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The Problem Of Traumatic Brain Injury In Humans.

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ABSTRACT

Traumatic brain injury is one of the most common types of damage and accounts for up to 50% of all types of injuries. Mortality from traumatic brain injury is 1% of total mortality. The frequency of traumatic brain injury and the severity of its consequences give this state great social significance. Traumatic brain injury mainly receives the most active and important socially and labor contingent of the population - people up to 50 years. It also determines large economic losses due to high mortality, frequent disability of the victims, as well as their temporary disability. There are five categories of traumatic brain injury outcomes: death, permanent vegetative state, severe disability, moderate disability and good recovery. Assessment of the outcome of traumatic brain injury can be carried out no earlier than 6 months after the trauma suffered. Of those admitted to hospitals with severe traumatic brain injury, about 60% survive. Of this group of patients, only 7% have a good recovery after discharge from the hospital. Almost all patients with severe traumatic brain injury have various neurological disorders. However, with the concussion of the brain, the vast majority of patients recover fully. The outcome of a brain contusion and open skull damage depends on the severity of the brain damage. In most cases, survivors have residual cerebral symptoms. Timely removal of a hematoma saves a sick life and in many cases leaves no symptoms. With severe brain damage and delayed medical care, mortality can reach 40-50%.

Keywords: brain brain, higher nervous activity, head injury, brain injury, skull damage.

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INTRODUCTION

The successful functioning of the human body is possible in the absence of injuries and various defects [1, 2]. Unfortunately, human activity is associated with a high risk of injury [3]. It is recognized that among the causes of death in young and middle age, injury takes the first place [4]. In this case, traumatic brain injury is one of the most common types of damage and accounts for up to 30% of all types of injuries. Mortality from traumatic brain injury is 1% of total mortality. The frequency of traumatic brain injury and the severity of its consequences give the problem great social significance. Traumatic brain injury mainly receives the most active and important socially and labor contingent of the population - people up to 50 years. It also determines large economic losses due to high mortality, frequent disability of the victims, as well as temporary disability [5].

Traumatic brain injury is a mechanical damage to the bones of the skull or intracranial formations, soft tissues, such as brain tissue, blood vessels, nerves, brain membranes. There are two groups of craniocerebral injuries - open and closed. In view of the great medical significance and danger to a traumatic brain injury in a person, the goal is to summarize the basics of the biosocial and medical aspects of traumatic brain injury described in the available literature.

CLASSIFICATION AND SYMPTOMS OF TRAUMATIC BRAIN INJURY

According to the severity of the lesion, there is a light, moderate severity and severe traumatic brain injury. The Glasgow Coma Scale is used to determine severity. In this case, the patient receives from 3 to 15 points depending on the level of impairment of consciousness, which is assessed by eye opening, speech and motor responses to stimuli. Light traumatic brain injury is estimated at 13-15 points, moderate - at 9-12, severe - at 3-8.

There are also isolated, combined (trauma is accompanied by damage to other organs) and combined (various traumatic factors affect the body) traumatic brain injury. Traumatic brain injuries are divided into closed and open.

With an open head injury, the skin is damaged, the aponeurosis and the bottom of the wound is bone or deeper tissue. Penetrating is considered an injury in which the dura mater is damaged. A particular case of penetrating trauma - otolikorreya as a result of fracture of the bones of the skull base. At the same time, if the dura mater is damaged, then an open wound is considered penetrating. With a closed head injury, the aponeurosis is intact, although the skin may be damaged [6].

All brain injuries are divided into:

- Concussion of the brain - an injury in which there is no persistent violations in the brain. All symptoms that occur after a concussion usually disappear over time (within a few days). Steady persistence of symptoms is a sign of more serious brain damage. The main criteria for concussion are the duration (from a few seconds to hours) and the subsequent depth of loss of consciousness and the state of amnesia. Non-specific symptoms - nausea, vomiting, pallor of the skin, cardiac abnormalities.
- Crush of the brain (hematoma, foreign body, air, injury site).
- Brain injury: mild, moderate and severe.
- Diffuse axonal damage.
- Subarachnoid hemorrhage.

At the same time, various combinations of types of craniocerebral injury can be observed: contusion and compression of hematoma, contusion and subarachnoid hemorrhage, diffuse axonal injury and contusion, contusion of the brain with compression of hematoma and subarachnoid hemorrhage [7].

Depending on whether the integrity of the skin of the skull and its tightness remain intact during an injury or if they are broken, the craniocerebral injuries are divided into closed and open. Traumatic brain injuries are traditionally divided into concussion, contusion and compression; conventionally, they also include a fracture of the base of the skull and fissure of the arch with the preservation of the skin.

Open craniocerebral injury includes fractures of the cranial vault, accompanied by injury of the adjacent soft tissue, fractures of the skull base, accompanied by bleeding or liquorrhea (from the nose or ear), as well as wounds of the soft tissues of the head with damage to the aponeurosis. With the integrity of the dura mater, an open head injury is attributed to non-penetrating, and in violation of its integrity - to penetrating.

Traumatic brain injury is divided into 3 stages: mild, moderate and severe. Mild brain injury includes concussion and mild brain injuries; moderately severe brain contusions; severe - severe brain contusions, diffuse axonal damage and compression of the brain [6].

According to the nature of the brain lesions, focal head injuries (arising mainly in shock-shock biomechanics), diffuse (acceleration-retardation occurring mainly due to trauma and deceleration) and its associated injuries are isolated.

Traumatic brain injury may be isolated (there are no extracranial lesions); combined (at the same time there are damage to the bones of the skeleton and / or internal organs), combined (simultaneously affect different types of energy - mechanical, thermal, radiation, chemical).

In terms of the onset of traumatic brain injury, it can be primary (when mechanical energy is not caused by any cerebral disorders immediately preceding it) and secondary (when mechanical energy is caused by the immediately preceding cerebral catastrophe causing the patient to fall, for example, with an epileptic seizure or stroke) [7].

Traumatic brain injury can be received for the first time or repeatedly, that is, to be first or second, third. During a traumatic brain injury, there are acute, intermediate, distant periods. Their temporal and syndromological characteristics are determined primarily by the clinical form of traumatic brain injury, its character, type, age, premorbid and individual characteristics of the victim, as well as the quality of treatment.

Concussion is characterized by a triad of symptoms: loss of consciousness, nausea or vomiting, retrograde amnesia. Focal neurological symptoms is absent.

Brain contusion is diagnosed in cases where cerebral symptoms are supplemented by signs of focal brain damage. Diagnostic boundaries between concussion and contusion of the brain and slight contusion of the brain are very unsteady, and in such a situation, the term "commutation-contusion syndrome" is most adequate, indicating the degree of its severity. Brain contusion may occur both at the site of injury and on the opposite side of the counter-strike mechanism. The duration of loss of consciousness during concussion is in most cases from a few to tens of minutes [8].

A brain contusion of a mild degree is characterized by turning off the consciousness up to 1 hour after the injury, complaints of headache, nausea, vomiting. In the neurological status, there is a rhythmic eye twitching when looking sideways (nystagmus), meningeal signs, reflex asymmetry. On radiographs, fractures of the bones of the cranial vault can be detected. In the cerebrospinal fluid there is an admixture of blood (subarachnoid hemorrhage).

A brain contusion of a moderate degree is characterized by a shutdown of consciousness for several hours. Expressed loss of memory (amnesia) on the events preceding the injury, the trauma itself and the events after it. Complaints of headache, repeated vomiting. Short-term disorders of respiration, heart rate, blood pressure are detected. There may be mental disorders. Marked meningeal signs. Focal symptoms manifested in the form of uneven pupil size, speech disorders, weakness in the limbs. When craniography often found fractures of the arch and base of the skull. When lumbar puncture - significant subarachnoid hemorrhage [9].

Brain contusion severe. It is characterized by a prolonged deactivation of consciousness (lasting up to 1-2 weeks). Gross violations of vital functions (changes in pulse rate, pressure level, respiration rate and rhythm, temperature) are detected. In the neurological status there are signs of damage to the brain stem - floating movements of the eyeballs, swallowing disorders, changes in muscle tone, etc. Weakness in the arms and legs up to paralysis, as well as convulsive seizures, can be detected. Severe contusion is usually accompanied by fractures of the fornix and base of the skull and intracranial hemorrhages.

The compression of the brain implies the development of a traumatic hematoma, often epidermal or subdural. Their timely diagnosis involves two unequal situations. With a simpler one, there is a "light period": a patient who has regained consciousness after some time begins to "load" again, becoming apathetic, lethargic, and then soporous. It is much more difficult to recognize a hematoma in a patient in a state of coma, when the severity of the condition can be explained, for example, by contusion of the brain tissue. The formation of traumatic intracranial hematomas with an increase in their volume is usually complicated by the development of a tentorial hernia — a protrusion of a brain squeezed by hematoma into the opening of the cerebellar branch, through which the brain stem passes. Its progressive compression at this level is manifested by a lesion of the oculomotor nerve (ptosis, mydriasis, divergent squint) and contralateral hemiplegia.

A fracture of the base of the skull is inevitably accompanied by contusion of the brain of varying degrees, characterized by the penetration of blood from the cranial cavity into the nasopharynx, into the periorbital tissue and under the conjunctiva, into the middle ear cavity (otoscopy reveals cyanotic eardrum color or rupture).

Bleeding from the nose and ears can be a consequence of local injury, so it is not a specific sign of a skull base fracture. Equally, the "symptom of glasses" is also often the result of a purely local trauma to the face. Pathognomonic, though not necessarily, the expiration of cerebrospinal fluid from the nose (rhinorrhea) and ears (otorrhea). Confirmation of the expiration of the cerebrospinal fluid from the nose is the "symptom of the teapot" - a clear increase in rhinorrhea when the head is tilted forward, and the detection of glucose and protein in the discharge from the nose, according to their content in the cerebrospinal fluid. A fracture of the pyramid of the temporal bone may be accompanied by facial paralysis and cochleovestibular nerves. In some cases, facial nerve paralysis occurs only a few days after the injury.

Along with acute hematomas, the trauma of the skull can be complicated by the chronically increasing accumulation of blood over the brain. Usually in such cases there is a subdural hematoma. As a rule, such patients - often elderly people with reduced memory, who suffer, in addition, with alcoholism - come to the hospital already in the stage of decompensation with compression of the brainstem. The trauma of the skull, which was many months ago, is usually not severe, is amnesized to the sick. Symptoms: impaired consciousness - stunning, stupor, coma. They indicate the presence of traumatic brain injury and its severity [10].

FIRST AID TO VICTIMS WITH SEVERE TRAUMATIC BRAIN INJURY

The results of the treatment of traumatic brain injury are largely dependent on the quality of pre-hospital care and the speed of hospitalization of the victim. It is hardly possible to find another type of injury, where the delay in the delivery of the patient to the hospital for an hour or two something significantly changed. Therefore, it is considered that the ambulance service, unable to deliver the victim with a severe traumatic brain injury to a neurosurgical hospital for several minutes, does not cope with their work. In many countries, patients with severe traumatic brain injury are transported to hospitals by helicopters [11].

Providing first aid at the scene, first of all, it is necessary to restore the airway. Along with oxygen deprivation (hypoxia), a frequent complication of traumatic brain injury is an increased accumulation of carbon dioxide in the body (hypercapnia). During transport, patients must breathe 100% oxygen. With multiple injuries accompanied by shock, simultaneously begin intravenous administration of Ringer's solution, reopolyglukine. Ischemia, hypoxia, or hypotension for a short period, even in cases of moderate traumatic brain injury, can lead to irreversible consequences. If a high spinal cord injury is suspected, the cervical spine should be immobilized.

Bleeding is necessary to stop the imposition of a tight bandage or rapid closure of the wound. Damage to the scalp, especially in the elderly, can lead to a dramatic weighting of the condition.

The generally accepted criteria for hospitalization for traumatic brain injury are:

- 1) a clear decrease in the level of consciousness,
- 2) focal neurological disorders (paresis of the limbs, uneven width of the pupils),
- 3) open fractures of the skull bones, bleeding or liquorrhea from the nose or ear canal,

- 4) epileptic seizures,
- 5) loss of consciousness due to trauma,
- 6) significant post-traumatic amnesia.

Caring for patients with severe traumatic brain injury is to prevent bedsores and hypostatic pneumonia (turning the patient in bed, massage, toilet skin, banks, mustard plaster, suction of saliva and mucus from the mouth, tracheal sanitation). It is necessary to monitor the possible development of complications of traumatic brain injury. These include violations of vital functions - a breakdown of the basic functions of life support (external respiration and gas exchange, systemic and regional blood circulation). In the acute period of craniocerebral injuries, among the causes of acute respiratory failure, pulmonary ventilation disorders prevail, associated with impaired airway patency caused by accumulation of secretions and vomitus in the nasopharyngeal cavity, followed by their aspiration into the trachea and bronchi, retraction of the tongue in patients with coma [12].

Dislocation process: temporal-tentorial inclusion, representing the displacement of the mediobasal divisions of the temporal lobe (hippocampus) into the slit of the necklace of the cerebellum and the insertion of the cerebellar tonsils into the large occipital foramen, characterized by the compression of the bulbar sections of the trunk.

Purulent-inflammatory complications are divided into intracranial (meningitis, encephalitis, and abscess of the brain) and extrahepatic (pneumonia). Hemorrhagic - intracranial hematomas, cerebral infarctions.

Mechanical trauma of the skull causes squeezed (transient or permanent) brain tissue, tension and displacement of its layers, transient sharp increase in intracranial pressure. The displacement of the medulla may be accompanied by a rupture of brain tissue and blood vessels, brain contusion. Usually, these mechanical disorders are complemented by complex dyscirculatory and biochemical changes in the brain.

Traumatic brain injury, depending on its severity and type, leads to various structural and functional brain damage at the subcellular, cellular, tissue and organ levels, and central regulation of the vital body functions. In response to brain damage, disorders of cerebral circulation, liquor circulation, and permeability of the blood-brain barrier occur. Due to excessive irrigation of brain cells and intercellular spaces, swelling and swelling of the brain develop, which, together with other pathological reactions, causes an increase in intracranial pressure [12].

The processes of displacement and compression of the brain, which can lead to the infringement of stem formations in the opening of the cerebellar branch or in the occipital-cervical dural funnel, are developed. This, in turn, causes further deterioration of blood circulation, metabolism and functional activity of the brain. An adverse secondary factor in brain damage is its hypoxia due to impaired breathing or circulation.

TREATMENT OF TRAUMATIC BRAIN INJURIES

All victims with concussion, even if the injury seems to be mild from the very beginning, must be transported to an on-duty hospital, where execution is performed, radiography of the bones of the skull is shown, for more accurate diagnosis, and if available, computed tomography of the brain can be performed.

Injuries in the acute period should be treated in the neurosurgical department. Patients with concussion are prescribed bed rest for 5 days, which then, taking into account the characteristics of the clinical course, is gradually expanded. In the absence of complications, discharge from the hospital is possible on the 7-10th day for outpatient treatment for up to 2 weeks [13].

Drug treatment for concussion of the brain is aimed at normalizing the functional state of the brain, removing headaches, dizziness, anxiety, insomnia.

Usually, the range of drugs prescribed for intake includes analgesics, sedatives and sleeping drugs. Analgesics (analgin, pentalgin, baralgin, sedalgin, maxigan) select the most effective drug in this patient. For dizziness, choose one of the available medicines. As sedatives, infusions of herbs (valerian, motherwort),

preparations containing phenobarbital (Corvalol, Valocordin), and tranquilizers (Elenium, Sibazon, Phenazepam, Nozepam, Rudotel) are used.

Along with symptomatic treatment for concussion of the brain, it is advisable to conduct a course of vascular and metabolic therapy in order to more quickly and completely restore the disorders of brain functions and prevent various post-communal symptoms. Appointment of vasotropic and cerebrotropic therapy is possible only 5-7 days after injury. Preferably a combination of vasotropic (cavinton, stugeron, theonikol, etc.) and nootropic (nootropil, aminolone, picamilon) drugs. The daily three daily intake of Cavinton is 1 tablet (5 mg) and nootropil 1 capsule (0.4) for 1 month [14, 15].

To overcome the frequent asthenic phenomena after concussion, multivitamins like Complivit, Centrum, and Vitrum are prescribed 1 tablet per day. Of the tonic drugs use ginseng root, extract of Eleutherococcus, lemongrass.

Concussion of the brain is never accompanied by any organic lesions. If any post-traumatic changes detected by computed tomography or magnetic resonance tomography are detected, it is necessary to speak of a more serious injury - a brain contusion.

The outcome of traumatic brain injury depends largely on the early assistance provided to the victim. Neurological status is assessed at this stage. Hypotension and hypoxia associated with traumatic brain injury are found in 50% of cases; hypotension accompanies systemic damage and may be due to hemorrhagic complications and a decrease in vascular tone with damage to the brainstem; hypoxia occurs with hemopneumothorax or with airway obstruction (usually upper). Causes of obstruction can be a coma and a recession of the tongue, the ingress of blood and aspiration masses into the respiratory tract.

Therapeutic measures are aimed at eliminating hypotension and hypoxia. Any patient with a traumatic brain injury should be considered as a patient with a full stomach, as there is a risk of aspiration of gastric contents into the tracheobronchial tree. Trained personnel at the scene should intubate the trachea, which reduces mortality in severe traumatic brain injury, and initiate intravenous infusion for the purpose of fluid resuscitation. Indications for tracheal intubation: obstruction of the upper respiratory tract, loss of protective reflexes of the upper respiratory tract, the inability of the patient to ensure the drainage of the respiratory tract, the need for mechanical support of respiration. Some authors highlight such indications as hypoxia, hypercapnia.

Spinal cord injury is recorded in 10% of traffic accidents. In order to avoid damage to the cervical spine, intubation is recommended when the head is in a neutral position. Intubation is facilitated by the administration of succinylcholine (1 mg / kg) and lidocaine (1.5 mg / kg intravenously). During the procedure, the method of traction of the head for the mastoid processes along the vertical axis of the body is used, which prevents over-bending and movement of the spine in the cervical region, while Selick is used to prevent aspiration and vomiting (pressure on the thyroid cartilage). During transportation, inhalation of 100% moistened oxygen is carried out, if necessary, assisted ventilation of the lungs is performed. The neck of the victim should be immobilized with a stiff collar. The victim is placed on a special board, which is tied with straps, which prevents the movement of the spine during transport. Board for immobilization should be radiopaque, which allows for the necessary research without shifting the victim [16].

At the scene, the correction of hypovolemic shock begins with the intravenous infusion of various solutions; after performing the catheterization of the peripheral vein, 500-1000 ml of isotonic solution, or 50-100 ml of 10% NaCl solution, or 250-500 ml of colloid solution are poured. The use of hypertonic NaCl does not cause an increase in intracranial pressure. At the prehospital stage, the volume of intravenous infusion is limited to avoid pulmonary edema, increased bleeding and increased intracranial pressure with a sharp rise in blood pressure. Mannitol is not used in the prehospital phase. According to numerous double-blind selective studies, dexamethasone and methylprednisolone, prescribed at the early stages of traumatic brain injury in appropriate doses, do not improve the clinical outcome.

In 40% of cases of traumatic brain injury, intracranial hematomas are diagnosed. Early surgical decompression is an imperative treatment method. With significant intracranial hemorrhage detected by computed tomography, delay with surgery during the first four hours increases mortality by up to 90%.

Clinical indications for surgery - a classic triad: impaired consciousness, anisocoria and hemiparesis. However, the absence of these symptoms does not exclude hematoma. A high probability of hematoma is observed in elderly patients, alcoholics, with injuries sustained during a fall, fracture of the skull bones (especially in the areas where meningeal vessels and venous sinuses pass).

At this stage, one of the most important tasks is the surgical reduction of intracranial pressure using the method of decompression craniotomy. The displacement of the median structures of the brain is a more reliable indicator for surgical intervention than the size of the hematoma. According to Ropper, the displacement of median structures by 8 mm is associated with coma; 6 mm - with deep stunning. The operation is shown with a shift of the median structures of more than 5 mm, an increase in intracranial pressure of more than 25 mm Hg. v.; serves as an indication for decompression craniotomy [17].

In view of the fact that there is a high risk of aspiration of gastric contents, to prevent aspiration, it is necessary to use crash induction — induction of anesthesia to rapid anesthesia (rapid segence induction) and taking Selick. Performing crash induction includes:

- preoxygenation with 100% oxygen for 3-5 minutes (with sustained independent breathing) [18];
- induction into anesthesia - a narcotic analgesic (5 µg / kg of fentanyl), intravenous anesthetic (5-6 mg / kg of sodium thiopental or 2 mg / kg of propofol). Doses of anesthetics depend on the depth of impairment of consciousness and the state of hemodynamics. The more pronounced disorders of consciousness and hemodynamics, the smaller the dose used. In patients with unstable hemodynamics, preference should be given to etomidate (0.2-0.3 mg / kg). Sodium thiopental and propofol are not indicated for patients with hypovolemia [19];
- rekurarization arduanom (10% of the estimated dose) for 5 minutes before the introduction of a muscle relaxant with a rapid onset of action (ditilina). The increase in intracranial pressure caused by ditilinom, short-term, single injection of this drug does not affect the outcome. In patients with paresis of the extremities (not earlier than 24 hours after traumatic brain injury), ditilin-induced hyperkalemia may occur, in such cases a relaxant of non-depolarizing type of action should be used;
- reception of Selick (pressure on the thyroid cartilage);
- tracheal intubation (laryngoscopy lasting less than 15 seconds). The position of the patient on the operating table with a raised head end at 30 degrees improves venous blood flow from the brain [20,21].

The issue of ventilatory support during anesthesia is very problematic. It should be said that hyperventilation has long become a routine method of treating patients with traumatic brain injury due to the fact that it causes vasoconstriction of arterioles of the brain and pia mater. It helps to reduce cerebral blood flow and volume, as well as intracranial pressure [22-25].

Known disadvantages of the method are hypoperfusion / ischemia (in already existing states of hypoperfusion) and inhibition of oxygen delivery due to the shift to the left of the oxyhemoglobin dissociation curve. When comparing patients who underwent hyperventilation with a decrease in PaCO₂ to 24 mm Hg. Art., with the control group, where RaSO₂ was reduced to 35 mm Hg. Art., revealed a significant difference in favor of the norm-ventilation, if we consider the clinical outcome 3-6 months after the injury. It has been proven that hyperventilation may have a beneficial effect in patients with increased cerebral blood flow, especially in young patients with predominant symptoms of cerebral edema with intact trunk function. Reducing intracerebral pressure effect of hyperventilation in patients with reduced cerebral blood flow (the late phase of traumatic brain injury, the acute phase in the elderly), if not completely absent, then very limited. Moreover, in such situations, hyperventilation can have a detrimental effect and cause further localized impairment of cerebral blood flow, which can fall below the ischemic threshold. It is usually recommended to continue artificial ventilation of the lungs in the postoperative period, since the maximum brain swelling occurs within 12-72 hours after injury [26-27].

The best method of anesthetic support in patients with traumatic brain injury should be considered to be the infusion of sodium thiopental at a rate of 4-5 mg/kg/hour. This method is especially useful for patients with severe traumatic brain injury and coma [28].

Nitrous oxide increases cerebral blood flow and the amount of air in the cranial cavity, so its use in its pure form during such operations is limited, although in a number of clinics they use N₂O in combination with

sodium thiopental infusion. This allows you to reduce the rate of infusion of the latter and, thus, to ensure the rapid awakening of the patient. When working with N₂O in this category of patients, ventilation should be carried out in the mode of moderate hyperventilation (PaCO₂ = 32 mmHg) and switched off before closing the dura mater [29,30].

An anti-depolarizing muscle relaxant is used to maintain myoplegia (vecuronium is preferred, but Arduan is widely used). Opioids are administered for the purpose of anesthesia during surgery. It has been established that fentanyl and sufentanil can increase intracranial pressure in traumatic brain injury. Maintaining blood pressure at a sufficient level with the use of opioids prevents the increase in intracranial pressure [31,32].

An important point during surgery before and after it is infusion therapy, which in patients with cerebral edema is somewhat different from that adopted in general anesthesiology and intensive care, although the general principles are preserved. Infusion therapy should ensure hemodynamic stability, prevent an increase in venous pressure in the cranial cavity, maintain a stable osmolarity of blood plasma and prevent the development of hyperglycemia and hypoglycemia. Perfusion pressure of the brain should be maintained at 80-90 mm Hg. Ast. [33,34].

During operations for the removal of acute epidural and subdural hematomas, especially with rapid decompression, there is a significant decrease in blood pressure, which can be aggravated by the initial hypovolemia and bleeding. In case of systemic injuries, patients are often hypovolemic, and the efforts of doctors should be directed at normalizing the volume of circulating blood. Hypovolemia can be masked by hypoxia, a sympathetic activation in response to an increase in intracranial pressure. To correct the initial hypovolemia, isotonic NaCl solution is poured to normalize arterial pressure, heart rate and diuresis. Hematocrit should be maintained at a level not lower than 30% in order to avoid brain ischemia. Isotonic solution of NaCl is the main and in most cases the only drug for patients with pathology of the cranial cavity. At the same time, it is important to remember that hypervolemia can increase swelling of the brain and contribute to an increase in intracranial pressure [35].

The anesthesiologist should strive for early awakening of the patient after surgery, which allows for an early neurological examination [36]. The presence of consciousness in the postoperative period greatly facilitates the monitoring of the patient and allows you to identify the development of complications earlier. Consciousness is the best criterion for assessing the patient's condition in the early postoperative period, however, early awakening of the patient should not be an end in itself. If the patient's condition allows, extubation is performed at the end of the operation. Along with stable hemodynamics, normal body temperature and adequate breathing, a necessary criterion for early extubation is the restoration of the patient's consciousness. If an increase in brain edema and an increase in intracranial pressure are expected and hyperventilation is supposed to be used to reduce it, we should not rush into extubation.

CONCLUSION

Traumatic brain injury is a severe and life-threatening pathology. There are five categories of traumatic brain injury outcomes: death, permanent vegetative state, severe disability, moderate disability and good recovery. Assessment of the outcome of traumatic brain injury can be carried out no earlier than 6 months after the trauma suffered. And admitted to hospitals with severe traumatic brain injury, 60% survive. Of this group of patients, only 7% have a good recovery after discharge from the hospital. Almost all patients with severe traumatic brain injury have various neurological disorders. With a concussion, the vast majority of patients recover completely. The outcome of a brain contusion and open skull damage depends on the severity of the brain damage. In most cases, survivors have residual cerebral symptoms. Timely removal of a hematoma saves a sick life and in many cases has no residual symptoms. With severe brain damage and delayed care, mortality can reach 40-50%.

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