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COMBAT-RELATED TRAUMATIC BRAIN INJURY: EPIDEMIOLOGY, CLASSIFICATION, PATHOGENESIS, AND CONTEMPORARY CARE PRINCIPLES

Juraev Anvar Mamatmurodovich
Specialized scientific and practical center for Neurosurgery and Neurorehabilitation at the Samarkand State Medical University

Abstract.
Background. Combat-related traumatic brain injury (TBI) in modern conflicts is dominated by blast and fragment mechanisms, frequent polytrauma, and a growing proportion of civilian casualties.
Objective. To summarize the contemporary evidence on combat TBI epidemiology, classification (soft-tissue, non-penetrating, penetrating injuries), key pathophysiological cascades (primary tissue disruption, secondary hypoxic–ischemic injury, infectious complications), and stage-based care principles across evacuation and definitive neurosurgical management.
Methods. A narrative review of international guidelines, military clinical practice guidance, and conflict-specific neurosurgical literature with emphasis on triage, neuroimaging, prevention of secondary insults (hypotension, hypoxia, intracranial hypertension), surgical debridement principles, antimicrobial and seizure prophylaxis, and early rehabilitation.
Results. Outcomes are strongly associated with trajectory and depth of injury (ventricular system, basal ganglia, brainstem involvement), time-to-imaging and time-to-surgery, standardized critical care (ICP/CPP targets where applicable), and systematic prevention of infection and seizures.
Conclusion. Improved routing, protocol harmonization, and multidisciplinary rehabilitation are pivotal to reducing mortality and long-term disability after combat-related TBI.
Keywords (EN): combat trauma; traumatic brain injury; penetrating brain injury; blast injury; neurosurgery; intracranial pressure; infection prophylaxis; rehabilitation.

БОЕВАЯ ЧЕРЕПНО-МОЗГОВАЯ ТРАВМА: ЭПИДЕМИОЛОГИЯ, КЛАССИФИКАЦИЯ, ПАТОГЕНЕЗ И СОВРЕМЕННЫЕ ПРИНЦИПЫ ОКАЗАНИЯ ПОМОЩИ

Жураев Анвар Маматмуродович
Специализированный научно-практический центр нейрохирургии и нейрореабилитации при СамГМУ

Аннотация.
Введение. Боевая черепно-мозговая травма (ЧМТ) в современных конфликтах характеризуется высокой долей минно-взрывных и осколочных поражений, множественностью ранений и значительным вкладом гражданских потерь.
Цель. Обобщить данные о структуре боевой ЧМТ, классификационных подходах (мягкие ткани, непроникающие и проникающие ранения), ключевых патогенетических механизмах (первичное разрушение, вторичное ишемически-гипоксическое повреждение, инфекционные осложнения) и принципах оказания помощи на этапах медицинской эвакуации.
Материалы и методы. Нарративный обзор современных клинических рекомендаций, военно-медицинских руководств и публикаций по опыту локальных войн/конфликтов с акцентом на нейрохирургическую тактику, профилактику осложнений, мониторинг и раннюю реабилитацию.

Результаты. Показано, что исходы определяются глубиной повреждения (вовлечение желудочковой системы, базальных ганглиев, ствола), своевременностью нейровизуализации, контролем вторичных факторов (гипотензия, гипоксия, внутричерепная гипертензия), адекватным хирургическим дебридментом, профилактикой инфекции и судорог.
Заключение. Оптимизация маршрутизации, стандартизация протоколов и мультидисциплинарная реабилитация являются ключевыми направлениями снижения летальности и инвалидизации при боевой ЧМТ.
Ключевые слова (RU): боевая травма; огнестрельное ранение; минно-взрывная травма; проникающая ЧМТ; вторичное повреждение мозга; инфекционные осложнения; внутричерепная гипертензия; реабилитация.

JANGOVAR BOSH MIYA SHIKASTLANISHI: EPIDEMIOLOGIYA, TASNIF, PATOGENEZ VA ZAMONAVIY TIBBIY YORDAM TAMOYILLARI

Jo’rayev Anvar Mamatmurodovich
Samarqand davlat tibbiyot universiteti huzuridagi Neyroxirurgiya va neyroreabilitatsiya ixtisoslashtirilgan ilmiy-amaliy markazi

Annotatsiya.
Kirish. Zamonaviy qurolli mojarolarda jangovar bosh miya shikastlanishi (BMS) koʻpincha mino-portlash va parcha-parcha jarohatlar, koʻp oʻchoqli shikastlanishlar hamda fuqarolar orasida yuqori talafotlar bilan kechadi.
Maqsad. Jangovar BMSning tuzilishi, tasnifi (yumshoq toʻqima, kirib bormaydigan va kirib boruvchi jarohatlar), patogenezning asosiy boʻgʻinlari (birlamchi destruksiya, ikkilamchi ishemik-gipoksik shikastlanish, infeksion asoratlar) va tibbiy evakuatsiya bosqichlarida yordam tamoyillarini umumlashtirish.
Material va usullar. Klinik tavsiyalar, harbiy-tibbiy qoʻllanmalar hamda lokal mojarolar tajribasiga oid maqolalar asosida narrativ tahlil; neyroxirurgik taktika, asoratlar profilaktikasi, monitoring va erta reabilitatsiyaga urgʻu berildi.
Natijalar. Prognozga shikast chuqurligi (qorincha tizimi, bazal tugunlar, miya ustuni zararlanishi), erta KT/MRT, ikkilamchi omillar nazorati (gipotenziya, gipoksiya, intrakranial gipertenziya), toʻliq jarrohlik ishlovi, antibiotik va antikonvulsant profilaktika hamda reabilitatsiya sezilarli ta’sir koʻrsatishi asoslandi.
Xulosa. Marshrutizatsiya va protokollarni standartlashtirish, koʻp tarmoqli jamoa yondashuvi va reabilitatsiya jangovar BMS oqibatlarini kamaytirishning tayanch yoʻnalishlaridir.
Kalit soʻzlar (UZ): jangovar shikast; oʻqotar jarohat; mino-portlash shikasti; kirib boruvchi BMS; ikkilamchi miya shikastlanishi; infeksion asoratlar; intrakranial gipertenziya; reabilitatsiya.

Introduction. Military conflicts, in addition to significant economic losses, cause large human losses among military personnel and civilians. The history of wars shows that during armed conflicts, the frequency of civilian casualties increases: for example, in the war between Germany and France (1870–1871), out of 100 killed, there were 98 military personnel and 2 civilians, during the First World War (1914–1917) - 52 and 48, respectively, during the Second World War (1939–1945) - 24 and 76, the Vietnam War - 2 and 98, during the confrontation on the Maidan in Kyiv (2013–2014) - 16 and 84. Battle injuries of the skull and GM are a set of injuries and wounds that occur during combat operations. In modern military conflicts, 50% of neurosurgical casualties have soft tissue head wounds, 28% have penetrating wounds, and 17% have non-penetrating wounds [1]. Almost 70% of casualties have mine-explosive injuries. During the military conflict in eastern Ukraine, head injuries were found in 37.5% of the wounded [2–4]. The type of weapon, the conditions of its use, the presence of protective clothing, etc. significantly affect the nature, localization, and severity of combat injuries [5]. A characteristic feature of the use of heavy, more “aggressive” weapons and their diversity is the multiplicity of wounds. During World War II, 54.6%

of Red Army soldiers had gunshot wounds to the head, of which 84.5% were shrapnel wounds, 15.5% were bullet wounds; 88.5% had single wounds, and 11.5% had multiple wounds. Gunshot wounds accounted for 67.9% of all combat head injuries [6]. During World War II, more than 70% of the wounded had a single wound requiring surgical treatment, while during the Vietnam War, 15% of the wounded had a single wound, 51.2% had 2 to 5 wounds, and 10% had 6 to 10 wounds requiring surgical treatment [2, 5]. Of the 296 victims with penetrating head injuries during the Karabakh conflict, 218 (73.5%) had blind wounds, and 78 (26.5%) had penetrating wounds. Initial surgical treatment was performed at the level of skilled medical care, but in 59% of cases, re-surgical intervention was necessary [7]. During the military operation in eastern Ukraine, out of 43% of wounded with combat head trauma who were treated at the Regional Clinical Hospital named after I.I. Mechnikov (Dnipropetrovsk), closed and open injuries, in particular, closed TBI, were found in 31% [8]. Modern weapons and tactics of warfare cause extremely severe injuries to both military personnel and civilians in the conflict zone. Head trauma is one of the most severe in terms of its consequences. In the structure of TBI, severe trauma accounts for 15–20%, moderate and mild — 80–85%. Mortality in severe TBI (8 points and less on the GKH) reaches 40–60%, moderate and mild — 10%, however, psychoneurological disorders at different times of the post-traumatic period are found in more than 50% of victims. Traumatism is not only a medical problem, but also an extremely important socio-economic problem. According to WHO,Trauma and violence are the main factors of violent death in peacetime [9]. Combat injuries of the skull and head include gunshot injuries (bullet, shrapnel, mine-explosive wounds, explosive injuries) and non-gunshot injuries (open and closed mechanical injuries) and their various combinations. Combat injuries of the skull and head are classified depending on the nature of the wounds of the soft tissues of the head, skull bones and head; type of wound canal; location of the wound; nature of the injury; condition of the head and its membranes, etc. [9]. Isolated, multiple and combined injuries (injuries) of the skull and head are distinguished. An isolated injury is considered a single injury. Simultaneous injury by one or more projectiles that injure the skull and head in several places is a multiple injury. Simultaneous damage to the skull and the brain, as well as the organ of vision, ENT organs or maxillofacial area is a multiple head injury. Simultaneous damage to the skull and the brain and other anatomical areas of the body (neck, chest, abdomen, pelvis, spine, limbs) is a combined TBI. Gunshot wounds to the skull and the brain are divided into three large groups: soft tissue damage - in 50% of cases, non-penetrating skull wounds - in 20%, penetrating - in 30%. Soft tissue injuries to the head are characterized by damage to the skin, aponeurosis, muscles or periosteum. With a gunshot wound to the soft tissues, there are no fractures of the skull bones, however, damage to the brain in the form of concussion, bruising or compression, in particular, a hematoma due to the energy of the lateral impact of the wounding projectile, is possible. Non-penetrating skull injuries are characterized by damage to soft tissues and bones while maintaining the integrity of the TOGM. Such injuries are usually accompanied by a contusion of the brain, subarachnoid hemorrhage, less often - compression of the brain by bone fragments, epi- or subdural hematoma. Despite fractures of the skull bones and microbial contamination of the wound, in most cases the TOGM prevents the spread of infection to the brain tissue. Penetrating skull and brain injuries are characterized by damage to the integument, bones, membranes and substance of the brain, severe course and high mortality (during World War II - up to 53%, local conflicts - up to 30%). The severity of penetrating injuries is determined by the structures through which the wounding projectile passes (cortex, subcortex, ventricles of the brain, basal ganglia, trunk) and the degree of their damage. Injuries to the brain stem and deep parts are extremely serious. In the presence of a penetrating wound, severe infectious complications most often occur: meningitis, meningoencephalitis, brain abscess (during World War II they were detected in 70% of such wounded, in local conflicts - in 30%) [10]. Closed TBI is divided into: brain concussion, mild, moderate and severe contusion; diffuse axonal injury, brain compression in the presence of depressed fractures, intracranial hematomas (epi- and subdural, intracerebral), progressive brain edema, and cerebrospinal fluid disorders. Almost 50% of soft tissue injuries of the head are accompanied by impaired consciousness of the victims due to brain damage of varying severity [2–4].Combat injuries of the skull and head include gunshot injuries (bullet, shrapnel, mine-explosive wounds, explosive injuries) and non-gunshot injuries (open and closed mechanical injuries) and their various combinations. 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In case of gunshot wounds of soft tissues, there are no fractures of the skull bones, however, damage to the brain in the form of contusion, crushing or compression is possible, in particular, by a hematoma due to the action of the energy of the lateral impact of the wounding projectile. Non-penetrating wounds of the skull are characterized by damage to soft tissues and bones while preserving the integrity of the brainstem. Such wounds are usually accompanied by crushing of the brainstem, subarachnoid hemorrhage, less often by compression of the brainstem by bone fragments, epi- or subdural hematoma. Despite fractures of the skull bones and microbial contamination of the wound, in most cases the brainstem prevents the spread of infection to the brainstem tissue. Penetrating wounds of the skull and brainstem are characterized by damage to the integument, bones, membranes and substance of the brainstem, severe course and high mortality (during World War II - up to 53%, local conflicts - up to 30%). The severity of penetrating wounds is determined by the structures through which the wounding projectile passes (cortex, subcortex, ventricles of the brain, basal ganglia, trunk) and the degree of their damage. Injuries to the trunk and deep parts of the brain are extremely severe. In the presence of a penetrating wound, severe infectious complications most often occur: meningitis, meningoencephalitis, abscess of the brain (during World War II they were detected in 70% of such wounded, in local conflicts - in 30%) [10]. Closed TBI is divided into: concussion of the brain, mild, moderate and severe contusion; diffuse axonal injury, compression of the brain in the presence of depressed fractures, intracranial hematomas (epi- and subdural, intracerebral), progressive edema of the brain, disorders of CSF. Almost 50% of soft tissue injuries of the head are accompanied by impaired consciousness of the victims due to brain damage of varying severity [2–4].In an isolated injury, one injury is detected. Simultaneous injury by one or more projectiles that injure the skull and head and neck in several places is a multiple injury. Simultaneous injury to the skull and head and neck, as well as the organ of vision, ENT organs, or maxillofacial region is a multiple head injury. Simultaneous injury to the skull and head and neck and other anatomical areas of the body (neck, chest, abdomen, pelvis, spine, limbs) is a combined TBI. Gunshot wounds of the skull and head and neck are divided into three large groups: soft tissue injuries - in 50% of cases, non-penetrating skull wounds - in 20%, penetrating - in 30%. Soft tissue injuries of the head are characterized by damage to the skin, aponeurosis, muscles, or periosteum. In case of gunshot wounds of soft tissues, there are no fractures of the skull bones, however, damage to the brain in the form of contusion, crushing or compression is possible, in particular, by a hematoma due to the action of the energy of the lateral impact of the wounding projectile. Non-penetrating wounds of the skull are characterized by damage to soft tissues and bones while preserving the integrity of the brainstem. Such wounds are usually accompanied by crushing of the brainstem, subarachnoid hemorrhage, less often by compression of the brainstem by bone fragments, epi- or subdural hematoma. 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Closed TBI also includes explosive contusion. Contusion is caused by the simultaneous relatively uniform impact of a mechanical factor on a large surface area during a shock air (water) wave that occurs during an explosion. Contusion can be caused by a fall from a height, a blow by loose soil during landslides. General damage to the body during contusion can be combined with local injuries. In severe contusion, ruptures of the lungs, liver, and kidneys may occur. Brain damage during contusion occurs frequently and is manifested by a concussion-contusion syndrome with or without focal neurological symptoms [5, 6, 11]. Brain contusion always causes psychoneurological disorders; 78% of concussed persons during World War II had impaired consciousness, in particular, in 26.6% - short-term, in 24.7% - up to 24 hours, in 14.3% - more than 1 day [2]. With mild concussion, the victim recovers 6–8 weeks after regaining consciousness. Many concussed persons remain with persistent psychoneurological disorders, in particular, cerebrasthenia, affective disorders, dysphoria, anxiety-depressive states, often - vestibular dysfunction, seizures, hysterical reactions, psychopathy, etc. Gunshot wounds of the skull and brain are classified according to various signs. By etiology - bullet, shrapnel and mine-explosive wounds are distinguished. They differ in the volume and nature of the injury, since bullets have greater kinetic energy than shrapnel, combined and combined injuries are characteristic of mine-explosive injuries. Closed and open mine-explosive head injuries were found in 72.5% of victims of combat TBI, bullet and shrapnel injuries - in 27.5% [2]. TBI causes areas of necrosis, hemodynamic disorders, cerebrospinal fluid circulation, general aseptic or septic processes, reactions to foreign bodies, processes of organization and scarring [12]. Primary traumatic injuries of the brain include fractures of the skull bones, contusion, crushing, intracranial hemorrhages, areas of necrosis, molecular shock in gunshot wounds, diffuse axonal injury, rupture of the brain tissue. Unresolved primary injuries cause hemodynamic disorders, cerebrospinal fluid circulation, hypoxia, edema of the brain tissue, etc. In the occurrence of secondary brain damage, an important role is played by arterial hypotension, hypoxia, hypovolemia, hypothermia, as well as the age of the victims, the state of hemodynamics, and the timeliness of providing specialized medical care. Selective sensitivity of individual groups of neurons (hippocampus, limbic system) to hypoxia after TBI causes the occurrence of secondary damage. Experimental studies show that during TBI, a cascade of reactive processes occurs, which progress over several days or weeks and cause irreversible changes in the brain. Delayed autodestructive processes are manifested by impaired blood flow and immune-inflammatory reactions [12]. The severity of neurological disorders is determined not only by the nature of neuronal damage, but also by the ratio of altered and unchanged neurons, which determines the course of TBI.The clinical course of the late period of TBI depends on the severity of destructive processes, which sometimes last for years. The compensatory capabilities of the brain are of great importance, in particular, intracellular regeneration, which contributes to the restoration of neurons and the preservation of certain interneuronal connections [12]. Primary and secondary injuries of the brain cause the occurrence of psychoneurological disorders, immunological changes, which determine the features of the course of the post-traumatic period. Penetrating wounds of the skull and brain can be through and blind, and by the location of the wound channel - tangential, segmental and diametrical (see figure). A tangential (tangential) wound occurs as a result of the superficial passage of a bullet or fragment and their damage to the bone, TOGM and the surface of the brain. It should be noted that in case of tangential wounds, despite the superficial localization of the wound channel and the insignificant amount of destruction of the GM substance, morphological and functional disorders that arise during the movement of the wounding projectile often spread to neighboring GM areas. This is due to the fact that the GM substance is a medium containing a large amount of fluid, located in a space limited by dense membranes and bones of the skull. The wound is considered segmental when the wounding projectile passes in the cranial cavity along one of the chords within one or two GM lobes, the wound channel is located at a certain depth of the GM, and it has a fairly large length. With any segmental wound, small fragments, bone fragments, hair, and sometimes pieces of headgear enter the wound channel. The destruction of the GM substance, as in any gunshot wound, is not limited to the area of passage of the projectile, but spreads in different directions in the form of the formation of hemorrhages and foci of GM tissue destruction at a considerable distance from the wound channel.passes in the cranial cavity along one of the chords within one or two lobes of the GM, the wound channel is located at a certain depth of the GM, and it has a fairly large length. With any segmental wound, small fragments, bone fragments, hair, and sometimes pieces of headgear enter the wound channel. 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In a diametrical wound, the wound channel is located deeper than in a segmental one, passing along the large chord (diameter) of the skull circumference. Diametrical wounds are the most severe, since the wound channel is located at a considerable depth, with damage to the ventricular system of the GM, trunk structures and vital formations. As a rule, diametrical wounds are accompanied by high mortality, victims die early due to direct damage to the vital centers of the GM. A diagonal wound is a type of diametrical, the wound channel passes along the diameter of the skull, but in a different plane, closer to the sagittal. The entrance hole of the wound channel is located on the face, jaws, neck, the exit - on the convexital surface of the skull; often such wounds are fatal due to primary damage to the GM trunk. In a blind skull wound, one entrance hole and a wound channel of different lengths are found, at the end of which there is a bullet or a fragment. By analogy with a penetrating wound, blind wounds are divided into simple, radial, segmental and diametric. The severity of a blind wound is determined by the depth of the wound channel and its size. Blind wounds with damage to the base of the GM are considered the most severe. Among penetrating wounds of the skull, the so-called "ricochet" wounds are distinguished, when, in the presence of one entrance hole in the wound channel, only fragments of the skull bones are found, and the wounding projectile, having hit the convex surface of the skull, causes damage and sharply changes its flight trajectory, moving away from the skull (external ricochet). With internal ricochet, the wounding projectile changes its trajectory when it touches the curved surface of the skull on the opposite side from the entrance hole of the wound channel [10]. More destructive GM injuries occur with open penetrating trauma. In a gunshot wound, several zones of damage are distinguished: - the zone of the wound channel; - the zone of primary necrosis, in which there is no viable GM tissue; - the zone of molecular shock, the dimensions of which depend on the kinetic energy of the wounding projectile and the destructive force transmitted to the surrounding tissues [6, 11]. Concussion foci in the form of hemorrhagic crushing are observed both around the GM wound and at a distance. The processes of tissue decay and wound cleansing can last 1–2 months. Over a long period of time, a scar forms in the GM tissue, which is accompanied by appropriate clinical manifestations [3, 6, 12]. Until recently, GM shock was considered an injury without structural changes, with temporary functional disorders. Recent studies show that concussion of the brain is characterized by a complex of interconnected dynamic destructive, restorative and compensatory-adaptive processes at the ultrastructural level in the synaptic apparatus, neurons and glial cells. The restoration of the ultrastructure of the cerebral cortex begins 24 hours after TBI and lasts up to 14 days, the first signs of normalization of the ultrastructural organization of the diencephalon-stem departments are noted 10–14 days after the injury, recovery lasts up to 4 months.Sometimes persistent pathological changes remain, which cause the emergence of various cerebro-visceral syndromes [12]. Cerebral infarction is characterized not only by functional, but also by irreversible micro- and macroscopic structural changes, the severity of which depends on the severity and localization of the injury. Diffuse axonal injury should be especially highlighted, which can be complete (axonotomy) or incomplete (intraaxonal injury). Incomplete axonal disruption can end with its restoration or transform into secondary axonotomy, which causes retrograde transneuronal and cascade degeneration. The reduction in mortality in TBI was facilitated by: - the introduction of artificial lung ventilation into clinical practice (in the 1960s); - unification of assessment of the condition of victims and provision of assistance (1970s); - introduction into clinical practice of neuroimaging methods (CT, MRI), cerebral angiography, pneumoencephalography, refusal to use diagnostic milling holes (1970s); - understanding the role of secondary mechanisms of brain damage in TBI (hypoxia, arterial hypotension, ICP and CPT levels, the possibility of their monitoring and correction (late 20th - early 21st centuries). The main principles of managing victims of TBI: maintaining the optimal CPT level, eliminating intracranial hypertension (ICH), preventing the occurrence of secondary brain ischemia, metabolic protection of the brain, replenishing its energy substrates, promoting regeneration and restoration of interneuronal connections. ICH occurs in 50% of victims of TBI in a coma with pathological changes in the brain according to CT, in 10-15% - without such changes. All patients with a level of consciousness of less than 8 points (according to the ECG) require ICP monitoring. The permissible level of ICP is less than 20 mm Hg (normal - 5-15 mm Hg), mean arterial pressure (BP) - higher 90 mm Hg. Sufficient is the level of ICP that provides CPT not lower than 70 mm Hg. CPT = mean BP (BPser.) – ICP; at CPT below 70 mm Hg. neuronal ischemia and secondary hypoxia of the cerebral hemisphere occur. The optimal level of CPT (above 80 mm Hg.) is achieved by increasing BPser. and decreasing ICP. BPser. = diastolic BP +1/3 of pulse pressure. So, at BP 120/80 mm Hg. BPser. is 93 mm Hg. During the treatment of victims of TBI, the rule of "three Gs" should be followed (hypertension, hypervolemia, close to normovolemia, hemodilution). The first and most important step is to ensure optimal oxygenation of the cerebral hemisphere, eliminate hypoxia and hypovolemia. Cerebral perfusion in the norm is almost 54 ml per 100 g of GM tissue per minute. When GM blood flow decreases to 10 ml/100 g per minute and less after 5 minutes, persistent anoxic depolarization of the cell membrane of neurons occurs due to energy starvation and inability to maintain intracellular ion homeostasis. At the same time, a large amount of the excitatory neurotransmitter glutamic acid is released from damaged neurons, which activates NMDA receptors of the membranes of neighboring neurons, triggers a pathological glutamate cascade and causes neuronal death.The use of magnesium preparations helps block NMDA receptors and the glutamate cascade, stopping secondary damage to the GM.

Perfusion pressure (PT) of the brain is one of the determining indicators of the consequences of TBI. PT depends on the values of ATser. and ICP. Without monitoring ICP, it is impossible to determine PT of the brain; changes in SiVO2 precede changes in PT. Therefore, monitoring ICP and determining SiVO2 and PT of the brain are mandatory during the treatment of victims of severe TBI. A certain metabolic protection of the brain is provided by the use of anesthetics: thiopental sodium, isoflurane and its analogues, propofol, sodium etomidate, which contribute to the reduction of perfusion of the brain tissue and its need for oxygen, however, in each specific situation, possible adverse reactions to drugs should be borne in mind. Elimination of hyperglycemia contributes to cerebroprotection by reducing the degree of damage to the brain; The optimal blood glucose level in TBI victims is 5.5–10 mmol/l, hypoglycemia of 2.2 mmol/l and less should be immediately eliminated. Recently, moderate hyperventilation has been used in TBI victims to correct ICP, which causes hypocapnia, reduced blood flow and blood filling of the brain. In the intensive care unit, hyperthermia is quite often observed in victims with severe TBI. Fever worsens the course of TBI and is associated with adverse consequences of treatment of victims with skull and brain injuries. Intravenous administration of paracetamol (infulgan), which also has an analgesic effect, is effective. Under normothermia, total ischemia causes brain damage after 5 minutes, under hypothermia, brain resistance to ischemia increases to 30 minutes. The therapeutic effect of hypothermia includes neuroprotection mechanisms, in particular, modulation of the release of excitatory amino acids, formation of free radicals, reduction of energy expenditure of organic phosphates, slowing down of HM metabolism (by 6% with a decrease in its temperature by every 1°C), reduction of oxygen consumption by HM tissue, accumulation of lactic acid, improving glucose absorption. The severity of the condition and level of consciousness of victims of TBI are assessed by ECG [13–15]. Assessment of the risk of intracranial complications in TBI is extremely important, which determines the principles of hospitalization, diagnosis and treatment of victims. Clinical signs of low risk of intracranial complications are the absence of neurological symptoms, headache, dizziness, presence of bruises, abrasions or superficial wounds of the head. Symptoms of medium risk of intracranial complications are loss of consciousness or a decrease in its level, progressive headache, post-traumatic epileptic seizures, alcoholic or other intoxication, vomiting, post-traumatic amnesia, signs of a skull base fracture, multiple trauma, severe facial skeleton trauma. Clinical signs of high risk of intracranial complications: decreased level of consciousness not associated with alcohol, medication, epileptic seizures or metabolic disorders, focal neurological deficit, negative dynamics of the wounded person's condition after GCS, penetrating or open TBI, cerebrospinal fluid, compressed skull fractures. The risk of intracranial complications in wounded people with mild TBI,as a rule, low, craniography data are negative, CT is performed only when the level of consciousness is reduced or focal neurological symptoms occur; outpatient symptomatic treatment is recommended. At medium and high risk of intracranial complications, hospitalization of victims, CT, treatment in accordance with the identified pathological changes are mandatory. The problems of treating severe TBI are given considerable attention, since the timely provision of qualified and specialized medical care reduces the risk of severe intracranial complications and mortality of victims. Psychoneurological consequences of TBI are observed quite often, in different periods of the disease. Based on many years of study of the mental state of victims with severe TBI, the following variants of disorders are distinguished [16]: - syndrome of suppression of consciousness; - syndrome of disintegration of consciousness; - cognitive and emotional-volitional disorders; - borderline mental disorders. In the distant period of TBI, the following are most often noted: - epileptic seizures; - open hydrocephalus (in 3.9% of victims with severe TBI); - post-concussion syndrome - residual effects of concussion and brain injury; - chronic traumatic encephalopathy; - Alzheimer's disease. Post-traumatic disorders depend not only on the multiplicity and severity of TBI, but also on the age of the victims. As the age of the victims increases, the frequency of occurrence of post-traumatic encephalopathy increases. Much attention is paid to the restoration of the victim's locomotor functions, his ability to self-care, and social adaptation. Unification of the assessment of the condition of victims with TBI contributes to the optimization of treatment tactics. The result of treatment of patients is assessed according to the Glasgow Outcome Scale (GOS) [13]. 1. Good recovery, including the restoration of working capacity. 2. P o m i r n a l i d i z a t i o n (loss of ability, therefore, preserved ability to self-care). 3. Gross disability (the patient requires external care, the possibility of contact is preserved). 4. Vegetative state (lack of contact). 5. Death. In 1980, F. Plum and JB Posner identified 8 types of consequences that detail the results of treatment of victims of TBI [14]. 1. Complete recovery. 2. Preservation of social adaptation with minor neurological and mental defects. 3. Restoration of the main types of previous household activities. 4. Significant disruption of labor and social adaptation, restoration of previous household activities at a reduced level. 5. Severe disability with partially preserved ability to self-care. 6. Severe disability, the patient requires external care. 7. Chronic vegetative state. 8. Death. The severity of psychoneurological disorders is determined by the type of brain injury (diffuse, focal, bruise, hematoma, gunshot wound), its localization, the occurrence of complications during the treatment of the victims. Combat open and penetrating TBI is considered a severe injury,which causes serious complications in any period of the disease due to gross destruction of the brain, high mortality of the victims. In the structure of skull and brain injuries in both peacetime and wartime, moderate and mild TBI prevail, which causes certain complications in the majority of victims, and in 33% - disability. The consequences of combat TBI, including the severity of the post-traumatic period, and the mortality of the victims, depend on the timing of the provision of emergency, qualified, specialized care, adequate rehabilitation, which should contribute not only to physical recovery, but also to the psychoneurological and social adaptation of patients. Assistance to victims of combat TBI is provided in accordance with the standards and protocols for managing patients with TBI [4, 17–19]. Modern methods of neuroimaging and neurophysiological studies allow us to objectify certain post-traumatic changes in the brain in dynamics. Often, changes in the brain according to CT and MRI in the acute period of TBI are much smaller than the real ones, both in closed and open gunshot injuries. CT is mandatory for victims of TBI with an average or high risk of intracranial complications. Given the frequent discrepancy between changes in the brain according to CT in the first hours after the injury and after 12–24 hours, the study must be repeated after 1 day or if the victim's state of consciousness deteriorates or neurological deficit deepens. In the absence of contraindications, MRI is recommended. In victims of mine-explosive TBI and acceleration injuries, a cervical spine examination is mandatory. Patients with impaired consciousness (8 points or less on the GCS) are intubated and hospitalized in an anti-shock ward or intensive care unit; It is advisable to monitor ICP (optimal level below 20 mm Hg) and CPT (80–90 mm Hg, however, not lower than 70 mm Hg). It is better to use external sensors for measuring ICP, with drainage of the lateral ventricles of the brain, parenchymal sensors can also be used, although they more accurately reflect changes in regional ICP. In victims with penetrating gunshot wounds, early surgical treatment of wounds is performed in parallel with intensive care measures, which is the main element of preventing complications in TBI [17]. In patients who undergo delayed or late primary surgical treatment of wounds, as well as with extensive skull decompression, installing sensors for measuring ICP, in our opinion, is inappropriate due to the high risk of infectious complications and the low informativeness of data on the level of ICP during extensive decompressive trepanation. In such a situation, the level of ICP can be roughly judged by the condition of the wound and the trepanation defect. In open and penetrating TBI, early administration of anticonvulsant drugs is mandatory. All victims of combat TBI with a score of 9–14 points on the GCG require neuroimaging and dynamic observation, and treatment in a specialized (neurological or neurosurgical) department.In the acute period of TBI, measures are taken to preserve the victim's life, prevent complications, and conduct early rehabilitation. In victims of severe TBI, various psychopathological changes are observed, in particular, a syndrome of depressed consciousness, cognitive, emotional-volitional, and borderline mental disorders. In 50–90% of patients, neurological disorders persist in the long-term post-traumatic period or new neurological syndromes arise [16]. Cognitive disorders (memory, speech, intelligence, praxis, gnosis) in the long-term period of TBI cause significant limitations in the lives of victims [20] due to impaired cognition, purposeful activity, and adaptation in social and personal life. Cognitive disorders are often more disabling than motor deficits. The emergence and progression of cognitive disorders is possible even after mild TBI. In 78 (50%) of 156 victims of mild TBI, cognitive impairments were detected after 2 months [6, 20]. During the rehabilitation of victims of combat TBI, a detailed study of the features of the course of cognitive disorders is necessary in order to improve the rehabilitation system. During the recovery period after TBI, replenishment of energy substrates of the brain is achieved by prescribing drugs containing cofactors of biochemical reactions of its energy supply: vitamins B1, B6, B9, B12, niacin (all nicotinic acid preparations). Drug treatment of victims of TBI should be aimed not only at preventing the occurrence of pathological changes, but also at restoring neuronal structures and networks. The drug Tivortin based on the essential amino acid arginine deserves attention, which is an active and versatile cellular stimulant, has an antihypoxic, membrane-stabilizing effect, and is an active regulator of intercellular metabolism and energy supply processes. L-arginine is the only substrate for the synthesis of nitric oxide (NO). The conversion of L-arginine to NO is a physiological process aimed at maintaining normal endothelial function. NO is a physiological angioprotector, helps to reduce leukocyte adhesion to the vascular wall and their damage to the endothelium, reduce smooth muscle cell proliferation, pathological vascular wall remodeling, platelet aggregation and mural thrombus formation. Given the importance of hemodynamic factors and blood oxygenation for neuronal function in TBI, it can be argued that tivortin infusion has a neuroprotective effect. Neurotrophic factors and molecules with similar effects are potential treatments for TBI. Neurotrophic factors play a key role in endogenous processes of protection and restoration of damaged neuronal structures [21, 22]. As shown by numerous experimental and randomized clinical studies, such a CNS-specific neurotrophic drug is Cerebrolysin [23, 24]. The results of a retrospective multicenter cohort study involving 7769 patients indicate that the consequences of TBI depend on its severity,The neuropeptide drug Cerebrolysin is recommended as an additional agent to the standard treatment regimen for TBI victims [25]. Cerebrolysin is the only available multimodal neurotrophic drug of European production (Austria) containing low-molecular neuropeptides capable of penetrating the blood-brain barrier [26]. Cerebrolysin stimulates the regenerative potential of BM tissue after TBI. With moderate damage to interneuronal connections, long-term regular use of multimodal neurotrophic drugs ensures the restoration of functional structures. The appointment of Cerebrolysin at a dose of 20–30 ml per day significantly improves the results of TBI treatment compared to those using standard methods when assessed on the 10th and 30th days. The clinical effectiveness of Cerebrolysin is due to its modeling effect on endogenous processes of protection against secondary damage - neuroplasticity and neurogenesis. The neuropeptides in Cerebrolysin act similarly to natural neurotrophic factors. It is thanks to this that the administration of Cerebrolysin helps to accelerate the functional recovery of patients and improve the consequences of TBI. According to experimental and clinical studies (in Alzheimer's disease, impaired cerebral circulation, TBI), the use of Cerebrolysin helps to improve cognitive function [26, 27]. The standard treatment regimen for TBI victims should be supplemented with multimodal agents, the safety and effectiveness of which have been clinically proven. Strategy for providing assistance to victims of combat TBI. 1. Unification of the severity of TBI, determination of the scope of diagnostic and treatment tactics. Victims with medium and high risk of intracranial complications are hospitalized in a multidisciplinary hospital (hospital), which has a specialized neurosurgical department. Such victims require consultations with a neurosurgeon, mandatory CT scan. Victims with a high risk of intracranial complications are hospitalized in the intensive care unit. 2. All victims with impaired level of consciousness (8 points or less on the SCG) are necessarily intubated. 3. Victims with penetrating and non-penetrating gunshot wounds to the head are given early (within 24 hours) primary neurosurgical treatment of wounds, antibiotics and anticonvulsants are prescribed. 4. Intracranial volumetric foci are removed, a sensor is installed to monitor ICP. 5. It is believed that all victims of combat TBI have hypovolemia, a risk of rapid secondary brain damage. 6. Monitoring of water, electrolyte and protein balance, blood saturation is mandatory. 7. Drug therapy is carried out to normalize breathing and systemic hemodynamics. 8. For the purpose of neuroprotection and improvement of neurotrophic capabilities of GM, early use of a neuroprotector with multimodal action is indicated - the drug Cerebrolysin at a dose of 10–30 ml intravenously drip for 10–14 days. 9.Study of structural and functional changes in the CNS according to neuroimaging and neurophysiological studies (CT, MRI, ultrasound diagnostics, electroencephalography), psychoneurological state of victims in dynamics.

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