

EXTREME STATES



General etiology of extreme states

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graph TD; A[General etiology of extreme states] --> B[Exogenous causes]; A --> C[Endogenous causes];
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Exogenous causes

- Physical (high and low temperature, electric current, radiation, etc.)
- Chemical (industrial poisons, acids, alkalis)
- Biological (toxins of microbes, parasites, etc.)

Endogenous causes

- Internal bleeding
- Cardiovascular failure
- Respiratory insufficiency
- Renal and liver failure
- Tumors, etc.

Pathogenetic types of collapse

cardiogenic

hypovolemic

vazodilation

Misallar:

- *Postinfarct*
- *Arrhythmic*
- *Cardiomyopathyc*

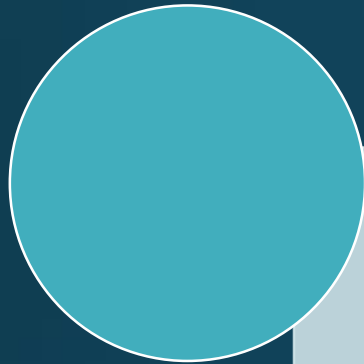
Hemorrhagic
Dehydration
Toxic-infectious
Orthostatic

Hypertermic
Orthostatic
Toxic

COLLAPSE

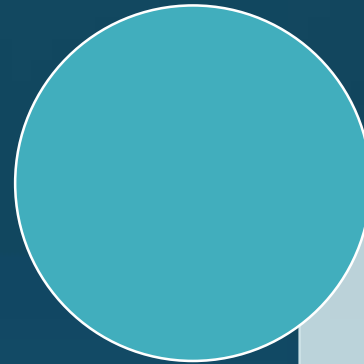
Stages of pathogenesis	Manifestations
Circulatory changes	Coronary insufficiency Decreasing of systolic volume and cardiac output Hypoperfusion of tissue Capillary trophic insufficiency
Changes in the nervous system	General inhibition Apathy Tremor of the fingers Convulsions Short-term loss of consciousness
Changes of respiratory system	Tachypnea Hypoxemia, hypercapnia
Disturbance of the excretory function of the kidneys	Oliguria, hyperazotemia
Hemodynamic disorders	Increasing viscosity of blood Hypovolemia Formation of blood clots

TYPES OF SHOCK



Shock associated with exogenous pain

Shock associated with endogenous pain



Shock associated with humoral factors

Psychoemotional shock

TYPES OF SHOCK

Septic

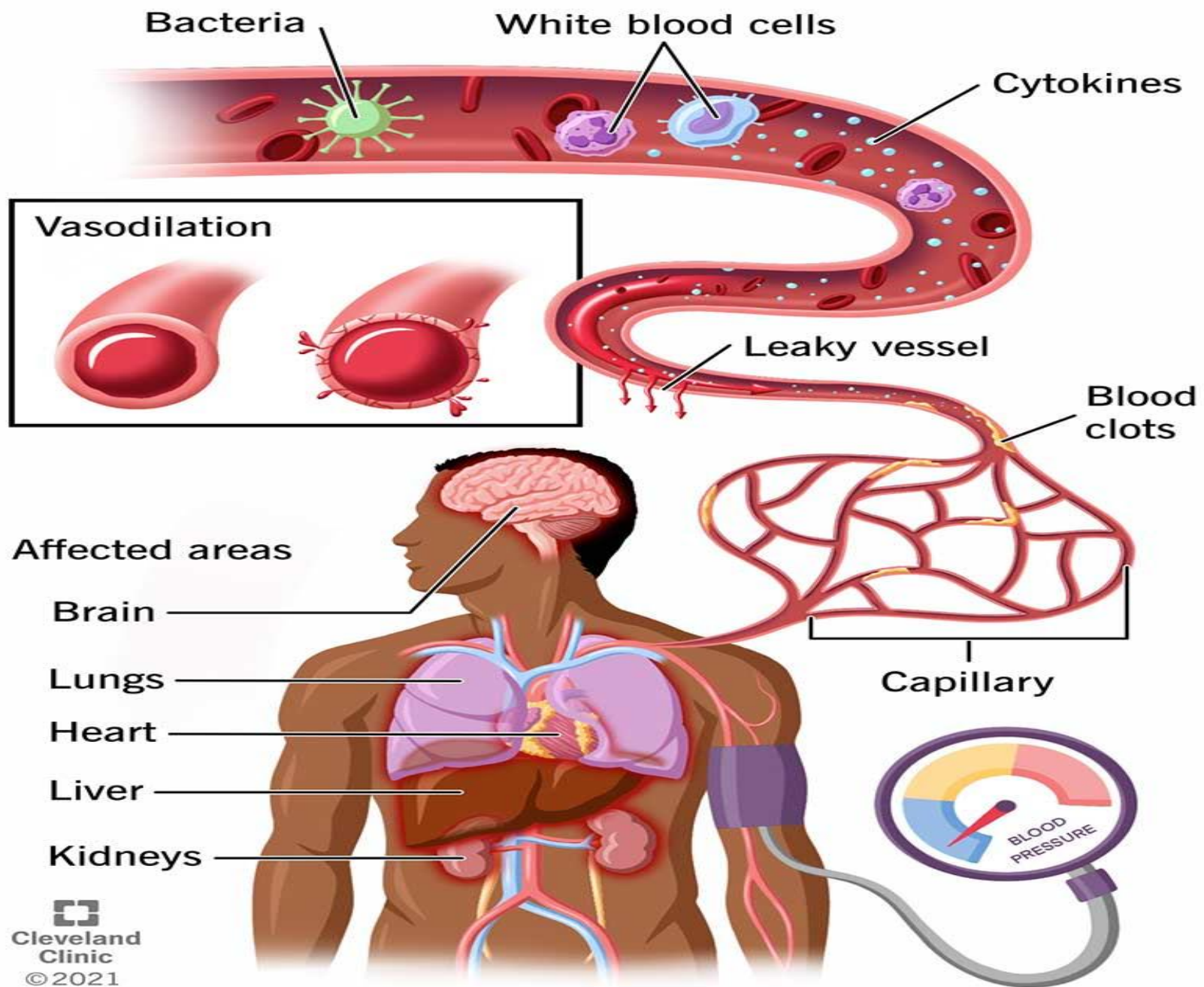
Anaphylactic

Cardiogenic

Traumatic

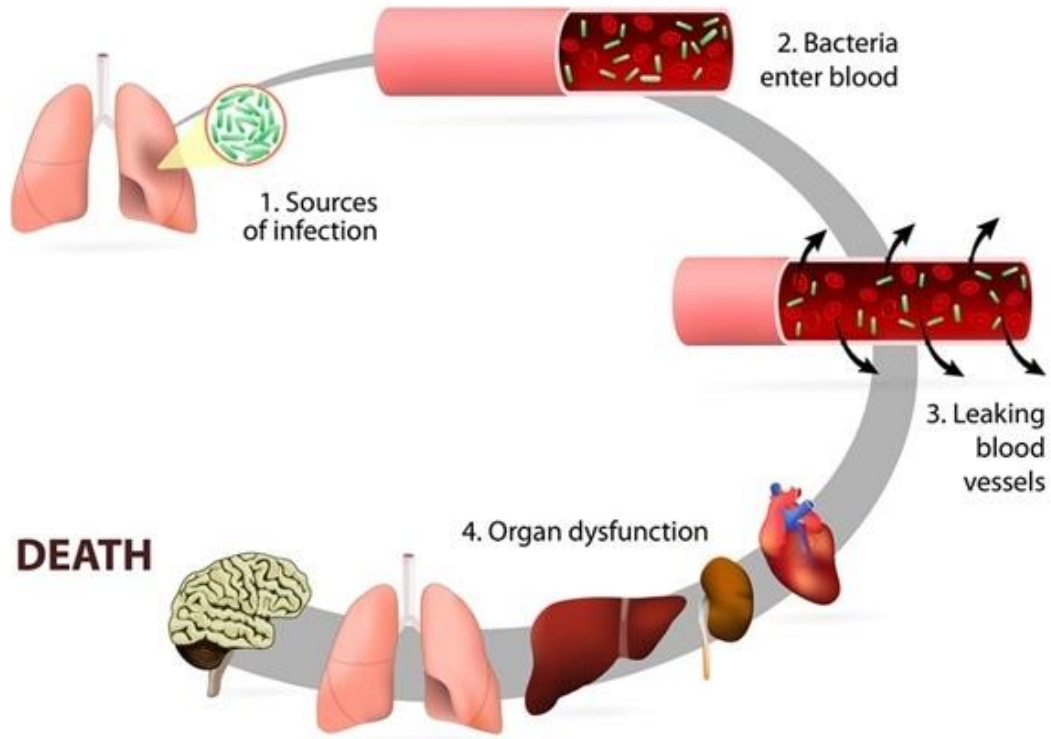
Transfusion etc.

Septic Shock



*Diagnostic examinations performed at the inpatient level
during emergency hospitalization*

- **Physical examination (measurement of body temperature, saturation, AT, pulse rate-min., *respiratory* rate)**
- **General analysis of blood; general analysis of urine; duration of coagulation; duration of bleeding**
- **Leukocyte index during intoxication; determination of glucose and ketone bodies in urine**
- **Biochemical analyzes (bilirubin, AST, ALT, alkaline phosphatase, total protein, albumin and its fractions, urea, creatinine, residual nitrogen)**



Symptoms of Sepsis



fever



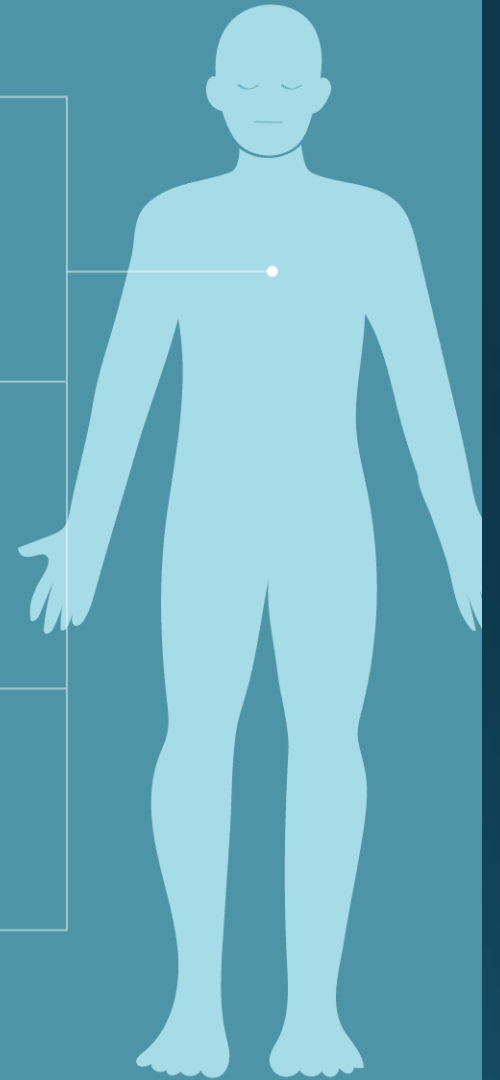
sweating



rapid heart rate
(tachycardia)



rapid breathing
rate (tachypnea)



Diagnostic examinations performed at the inpatient level during emergency hospitalization

- **Indicators of blood acid-alkaline balance (pH, BE, HCO₃, lactic acid)**
- **Electrolytes in the blood (potassium, sodium, calcium)**
- **Coagulogram (PT, thrombin time, fibrinogen, D-dimer, blood group according to the ABO system, blood rhesus factor)**
- **ECG; X-ray of the chest, USM of the organs of the abdominal cavity and kidneys.**

Potential markers of sepsis

General settings:

- **Body temperature $>38.0^{\circ}\text{C}$ or $<36^{\circ}\text{C}$**
- **Heart rate >90 beats per minute**
- **Tachypnea (respiratory rate >20 per minute)**
- **Altered mental status**

Potential markers of sepsis

Indicators of inflammation:

- **Leukocyte formula** >12 g/l or <4 g/l, immature forms more than 10%;
- **If the concentration of C-reactive protein in the plasma (normal up to 1 mg/l) is more than 10 mg/l, acute inflammation, if it is more than 100 mg/l, bacterial sepsis;**
- **A procalcitonin level (normally less than 0.1ng/l) ≥ 10 ng/l is characteristic of septic shock, with a high risk of lethal outcome.**

In today's clinical practice, procalcitonin (PCT) has developed into a promising new biomarker for early detection of (systemic) bacterial infections. In 1993, Assicot et al. demonstrated a positive correlation between high serum levels of PCT and patients with positive findings for bacterial infection and sepsis (eg, positive blood cultures). Further, they demonstrated that PCT did not elevate in viral infections and that serum levels of PCT would decrease following administration of appropriate antibiotic therapies. Current inflammatory biomarkers, such as C-reactive protein (CRP), lack the specificity required to diagnose bacterial versus non-bacterial infections accurately. Therefore, PCT assays with a specificity of 79%, have since been developed and utilized to more accurately determine if a bacterial species is the cause of a patient's systemic inflammatory reaction.

Anaphylactic shock

The severity of anaphylactic shock is determined by the severity of hemodynamic disturbances.

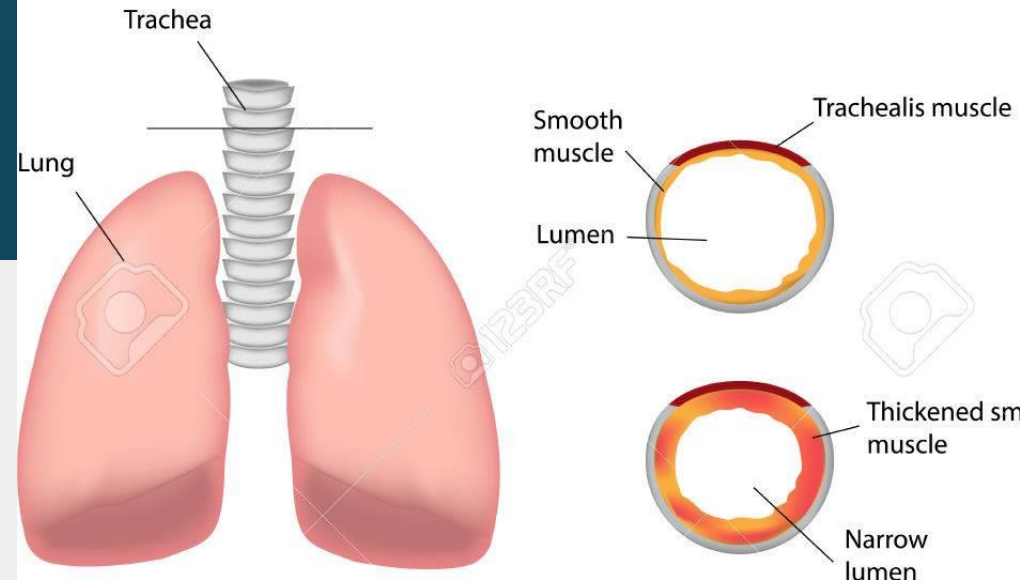
The duration of development of degree I (mild) is from a few minutes to 2 hours. Systolic AT decreases to 90 mm.c. milk. The patient's consciousness is clear, itchiness, urticaria, headaches, dizziness, feeling of heat in the head area, ringing in the ears, tachycardia, increased weakness are noted. This condition is easily resolved with anti-shock therapy.

Degree II (moderately severe) systolic BP is lower than 90-70, diastolic - 40 mmHg. Fainting is not noted. Tachycardia, arrhythmia, acute weakness are noted. Asphyxia, vomiting, involuntary urination and defecation may occur due to laryngeal edema and bronchospasm.

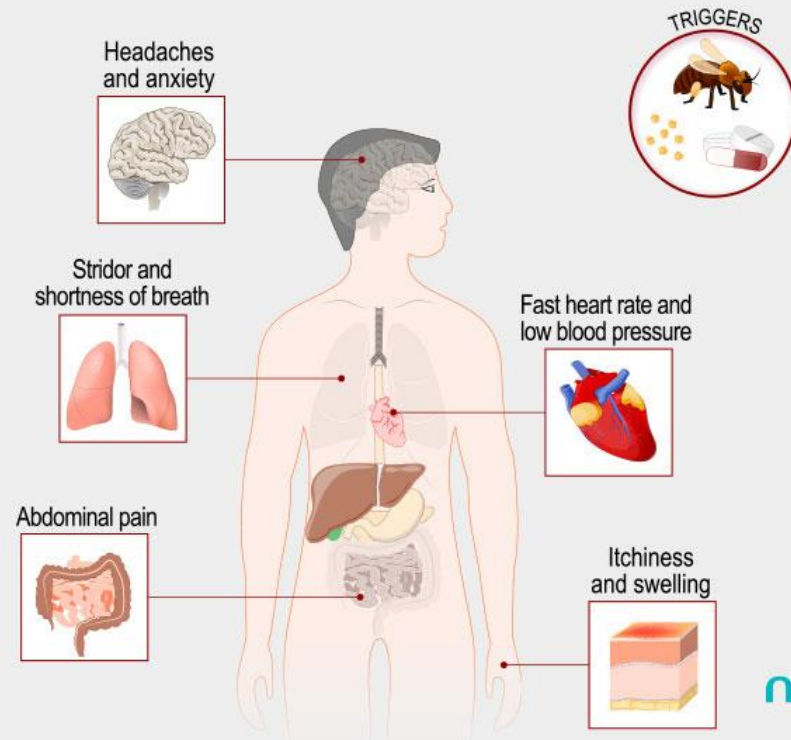
III degree (severe) is manifested by loss of consciousness, acute respiratory and cardiovascular failure (dyspnea, cyanosis, stridorous breathing, threadlike pulse, systolic BP below 60 mm Hg, diastolic BP may not be determined. Shock Antiretroviral therapy often has little effect.

IV degree (very severe) fulminant collapse (paleness, cyanosis, threadlike pulse, sharp drop in blood pressure to zero), comatose state (loss of consciousness, involuntary urination and defecation, dilated pupils and lack of reaction to light), in the end, cardiac and respiratory activity stops.

Anaphylactic Shock



Anaphylaxis



Basic and additional diagnostic criteria:

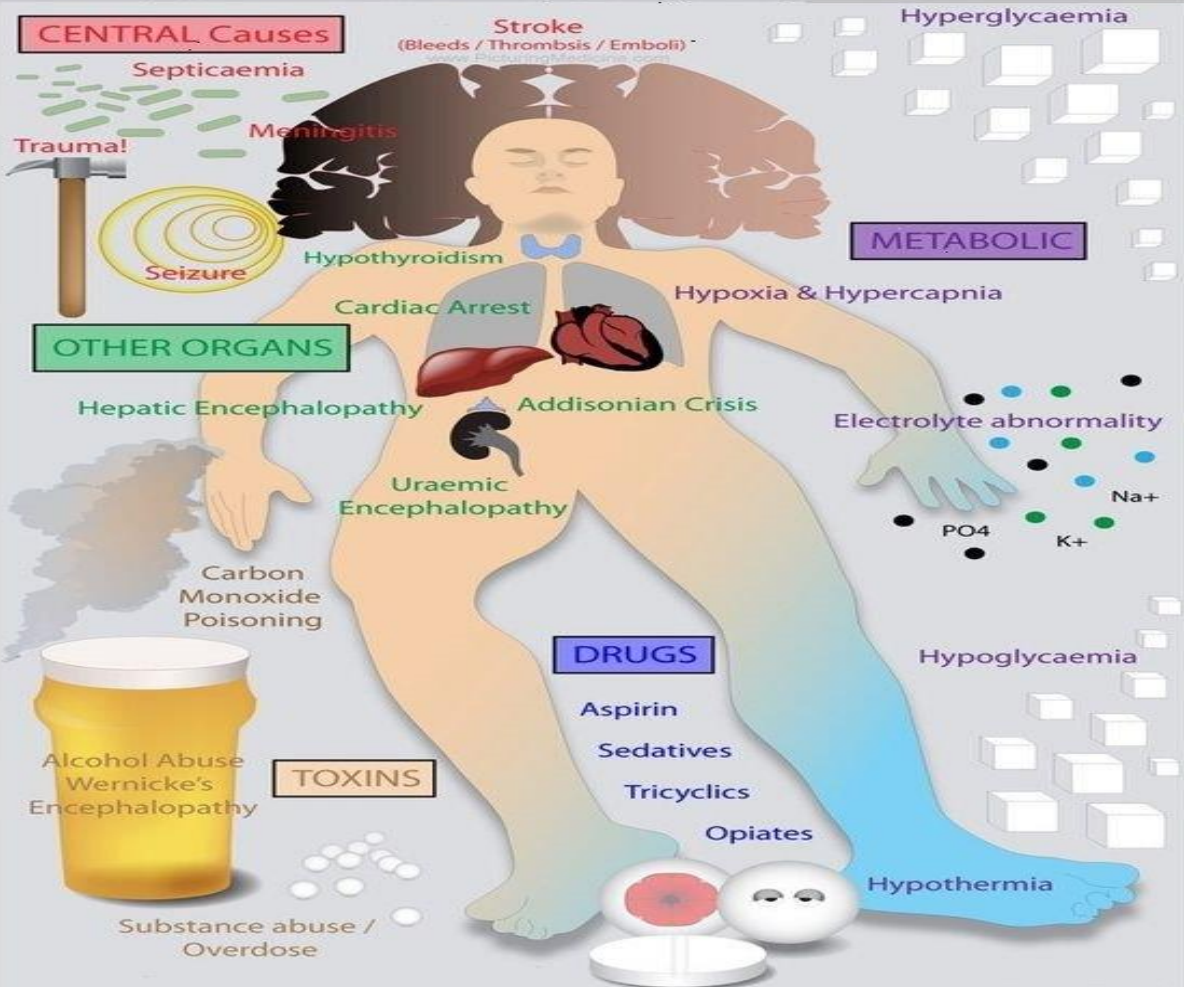
- Assessment of the level of consciousness (drowsiness, loss of consciousness)
- Assessment of the condition of the skin and mucous membranes (pale and sometimes cyanotic, erythema, rash, edema, rhinitis, conjunctivitis symptoms)
- Assessment of swallowing and breathing (difficulty swallowing, signs of acute respiratory failure)
- Determination of the nature of the pulse (thread-like and rhythm disturbances) and arterial pressure (decrease to 30-50 mm Hg)
- Determination of symptoms such as vomiting, involuntary urination and / or defecation, bloody discharge from the uterus.

COMA

Primary cerebral coma is based on the cessation of brain functions due to **primary brain damage**.

- Traumatic coma develops as a result of damage to the CNS during head trauma;
- Epileptic coma can occur during epileptic seizures;
- Apoplectic coma develops as a result of acute disruption of cerebral blood circulation;
- Meningeal coma is caused by intoxication during infectious meningitis;
- Apoplectic coma can develop as a result of secondary disruption of cerebral blood circulation during myocardial infarction;

Coma (unroutable and unresponsive state)



Coma of endocrine origin - deficiency or excess of hormone synthesis, caused by metabolic disorders due to taking too many hormonal drugs.

- Acute lack of insulin during diabetic coma-diabetes results in ketoacidosis, plasma hyperosmia, significant hyperglycemia.
- Hypocorticotoid coma is associated with acute adrenal insufficiency.
- Hypopituitary coma is associated with a sharp decrease in the secretion of pituitary hormones;
- Hypothyroid coma develops due to a sharp decrease in the secretion or utilization of thyroid hormones

***Toxic coma* can develop due to the effects of exogenous poisons, liver or kidney failure, endogenous intoxications, toxicoinfections, pancreatitis, various infectious diseases. The following types are distinguished:**

- Barbiturate coma occurs during poisoning with derivatives of barbituric acid (phenobarbital, luminal);
- A carbon monoxide coma occurs during CO poisoning;
- Cholera coma develops as a result of poisoning with bacterial toxins during cholera;
- Eclamptic coma occurs during an eclamptic seizure;
- Hyperlactatacidemic or lactacidotic coma is caused by a sharp increase in the amount of lactic acid in the blood during diabetes;
- Hepatic coma occurs as a complication of liver failure;
- Uremic coma occurs during kidney failure.

Hypoxic or anoxic coma is caused by a sharp weakening of cellular respiration due to the inability of oxygen to enter the cells sufficiently or the blockade of tissue respiration enzymes. This type of coma belongs to:

- Anemic coma develops against the background of severe anemia;
- Asthmatic coma-bronchial develops during an asthma attack or asthmatic condition.
- Respiratory coma occurs during external respiratory failure. It is related to hypoxia, hypercapnia and uncompensated acidosis, and develops as a result of gas exchange disturbances in the lungs.

Developing comas associated with the loss of electrolytes, water and energy substances:

- Hunger coma develops during acute alimentary dystrophy;
- Hemolytic coma occurs during acute massive hemolysis;
- Malaria coma develops during a malarial paroxysm;
- Chlorpenic (hypochloremic) coma - occurs when chlorine is lost from the body (incessant vomiting, improper treatment with diuretics, long-term salt-free diet, polyuria stage of renal failure, swishes in the small intestine).

Thermal coma can occur during heatstroke.

Signs of a Diabetic Coma

Ways to Prevent It




 You feel shaky or tired

 You feel like you're starving or parched

 You become erratic


 You may feel faint, or your pulse races

 You sweat profusely or need to urinate a lot

 You start having seizures or feeling nauseous

Carry around a snack 

Plan your meals wisely 

Make sure your loved ones know the warning signs 

Monitor your medication regimen 



The severity of coma according to Glazko scale

Signs	Degree of dysfunction	Scores
Opening of eyes	Confused	4
	Upon request	3
	In response to pain irritation	2
	Is absent	1
Verbal reactions	Oriented	5
	Confused consciousness	4
	Soporuous state	3
	No consciousness	2
		1
Motor response to irritation		6
	Follows the requirements	5
	In response to pain irritation	4
	Unfocused reactions in response to pain	3
	Flexion synergy	2
	Extensor synergizm	1
	Is absent	0
Maximum score		15
Minimum score		3