

# Cardiogenic shock (pediatrics)

## 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.*

Characteristics of congestive heart failure

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

## 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

#### Under construction

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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 analgesedation 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)!*

## Links

### Source

- HAVRÁNEK, Jiří: *Shock*. (edited)

### Related Articles

- Shock (Pediatrics)
- Shock
- Cardiogenic shock