

Shock (pediatrics)

Shock is defined as a disproportion between the need and supply of oxygen to the tissues. It is a micro and/or macrocirculation disorder that leads to failure of tissue perfusion, oxygen consumption and energy metabolism of cells. Insufficient oxygen supply leads to a shift of aerobic metabolism to a less efficient anaerobic metabolism, lactic acidosis occurs. The Brain does not have the capacity for anaerobic metabolism, and that is why it is seriously affected when there is a lack of oxygen.

The most common form of **shock in children** is hypovolemic and septic shock.

As stated above, the measure of shock is perfusion impairment. The following table gives the answer to the question of which clinical condition can already be considered as shock.

Symptomatology of reduced perfusion (Heart Diseases in Infants, Children and Adolescents 1994)

	Reduced perfusion	Significantly reduced perfusion (preshock state)	Severely reduced perfusion (shock state)
CNS	0	restlessness, anxiety or apathy	agitation/confusion, severe impaired consciousness to coma
Respiration	0	mild tachypnea	marked tachypnea
Metabolism	0	compensated MAC	decompensated MAC
GIT	0	reduced motility	ileus
Kidneys	increased osmolality of urine	oliguria	anuria
Skin	slowed capillary return	cool acres	cool, marbled to cyanotic acres
Cardiovascular system	tachycardia	marked tachycardia, reduced peripheral pulsation	pronounced tachycardia, hypotension, palpable only pulsations over the large arteries

Oxygen delivery (oxygen delivery, DO_2)

Oxygen delivery (DO_2) is directly proportional to cardiac output and the oxygen content in arterial blood (arterial oxygen content, CaO_2). For pediatrics, we always choose indexed values, i.e. values related to the body surface.

- $DO_2 \text{ (index)} = CI \times CaO_2 \times 10$
- $CI = HR \times SV$
- $CaO_2 = (Hb \times 1.34 \times SaO_2) + (0.003 \times PaO_2)$
- $CvO_2 = (Hb \times 1.34 \times SvO_2) + (0.003 \times PvO_2)$
- $a - v DO_2 = CaO_2 - CvO_2$
- DO_2 = oxygen delivery, represents oxygen delivered by tissue per minute, reference values $DO_2 = 550\text{-}650 \text{ ml/min/m}^2$
- SV = stroke volume = pulse volume
- HR = heart rate = heart rate
- CI = cardiac index = cardiac output related to a unit of body surface area)
- CaO_2 = oxygen content in arterial blood, reference values $CaO_2 = 17 - 20 \text{ Jr.}$
- CvO_2 = oxygen content in mixed venous blood, reference values $CvO_2 = 12\text{-}15 \text{ ml}$
- SaO_2 = saturation of arterial blood O_2 , it is reported as $SaO_2 / 100$
- SvO_2 = saturation of mixed venous blood, it is given as $SvO_2 / 100$
- PaO_2 = partial pressure of oxygen in arterial blood, it is given in torrs
- PvO_2 = partial pressure of oxygen in mixed venous blood, it is given in torr
- $a - v DO_2$ = arteriovenous oxygen content difference (oxygen content difference), reference values $a - v DO_2 = 3 - 5 \text{ ml/dl}$
- Hb = hemoglobin, it is given in the amount of g/dl

Oxygen consumption (oxygen consumption, oxygen uptake, VO_2)

The rate of O_2 consumption is VO_2 (oxygen consumption, oxygen uptake), reference values $VO_2 \text{ (index)} = 120 - 200 \text{ ml/min/m}^2$

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

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

- oxygen content in mixed venous blood CvO_2
- O_2 extraction (oxygen extraction, O_2ER), i.e. the ratio between the amount of consumed and delivered oxygen VO_2 / DO_2 , which is expressed as a percentage. Normal extraction values are around 25%, but with significantly increased tissue demand or reduced perfusion, O_2 extraction can rise to 50%. As part of the shock states, we try to keep the oxygen extraction below 30%.

$$\text{O}_2\text{ER} = \text{VO}_2 / \text{DO}_2$$

Both CvO_2 and O_2ER depend on the mixed venous blood saturation values of SvO_2 and cardiac CO output. CO/CI depends on heart rate value and stroke volume (the latter is determined by preload, afterload and contractility). Increasing heart rate, improving myocardial contractility and relaxation in diastole, optimizing preload and afterload increase CO/CI. Oxygen carrying capacity can be improved by optimizing hematocrit. For critically ill children, but in a stable condition, we consider a hemoglobin value of 70 g/l as the borderline for transfusion. By improving all these parameters, DO_2 can be increased. In some specific situations (fever, high flow stage sepsis, trauma, thyrotoxicosis) metabolic needs can exceed even normal DO_2 .

Basic physiological calculations of ventilation

	unit	standard
C_aO_2	ml	17-20
C_vO_2	ml	12-15
$\alpha\text{-vDO}_2$	ml/dL	3-5
DO_2 (index)	ml/min/m ²	550-650
VO_2 (index)	ml/min/m ²	120-200
O_2ER	%	20-35

With insufficient supply of O_2 , some cells can cover their energy needs by anaerobic glycolysis, i.e. by converting glucose into lactic acid. However, the energy efficiency is negligible (2 ATP per glucose compared to 36 ATP in oxidative combustion). The dissociation of lactic acid into H^+ and lactate then leads to the development of MAC. The lack of energy first causes the limitation of cell function and finally their irreversible damage. Likewise, shock is a condition caused by a severe and extensive reduction in effective tissue perfusion leading first to reversible, then irreversible cell damage. Effective tissue perfusion can be reduced globally, i.e. by reducing the minute cardiac output or increasing inefficient regional perfusion based on blood flow distribution disorders or substrate utilization disorders at the cellular level.

Factors that determine the effectiveness of tissue perfusion can understandably cause shock even if they are severely affected in isolation. In most cases, especially in later forms of shock, these are manifestations of multifactorial damage. **Determinants of effective tissue perfusion** can be classified into 4 main categories:

1. quantities affecting the performance of the heart muscle;
2. effective blood volume;
3. factors affecting vascular resistance and permeability (and thus the distribution of circulating blood volume);
4. factors affecting the availability of oxygen at the cellular level.

From a practical point of view, it should be noted that shock can be present with normal, decreased or increased cardiac output, with normal, decreased or increased BP.

In children, at first it is often hypodynamic shock = low flow with reduced CO/CI and, conversely, high peripheral systemic resistance (the exception is the initial phase of septic shock, hepatic failure, thyrotoxic crisis, etc.).

Physiology and Pathophysiology Notes

Vascular tone control

Vasomotor tone of vessels is affected by several mechanisms: nervous and humoral factors, composition of blood gases, local metabolic regulation, function of the endothelium and smooth muscles of the vascular media.

A mechanism that regulates vascular resistance in one region may be completely without effect in another region. E.g. in the context of hypovolemic shock, the perfusion of the heart and brain is preserved, and on the contrary, it is reduced in the muscles, skin and splanchnic.

Neuromodulation of vascular tone

Receptors to which noradrenaline, acetylcholine or neuropeptides bind are represented throughout the circulation. However, the distribution of receptors is organ-specific, allowing rapid and coordinated redistribution of blood flow in response to hypoxia, postural changes, and hemorrhage. In all organs, the nerve endings of efferent nerves also contain nonadrenergic and noncholinergic peptides, e.g. neuropeptide Y, VIP (vasoactive intestinal peptide), substance P, calcitonin gene-related peptide (CGRP). Most of these peptides, with the exception of neuropeptide Y, lead to vasodilation and help regulate regional perfusion.

Humoral regulation of vascular tone

Humoral factors that regulate vascular tone include the renin-angiotensin-aldosterone system (RAAS), ADH, bradykinin, histamine, serotonin, thyroxine, natriuretic peptides, and a number of others. These factors affect vascular tone in a direct and indirect way. These factors tend to decrease in concentration during hypertension,

congestive heart failure or shock, and their antagonists are often used in the therapy of these conditions. Certain factors such as histamine, serotonin, thyroxine probably affect vascular resistance only in pathological conditions and do not apply in physiological conditions.

Angiotensin plays a special role in blood pressure homeostasis. Hypovolemia leads to increased production of renin in the kidney, which converts angiotensinogen to angiotensin I. Angiotensin I is converted to active angiotensin II by angiotensin-converting enzyme (ACE) in the endothelium, especially in the pulmonary bed. However, angiotensin II can be produced directly from renin locally in the heart and vessel wall. Angiotensin II causes generalized vasoconstriction in the systemic and pulmonary circulation, but locally stimulates the release of vasodilating prostaglandins in the kidneys and lungs.

Aldosterone was primarily known for its effect on sodium and potassium balance. Its concentration increases with the release of renin. In patients with congestive heart failure, we find its high concentrations both due to dilutional hyponatremia and reduced degradation in the liver. High concentrations, which are a sign of the body's initially compensatory reaction overshooting, are harmful to the cardiovascular system. Inhibition of aldosterone by spironolactone appears to be of great benefit in the therapy of heart failure.

ADH (antidiuretic hormone, vasopressin) has an antidiuretic effect and at the same time causes vasoconstriction, low concentrations of ADH lead to vasodilation in the coronary, cerebral and pulmonary basins. The concentration of ADH decreases in septic shock, on the contrary, it increases in hypovolemia, congestive heart failure and liver cirrhosis. Selective ADH antagonists allow excretion of free water without ion excretion and are useful in the treatment of hypervolemia in patients with congestive heart failure, cirrhosis, or SIADH. **Bradykinin is a potent vasodilator in the pulmonary and systemic circulation. It is released locally from kallikrein by the action of proteolytic enzymes as a result of tissue damage.**

Histamine is also released from mast cells in response to tissue damage. It is a potent vasodilator in the systemic circulation, but leads to vasoconstriction in the pulmonary circulation. It also increases vascular permeability.

Natriuretic peptides are released from the heart as it distends in congestive failure. They cause vasodilation and increase natriuresis. ANP (atrial natriuretic peptide) is released especially in the atria, BNP (brain natriuretic peptide) from the ventricles and C-natriopeptide from the cardiac endothelium. Recombinant BNP (nesiritide) is more effective than dobutamine in the treatment of acute severe congestive heart failure.

Serotonin causes vasodilation or vasoconstriction depending on the type of serotonin receptor affected.

Effect of blood gases on vascular tone

The values of $p\text{aO}_2$ and $p\text{aCO}_2$ are dependent on the quality of tissue perfusion. Hypoxia and hypercapnia that accompany hypoperfusion are associated with a vasodilator effect.

Local metabolic regulation of vascular tone

Local metabolic regulation of vasomotor tone represents an ideal homeostatic mechanism. With its help, the metabolic needs of tissues directly affect local perfusion. E.g. adenosine, which accumulates locally during high tissue metabolism and marginal tissue oxygenation, leads to vasodilation in the coronary basin, striated muscle, splanchnic, and cerebral circulation.

Regulation of vascular tone through the endothelium

The vascular endothelium plays a prominent role in the regulation of vascular tone. In addition to influencing vasoactive eicosanoids and their role in angiotensin metabolism, the endothelium produces a number of vasoactive substances. **Nitric oxide** (NO; a potent vasodilator) and **endothelins** are among the most important. Endothelins (ET-1, ET-2, ET-3) represent a family of vasoactive substances. ET-1 is a potent vasoconstrictor, otherwise the effect of endothelins depends on acting on two types of receptors: ET-A receptors located in vascular smooth muscle mediate vasoconstriction, ET-B on endothelial cells mediate vasodilation. Endothelin antagonists, such as bosentan, are beginning to be used therapeutically.

Regulation of vascular tone through smooth muscle of the vascular media

Changes in vascular smooth muscle tension are a response to distension or an increase in transmural pressure. An increase in vascular flow leads to local vasoconstriction. The opposite reaction is caused by a decrease in vascular flow.

Autoregulation

In all organs, if the perfusion pressure is suddenly increased or decreased while oxygen consumption is maintained constant, the flow rate will increase or decrease temporarily, but then return to the previous value. This phenomenon is called **autoregulation**.

The myogenic tonic response partially explains this phenomenon, but it is not the only mechanism. Some scientists believe that tissues have oxygen sensors that respond to transient increases or decreases in oxygen supply. Other researchers argue that the process of autoregulation is mediated by increased or decreased release of nitric oxide, which is transported to the tissues via hemoglobin as S-nitrosohemoglobin or by the release of ATP from erythrocytes.

Some autoregulatory mechanisms are specific to individual microcirculations (eg, renal). Self-regulatory mechanisms differ in individual organs.

Pulmonary circulation

In the fetus, the pulmonary circulation has the character of a systemic circulation, the pulmonary arteries have a strongly developed medial smooth muscle. This is the reason for the high pulmonary resistance in the fetus even early postnatally. After birth, within a few weeks, the musculature of the mediastinum involutes and progressively decreases the resistance of the pulmonary canal. During the first 24 hours after birth, the pulmonary arterial pressure drops to a value of approx. 50% of the mean arterial pressure, and the pulmonary circulation remains at low pressure with low vascular resistance. Due to the intimate relationship between small pulmonary vessels and alveoli, intra-alveolar pressure affects pulmonary flow, especially in patients with artificial pulmonary ventilation.

The most important factors that influence pulmonary vascular resistance in the postnatal period are the **oxygenation** rate and the **pH value**. When the oxygen tension in the alveoli decreases, hypoxic pulmonary vasoconstriction develops in a given lung segment. The goal is to redistribute blood flow to well-ventilated areas of the lung and thus maintain a favorable ventilation/perfusion (V/Q) ratio. This phenomenon is highly specific to the pulmonary circulation, as the blood vessels of other organs (including the CNS) respond to hypoxia by vasodilation. Acidosis potentiates hypoxic pulmonary vasoconstriction, alkalosis reduces it. The actual mechanism of the pH-mediated response of the pulmonary vascular bed is not fully understood, but it appears independent of pCO₂. The mechanism of alveolar hyperoxia and alkalosis is often used to induce pulmonary vasodilation in patients with pulmonary hypertension. Hypocapnia and RAL in turn lead to vasoconstriction in the systemic circulation, which may have adverse consequences in CNS and cardiac perfusion.

Selective pulmonary vasodilators are oxygen and inhaled nitric oxide (iNO).

Coronary circulation

The right and left coronary arteries arise from the sinus of Valsalva and run along the surface of the heart. Perfusion of the heart takes place during diastole. In tachycardia, diastole shortens, myocardial perfusion decreases and ischemia may occur. Under normal circumstances, right ventricular perfusion takes place even during systole due to the low pressures. The coronary circulation also exhibits autoregulation. An increase in pressure causes vasoconstriction, a decrease in pressure leads to vasodilation. When the pressure drops below 40 torr, the autoregulation mechanism is no longer effective and ischemia develops.

Renal circulation

Approximately 20% of cardiac output flows through the kidneys, although the weight of the kidneys represents approximately 0.5% of the total body weight. The reason is to promote sufficient glomerular filtration to maintain water and solute homeostasis. At the end of the arterial river we find afferent arterioles, which open into the capillary network within the glomerulus. Glomerular capillaries form in the outflow part into an efferent arteriole, which subsequently creates a secondary capillary system (peritubular capillaries). The increased hydrostatic pressure inside the glomerular capillaries promotes filtration, while the much lower pressure inside the peritubular capillaries aids reabsorption. Changes in the resistance of afferent and efferent arterioles allow for dynamic changes in renal function in response to fluid and solute needs.

Renal flow is determined by the difference between renal arterial pressure (corresponding to systemic arterial pressure) and renal venous pressure. Renal vasomotility is influenced by both external factors (sympathoadrenal system, natriuretic peptides, RAAS) and internal factors that are responsible for autoregulation of renal flow in response to changes in renal perfusion pressure (RPP). **Glomerular filtration** is given by glomerular filtration pressure (glomerular filtration pressure, GFP). GFP depends on RPP and the balance between arterial tone of afferent and efferent arterioles. Specifically, vasoconstriction of the vas efferens increases glomerular filtration, vasoconstriction of the vas afferens decreases glomerular filtration.

Endothelial function

The endothelium performs a number of functions:

- Endothelial cells play an important role in the body's defenses - they enable the adhesion and subsequent extravasation of leukocytes through molecules - selectins, adherins, integrins.
- The endothelium is intimately linked to the function of the coagulation system. It has the ability to produce procoagulant factors (platelet activating factor = PAF, von Willebrand factor, fibronectin, ff. V and X) and anticoagulant factors (heparan, dermatan sulfate, thrombomodulin) and by producing NO and PGI₂ it inhibits platelet aggregation and degradation.
- The endothelium regulates capillary permeability by producing endothelin 1 (ET-1), which increases permeability, and by producing PGE₁, which decreases permeability.

Relationship between flow, pressure and vascular resistance

From the point of view of the diagnosis of the shock state syndrome, the parameter of perfusion efficiency with subsequent manifestations of organ dysfunction is absolutely essential.

Organ perfusion (flow) is determined by the pressure of flowing blood and vascular resistance. Under normal circumstances, a sufficient pressure gradient is present and vasomotor control regulates individual organ perfusion according to metabolic need. Under resting conditions, only part of the vascular system is open. In most cases, the onset of shock syndrome is linked to a drop in pressure and subsequent failure of organ perfusion. However, the level of blood pressure is not the only determinant of perfusion. With high blood pressure, but at the same time high vascular resistance, tissue perfusion is also not sufficient.

Thus, the severity of the shock state is primarily determined by the depth of the tissue perfusion disorder. Good tissue perfusion ensures an adequate supply of nutrients and oxygen at the cellular level. However, we must always relate tissue perfusion to the current needs of the organism. In conditions with hyperkinetic circulation (thyrotoxicosis , high flow phase of sepsis , liver failure), even "normal" perfusion may be insufficient, as the tissues show a higher need for oxygen and energy substrates than the organism is able to provide at the given moment. Simply put, demand for O₂ exceeds supply. **The parameters of adequate oxygen supply** are:

- absence of hypotension ,
- warm periphery with good capillary return,
- diuresis > 1 ml/kg/hour,
- normal consciousness _
- lactate < 2 mmol/l,
- With vc O₂ > 70%.

The decisive parameter determining the regional perfusion Q is the blood flow generating the dynamic blood pressure. According to **Poiseuille's law** :

$$Q = (P_{in} - P_{out}) / R$$

where Q is tissue flow, P_{in} is input pressure, P_{out} is output pressure, R is resistance. In the case of a simple tube, this is determined by the diameter of the tube, its length, it is inversely proportional to the fourth power of the radius and directly proportional to the value of the viscosity of the flowing fluid.

The severity of the shock state is determined primarily by the depth of the tissue perfusion disorder.

Thus, regional perfusion is determined by blood pressure and regional resistance. The resistance of various areas of the systemic circulation and the cardiac output determine the value of the systemic arterial pressure. Local factors controlling regional perfusion may have different effects than control mechanisms regulating systemic arterial pressure. For example, hypoxia leads to vasoconstriction by activating central baroreceptors, but vasodilation occurs in the periphery. If we take into account the whole body perfusion Q_{co} and neglect P_{out} (the venous pressure is small compared to the value of the arterial pressure), we get the equation:

$$P_a = Q_{co} \times R_{sv}$$

where P_a is arterial pressure, Q is cardiac output, R_{sv} is systemic vascular resistance. For a more accurate determination of tissue perfusion, however, we take venous pressure into account (P_{out} = CVP) in a situation where we want to define the perfusion pressure parameter = perfusion pressure PerP. This corresponds to the difference between mean arterial pressure MAP and central venous pressure CVP. So:

$$PerP = MAP - CVP$$

Limit values of perfusion pressure in cm H₂O (mm Hg - rounded values)

Children's age	Perfusion pressure in cm H ₂ O (mm Hg)
newborns	55 (40)
infants	60 (45)
toddlers	65 (50)
preschoolers	65 (50)
school children	65 (50)

However, perfusion pressure is not the only important parameter, it is necessary to simultaneously maintain S_{vc} O₂ > 70% with the help of transfusion or inotropic support, lactate level < 2 mmol/l, good peripheral perfusion, diuresis > 1 ml/kg/h.

In conditions with **intra-abdominal hypertension** (ascites , ileus) the perfusion pressure is equal to the difference MAP - IAP (intra-abdominal pressure). The relationship between flow, pressure and resistance can also be applied to individual organs. In the kidneys, for example, renal flow Q = (mean renal arterial pressure - mean renal venous pressure) / renal vascular resistance.

Some organs, as already mentioned above, have the ability of **vasomotor autoregulation** , which maintains blood flow even at low blood pressure. This works up to a certain critical point where the perfusion pressure is reduced below a value where sufficient flow can still be maintained in the organ. The purpose of shock treatment is

to maintain the perfusion pressure above a given critical point (but be careful - the critical point is not a fixed value, it is strictly individual).

The kidneys are a textbook example: the kidneys need the second highest blood flow. At the same time, accurate determination of diuresis and creatinine clearance is very easy and enables the quality of renal perfusion to be assessed. And it is the quality of renal perfusion that provides a picture of perfusion in other visceral organs as well. The kidneys thus represent a kind of "window" into organ perfusion. Therefore, an accurate assessment of diuresis in every critically ill patient is absolutely essential!

If hypotension occurs, it is the result of low cardiac output or low vascular resistance. From this point of view, it is possible to divide shock states into only two basic categories - **shock with low cardiac output** and **shock with low systemic vascular resistance** .

Age-Specific Vital Signs and Laboratory Values (Pediatric Critical Care 2005)

Age	Heart rate (beats per minute)	Respiratory rate (breaths per minute)	Leukocytes (leu x 10/3 in ml)	Systolic BP (mm Hg)
0 days - 1 week	> 180 x < 100	> 50	> 34	< 65
1 week - 1 month	> 180 x < 90	> 40	> 19.5 x < 5	< 75
1 month - 1 year	> 180 x < 90	> 35	> 17.5 x < 5	< 100
25 years	> 140	> 22	> 15.5 x < 6	< 94
6 - 12 years	> 130	> 18	> 13.5 x < 4.5	< 105
13 - 18 years	>110	> 14	> 11 x < 4.5	< 117

Note: the values shown represent the 5th or 95th percentile for the given age group. Any shock state can result in a systemic inflammatory response (SIRS). Unattenuated cascades of cytokines, complement and coagulation lead to a violation of the integrity of the vascular wall and an increase in the adhesiveness of the endothelium. The result is then extravasation, vasodilatation, thrombosis , tissue hypoxia. Lactic acidosis is an expression of mitochondrial hypoxia.

Shock classification

Shock classification often describes its own cause of shock condition, i.e. bleeding, trauma, sepsis etc. This terminology connected with the underlying cause is, of course, acceptable. However, using the selected criteria, it is possible to determine the shock states of the 5 main categories and to distinguish:

- **Hypovolemic shock**
- **Distributional shock**
- **Obstructive shock**
- **Cardiogenic shock**
- **Dissociative shock**

According to the Nelson Textbook of Pediatrics 2007, septic shock is excluded from the distribution shock group as a separate type of shock. This is due to its mixed nature of pathogenesis, where in addition to a distribution disorder we find hypovolemia ("third spacing") and cardiogenic depression (the effect of endotoxin, cytokines, etc.). Even outside of the above division, this only proves that in the clinical picture we often distinguish '*mixed shock*'. It is a combination of two, sometimes three basic types. One type usually predominates in this mixed image. A typical example is traumatic shock, which is most often a combination of hypovolemic and distributional shock, but depending on the nature of the injury, it can also be a cardiogenic or obstructive shock.

Hemodynamic determinants of shock states (Fuhrman, Zimmerman – Pediatric Critical Care, 1998)

TYPE OF SHOCK	Cardiac index	SVRI	MAP	Pulmonary Capillary Wedge Pressure	CVP
Hypovolemic	↓	↑	↔ or ↓	↓↓↓	↓↓↓
Cardiogenic - systolic dysfunction	↓↓	↑↑↑	↔ or ↓	↑↑	↑↑
Cardiogenic - diastolic dysfunction	↔	↑↑	↔	↑↑	↑
Obstructive	↓	↑	↔ or ↓	↑↑	↑↑
Distributional	↑↑	↓↓↓	↔ or ↓	↔ or ↓	↔ or ↓
Sepsis - early phase	↑↑↑	↓↓↓	↔ or ↓	↓	↓
Sepsis - late phase	↓↓	↑↑	↓↓	↑	↔ or ↓

Hypovolemic shock

Pathogenesis and characteristics

Hypovolemic shock is *the most common shock condition in children*. Hypovolemic shock is an absolute loss of effective circulating volume. Hypovolemia leads to a reduction in preload, subsequently stroke volume and cardiac output. Activation of peripheral and central baroreceptors results in the release of catecholamines, vasoconstriction and tachycardia. These compensatory mechanisms are effective for an acute loss of 10-15% blood volume. If the loss exceeds 20-25%, these mechanisms cease to be effective, the CO/CI decreases. Blood pressure is often normal due to an extensive increase in peripheral vascular resistance. The extraction of oxygen in the tissues increases, i.e. the arteriovenous difference expands. When compensatory mechanisms fail, tissue hypoxia and lactate MAC develop. hypotension, impaired consciousness, oligoanuria occurs. The terminal phase is characterized by myocardial dysfunction and cell death.

From a practical point of view, it is important to emphasize that blood pressure decreases only in the pre-terminal phase, after all compensatory mechanisms have been exhausted, so hypotension is in no case an early marker of the severity of the condition. On the contrary, attention should be paid from the beginning to clinical signs such as tachycardia, cold akra with weakened pulsations and prolonged capillary return, reduced diuresis.

Uncomplicated and timely treated hypovolemic shock does not lead to the development of capillary leak syndrome. However, patients with burns, with trauma of soft tissues are at risk. Also, severe and prolonged hypovolemic shock leads to capillary wall damage.

Hypovolemic shock is characterized by: a high systemic vascular resistance index (SVRI), a decrease in CVP and CI, widening of the AV difference, and late-onset hypotension. Tachycardia, low systolic pressure, and its increase with liver compression predict a good response to volume expansion.

Etiology

- dehydration
- bleeding
- sequestration ECT: paralytic ileus, burns

Therapy

The primary goal is to replenish fluids - crystalloids, colloids or blood. The total amount of fluids administered usually exceeds the absolute volume loss, as the capacity of the vascular space increases and cell membrane dysfunction occurs. As part of the treatment of hemorrhagic shock, administration of a greater proportion of colloids compared to crystalloids, especially plasmas and sufficient substitution with erysma is again in the forefront of interest. A recent recommendation that is already appearing for pediatric patients is the tactic of permissive hypotension, i.e. giving just enough fluid to ensure sufficient tissue perfusion, no more.

Distributive shock

Distributive shock (pediatrics)

Obstructive shock

Pathogenesis and characteristics

Obstructive shock is characterized by obstruction of the outflow of blood from the heart (right ventricular, left ventricular, biventricular obstruction). It is caused by the inability to generate adequate CO/CI despite normal intravascular volume and myocardial function. Filling pressures are elevated, cardiac output is reduced. Obstructive shock in resuscitation care is most commonly encountered with pneumothorax, aggressive artificial pulmonary ventilation, a. pulmonalis embolization, asthmatic stasis, cardiac tamponade, and pulmonary or systemic hypertension. APV has the same effect on preload and subsequently contractility as hypovolaemia and leads to a leftward shift of the Frank-Starling curve. Obstructive shock is characterized by hypotension/hypertension, decreased CO/CI, rise in CVP and PAWP, increase in SVRI.

Etiology

- venous obstruction
- tension pneumothorax
- artificial pulmonary ventilation
- asthma bronchiale
- constrictive pericarditis
- cardiac tamponade
- coarctation of the aorta
- pulmonary embolism
- pulmonary hypertension
- aortic dissection

Cardiac tamponade

Cardiac tamponade is defined as *hemodynamically significant compression of the heart in the pericardial envelope*. The cause is transudate or exudate (*hydropericardium*), blood (*hemopericardium*), or gas in the pericardium (*pneumopericardium*).

The clinical presentation of tamponade is insidious, especially when it occurs against a background of an underlying cause such as malignancy, collagenosis, renal failure or pericarditis.

In the initial phase, the symptomatology is non-specific. When CO/CI decreases, the symptomatology is similar to congestive heart failure, but there is no evidence of congestion on lung X-ray. Physical findings suggestive of tamponade are pulsus paradoxus, narrowing of pulse pressure, a flutter murmur over the pericardium, or weakening of the oesophagus and distension of the jugular veins. The method of choice in diagnosis is echocardiography.

The symptomatology of cardiac tamponade is similar to congestive heart failure, however, there is no evidence of congestion on lung X-ray

. Template:Good Example If not addressed immediately (pericardiocentesis), electromechanical dissociation and death of the patient occurs. Pericardiocentesis is performed under echocardiographic control, blinded only as a last resort. The definitive solution, if needed, is surgical drainage of the pericardium.

Medical therapy cannot replace drainage, but may help us gain more time if pericardiocentesis or surgical drainage are not immediately available. We choose volum-expansion to maintain venoatrial gradients, and we administer inotropics, but these have little effect. Drugs such as diuretics or digoxin are contraindicated. If the patient is on UPV it is necessary to reduce PIP and PEEP.

Cardiogenic shock

Pathogenesis and characteristics

Cardiogenic shock is heart failure of various etiology, most often with a decrease in systolic volume. The hemodynamic picture of cardiogenic shock is essentially the same as that of hypovolemic shock with one very important exception: *the filling pressures of the heart chambers are increased* (similar to obstructive shock).

Clinically the most striking sign of left-sided failure is pulmonary edema and a cold periphery with impaired perfusion (impaired consciousness, oliguria), right ventricular failure in childhood is dominated by hepatomegaly and swellings can form, especially periorbitally. In children, it is most often a case of *bilateral failure*.

The most important **diagnostic** method, which will help to correctly determine the severity of cardiogenic failure, is echocardiography with Doppler imaging and, of course, electrocardiographic examination (in children, 12-lead is practically necessary to distinguish possible artifacts). Based on the mentioned examinations, it is possible to determine the optimal therapy and conditions for further monitoring. These also include the decision to indicate the introduction of invasive monitoring of minute cardiac output, monitoring of oxygenation parameters and pressures in the pulmonary basin. Stabilization of the circulatory situation is also a key prerequisite for successful therapy of the underlying disease in the event that cardiogenic failure is its complication.

In the conditions of resuscitation care, cardiogenic shock is most often caused by tachyarrhythmia or secondary impairment of cardiac functions (sepsis, hypoxia, prolonged hypovolemia, stage cardiopulmonary resuscitation) . Heart failure is the terminal stage of shock states of any etiology. The causes are not fully elucidated, but the influence of specific toxic substances with a direct cardiodepressive effect, myocardial edema, dysfunction of adrenergic receptors, altered movement of calcium in the sarcolemma, impaired coronary flow due to impairment of systolic and diastolic heart function is assumed.

UPV application of positive pressure significantly affects cardiac output. Within the left ventricle, UPV reduces both preload and afterload. Thus, UPV can have an adverse hemodynamic impact in patients with hypovolemia, when the decrease in cardiac output is potentiated. Conversely, in patients with fluid overload or left-sided heart failure, UPV has a favorable hemodynamic effect due to the reduction of preload and afterload. Within the right ventricle, the situation is more complicated. UPV also reduces right ventricular preload, but the effect on afterload is a function of pulmonary vascular resistance. This can be reduced or increased due to UPV, i.e. right ventricular afterload during UPV can be reduced or increased.

When choosing a ventilation mode, we always take into account its potentially negative effects on circulation, including regional perfusion disorders. Events that significantly affect myocardial contractility include the entire complex of mediators systemic inflammatory response of the organism to stress.



For more information see Heart Failure (Paediatrics).

Cardiogenic shock is characterized by: hypotension, decreased CO/CI, rise in CVP and PAWP, increase in SVRI.

Anamnesis	Physical Finding	X-ray of the chest
<ul style="list-style-type: none"> high respiratory effort long feeding time not thriving significant sweating frequent respiratory infections 	<ul style="list-style-type: none"> tachycardia, tachypnea irregular heart rhythm cold acra, weak peripheral pulse physical findings on the lungs: wheezing, crackles dyspnea cough cyanosis sweating hepatomegaly distention of jugular veins peripheral edemas hypotension 	<ul style="list-style-type: none"> cardiomegaly pulmonary venous congestion hyperinflation

Etiology

When dividing by etiology, it is possible to take into account three basic categories:

myopathy

- infective myocarditis
- cardiomyopathy
 - idiopathic x familial dilated cardiomyopathy
 - anthracyclines
 - neuromuscular diseases (m. Duchenne, spinal muscular atrophy, Friedreich's ataxia)
- hypoxic-ischemic cause
- depression of function caused by e.g.
- pharmacological depression (calcium channel blockers, anesthetics)
- in children rarely myocardial infarction (abnormal spacing of coronary arteries, m. Kawasaki)

mechanical involvement of the myocardium

- valve failure (febris rheumatica)
- hypertrophic cardiomyopathy
- VVV cardiac

arrhythmia

- AV Blocks
- tachyarrhythmia: supraventricular, ventricular

Cardiomyopathy

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Last update: Friday, 10 Nov 2023 at 1.12 pm.

Patients with **dilated cardiomyopathy** may be in shock condition.

- Myocarditis** is one of the most common causes of dilated cardiomyopathy in previously healthy children.

Clinical manifestations of myocarditis are multifaceted.

- May be in the foreground
 - myocardial dysfunction,
 - dysrhythmia
 - or may be clinically "silent" cases.

The most common symptoms are

- tachycardia and
- tachypnoe,

The most common life-threatening dysrhythmias are

- supraventricular and
- ventricular tachycardia.

Rarely can we encounter rhythm disorders - AV blocks,

- which lead to bradycardia and hypotension and are also extremely serious.

Approach to a patient with myocarditis or other forms of dilated cardiomyopathy is the same as for patients in cardiogenic shock, but the response to traditional inotropic therapy may not be sufficient. In addition, infusion of catecholamines in these cases can lead to the development of severe dysrhythmias.

- Recommended for diagnosed myocarditis
 - corticoid therapy or better HDIVIG at a total dose of 2 g / kg (1 g / kg / day for 2 days).
 - These medicines may modulate the inflammatory response.
- Rescue therapy is ECMO.

Hypoxic-ischemic impairment

Shock following a severe hypoxic-ischemic event (drowning, ALTE, prolonged CPR) is most often cardiogenic. Shock is characterized by low CO/CI, elevation of both right and left ventricular filling pressures (increased CVP and PAWP), increased SVR and PVR, and increased oxygen extraction index. In most patients, arterial pressure is elevated due to increased peripheral vascular resistance. Studies have well documented the development of both systolic and diastolic myocardial dysfunction after successful cardiopulmonary resuscitation.

Therapy

In the therapy of cardiac arrhythmias, it is necessary to choose an antiarrhythmic drug with the least cardiodepressive effect, reduce the metabolic demands of the organism and the myocardium by effective analgesia and therapy of febrile states. A basic condition for the adjustment of myocardial function is also the care of the internal environment, especially acid-base balance, blood gases and electrolyte balance with a focus on the prevention of disturbances in the levels of potassium, calcium and [[magnesium]] at. Therapy with inodilators and inoconstrictors must always be titrated and flexible. The sufficiency of cardiac output is a relative quantity and must always be related to a specific metabolic situation and type of disease.

When developing a shock state in the youngest children (newborns, infants < 4 months), we must first think of sepsis or heart failure (unrecognized VVV or prolonged tachydysrhythmia)!

Dissociative shock

Dissociative Shock (Pediatrics)

Monitoring/diagnostic management

Circulation and ventilation

It is necessary to monitor the heart rate, blood pressure invasively – IBP (monitor MAP and perfusion pressure values), via CVK then CVP and S_{vO_2} , continuous ECG and pulse oximetry monitoring.

Indications for the introduction of a Swan-Ganz catheter are extremely rare in pediatrics (severe form ARDS using PEEP > 10 cmH₂O, monitoring of patients after some corrections [[Congenital heart defects|VVV heart]]). A Swan-Ganz catheter is also considered in patients who remain in shock despite pressure-correcting therapy but S_{vO_2} is < 70%. Due to the invasiveness and risk of introducing a pulmonary catheter, semi-invasive options for measuring cardiac output are clearly preferred today, e.g. the PiCCO method, which also enables the determination and calculation of other hemodynamically important parameters.

We intermittently check blood gases and ABR from arterial blood (arterial line). The advantage is etCO₂ monitoring during UPV, which allows to reduce the frequency of blood sampling.

As part of the ventilation, we then monitor the respiratory frequency and, when applying UPV, a number of parameters depending on the use of pressure or volume ventilation. But we always follow $P_{Fi} = pO_2 / FiO_2$, oxygenation index = $(FiO_2 \times P_{maw}) / pO_2$, lung compliance and resistance, Vd/Vt parameter.

Standard examinations are chest X-ray, echocardiography and ECG (12-lead recording). In the intensive care environment, this is the so-called **bed-side monitoring**.

Non-invasive blood pressure monitoring (NIBP)

For the measurement of **non-invasive monitoring of blood pressure**, we have a classical auscultation method with mercury tonometer, Doppler technique and oscillometric determination.

The **Auscultation method** has a disadvantage for the youngest children, for the non-cooperating and, if necessary, for frequent measurements.

Doppler technique is suitable for young children and for conditions with impaired perfusion. A small Doppler probe is located above the radial or brachial artery. Blood movement is excellently sensed by sensitive ultrasound. The cuff placed on the upper arm is inflated until the Doppler signal disappears completely. It is then slowly released. Systolic pressure is subtracted when the first Doppler signal appears, diastolic pressure is subtracted when signal length and quality decrease. The correlation with the pressure measured directly intraarterially is good, but the method is not suitable for continuous measurement.

The oscillometric method is easy to implement. The principle is that when the cuff is inflated, the blood flow in the artery causes oscillations. If the cuff pressure begins to drop, the device registers systolic and diastolic blood pressure and mean arterial pressure.

It always depends on the adequate width of the cuff: too narrow a cuff leads to the measurement of falsely high values, too wide cuff to measure falsely low values of BP (in this case, however, the significance of the error is only small).

All techniques have limitations in conditions with a significant decrease in cardiac output, in severe hypotension or systemic vasoconstriction, in conditions with generalized edema, in extreme obesity.

Pulse pressure (PulP)

Pulse pressure (pediatrics)

Invasive blood pressure monitoring (IBP)

Arterial cannulation, in addition to the benefit of continuous blood pressure monitoring, may be helpful in assessing blood pressure. A normal arterial pulse curve has a sharp rise during the rapid ejection phase. It is followed by a phase of slow ejection, which appears as a plateau with a subsequent drop in arterial pressure. A dirotic finding indicates the end of ejection and closure of the aortic valve. The subsequent drop in arterial pressure during diastole is attributed to aortic run-off (see pulse pressure). We see a reduction in pulse amplitude (similar to pulse pressure) in patients with reduced cardiac output. A flat onset of the curve shape during the fast ejection phase is indicative of a contractility disorder. We see an increase in pulse amplitude in conditions with hyperkinetic circulation.

Hemodynamics

In addition to routine methods such as CVP or IBP measurements, modern thermodilution methods and the ability to **analyze the arterial pressure pulse curve** (eg. the PiCCO method) make it possible to determine more detailed hemodynamic parameters. Thermodilution methods are proving to be more accurate than ultrasound determination of cardiac output (EBM data). For the needs of pediatrics, the most important is the indexed values of individual parameters, which are related to the body surface and thus allow comparison between the values of patients of different age groups.

Parameters defining the preload

In addition to CVP (pressure parameter defining the right ventricular preload), which is the most commonly used preload marker, we can monitor a number of other parameters in more detailed hemodynamic measurements:

- *global end-diastolic volume* (GEDVI) indicates the volume of blood contained in all 4 cavities of the heart at the end of diastole,
- *intrathoracic blood volume* (ITBVI) indicates the volume of blood contained in all 4 heart cavities at the end of diastole + blood volume in the pulmonary vessels ($ITBVI = 1.25 \times GEDVI$).

ITBVI and GEDVI show greater sensitivity and specificity in determining cardiac preload than standard CVP and PAWP, but also right ventricular end-diastolic volume calculated by echocardiography. Another advantage of ITBVI and GEDVI is that they do not interfere with artificial lung ventilation. Indexed values must be used for children.

In patients with UPV, we can use another parameter of hemodynamics – (*stroke volume variation*, SVV – dynamic parameter). SVV reflects changes in cardiac preload depending on UPV cycles. An increase in the SVV value predicts the need for volume expansion.

Parameters defining afterload

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

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

$$PerP = MAP - CVP$$

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

Where $PerP = \text{perfusion pressure}$, is the difference between mean arterial pressure and central venous pressure. The indexed value of SVR related to body area is SVRI:

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

Based on these relationships, it is possible to increase cardiac output by reducing vascular resistance. At the same time, however, it follows that good blood pressure may not indicate good cardiac output (vascular resistance may increase with decreasing cardiac output).

The same applies to pulmonary vascular resistance :

$$\text{PVR} = (\text{MPAP} - \text{PAOP}) \times 80 / \text{CO}, \text{ resp. } \text{PVRI} = (\text{MPAP} - \text{PAOP}) \times 80 / \text{CI}$$

MPAP is *mean pulmonary artery pressure* and PAOP is *pulmonary artery opening pressure*; (Cave!: confused with a pressure wedge. PAWP pulmonary, Pulmonary artery wedge pressure).

Extravascular lung water

Extravascular lung water (EVLW) indicates the volume of free water in the lungs and allows bedside quantification of the severity of pulmonary edema. In addition to pulmonary edema, it correlates with the severity of ARDS or the length of UPV. It is a better indicator of pulmonary edema than a chest x-ray.

Contractility

Contractility is the intrinsic inotropic activity of the myocardium independent of preload and afterload. Its exact determination is very difficult. It is affected by ionized calcium, compliance and the supply of myocardial energy substrates. An indicator of contractility is the ability to develop pressure per unit time, in practice the following is used:

- maximum ventricular elastance index according to Sugi and Sagawi,
- heart rate values left resp. right ventricle: LVSW resp. RVSW (left / right ventriculus stroke work),

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

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

- 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 in the simplest way from the steepness of the rise of the pulse curve during direct measurement of arterial pressure.

Cardiac output

Within the possibilities of more detailed hemodynamics, 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 [cardiac frequency]] (heart rate) :

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

By recalculation to the body surface, we obtain the cardiac index = CI.

CO calculation using Fick's formula :

$$\text{CO} = [\text{VO}_2 / (\text{C}_a\text{O}_2 - \text{C}_v\text{O}_2)] \times 10$$

Pulmonary wedge blood pressure measurement (PAWP)

We measure the PAWP (pulmonary artery wedge pressure) value with a **Swan-Ganz catheter**. It is the result of pulmonary resistance and left heart function. Its values are close to the pressure in the left atrium. Used to accurately determine CI (cardiac index). It has a rare application in pediatrics.

Reference values: 6-16 cm H₂O (ideally 7-15 cm H₂O)

Selected hemodynamic parameters

	parametr	jednotka	norma
cardiac output	CI (cardiac index)	l/min/m ²	3,0-4,5 (5,5)
preload	CVP (central venous pressure)	mm Hg	3-10
lung	EVLW (extravascular lung water)	ml/kg	3,0-7,0
afterload	SVRI (systemic vascular resistance index)	dyne.s.cm/5.m/2	800-1600
contractility	EF (ejection fraction)	%	55-75

Consciousness

Within the framework of the shock state, the "impairment of consciousness" can be expressed in many different ways, both qualitatively and quantitatively. Classification scales are used for objectification: Beneš score and above all Glasgow coma scale (GCS).

We monitor the state of the pupils, stem reflexes (nasopalpebral, corneal), the state of muscle tone, possibly complete neurological monitoring as needed. If the condition requires "absolute" monitoring of CNS function, we use continuous EEG, intraparenchymatous measurement of intracranial pressure (ICP), multimodal intraparenchymatous sensors (monitor pH, pCO₂ and pO₂), monitoring of blood saturation in the jugular bulb S_{vj}O₂, transcranial Doppler ultrasonography, spectroscopy using near infrared radiation (near infrared spectroscopy = NIRS), ev. microdialysis. When imaging methods are indicated, we prefer CT and MRI.

Laboratory

As part of **biochemical monitoring** we investigate: KO + diff. (possibly also blood group), creatinine, urea, iontoqram, liver tests, S-amylase, glycemia, albumin, lactate, S-osmolality and hemocoagulation.

We are interested in chemistry and sediment, urinary osmolality, waste ions, creatinine and urea from **urine examination**. Hyperosmolar urine with low natriuresis is demonstrated in the case of a deficit of effective circulating volume or, conversely, hypoosmolar urine with high natriuresis in acute renal failure (shock kidney). Evidence of microalbuminuria is a marker of endothelial damage. A fundamental examination before starting ev. antibiotic therapy is collection by cultures (blood culture, urine, CSF, purulent collections - pleural exudate, joint effusion, puncture of abscess, etc.). From a general point of view, we demonstrate a lactate MAC, when lactate is > 2 mmol/l, a widening of the anion gap and a decrease of bicarbonate. Unfortunately, the specificity of hyperlactacidemia is not high, a simple lactate value does not reveal regional perfusion disorders, the lactate level also depends on hepatic production. Considering these aspects, the modern method gastric tonometry appears to be more advantageous for assessing organ perfusion.

The value of "glycemia" can be increased (more often) or decreased. Hyperglycemia is caused by insulin receptor resistance to insulin.

Changes in serum osmolality and blood biochemistry are dependent on the precipitating cause of the shock state. A sudden decrease in leukocytes can indicate a violation of the integrity of the vascular wall. The finding of hypophosphatemia indicates a major disorder of intracellular metabolism, as phosphorus is a valuable intracellular ion.

It is essential to monitor "diuresis", in the case of a shock state always an hourly diuresis with a 6-hour fluid balance. This means the unconditional insertion of a urinary catheter. A good diuresis is an excellent reflection of the adequacy of organ perfusion. But beware – sufficient diuresis can be misleading in the polyuric type of acute renal failure. We also monitor peripheral and central body temperature, as well as inflammatory markers (especially CRP and procalcitonin) as part of comprehensive diagnostics.

Gastrointestinal tract

We always insert a Nasogastric tube (NGS). At first, we use it to decompress the GIT and suction the stomach contents to prevent possible aspiration. The implementation of NGS is absolutely essential in patients with suspected sudden abdominal events where we must not give anything p.o., or in patients after drowning where there is a high risk of aspiration.

Gradually, NGS is used as a way of enteral nutrition. In the case of gastric atony, it is necessary to implement enteral nutrition via a nasojejunal tube (in this case, bolus feeding can no longer be used, but continuous feeding - usually 21 hours with a three-hour break).

The basis is the monitoring of peristalsis, evaluation of residues in the probe, registration of the number and nature of stools. Stool examination is used for culture, proof of occult bleeding or proof of clostridial antigen and toxin (*Clostridium difficile*). The most important imaging examination is undoubtedly sonography.

As part of liver function, we monitor complete liver tests (bilirubin direct and indirect, transaminases, GMT, ALP, LDH, cholinesterase), ammonia, coagulation (especially Quick and fibrinogen), albumin, glycemia and urea.

The modern method of gastric tonometry is suitable for assessing organ perfusion. Its advantage is the detection of regional hypoperfusion affecting the digestive tract (as a prototype of splanchnic circulation), the advantage is also continuous measurement. However, this method assumes that hypoperfusion of the splanchnic region will precede systemic perfusion. The disadvantage of this method is its relative invasiveness.

Methods monitoring regional perfusion

The values of serum lactate or the values defining MAC are a reflection of the global situation and, moreover, their results are mostly limited by the collection of venous blood. Methods defining regional perfusion and at the same time minimally invasive are in the foreground: gastric tonometry, NIRS (near-infrared spectroscopy), rectal tonometry, sublingual capnometry. All these methods are in the research stage and their routine use is not part of the article. recommended.

Main principles of care for patients in shock

Shock is defined as a syndrome with inadequate tissue oxygenation. Therapeutic efforts therefore try to establish a balance between the supply and the actual need for oxygen. Oxygen consumption is reduced by intubation, mechanical ventilation, sedation, myorelaxation, control of hyperpyrexia. On the other hand the oxygen supply is increased by oxygen therapy with either non-invasive or invasive airway management.

- CVP 5 to 10 cm H₂O
- PAWP 7 to 15 cm H₂O
- age-appropriate values MAP and PerP
- CI 3 to 6 l/min/m²
- SvcO₂ > 70 %
- O₂ER < 30 %
- minimization of myocardial damage – physiological standards of AST, troponin, CK-MB, ECG, echokardiography
- adequate airiness of the lungs
- lactate < 2 mmol/l

Organ dysfunction criteria:

Cardiovascular system	Respiratory system	CNS
<p>decreased BP < 5th percentile for age or sBP < 2 SD despite bolus volume expansion > 40 mL/kg/1 hr.</p> <p>or</p> <p>the need for inotropic support to maintain BP within the physiological range</p> <p>or</p> <p>two of the following criteria: otherwise unexplained MAC with BE -5 mmol/l; lactate increase > 4 mmol/l; capillary return > 5 seconds; peripheral and central temperature difference of > 3 degrees C.</p>	<p>PFi < 300 in the absence of cyanotic heart disease</p> <p>or pre-existing lung disease</p> <p>or</p> <p>pCO₂ > 65 torr or > 20 torr compared to the patient's normal value</p> <p>or</p> <p>need FiO₂ > 0.50 to maintain SaO₂ > 92%</p> <p>or</p> <p>the need for non-elective non-invasive or invasive ventilation</p> <p>PFi < 300</p>	<p>the GCS < 11 p.</p> <p>or acute decrease of the GCS > 3 p.</p>

Organ dysfunction criteria II.:

Hematopoiesis	Kidneys	Liver
<p>thrombocytes < 80,000 or a decrease of > 50% from the highest value recorded in the last 3 days (for patients with chronic hematological or oncological diseases)</p> <p>or</p> <p>INR > 2</p>	<p>an increase in S-creatinine > 2x over the upper limit or a double increase in the value compared to the normal value of the given patient</p> <p>or</p> <p>oligoanuria < 0,5 ml/kg/hod.</p>	<p>total bilirubin > 4 mg/dl (does not apply to newborns)</p> <p>ALT increase > 2x over the upper limit</p>

It is necessary to think about the possible **complications of shock conditions:**

- ARDS
- DIC
- acute renal failure
- acute liver failure
- myocardial ischemia
- edema of CNS
- rhabdomyolysis
- pancreatitis
- sepsis
- metabolic disorders

The aforementioned complications are a sign of the development of MODS (multiple organ dysfunction syndrom) and they significantly increase morbidity and mortality of the patients.

right ventricular preload	<ul style="list-style-type: none"> ▪ CVP ▪ size of liver ▪ echocardiography -> right ventricular end-diastolic volume
left ventricular preload	<ul style="list-style-type: none"> ▪ PAWP ▪ pulmonary edema (chest X-ray , EVLWI = extravascular lung water index) ▪ echocardiography -> left ventricular end-diastolic volume
global preload parameters	<ul style="list-style-type: none"> ▪ GEDVI ▪ ITBVI
afterload	<ul style="list-style-type: none"> ▪ SVRI (Systemic Vascular Resistance Index) ▪ PVRI (Pulmonary vascular resistance Index) ▪ MPAP (Mean pulmonary arterial pressure) ▪ MAP
contractility	<ul style="list-style-type: none"> ▪ maximum ventricular elastance index according to Sugi and Sagawi ▪ ejection fraction (echocardiography) ▪ GEF ▪ CFI ▪ pulse work of the left (LVSW) and right (RVSW) ventricle ▪ the steepness of the rise of the pulse curve
tissue perfusion	<ul style="list-style-type: none"> ▪ diuresis ▪ perfusion pressure ▪ lactate ▪ gastric tonometry
cardiac output	<ul style="list-style-type: none"> ▪ CO/CI (PiCCO x Fick's principle) ▪ echocardiography -> ejection fraction ▪ SvcO₂

Symptomatology of shock states

Resting tachycardia is characteristic (the heart rate must always be assessed according to the child's age and body temperature), a poorly palpable pulse on the small arteries of the leg, possibly cyanosis. Blood pressure can also be increased in a certain phase of shock, the cause being a pronounced α -mimetic reaction during the centralization of circulation. Especially in children, shock often occurs under the image of *low flow* , i.e. with an increased SVRI and a decrease in CI (this is typical, for example, of hypovolemic shock, burn trauma). We register hypotension when the effective circulating volume drops by 20-30% of the appropriate value. Physical examination shows cool, map-like skin and capillary refill time > 3 seconds. A valuable sign of the quality of organ perfusion is the previously mentioned monitoring of hourly diuresis, which drops into the zone of oligoanuria when the kidneys are hypoperfused.

In the initial stages of shock, RAL with hyperventilation is present, gradually transitioning to lactate MAC , when we clinically observe raspberry-red mucous membranes and also hyperventilation = Kussmaul breathing (if the patient still has enough energy), as a respiratory compensation of the metabolic disorder.

- Severe MAC with pH < 7.2 reduces cardiac contractility, lowers the threshold for the onset of arrhythmias , and causes dilation of arterioles and thus hypotension with compensatory tachycardia.
- In hypovolemic shock, signs of dehydration predominate , i.e. dry mucous membranes, absent tears during crying, reduced skin turgor, haloed and sunken eyes, sunken large fontanelle, non-palpable liver in newborns and infants.
- In cardiac failure, on the other hand, we can notice peripheral edema and the most typical symptom in children is hepatomegaly.
- As part of the CNS function, we record restlessness, behavioral change, impaired consciousness, which we objectify with the Glasgow coma scale.
- It is also important to determine and compare the peripheral temperature (measured on the dorsum of the leg) and the central temperature measured in the anus with a rectal sensor. A difference between central and peripheral temperature > 8 °C is a sign of shock circulation. A difference between central and peripheral temperature > 2 °C indicates increased α -mimetic activity.

Therapeutic interventions

Patient Assurance

The basic step in approaching a patient in a state of shock is to ensure the patency of the airways, administer 100% oxygen, ventilate with an ambuvac mask if necessary or intubate the patient and, if possible, start UPV as soon as possible. . Regardless of the etiology of the shock state, quick decisions should always be made for ventilatory and circulatory support. The introduction of UPV in shock states is not generally applied only on the basis of a diagnosis of global respiratory insufficiency, but hypermetabolism, hyperkinetic circulation, resistant metabolic acidosis, impaired consciousness and extreme work of breathing may lead to a decision on adequate provision of the child. We therefore indicate early intubation and UPV (in general, sooner rather than later). It is necessary not to leave the child in respiratory distress for too long. UPV allows redistribution of cardiac output from the respiratory muscle area toward vital organs, plus positive pressure UPV reduces afterload and can increase stroke volume. The disadvantage of UPV in patients with hypovolemia is that during positive pressure ventilation preload continues to decrease and hypotension can manifest.

If the anamnesis or clinical examination indicates pneumothorax or hemothorax, we will consider the urgency of performing a pleural puncture. At the same time, it is necessary to ensure circulation, i.e. ensure intravenous (2 IV lines are ideal) or intraosseous access. In neonates, we prefer umbilical vein cannulation. For children > 6 years old, an alternative is when it is impossible to provide i.v. entry cannulation of the central venous course, if an experienced doctor who controls the technique is available and the patient is in an environment where complications arising from the cannulation can be dealt with urgently. After the basic securing of entry into the circulation, the next step is the elective securing of the CVK and arterial line. The goal is to achieve CI 3.3-6 l/min/m² and oxygen consumption VO₂ (oxygen consumption) > 200 ml/min/m² /vul>. The recommended Hb value for shock states is approx. 100 g/l, Ht 0.30-0.40.

Volume therapy

Volume therapy in shock (pediatrics)

Inoconstriction and inodilation treatment

The basic goal of the administration of these substances is to increase tissue perfusion and maintain perfusion gradients, however, a prerequisite for their effect is sufficient filling of the vascular bed. The administration of inodilating substances in a hypovolemic patient can cause serious complications resulting from hypotension or tachyarrhythmia. Administration of inoconstrictive substances is not effective in normal doses. Vasopressors should be titrated according to perfusion pressure or systemic vascular resistance so that diuresis and physiologic creatinine clearance are optimal.

It should be noted that if the shock is complicated by myocardial dysfunction, then preparations with a positive inotropic effect (increasing contractility) can reduce preload and afterload, improve myocardial oxygen supply by increasing coronary perfusion pressure. Coronary flow is also improved by lengthening the diastolic phase while lowering the heart rate. However, if a drug with a positive inotropic effect is administered to a patient with normal cardiac contractility, the result may be increased myocardial oxygen consumption.

We also ensure normal reactivity of the myocardium and vascular system by maintaining normal acid-base ratios and electrolyte levels, especially potassium, magnesium and calcium. Inoconstrictors or inodilators are usually administered with a linear dispenser. When dealing with circulatory complications in critically ill patients, we use one or two substances, exceptionally a larger number. The effect on individual receptors is in some cases dose-dependent (e.g. dopamine, adrenaline) and their introduction into the systemic circulation should be completely separated from other substances. We preferably use multi-channel central venous catheters for this purpose. Catecholamine solutions must be protected from light and we require intra-arterial BP measurement when administered. Administration into peripheral veins causes early reactive inflammation. Only dobutamine, other catecholamines can only be administered into the peripheral watercourse for a short time and with maximum dilution.

From a clinical point of view, it is possible to divide the group of inotropic substances into substances that are **inoconstrictive** (noradrenaline, adrenaline, dopamine) and substances that are **inodilatory** (dopexamine, dobutamine, isopreterenol). A specific group of inotropic substances are phosphodiesterase III blockers (PDE III) = inodilators in the narrower sense of the word. Catecholamines stimulate α -1, α -2, β -1, β -2 and dopaminergic = α -receptors and lead to an increase in cAMP (cyclic adenosine monophosphate), PDE III inhibitors increase cAMP by preventing its degradation inside cells.

Mechanism of action

Adrenergic receptors are represented by 8 gene subtypes, but from a practical point of view we distinguish α -1, α -2, β -1, β -2 and α -1 and α -2 receptors.

Both **β -1** and **β -2** receptors are located in the ventricular myocardium muscle and the atrial muscle. In addition, β -2 receptors are located on the presynaptic endings of sympathetic nerves and stimulate the release of neurotransmitters. In the smooth muscle of blood vessels, activation of β -2 receptors leads to vasodilation, in the smooth muscle of bronchi to bronchodilation (through the mechanism of smooth muscle relaxation). β -2 receptors

in the SA node are responsible for the positive chronotropic effect. β -1 stimulation of the myocardium increases not only inotropy (force of contraction), but also varying degrees of chronotropy (increased heart rate), dromotropy (increased conduction velocity) and bathmotropy (increase in irritability).

α -1 receptors are mainly found in the smooth muscle of blood vessels, where they cause vasoconstriction. However, α -1 receptors are also found in the muscle of the myocardium. Their irritation has a positive inotropic effect, but does not affect the heart rate. α receptors were originally differentiated with respect to their location on nerve endings. The postsynaptic receptor was designated as α -1 and the presynaptic receptor as α -2. Stimulation of the α -1 receptor leads to the contraction of smooth muscle, while stimulation of the **α -2** receptor inhibits the release of noradrenaline from presynaptic granules, thus promoting vasodilation.

Dopaminergic (delta) receptors are divided like others into postsynaptic d -1 and presynaptic d -2. d -1 receptors are located in the smooth muscle of renal, splanchnic, coronary and cerebral vessels. Their activation leads to vasodilation. d -2 receptors inhibit the release of noradrenaline from sympathetic endings.

The mechanism of action of **phosphodiesterase blockers** is based on the fact that normally cAMP is inactivated by phosphodiesterase, which causes its conversion to AMP. Inhibition of phosphodiesterase increases cAMP concentration and enhances β -receptor mediated activity.

Disorders of receptor function

As part of the receptor disorder, the mechanism of reducing the sensitivity of receptors is best described on the principle of agonist-mediated desensitization. Within seconds to minutes after agonist binding to the receptor, uncoupling may occur due to receptor phosphorylation (phosphorylation involves multiple mechanisms). In addition to agonist-mediated desensitization, there are other factors involved in so-called down-regulation: endotoxin, TNF, congestive heart failure. Another mechanism of down-regulation of receptors is their sequestration inside target cells and their subsequent degradation.

Inoconstrictors

Adrenaline

Adrenaline is produced in the adrenal medulla (tyrosine \rightarrow DOPA \rightarrow dopamine \rightarrow noradrenaline \rightarrow adrenaline). Adrenaline is a potent, directly acting α -1, β -1 and β -2 receptor agonist.

Adrenaline in low concentrations first affects β -2 receptors. It potentiates the activity of the SA node, increases the heart rate, helps vasodilation, i.e. a decrease in SVRI and decreases diastolic blood pressure. A decrease in SVRI further increases the direct chronotropic effect of adrenaline. Unfortunately, the increased consumption of oxygen by the myocardium is a disproportionate increase in inotropy and thus decreases myocardial performance. As the concentration increases, the α -1, β -1 component rapidly enters. Stimulation of α -1 receptors leads to an increase in SVRI (significantly in the area of the splanchnic) and at the same time pulmonary vascular resistance. High doses of adrenaline or its use in patients with myocarditis or infarction can lead to the development of severe atrial and ventricular dysrhythmias.

In practice, the combination of the β -2 effect, which lowers diastolic pressure, and the α -1 effect, which increases systolic pressure, increases the pulse pressure value.

During stress, when a large amount of adrenaline is flushed out, receptors can be desensitized very quickly, even before exogenous adrenaline administration begins.

Adrenaline is intended for the treatment of shock in connection with myocardial dysfunction, especially in patients after successful cardiopulmonary resuscitation or after a hypoxic-ischemic insult. In septic patients, where there was no improvement in the condition after volume expansion, continuous infusion of adrenaline can be beneficial. Adrenaline is most useful in conditions with hypotension, low CI and high SVRI (cold shock = low flow). At low doses of 0.005–0.1 μ g/kg/min, SVRI slightly decreases, but heart rate, blood pressure, and cardiac output increase. In medium doses of 0.1–1.0 μ g/kg/min. α -1 adrenergic activity begins to predominate and the further increase in CO balances the still persistent vasodilation (induced by the activation of β -2 receptors), which, as already mentioned, leads to a decrease in diastolic pressure. In very high doses (> 1 –2 μ g/kg/min.), vasoconstriction by activation of α -1 receptors predominates, splanchnic perfusion is significantly reduced, afterload increases, and myocardial function may decrease with elevation of serum [[lactate]]at.

As part of cardiopulmonary resuscitation, when we administer bolus high doses, we use precisely α -1 activity, which brings massive vasoconstriction everywhere, except for the coronary and cerebral blood vessels, at the same time leading to an increase in SF, BP and vascular resistance. Adrenaline is administered as a bolus dose of 0.01 mg/kg (10 μ g/kg). Previously recommended subsequent 10-fold higher doses (so-called high dose epinephrine) are no longer recommended. The same dose is given intraosseously, 0.1 mg/kg is given intratracheally. Adrenaline has a number of side effects. Within the CNS it leads to anxiety, nausea. High doses can cause myocardial ischemia, arrhythmias. Although ventricular tachycardia is rare in childhood, it occurs more often with concomitant myocarditis, hypokalemia and hypoxemia. Adrenaline also has significant metabolic effects: stimulation of β -2 receptors, which are associated with Na-K-ATPase in muscles, leads to hypokalemia (infusion of 0.1 μ g/kg/min. leads to a decrease in potassium by 0.8 mmol/l). β -adrenergic mediated suppression of insulin results in hyperglycemia. Adrenaline is degraded by monoamine oxidase or catechol-o-methyltransferase. The recommended dosage is 0.005–2.0 μ g/kg/min, as part of cardiopulmonary resuscitation we administer 10 μ g/kg i.v. as a bolus. Adrenaline is stable when diluted to 5% glucose or 1/1 FR.

Indications:

- shock in association with myocardial dysfunction, especially in patients after successful cardiopulmonary resuscitation or after a hypoxic-ischemic insult.
- sepsis, where the condition did not improve after volume expansion, dopamine or dobutamine and high SVRI (low flow) persists.
- conditions with hypotension, low CI and high SVRI.
- cardiopulmonary resuscitation

Noradrenaline

Noradrenaline is a potent inotropic substance with a direct effect on β -1 and α -1 receptors. It has a powerful vasoconstrictive effect, as α -adrenergic stimulation is not opposed by the β -2 effect. Noradrenaline does not increase the heart rate, as it reflexively reduces the activity of the SA node through the vagus nerve and thus eliminates the expected β -1 chronotropic effect. Noradrenaline is also powerful inotropic effect. It mainly increases diastolic BP and diuresis. An increase in afterload tends to increase oxygen consumption in the myocardium, however noradrenaline reflexly reduces heart rate and thereby reduces myocardial oxygen consumption and improves coronary flow in diastole. It has no β -2 agonist effect. It is one of the most widely used drugs in the treatment of circulatory insufficiency in resuscitation care. It is the vasoconstrictor of first choice today. Noradrenaline improves perfusion in severely hypotensive children with low SVRI and normal or elevated CI. Typical choices are septic or anaphylactic shock. Noradrenaline, like other catecholamines, should be administered only after volume depletion has been completed, ideally in patients where both SVRI and CO/CI can be assessed. In children, noradrenaline is recommended for the high flow form of shock, which is refractory to volume expansion and dopamine. On the other hand, norepinephrine can increase blood pressure without improving organ perfusion. Typical cases are low CI, insufficient volume expansion, increase in PAWP. The use of high doses of norepinephrine, which increase pressure but do not improve organ perfusion, may contribute to the development of MODS. In general, however, the limitation of upper doses of noradrenaline/adrenaline is the occurrence of adverse effects, i.e. myocardial ischemia, tachycardia and arrhythmias. In case of extravasation, we quickly infiltrate the affected tissue with phentolamine (5 to 10 mg in 10 ml 1/1 FR). The recommended dosage is 0.01 to 1.0 μ g/kg/min. The wide range of recommendations is due to the need for titration of continuous noradrenaline administration. Noradrenaline is stable when diluted to 5% glucose.

Indications:

- the most frequently used drug in the treatment of circulatory insufficiency in resuscitation care, it is today the vasoconstrictor of first choice
- severe hypotension with low SVRI and normal or elevated CI (septic or anaphylactic shock)
- high flow form of shock that is refractory to volume expansion and dopamine.

Dopamine

Dopamine is a central neurotransmitter, it is also found in sympathetic nerve endings and in the adrenal medulla, where it is a rapidly usable precursor for the formation of noradrenaline. Dopamine affects D1 and D2 receptors (dopa receptors), which are located in the brain and vascular bed kidney, splanchnic and [heart]. Depending on the dose, it also stimulates α + β receptors, but the affinity for these receptors is lower. Stimulation of D-1 receptors leads to vasodilation, increased perfusion, and can increase the excretion of solutes and water in the kidneys. However, meta-analytic studies confirm that so-called renal doses of dopamine of 2.5 to 5 μ g/kg/min. they are not recommended because their protective effect on increasing renal perfusion has not been confirmed (*Intensive Care Med* 2002). By influencing D-2 receptors, dopamine regulates the release of aldosterone and prolactin and also affects the renal clearance of solutes. The fact that newborns and infants show lower sensitivity to dopamine is a tradition, but not definitively confirmed. Dopamine is recommended as the drug of first choice in children in septic shock where volume expansion has failed, dopamine is suitable in children with mild myocardial dysfunction and hypotension after cardiopulmonary resuscitation. Severe contractility or vasomotor impairment requires the use of other catecholamines. Children with primary myocardial dysfunction and in the absence of hypotension benefit more from administration of dobutamine or milrinone. At a dose below 5 μ g/kg/min, the effects are dominated by influencing D-1 receptors, at a dose of 5 to 10 μ g/kg/min, β -1 shows adrenergic effects, at doses of 10 to 15 μ g/kg/min, it has a mixed α + β effect. Dose increase to > 15 μ g/kg/min. leads to increased stimulation of α -1 receptors, increasing dose > 22-25 μ g/kg/min. is no longer relevant and it is necessary to choose another inotropic agent. In shock state with hypotension, we start administration at a rate of 5 to 10 μ g/kg/min., increasing the infusion rate in steps of 2 to 5 μ g/kg/min. We assess the effect of the treatment according to the difference in central and skin temperature, capillary return, diuresis. When doses > 25 μ g/kg/min are required, SVRI (predominance of α -receptor stimulation) increases more significantly than cardiac output. We refer to this condition as dopamine-resistant. The next step is the use of noradrenaline for high flow form (warm shock) or adrenaline for low flow (cold shock). Disadvantages of dopamine include its proarrhythmogenic effect, tachycardia and increased myocardial oxygen consumption, hypertension. With the exception of bipyridines, all inotropic agents increase myocardial oxygen consumption because they increase myocardial workload. The effectiveness of dopamine is significantly limited in patients with a depleted supply of endogenous catecholamines. Dopamine and other β -agonists decrease PaO₂ by interfering with alveolar pulmonary vasoconstriction (exacerbating the V/Q imbalance). In case of extravasation, we quickly infiltrate the affected tissue with phentolamine (5 to 10 mg in 10 ml 1/1 FR). The recommended dosage is 5 to 20 μ g/kg/min. Dopamine is stable when diluted to 5% glucose or 1/1 FR.

β -agonists have a hypokalemic effect (by affecting Na-K-ATPase) and reduce PaO₂ (the vasodilatation induced by them in the pulmonary basin interferes with the mechanism of hypoxic alveolar vasoconstriction => deepening of the V/Q disparity when the P-L shunt increases).

Indications:

- drug of first choice in children in septic shock where volume expansion has failed
- suitable for children with mild myocardial dysfunction and hypotension after cardiopulmonary resuscitation

Inodilators

Dobutamine

Dobutamine is a synthetic analogue of dopamine. It has no dopaminergic activity. It is a potent inodilator with inotropic β -1 and vasodilatory + chronotropic β -2 activity affecting arteriolar and venous channels. Its great advantage is that it does not have its own proarrhythmogenic effect and practically does not have its own toxic effect. In septic shock, we administer dobutamine if myocardial dysfunction prevails. However, usually the main concern is the regulation of vascular tone, and SVRI-increasing drugs are preferred. In myocardial dysfunction, dobutamine alone or in combination with dopamine increases CO and subsequently blood pressure. However, dobutamine is most often combined with noradrenaline in conditions with myocardial dysfunction associated with a high flow form of shock (sepsis) or ARDS. Dobutamine and noradrenaline are currently the most frequently used combination of vasoactive substances in resuscitation care. In children with myocardial dysfunction, dobutamine increases systolic volume and CO, without a significant increase in heart rate. Dobutamine leads to a decrease in SVR and PVR. These mechanisms explain the increase in pulse pressure.

Indications for the administration of dobutamine in pediatrics are conditions of congestive heart failure with low CI and normal or slightly reduced blood pressure (viral myocarditis, drug-induced cardiomyopathies, [[myocardial infarction|myocardial infarctions]] -m. Kawasaki, abnormal distance of the left coronary artery)

In myocardial failure, we start with dobutamine and ensure adequate intravascular volume according to CVP. Simple volume expansion is not appropriate here. Dobutamine is the inodilator of choice today. Dobutamine can also be administered as a single catecholamine into a peripheral vein.

Adverse effects include marked tachycardia, which may increase oxygen consumption and require dose reduction or change to another agent. Rarely, it may cause atrial or ventricular dysrhythmias, especially in patients with myocarditis, electrolyte imbalance, or at high doses. Dobutamine, like other inotropic agents, must be administered with caution in patients with left ventricular outflow obstruction (hypertrophic aortic stenosis).

The recommended dosage is 2-20 $\mu\text{g/kg/min}$. Children < 1 year may be less responsive to dobutamine or delta doses of dopamine. If doses > 22 $\mu\text{g/kg/min}$. do not lead to an improvement in the hemodynamic state, we are considering changing to adrenaline. Dobutamine is stable when diluted to 5% glucose or 1/1 FR.

Indications:

- septic shock if myocardial dysfunction predominates
- in combination with noradrenaline in conditions with myocardial dysfunction in connection with high flow form of shock (sepsis) or ARDS
- conditions of congestive heart failure with low CI and normal or slightly reduced blood pressure (viral myocarditis, drug-induced cardiomyopathy, myocardial infarctions - Kawasaki muscle, abnormal distance of the left coronary artery)
- in case of myocardial failure, we start with dobutamine and ensure adequate intravascular volume according to CVP values

Dopamine and dobutamine are drugs that increase systolic volume.

Phosphodiesterase III blockers

Phosphodiesterase III blockers (PDE III) are divided into bipyridine (amrinone and milrinone) and imidazole (enoximone and pyroximone) preparations. They do not belong to catecholamines, their effect is through selective inhibition of phosphodiesterase III, they do not act on adrenergic receptors or lead to inhibition of Na-K-ATPase. Their effect is similar to dobutamine, i.e. especially the β -2 effect. They increase myocardial contractility, have a vasodilating effect, and improve diastolic function (lusitropic effect). The disadvantage is a whole range of side effects, led by a high proarrhythmogenic effect, the result of which can be systemic hypotension with ventricular tachycardia.

When using phosphodiesterase III blockers, most experts recommend continuous infusion to achieve steady state. Because these drugs have a long half-life, their infusion should be stopped at the first signs of tachyarrhythmia, hypotension, or an excessive decrease in SVR, especially if liver or kidney dysfunction occurs at the same time. The hypotensive effects of phosphodiesterase III blockers can be eliminated by replacing co-administered adrenaline with noradrenaline. Milrinone, as a newer agent, has fewer side effects than amrinone, and is a more selective PDE III inhibitor.

Indications for amrinone/milrinone in children are:

- normotensive patients with low CI but high SVRI despite epinephrine or nitrate infusion
- low cardiac output in dilated forms of cardiomyopathy when other inotropic support fails
- patients with down-regulation of β -1 and β -2 receptors
- with toxic effects of nitrates
- conditions with severe heart insufficiency refractory to other treatment
- postoperative conditions in cardiac surgery

Drug affecting venous return (preload)

Administering preload = diuretics and venodilators in heart failure with reduced contractility will improve cardiac performance by reducing ventricular size and reducing wall tension. First of all, we reduce preload by restricting fluids and administering diuretics.

Diuretics

Diuretics relieve symptoms of pulmonary congestion and peripheral edema. We most often use *furosemide* in a dose of 0.5–2 mg/kg i.v. as a bolus according to diuresis, or continuously up to a maximum total dose of 10 mg/kg/day. By directly acting on the loop of Henle, it causes the excretion of ions Na, K, Cl and body water. It has a quick and short-term effect.

During long-term diuretic treatment, when there is a risk of developing secondary hyperaldosteronism and hypokalemia, spironolactone is indicated in a dose of 1-3 mg/kg/day divided into 3 doses. Spironolactone is a competitive aldosterone inhibitor acting on the distal renal tubule. It has a very weak diuretic effect by itself, but potentiates the effect of other diuretics. It partially antagonizes the loss of K ions. In combination with ACE inhibitors or excessive potassium substitution, it causes hyperkalemia.

The administration of diuretics and venodilators has adverse effects in patients with reduced myocardial contractility and circulating volume deficit or insufficient ventricular filling!

A factor affecting preload and afterload

The common denominator for this group of drugs is *reduction of peripheral vascular resistance*. They have a combined effect on veins and arteries. It should be emphasized that high peripheral vascular resistance is a frequent symptom during shock states in children. We are talking about the fact that *hypodynamic shock is typical for children*. Affecting the resistance and capacity of the systemic vascular bed has an effect on cardiac performance. An increase in peripheral vascular resistance with unchanged preload and contractility decreases cardiac output. The use of vasodilators and other drugs with a relaxing effect on the smooth muscle of peripheral vessels can modify cardiac performance in heart failure. Peripheral vascular vasodilatation reduces myocardial afterload. By increasing the capacity of the systemic flow, the preload of the myocardium also decreases and the filling volume of the heart decreases. However, the reduction of peripheral resistance carries the risk of systemic vasodilation, which in the case of subclinical or unrecognized hypovolemia can lead to life-threatening hypotension. Simultaneously with the reduction of SVR, the regulatory mechanisms of fluid redistribution are disrupted. When using vasodilator therapy, it is advisable to monitor filling and systemic pressures. Medicines that reduce high SVR include sodium nitroprusside, nitroglycerin and ACE inhibitors, and to a lesser extent dehydrobenzperidol or chlorpromazine.

Sodium nitroprusside

Nitroprusside is a fast-acting peripheral vasodilator. It has a direct vasodilating effect on arterioles and veins. It primarily reduces afterload and thus increases cardiac output. The result is reduced filling of the left ventricle, reduction of pulmonary congestion, reduction of volume and pressure in the left ventricle, better emptying of the left ventricle in systole, reduced oxygen consumption by the myocardium. Its effect is tied to its immediate administration, i.e. after stopping the infusion, the effect is immediately lost. When using it, invasive blood pressure monitoring is absolutely necessary. Prolonged administration may lead to a rise in serum cyanide levels; their control is necessary. During intoxication, disorders of consciousness, MAC appear. The recommended dose is 0.5–10 µg/kg/min, the dose is titrated according to the effect. As a rule, we start with a low dose and, depending on the effect, increase the dose by approx. 0.5 µg/kg/min after 10 minutes. Nitroprusside can be combined with dopamine or dobutamine because they have a synergistic effect on increasing cardiac output. Due to its drastic effect, which can also be associated with serious complications, we only use nitroprusside in the most severe cases.

Nitroglycerin

Venodilators are indicated for elevated end-diastolic pressure. The main representative is nitroglycerin. It has a direct venodilating effect, it dilates the smooth muscle of the vascular wall, predominantly systemic veins and coronary arteries. It reduces venous return and reduces congestion in the systemic and pulmonary basins. In low doses, it leads to venodilatation and reduction of preload. High doses cause more pronounced vasodilation in the pulmonary basin (cave: congestion!), dilation of arterioles and reduction of afterload. Pharmacological effects depend mainly on the state of the intravascular volume, less on the dose (hypovolemia increases the risk of hypotension). Usual doses are 0.25–5 µg/kg/min continuously i.v.

ACE inhibitors

ACE inhibitors lead to vasodilation and reduction of aldosterone secretion. The result is increased excretion of sodium, which leads to a decrease in systemic peripheral resistance, a decrease in EDP and an increase in cardiac output. Another positive effect is the ability to remodel the hypertrophic myocardium of the ventricles. A

representative is e.g. enalapril, doses p.o. 0.15–0.5 mg/kg/d in 1–2 doses, for i.v. treatment 5–10 µg/kg/dose 1–3 times within 24 hours

Steroids

Administration of hydrocortisone should be reserved for conditions unresponsive to adequate treatment with volume expansion and inotropes or situations with suspected or proven adrenal insufficiency. Children with septic shock and purpura, with previous chronic corticoid therapy and with adrenal or pituitary abnormalities are a risk group. The exact definition of adrenal insufficiency is not formulated, in case of septic shock resistant to catecholamines, the finding of a cortisol level < 500 nmol/l is considered to be its sign. The optimal dosage of steroids in children is not formulated, the most often recommended doses vary from 1 to 2 mg/kg of hydrocortisone as stress doses, alternatively 200 mg/d divided into 3–4 doses regardless of body weight. One of the recent recommendations for the administration of hydrocortisone: 0.18 mg/kg/hour. continuously. Recent meta-analyses have confirmed that methylprednisolone-type steroids in high doses, i.e. 30 mg/kg, are ineffective or even harmful in shock states.

Metabolic support

In cardiogenic shock, we administer fluids at a dose of 80–100% of the normal daily requirement, more precisely according to CVP and PAWP values. For other types of shock, we initially increase the daily fluid requirement to 150–200% of normal, and a significantly positive water balance is not unusual during the first day of therapy. bicarbonate therapy is chosen in a situation of severe MAC (pH < 7.1, HCO₃ < 8) despite adequate volume expansion.

Other therapies

The finding of hypocalcemia can lead to a picture of left ventricular dysfunction, which is completely reversible after calcium correction. Especially in the smallest children, where glycogen reserves are reduced, we can find hypoglycemia. In general, the last option, the so-called rescue therapy, is ECMO (extracorporeal membrane oxygenation).

Complications of shock states

As part of the shock, we can find various multisystem dysfunctions. Their diagnosis is as important as their treatment. Possible complications of any shock state are:

- acute tubular necrosis
- ischemia of intestine: NEC, perforation
- myocardial ischemia
- CNS damage: intracranial hypertension, convulsions
- pancreatitis
- DIC
- rhabdomyolysis
- metabolic disorders
- MODS

Links

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