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Chapter 2
LIFE-THREATENING CONDITIONS

2.1. Terminal state

According to the classification proposed by the Russian academician V.A. Negovsky, the process of dying includes 3 stages.

1. *Pre-agonic condition:* general inhibition and motor excitement while the mental state is confused. Blood pressure cannot be determined. The pulse is palpated only on the carotid and femoral artery. Pronounced dyspnea alternating with bradypnea, cyanosis, pallor, anuria. At the end of the pre-agony the excitability of respiratory center decreases — there is a terminal pause lasting from a few seconds to 3 or 4 minutes (breathing ceases, bradycardia sets in, the pupils are dilated, corneal and pupillary light reflexes disappear).
2. *Agony*: the last short flash *of life*. After a possible very short recovery, consciousness and eye reflexes subside completely. Pulse on large arteries becomes markedly weaker; ECG shows signs of hypoxia and cardiac arrhythmia. Pathological breathing is observed, which can be of two types: convulsive with a large amplitude (2–6 respiratory movements per minute) or weak, rare, superficial, with a small amplitude. Agony ends with the last breath and heart contraction, and clinical death follows.
3. *Clinical death* is marked by cessation of cardiac and respiratory activity, and by a sudden depression of brain functions presenting in the form of a triad of clinical signs, usually easily detectable:
   - asystole (no pulsation on the carotid and femoral artery);
   - apnea;
   - coma (unconsciousness), dilation of pupils with no light reflex.

Causes of cardiac arrest may be classified into 2 groups: cardiogenic and non-cardiogenic. The first group includes mechanical injury to the heart, embolism of coronary arteries, acute coronary syndrome (ACS) , severe cardiac arrhythmia; the second group covers cases of primary accidents in respiratory system, metabolism, and neuroendocrine system. For instance, there are records of cases of cardiac arrest at the height of a strong psycho-emotional crisis due to hyperadrenia. Such arrest of a potentially healthy heart is a most favorable option for successful resuscitation. Reversibility of pathological changes is uncertain if clinical death has followed from multiple injuries, severe brain injury, previous hypoxia, or vast blood loss with a prolonged period of pronounced hypovolemia. In these cases, the compensation capacity of the body has already been largely exhausted before resuscitation.

Immediately after cardiac arrest and respiratory failure, metabolism sharply reduces, but does not stop completely due to anaerobic glycolysis. For this reason,

clinical death is a reversible condition, its duration is determined by how long the cerebral cortex viability persisted while blood circulation and respiration had subsided completely. Yet, clinical death can be reversed only if resuscitation procedure is successful.

One or another type of hypoxia underlies the dying process.

The brain is an organ most sensitive to hypoxia. Despite the compensatory body reactions (e.g., centralization of blood circulation in critical conditions), the brain functions are already destroyed at the pre-agonic stage, which manifests as consciousness disorder. With hypoxia building up, it leads to disappearance of corneal reflex, dilation of pupils, vasomotor and respiratory center disorder.

Cerebral cortex cells can sustain in the absence of blood circulation for 3–5 minutes, and then finally die. This is followed by so-called *social death*. Resuscitation measures at this point may successfully revive reflexes and spontaneous breathing, but consciousness is lost irreversibly. After 5 to 7 minutes brain death sets in, an irreversible destruction of all brain structures, including the midbrain, cerebellum and stem. Cardiac activity can still be restored, but spontaneous breathing is no longer restored. Thus, biological life of the body can still be maintained with artificial lung ventilation, but there is no chance for recovery.

Thus, in normal conditions, the duration of clinical death is 5 to 7 minutes, which leaves a very tight time frame for resuscitation. However, in conditions of hypothermia, when the level of metabolism, and hence the tissue need for oxygen is significantly reduced, the period of clinical death may be prolonged. In some cases, it may be as long as an hour.

Biological death is an irreversible condition that occurs after clinical death, where it is impossible to revive the body by any means. All the body tissues die, and the first to die, as noted previously, is the brain followed by the heart, lungs, liver, kidneys and lastly the skin. Objective signs of biological death are hypostatic spots, lowering body temperature and rigor mortis.

2.2. Shock

Classification and basis of pathogenesis

"Shock is easier to recognize than to describe, and easier to describe than to define" — this famous saying very accurately describes the many not so successful attempts to give a comprehensive description of shock (originating from Old French for "to blow, to shove"). Common definitions mention the impact of a strong pathological stimulus, disruption of the central nervous system and metabolism, multi-organic failure syndrome, decompensation of vital functions — and all these changes are there. However, the majority of definitions of shock rightly indicate an impairment of blood supply to tissues as the main factor ("critical reduction of tissue blood flow", "hypo-circulation syndrome", "reduced tissue perfusion", etc.).

Shock can be defined as a condition in which oxygen delivery to tissues is not adequate to their needs, leading to tissue hypoxia, multiple organ dysfunction, and metabolic disorders.

Reasons that can cause shock are very diverse: mechanical or thermal trauma, bleeding, heart disorder, pulmonary embolism, allergic reactions (anaphylaxis), intoxication, hormonal disorders, injury to the central nervous system, overdose of neurotropic drugs (narcotic drugs, sedatives), pain. Nevertheless, despite the striking dissimilarity of these causes, they all cause the same type of condition called shock.

One of the most common classifications of shock is based on the type of circulatory disorders of different causes.

Hypovolemic shock is caused by a loss of a significant amount of blood (due to bleeding) or plasma (due to edema or dehydration). This loss is called absolute hypovolemia. It should be remembered that even in the absence of massive external bleeding, acute blood loss could develop due to bleeding in body cavity and formation of retroperitoneal and intermuscular hematomas. The volume of blood loss in closed bone fractures can be significant and range from 1500 to 2000 ml in pelvic displaced fracture; from 800 to 1000 ml in femur (diaphysis) fracture; from 350 to 650 ml in fracture of both shins; from 200 to 500 ml in humerus fracture; from 100 to 150 ml in rib fracture.

Cardiogenic shock is caused by a decrease in the heart's ability to pump blood into great vessels, which most often happens at myocardial infarction or cardiac arrhythmia.

Obstructive shock is caused by decreased permeability of great vessels due to arterial thrombosis or thromboembolism.

Distributive shock is caused by blood deposition in peripheral vessels because of microvasculature block or vasodilation. This may be due to drug-related, hormonal, or neurogenic (including pain) factors, intoxication, allergic reactions. Blood does not leave the vascular bed, but a part of it stops the small peripheral vessels, not actually participating in bloodstream. Thus, a sort of "bleeding into one's own vessels" happens. This condition is known as *relative hypovolemia*.

When hypovolemia (both absolute and relative) reaches certain limits (deficit of circulating blood volume up to 10%), venous vessels adapt to the changed blood volume. With the depletion of venous motor function, central venous pressure decreases, and venous return goes down. This leads to a decrease in blood volume entering blood vessels of the circulatory system on heart contractions ("*reduced cardiac output syndrome*").

Low cardiac output syndrome underlies the pathogenesis of all types of shock.

Thus, all the dissimilar causes of shock lead in different ways to the same type of state — low cardiac output syndrome. The syndrome occurs if:

- volume of blood or plasma in circulatory bed decreases (absolute hypovolemia, hypovolemic shock);
- contractility of the heart reduces (cardiogenic shock);
- permeability of great vessels reduces (obstructive shock);
- blood is deposited in peripheral vasculature (relative hypovolemia, distributive shock).

From here on the pathological process develops in a common way.

Low cardiac output syndrome leads to a decrease in organ blood flow. Under these circumstances, adaptive reactions aim primarily to maintain adequate blood supply to vital organs (heart and brain) at the expense of peripheral vasoconstriction, ensuring blood pressure above the critical level (the phenomenon of *centralization of blood circu-*

lation). The longer the reaction continues, the less practical it becomes. Increasing stasis and blood deposition in venules and capillaries cause or exacerbate relative hypovolemia.

Due to tissue hypoxia and stimulation of anaerobic metabolism, the number of under-oxidized intermediary products increases, and metabolic acidosis develops. Blood corpuscles stack and form the so-called "rouleaux" (*sludge syndrome*). As a consequence of decreased volume rate of blood flow, tissues have time to "pick up" almost all oxygen from blood, which lowers oxygen content in venous blood from 14–15 to 4–5%. The body tries to saturate blood with oxygen by increasing the number of respiratory movements and chest excursions, yet hyperpnea and inclusion of auxiliary muscles in breathing lead to an increased need of oxygen for respiratory muscles. In addition, the emergence of functional vascular shunts in lungs does not allow saturation of the blood with oxygen.

Reduced organ blood flow after the centralization of blood circulation has started leads to multi-organ failure syndrome:

- skin — coldness, cold sticky sweat, chills;
- kidneys — oliguria; later – anuria, "shock kidney", uremia;
- intestinal tract — ischemic focal necrosis, "stress" ulcers, hemorrhages, reduced barrier function of mucous lining with developing bacteremia and intoxication;
- lungs — hyperpnea, "shock lung", respiratory failure;
- liver — focal necrosis in the center of hepatic lobules, detoxification function impairment (Fig. 2.1).

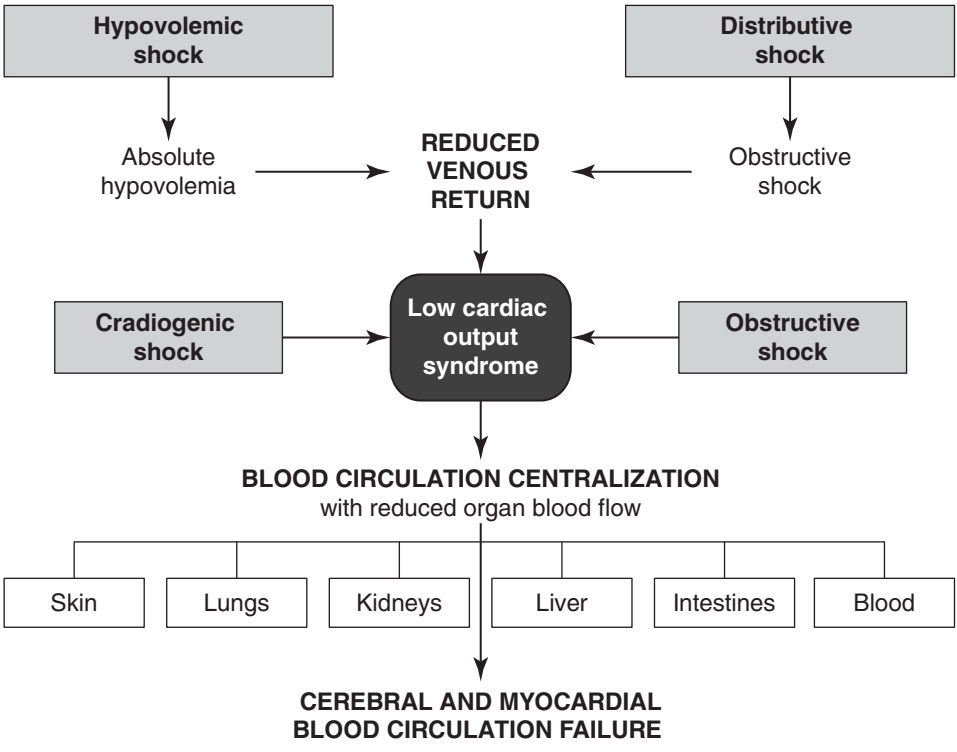

Fig. 2.1. Pathological disorders in shock development

With the mechanism of circulatory centralization depleting, homogenous distribution of blood throughout the body returns. Developing concurrent hypoxia of heart muscle leads to decreased contractility, exacerbating the low cardiac output syndrome, and hypoxia of brain — to central regulation of vascular tone disorder, which contributes to relative hypovolemia augmentation.

In case of mechanical trauma, various etiological factors are combined in shock development: hypovolemic syndrome due to blood or plasma loss, afferent impulses (related and not related to pain) from the lesion, injury to vital organs, autotoxemia, psycho-emotional stress. Thus, a shock that develops as a result of mechanical trauma (sometimes referred to as traumatic shock) is actually a *combined* one with predominant hypovolemic component.

Late or incomplete elimination of the following 4 factors has the most pronounced negative impact on the prognosis:

1) hypovolemia;
2) respiratory failure (necessarily developing in severe shock);
3) bleeding;
4) pain syndrome.

Criteria for assessing shock severity. For reliable assessment of a shocked victim's state, it is necessary to control cardiac output, acid-base balance and a number of other parameters. However, such examinations at the prehospital stage are difficult or impossible. Before evacuation to hospital, it is important to determine early whether shock has developed or not, and if shock is present – to establish its severity quickly and without the use of complex equipment. Several indications of the hemodynamics adequacy as a whole will help to achieve that.

Systolic BP level is an extremely important indicator that indirectly characterizes the blood perfusion volume of internal organs, including brain, cardiac muscle, kidneys.

Systolic BP below 60 mm Hg, as a rule, indicates a "failure" in the mechanism of blood circulation centralization with brain hypoxia, myocardial ischemia, and oligoanuria developing. However, BP alone cannot reliably mirror the state of organ and tissue blood flow in other parts of the body; it must be used in combination with other clinical evidence of shock. At the same time, popularity and easy access to BP measure often make doctors exaggerate the importance of this indicator for prognosis and assesment of hemodynamic state, prompting to treat not the patient, but his "blood pressure".

> BP surge is a relaxing sign only if combined with normalization of central venous pressure and hourly diuresis, skin getting warm and pink.

Shock index (sometimes called Allgower index) is calculated as the ratio of pulse rate to systolic BP. Normally, shock index is between 0.5 and 0.6. When the value is over >1.5, shock is considered irreversible.

Central venous pressure is detected with catheter inserted into central vein. This indicator characterizes venous return: the ability of cardiac muscle to cope with this blood flow. Normal value of central venous pressure is between 60 and 140 mm Aq.

Diuresis per hour is indicative of organ blood flow, which decreases when centralization of blood circulation starts. Lower limit of normal in the absence of in-

fusion therapy is 30 ml/h, with ongoing infusion – 50 ml/h. Since hourly urine output has a high diagnostic and prognostic value, it must be measured in all severe injuries.

For all victims with severe trauma constant bladder catheterization is indicated

Limb skin color and temperature characterize peripheral blood flow. Cold pale skin and pale nails are often a sign of hypovolemia with centralization of blood circulation.

Warm skin and pink nails indicate good peripheral blood flow and are a favorable sign even when BP is still low.

Mottled skin (*livedo reticularis*) and cyanotic nails that become white for a long time upon pressure indicate a transition from peripheral vascular spasm to paresis, which is a dangerous sign of imminent irreversible impairments. Normally, the time needed for nail bed capillaries to refill after pressure is no more than 2 seconds. Exceeding this time is called a *sign of "spots"*.

Rectal skin temperature gradient is an objective and very sensitive criterion for evaluation of peripheral hemoperfusion. This indicator is defined as the difference between temperature in rectal lumen at 8–10 cm, and the temperature of skin at distal point in systemic circulation (on the back of the foot at the base of the first finger). Normally, the gradient is 3 to 5 °C. It is necessary to note that this indicator makes it possible to objectively assess the state of microcirculation in hypotension, normotension and hypertension. Monitoring the dynamics of rectal dermal temperature gradient allows controlling the effectiveness of anti-shock therapy and has a great prognostic value. As an additional measure, rectal and axillary temperature can be compared. If the latter is lower than the former by more than 1 °C, perfusion of peripheral tissues is impaired.

Temperature of mixed venous blood. In shock, circulation flow slows down, and the temperature of venous blood may drop to 30 °C. It increases with improvement, and decreases upon negative changes.

Effect of anti-shock therapy. Absence of pressor response to intravenous administration of norepinephrine (Noradrenaline) (15 mg in 500 ml solution) or to intra-arterial blood or blood substitute pumping indicates irreversible shock and suggests an unfavorable prognosis.

Depending on diagnostic capabilities, a small and a specialized program for monitoring general condition of the victim is determined.

The small program is open to any doctor with the simplest equipment, and can be implemented at both hospital and in pre-hospital stage. It includes measuring the following parameters:

- BP;
- central venous pressure (if central vein is catheterized);
- respiration rate;
- hourly diuresis;
- intensity of blood flow — skin color, body temperature, capillary nail refill test.

Specialized program includes determining a number of laboratory and functional parameters (biochemical and gas composition of blood, acidic-base condition, co-

agulogram, monitoring of vital organs and systems, peripheral resistance of vascular bed, etc.), which is available only in specially equipped hospitals (usually in intensive care units).

Shock severity

As early as in XIX century N. Pirogov described the erectile and torpid phase of shock. The erectile phase is marked by motor and speech excitation, increase or preservation of systolic BP ("...the wounded one moans and cries loudly, his face convulsively twists, becomes pale, blue and swollen from the cry, the pulse is tense and quick, breathing short and frequent"). At the same time, the erectile phase is quite rare. It is detected only in 8.5 to 10.0% of patients at prehospital stage, and in 4 to 5% — at the in-hospital stage, which does not allow considering the erectile phase a reliable diagnostic sign of developed shock.

In contrast, the torpid phase of shock is characterized by retardation, decrease in systolic BP (N. Pirogov called such victims "stiff", as they "...do not cry, do not complain, are not concerned about anything; body cold, face pale, gaze fixed, pulse and respiration scarcely perceptible, wounds and skin are almost completely insensitive"). The torpid phase is attributable to any severe shock. Yet, it combines states of various pathological nature, of different prognosis and necessary therapy. Thus, in practice this classification alone is not practical.

Until lately, 4 degrees of shock severity were distinguished based on systolic BP level, state of consciousness and shock index. However, this division does not fully reflect the nature of body changes, is not prognosis-oriented, and does not meet the needs of diagnostic triage and determining victim transportability in mass casualty emergencies. In accordance with the current classification, shock progress has 3 phases: compensated shock corresponding approximately to severity I or II according to the "traditional" classification), decompensated reversible shock (corresponds to approximately severity III), and decompensated irreversible shock (corresponds to degree IV).

Compensated shock. Deficient volume of circulating blood is compensated by centralization of blood circulation: even a 25% decrease in blood volume may not be accompanied by BP decrease. Central venous pressure index decreases insignificantly. At the same time, peripheral hemoperfusion noticeably decreases. Pale skin and mucous membranes and "spots" symptom are observed. Shock index is 0.7 to 1.2; rectal and skin temperature gradient does not exceed 7 °C. Diuresis may drop 1.5 to 2 times compared to normal, but does not go below critical level of 30 ml/h. Signs of brain or myocardial hypoxia are absent, since centralization of blood circulation is still effective.

Decompensated reversible shock. High peripheral vascular resistance is no longer able to compensate for the small cardiac output, which leads to a decrease in systemic BP. Hypotension indicates inefficiency of blood circulation centralization. Deficient volume of circulating blood is 30–40%. Organ blood flow in the brain, heart, liver, kidneys, intestines is impaired with signs of hypoxia. Heart tones become muffled suggesting ischemia (determined by ECG) and worsening myocardial contractility. Oliguria is associated with impaired renal blood flow and reduced hydrostatic pressure. Consciousness impairment (stupor), hyperpnea, cold

limbs are observed; tachycardia increases to 120 beats per minute. Shock index is 1.3 to 1.5; rectal and skin temperature gradient is 7 to 16 °C. Metabolic disorders aggravate, decompensated acidosis develops, which demands correction. Sensitivity of vessels to endo- and exogenous pressor amines decreases.

Decompensated irreversible shock. Impairments that began during the period of blood circulation decompensation aggravate further. The deficient volume of circulating blood is over 40%. A dangerous sign is appearance of acrocyanosis while the skin is generally pale, along with hypotension and anuria. The term "irreversible" is conventional to an extent, yet the pathological changes in the body are so deep already that chances of their elimination and rehabilitation are rather theoretical, as a rule. If blood circulation decompensation persists for a long time (over 12 h) and tends to deepen, the shock should be considered irreversible. Shock index is over 1.5 by that time, rectal and skin temperature gradient is over 16 °C, venous blood temperature is below 30 °C, the patient is unconscious, there is no pressor reaction to therapy (table 2.1).

Particular attention should be paid to timely detection of decompensated reversible shock, since this condition is a kind of "border between life and death". Even a slight further deterioration renders the shock irreversible, and the prognosis becomes dismal. It is with decompensated reversible shock that the therapy necessary should be carried out as intensively as possible, while carefully avoiding any actions that are not absolutely mandatory (manipulations, operations, transportation and even position changing) and can aggravate the severity of the victim's condition even in a slightest and briefest manner.

> As a rule, shock becomes irreversible if comprehensive care is not provided within 2 to 4 hours.

Some features of shock progress

In the first hours of injury to a lower body part, acute blood loss (absolute hypovolemia) is the leading factor in the development of shock, while toxemia takes up increasingly later on. At chest injuries, impairment of gas exchange and heart pumping function, irritation of the extensive receptor field are essential in addition to blood loss. Combined injuries with dominant traumatic brain injury are especially difficult for shock diagnosis. BP can remain normal or even elevated for a long time at such severe lesions.

> Normotension in head trauma and restless behavior often indicate progressing brain hypoxia. The doctor should be troubled rather than comforted by it!

Various groups of victims display different aspects of shock progress.

Elderly people usually suffer from a number of chronic diseases, which results in significantly reduced compensatory capabilities. Shock develops at a much smaller total circulating blood volume deficit than with younger people. In addition, most elderly people have hypertension, so this should be assumed in the absence of information about "working" values of the victim's BP.

Table 2.1. Essential indications when assessing shock severity

Shock severity			Systolic BP	Shock index	Deficiency of circulating blood	Deficiency of circulating blood decrease	Venous blood temperature	Rectal and skin temperature gradient	Diuresis	Skin	Consciousness
Traditional classification		Current classification									
I		Compensated	>70	0.7–1.2	10–25% (350–1300 ml)	± (insignificant)	Negligibly lower		Low, but no more than 30 ml/h	Pallor, "spots" sign	Persists
II											
III		Decompensated reversible	50–70	1.3–1.5	30–40% (1300–1800 ml)	± (insignificant)	30 to 34°C		Oliguria (<30 ml/h)	Mottled, chillness	Stupor
IV		Decompensated irreversible	<50	>1.5	>40% (1800–2500 ml)	+++ (pronounced)	<30°C		Anuria	Acrocyanosis	Absent

"Normal" BP level with an elderly victim against the background of chronic hypertension can lead to erroneously good assessment of their condition.

With children, the peculiar feature of the body is the ability to maintain normal BP even after a serious injury. However, prolonged and persistent centralization of blood circulation in the absence of appropriate therapy is suddenly replaced by decompensation of hemodynamics, which is much more difficult to correct in children than in adults.

The younger the child, the more unfavorable is hypotension as a prognostic sign of shock.

In pregnant women, when centralization of blood circulation starts, the body tries to maintain its own homeostasis primarily at the cost of fetal homeostasis. In this context, anti-shock therapy aimed at combating hypotension and hypoxia should be carried out as soon as possible after injury, before the fetus sustains irreversible changes. In shock management, the volume of infusion should exceed the "estimated" volume by at least 25%, taking into account elevated total volume of circulating blood in late pregnancy. Approximately 10% of heavily pregnant women have hypotension in dorsal position.

Pregnant accident victims should be transported in "half-rolled on the left side" position.

2.3. Acute respiratory failure

Respiratory failure is the inability of respiratory system to maintain adequate gas exchange.

Upon an injury, an obstacle preventing oxygen delivery to tissues and removal of carbon dioxide can occur at any stage of gas exchange (in the chain: external respiration — blood — circulatory system — tissue respiration). These are the types of hypoxia:

- *respiratory hypoxia* developing as a result of airway obstruction, mechanical injury to lungs, violated sealing of pleural cavity or chest frame, or inhaling liquid or toxic substances;
- *hemic (isotonic) hypoxia* due to blood loss, or reduction in amount of hemoglobin;
- *circulatory (stagnant) hypoxia* resulting from disorders of blood circulation inevitable at shock, especially in microcirculation system;
- *histotoxic hypoxia* developing due to the appearance of toxic substances in the blood (endogenous, bacterial toxins).

A combination of various gas exchange disorders arising from injuries is called traumatic syndrome of acute respiratory failure.

The causes of acute respiratory failure at injuries can be pneumothorax, hemothorax, forced chest compression (traumatic asphyxia, sometimes called Olivier syndrome), spinal cord injury associated with spine trauma, aspiration of blood or vomit, etc. In the development of acute respiratory failure, general pathophysiological mechanisms are involved. Behind them are defects in locomotor respiratory system,

airway obstruction at various stages, retraction of lungs due to compression (excess atmospheric pressure, blood leaking into pleural cavity), hemorrhage into parenchyma, interstitial edema, diffusion disorders due to thickening of alveolar-capillary membrane (vascular-alveolar bypass). Any of these leads to severe hypoxia, aggravated by an increase in the oxygen "breathing cost" due to increased load on respiratory muscles.

Disorders of pulmonary gas exchange at trauma and shock occur mainly due to changes in the optimal ratio of ventilation and blood flow, increased venous blood shunting in lungs, leading to impaired blood oxygenation. Disorders of alveolar blood perfusion leads to an increase of the so-called physiological "dead space". The blood flowing into lungs bypasses alveoli thus ceasing to be saturated with oxygen, to release carbon dioxide, and showing a composition similar to venous blood. It has been established that at pulmonary "shunt" of 50% (the normal values are 5 to 7%), mortality of victims with mechanical injuries reaches almost 100%.

Respiratory failure syndrome occurs in any serious condition associated with severe disorder of general circulation. This abnormality is called acute respiratory distress syndrome (ARDS). In Russian literature the following terms are often used: respiratory distress syndrome of adult, acute respiratory distress syndrome, or the "shock lung". All of these terms are synonyms and describe the same state.

> Any serious injury is accompanied by respiratory system disorder, even if the chest is not injured.

Normally, the inner surface of the alveoli is covered with a layer of *surfactant* – a surface-active agent that stabilizes alveolar volume by reducing surface tension and thus ensuring normal gas exchange. The film of surfactant should be constantly renewed, which is the function special cells located in the alveoli walls and producing the surfactant (Fig. 2.2).

Various factors can lead to development of respiratory distress syndrome. They can be divided into 2 groups.

Primary pulmonary lesion develops when surfactant layer covering the alveoli from the inside erodes at first. The cause of such destruction can be aspiration (of water, blood, vomit), inhalation of toxins and irritants, pulmonary infection (including pneumonia), as well as lung trauma (injury or contusion).

Secondary pulmonary lesion is associated with depression or cessation of surfactant production, whereby alveolar surfactant layer is not renewed. The cause may be drug-related (including narcotics overdose), immunological reactions, pulmonary embolism, fat embolism, sepsis, transfusion complications, and body's response to any extrapulmonary processes associated with impairment of blood flow to organs (including the consequences of severe trauma outside the chest).

At respiratory distress syndrome, interstitial pulmonary edema develops with bronchospasm events, increased pressure in pulmonary circulation, loss of elastic properties of pulmonary parenchyma. Alveoli are filled with edematous fluid, which leads to a severe form of acute respiratory failure due to impairment of the alveolar diffusion of gases. Lung ventilation work increases significantly, the "dead space" grows, and arterial hypoxemia resistant to oxygen therapy aggravates.

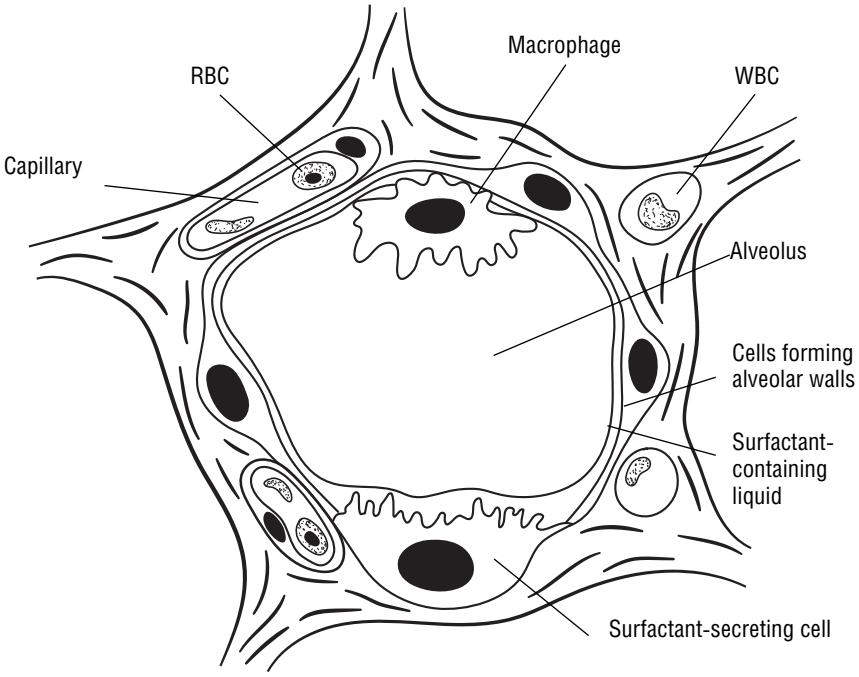

Fig. 2.2. Alveolus

Mortality associated with "shock lung" amounts to 50 to 75% according to various estimates.

There are two main body mechanisms of compensation of acute respiratory failure: stimulation of external respiration and increasing oxygen absorption by tissues. Shortness of breath is the first compensatory response, since it is energetically more profitable for the body to increase inhalation depth at first. At the same time, breathing becomes more frequent (tachypnea), which decreases respiratory volume and lung capacity. Another compensatory mechanism is to increase oxygen consumption and utilization by cells. It is implemented by increasing the coefficient of oxygen utilization and shifts promoting dissociation of oxyhemoglobin in tissues.

In the development of acute respiratory failure of any etiology, it is customary to differentiate the compensated and decompensated stage.

Compensated stage is marked by skin pallor, moderate participation of auxiliary muscles in breathing process, shortness of breath with no more than 30 respiratory movements per minute, emergence of scattered, mostly dry rales at auscultation; tachycardia up to 100 beats per minute.

Decompensated stage occurs when pathological factors remain untreated. The indications are: severe general condition; acute pallor and cyanosis of skin, visible mucosa and subungual hematoma; frequency of respiratory movements increase to over 30 per minute; active participation of auxiliary muscles and nasal alae in respiratory act; rough dry and wet rales audible at a distance; pronounced tachycardia; and frequently arrhythmia, hypotension. In acute blood loss, arterial hypoxemia can also occur without cyanosis, which requires a 5% concentration of reduced hemoglobin,

while such conditions often do not occur due to the loss of hemoglobin with blood. Hypercapnia in injuries is rare.

A great help in diagnosing is X-ray examination. Even in the early stages, it allows detecting signs of the onset of "shock lung" (intensified vascular pattern, signs of venous hypertension, etc.).

Fat embolism. This condition is considered a pulmonary abnormality developing as a result of injury to fat depots. The main source of embolic fat are traumas and pathological conditions. The most common causes of fat embolism are:

- bone injury;
- soft tissue wounds (especially of adipose tissue);
- surgical interventions (primarily orthopedic surgery and liposuction);
- ruptures of parenchymal organs;
- some morbidities (osteomyelitis, fatty degeneration of blood clots, jaundice, diabetes, etc.).

Droplets of fat enter vascular bed directly (in vessel injury) or as a result of transudation, which upon a fracture is promoted by to repeated manipulations on bone fragments, insufficient immobilization or development of intense hematoma in the injured area.

The majority of fat droplets enter the lungs leading to embolism; and only a small part of them go into other organs (brain, kidneys, myocardium).

The lung responds to the presence of emboli by lipase secretion, which influences fat hydrolysis glycerol and free fatty acids. Surfactant is injured last ("primary pulmonary lesion"), which leads to alveoli dysfunction, disruption of the mechanism of pulmonary gas exchange, development of respiratory distress syndrome ("shock lung") and ultimately – to severe respiratory failure.

2.4. Coma

Coma is a profound depression of the functions of central nervous system characterized by loss of consciousness and insensitivity to external stimuli. This condition may be caused by various injuries or metabolic disorders. Comas can be exogenous (caused by external factors) and endogenous (caused by a violation of activities of various physiological systems with severe metabolic disorders).

The causes of coma may be different.

Exogenous coma may be:

- traumatic (brain injury);
- hypo- and hyper-thermal (hypothermia, heat and sunstroke);
- exotoxic (poisoning with drugs, carbon monoxide, or infectious intoxication);
- alimentary (severe starvation);
- hypoxic (asphyxia).

Endogenous coma can be:

- hypoxic, anemic (disorders of circulatory system);
- diabetic, thyrotoxic (disorders of the endocrine system);
- uremic (disorders of excretory system);
- hepatic (disorders of liver functions);
- endotoxic (in ischemic tissue lesion).

Despite the variety of coma causes, this condition is characterized by common mechanisms of consciousness and brain reflex activity disturbance.

> The immediate cause of coma of any etiology is a discrepancy between the brain's need for oxygen and energy substrate (glucose), and the possibility to meet these needs.

Coma is often accompanied by cerebral edema. Dislocation of brain structures and their compression due to edema are the most important causea of vital organ dysfunction.

Although the word "coma" comes from the Greek *kõma*, meaning "deep sleep", the origin, development and significance of this state is fundamentally different from sleep. Pathological deep sleep can be interrupted by sufficiently strong external stimuli, and then consciousness returns; after cessation of irritating effects sleep can resume. In contrast, a patient who is in coma does not regain consciousness even with intense external stimuli.

However, coma should be distinguished from *stupor*, where there is a kind of cataplexy and disorder of many reflex reactions, but consciousness is preserved. It is also necessary to tell apart coma and shock. Shock is characterized by a two-phase change in the state of central nervous system and physiological functions (a period of excitation alternates with a period of retardation). In coma, there is typically an ever-increasing inhibition of brain functions and the activity of physiological systems.

The development of coma is preceded by a pre-coma state, or stupor, which is characterized by mental confusion with recurrent elucidation. Patients react to strong impacts, just as pain stimuli. While conscious, patients are indifferent to the environment; respond to questions with monosyllabic, sometimes inadequate answers.

Coma is characterized by complete loss of consciousness: reaction to various reflexes, including pain, is absent. With aggravation of coma severity, reflexes such as tendon, pupillary and corneal do not work. Pathological forms of breathing are common (e.g., Kussmaul breathing), cardiac activity weakens, cardiac hypotension occurs, body temperature decreases. Comatose state with motor excitation attacks, delusions and hallucinations, central paralysis events is possible. Coma can last for a quite significant time (several hours and even days). In the case of an unfavorable course of coma, it can go into a terminal state.

Essential treatment is to eliminate brain hypoxia combined with artificial replacement of impaired vital functions. Artificial lung ventilation, hypothermia (to reduce the need for oxygen in cells), parenteral nutrition, dehydration, and preventive measures against complications (such as bedsores, hypostatic pneumonia, urinary infection, stress ulcers, drying of the cornea, etc.) are used.

If the treatment was effective, further improvement with a gradual return of contact is possible. However, after a total brain lesion, memory disorders, attenuation of concentration, loss of initiative, personality change, and regression of intelligence often persist. Full recovery is rarely possible and solely in young patients.