delivered to tissues per minute, reference values $DO_2$ = 550–650 ml/min/$m^2$

***SV = stroke volume***

***HR = heart rate***

***CI = cardiac index*** → cardiac output per unit body surface area

***$CaO_2$*** = oxygen content in arterial blood, reference values $CaO_2$ = 17–20 ml

***$CvO_2$*** = oxygen content in mixed venous blood, reference values $CvO_2$ = 12–15 ml

***$SaO_2$*** = $O_2$ saturation in arterial blood, is referred to as $SaO_2$/100

***$SvO_2$*** = saturace smíšené žilní krve, je uváděna jako $SvO_2$/100

***$PaO_2$*** = partial pressure of oxygen in arterial blood, is given in torr

***$PvO_2$*** = partial pressure of oxygen in mixed venous blood, is given in torr

***a–v $DO_2$*** = arteriovenous oxygen content difference (oxygen content difference), reference values a–v $DO_2$ = 3–5 ml/dl

***Hb*** = hemoglobin, given in quantity of g/dl !

**Oxygen consumption (oxygen uptake, $VO_2$)**

The measure of O2 consumption is VO2 (oxygen consumption, oxygen uptake), reference values **$VO_2$ (index) = 120–200 ml/min/$m^2$**

$$VO_2 \text{ (index)} = CI \times (CaO_2 - CvO_2) \times 10$$

The basic task of the cardiopulmonary unit is to ensure the **balance between $VO_2$ and $DO_2$.** The balance is determined by :

- *oxygen content in mixed venous blood $CvO_2$*
- $O_2$ extraction ($O_2$ER), i.e. the ratio between the amount of oxygen consumed and delivered $VO_2$/$DO_2$, expressed as a percentage.

Normal **extraction** values are around **25%**, but with significantly increased tissue demand, $O_2$ extraction can rise to 50%. In shock conditions, we try to keep $O_2$ extraction below 30%.

$$O_2ER = VO_2 / DO_2$$

Both $CvO_2$ and $O_2$ER depend on the values of mixed venous blood $SvO_2$ saturation and cardiac CO output. CO/CI depends on the values of heart rate, heart volume, preload, afterload and contractility. Increasing heart rate, improving myocardial contractility and relaxation in diastole, and optimizing preload and afterload increase CO/CI. Oxygen carrying capacity can be improved by optimizing hematocrit. By improving all these parameters, $DO_2$ can be increased. In some specific situations (fever, sepsis, trauma, thyrotoxicosis), metabolic needs may exceed even normal $DO_2$.

| Basic physiological calculations of ventilation | | |
|---|---|---|
| **Parameter** | **Unit** | **Standard** |
| $CaO_2$ | ml | 17–20 |
| $CvO_2$ | ml | 12–15 |
| a–$vDO_2$ | ml/dl | 3–5 |
| $DO_2$ (index) | ml/min/$m^2$ | 550–650 |
| $VO_2$ (index) | ml/min/$m^2$ | 120–200 |
| $0_2ER$ | % | 20–35 |

## Hemodynamics

### Minitoring Options

#### PiCCO System

The PiCCO (Pulse Contour Cardiac Output) system is a **less invasive method** than the Swan-Ganz pulmonary artery catheter - it requires the insertion of a central venous catheter and a thermodilution arterial catheter (inserted via the a.axillaris or a.radialis or, more commonly, a.femoralis) to determine cardiac output, without the need for pulmonary artery catheterization. With this system, in addition to cardiac output, **preload volume parameters can be determined and pulmonary oedema quantified**.

Cardiac output is measured intermittently by transpulmonary thermodilution and continuously by heart rate curve analysis. During the three bolus thermodilution measurements, the shape of the heart rate curve is analysed and calibrated; cardiac output is then continuously monitored by comparing these "calibrated" curves with several consecutive heart rate curves. Since a regular heart rhythm is required, **the system fails in the presence of arrhythmias** (e.g. atrial fibrillation).

**In case of sudden fluctuations** in hemodynamics, it is **necessary to re-calibrate** using thermodilution (standard calibration is performed after at least 6 hours).

The PiCCO system uses thermodilution curve analysis and knowledge of individual volumes (end-diastolic volumes of both chambers and atria) from thermodilution measurements between the application site and the detection of the tracer (solution of known temperature). Further, from the volumes determined by thermodilution techniques between the site of application and detection, the "**extravascular lung water**" (EVLW) can be calculated to quantify pulmonary edema. This is the difference between total lung fluid content (pulmonary thermal volume, PTV) and intravascular lung fluid (pulmonary blood volume, PBV).

#### LiDCCO System

A variant of this system is a **system using lithium chloride dilution** (LiDCO) instead of thermodilution. Calibration is performed by detecting the presence of LiCl in peripheral arterial blood (a.radialis) after its bolus administration into the venous part of the vasculature. **Cardiac output is** continuously **monitored by subsequent comparison of heart rate curves**.

In addition to routine methods such as CVP or aTK measurements, modern thermodilution methods and the possibility of arterial pressure pulse curve analysis (e.g. the PICCO method) allow more detailed hemodynamic parameters to be determined. For paediatric purposes, the most important are the indexed values of the individual parameters, which are related to the body surface and thus allow comparison between the values of different patients.

### Parameters defining preload

In addition to CVP (the pressure parameter defining right ventricular preload), which is the most commonly used marker of preload, a number of other parameters can be monitored as part of more detailed haemodynamic measurements:

- **global enddiastolic volume** (**GEDV**) indicates the volume of blood contained in all 4 cavities of the heart at the end of diastole
- **intrathoracic blood volume** (**ITBV**) indicates the volume of blood contained in all 4 cavities of the heart at the end of diastole + the volume of blood in the pulmonary vessels

ITBV and GEDV **show greater sensitivity and specificity** to determine cardiac preload than standard CVP and PAWP filling pressures, but also than right ventricular end-diastolic volume calculated by echocardiography. Another advantage of ITBV and GEDV is that **they do not interfere with artificial pulmonary ventilation**. **In children**, as mentioned above, the **indexed values**, i.e. GEDVI and ITBVI, should be used.

n patients on UPV we can use another hemodynamic parameter - **stroke volume variation** (**SVV** - dynamic parameter). SVV reflects changes in cardiac preload in relation to UPV cycles. A rise in SVV may predict the need for volume expansion

### Parametry defining afterload

In practice, the **systemic and pulmonary vascular resistance** are evaluated (based on Ohm's law) as a determinant of afterload. Knowing the values of CO, we can calculate **the value of systemic vascular resistance** (**SVR**):

$$SVR = (MAP - CVP) \times 80 / CO$$

$$PP = MAP - CVP$$

$$SVR = (MAP - CVP) \times 80 / CO = PP \times 80 / CO$$

- **PP** = perfusion pressure; difference between mean arterial pressure and central venous pressure

The indexed SVR value related to body surface area is **SVRI** :

$$SVRI = (MAP - CVP) \times 80 / CI = PP \times 80 / CI$$

Based on these relationships, it is therefore possible to increase cardiac output by reducing vascular resistance, but it also follows that good blood pressure does not necessarily mean good cardiac output - vascular resistance can rise at the same time as cardiac output falls!

By analogy, **for pulmonary vascular resistance**, :

$$PVR = (MPAP - PAOP) \times 80 / CO$$

respectively,

$$PVRI = (MPAP - PAOP) \times 80 / CI$$

**MPAP** is the **mean pulmonary artery pressure** and **PAOP** is **pulmonary artery opening pressure**. ⚠.

### Extravascular pulmonary water

*Extravascular lung water* (**EVLW**) and its index EVLWI indicate the **volume of free water** in the lungs and allow bedside quantification of the severity of pulmonary edema. In addition to pulmonary edema, it correlates with the severity of ARDS or the duration of UPV. It is a **better indicator of pulmonary oedema** than chest X-ray.

| Hemodynamic parameter | | | |
|---|---|---|---|
| | **Parameter** | **Unit** | **Standard** |
| **Cardiac Output** | CI (cardiac index) | l/min/$m^2$ | 3,0–4,5 (5,5) |
| | SVI (stroke volume index) | ml/$m^2$ | 30–60 |
| | SvcO2 | % | 65–75 % |
| **Preload** | CVP (central venous pressure)<br>*pressure parameter* | cm $H_2O$ | 3–10 |
| | GEDVI (global enddiastolic volume index)<br>*volume parameter* | ml/$m^2$ | 390–590 |
| | ITBVI (intrathoracic blood volume index)<br>volume *parameter* | ml/$m^2$ | 550–850 |
| | SVV (stroke volume variation)<br>dynamic *parameter* | % | ≤ 10 |
| | PPV (pulse pressure variation)<br>dynamic *parameter* | % | ≤ 10 |
| **Lungs** | EVLWI (extravascular lung water index) | ml/kg | 3,0–7,0 |
| **Afterload** | MAP | torr (mmHg) | dle věku |
| | SVRI (systemic vascular resistance index) | dyne.s.cm/5.m/2 | 800–1600<br>1600–2400 (u dospělých) |
| | PVRI (pulmonary vascular resistance index) | dyne.s.cm/5.m/2 | 250–430 |
| **Contractility** | CFI | l/min | 4,5–6,5 |
| | GEF | % | 25–35 |
| | EF (ejacuataion fraction) | % | 55–75 |

### Contractility

Contractility is the **intrinsic inotropic activity of the myocardium independent of preload and afterload**. It is influenced by ionized calcium, compliance and myocardial delivery of energy substrates.

An **indicator** of contractility is **the ability to exert pressure per unit time**, in practice it is used :

- values of left and right ventricular stroke work: **LVSW** and **RVSW** (left/right ventricular stroke work)

$$\mathbf{LVSW = 0{,}0136 \times SV \times (MAP - PAOP)}$$

$$\mathbf{RVSW = 0{,}0136 \times SV \times (MPAP - CVP)}$$

- global ejection fraction (**GEF**) and cardiac function index (**CFI**) derived from parameters measured by the PiCCO system;
- the level of myocardial contractility can also be estimated from the steepness of the rise in the pulse curve during direct measurement of arterial pressure.

### Cardiac Output

Within the limits of more detailed haemodynamics we are able to determine the **stroke volume (SV)**. Based on this value, we can calculate **cardiac output (CO)**, which is the product of stroke volume and heart rate:

$$\mathbf{CO = HR \times SV}$$

**By converting to body surface area we get cardiac index = CI.**

**Calculation of CO using Fick's formula:**

$$\mathbf{CO = \{VO_2 / (CaO_2 - CvO_2)\} \times 10}$$

### BP measurements in pulmonary artery wedging, PAWP

The **PAWP value is measured with a Swan-Ganz catheter**. It is the resultant of pulmonary resistance and left heart function. Its values are close to the left atrial pressure. **It is used to determine the exact CI**. It has a rare application in paediatrics.

- **reference values**: 6–16 cm $H_2O$ (ideally 7–15 cm $H_2O$)

**Indications for Swan-Ganz catheter insertion:**

- unclear intravascular volume
- PEEP > 12 cm $H_2O$
- heart failure
- need for intensive inotropic myocardial support

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