



## EPIDEMIOLOGY OF TRAUMATIC BRAIN INJURY

*Aripov Dilshodbek Murodilloevich*

*<https://orcid.org/0009-0004-1840-0819>,*

*Bukhara State Medical Institute named after Abu Ali ibn Sino, Uzbekistan,  
Bukhara*

**Annotation:** *Traumatic brain injury (TBI) is one of the most important complex multidisciplinary problems in the field of health care. This article provides information on the modern principles of traumatic brain injury. The review examines in detail the epidemiology, pathogenesis, and mechanisms of neurological complications after trauma.*

**Keywords:** *brain injury, compensatory-adaptive mechanisms, pathogenesis, epidemiology.*

## BOSH MIYA TRAVMATIK SHIKASTLANISHI EPIDEMIOLOGIYASI

*Aripov Dilshodbek Murodilloevich*

*<https://orcid.org/0009-0004-1840-0819>,*

*Abu Ali ibn Sino nomidagi Buxoro davlat tibbiyot instituti, O'zbekiston,  
Buxoro*

**Annotatsiya:** *Bosh miya shikastlanishi (BMSH) sog'liqni saqlash sohasidagi eng muhim murakkab multidissiplinar muammolardan biridir. Ushbu maqolada bosh miya travmatik shikastlanishining zamonaviy tamoyillari haqida ma'lumotlar keltirilgan. Sharhda bosh miya shikastlanishi epidemiologiyasi, patogenezi, travmadan keyingi nevrologik asoratlarning mexanizmlari batafsil ko'rib chiqiladi.*

**Kalit so'zlar:** *bosh miya shikastlanishi, kompensatsion-adaptiv mexanizmlar, patogenezi, epidemiologiya.*

## ЭПИДЕМИОЛОГИЯ ЧЕРЕПНО-МОЗГОВОЙ ТРАВМЫ

*Арипов Дилшодбек Муродиллоевич*

*<https://orcid.org/0009-0004-1840-0819>,*



*Бухарский государственный медицинский институт имени Абу Али ибн Сино, Узбекистан, г. Бухара*

**Аннотация:** *Черепно-мозговая травма (ЧМТ) является одной из важнейших сложных мультидисциплинарных проблем в сфере здравоохранения. В данной статье представлены сведения о современных принципах лечения черепно-мозговой травмы. В обзоре подробно рассматриваются эпидемиология, патогенез и механизмы развития неврологических осложнений после травмы.*

**Ключевые слова:** *черепно-мозговая травма, компенсаторно-приспособительные механизмы, патогенез, эпидемиология.*

**Relevance of the study.** Traumatic brain injury (TBI) is one of the most important complex multidisciplinary problems in the field of health. According to the World Health Organization, it has an average annual growth rate of 2%. In this, the injured are most often aged 20 to 60, that is, those of working age. In the UK, 1 million people require medical care with a TBI every year. TBI accounts for 10% of deaths and 16% of disability worldwide, which is significantly more than malaria, tuberculosis and HIV/AIDS combined. More than 5 million people die from TBI every year in the world [WHO, 2023].

TBI is one of the leading causes of death in the population under 45 years of age and is a leading cause of disability. Traumatic brain injury is the third leading cause of death in the world after cardiovascular diseases and cancer. Central nervous system dysfunction accounts for 30-40% of injuries, and disability accounts for 25-30%. The most common causes of traumatic brain injury are road traffic accidents, followed by criminal, sports and industrial mechanical injuries [Byvaltsev V. A., 2018].

The United Nations General Assembly adopted an ambitious goal to halve the number of deaths and injuries from road traffic accidents worldwide by 2030 (A/RES/74/299) [WHO, 2022].



Currently, there is an annual increase in injuries worldwide, which is associated with the rapid growth in the number of vehicles and speeds. Traumatic brain injury is often observed in transport accidents (up to 70%). The frequency and severity of traumatic brain injuries, high mortality (up to 26.8-81.5%) determine the urgency of this problem and require further development of methods for treating TBI and its complications. Even with isolated TBI, the mortality rate is 39%, and with combined injuries it reaches 68% and above. The medical and social aspect of this problem has not been worked out either. Traumatic brain injuries are most often observed between the ages of 20 and 50, that is, during the period of greatest human labor activity, 1.5 times more often in men than in women. Men suffer more severe injuries, and among them the mortality rate is 3 times higher. In this regard, the problem of TBI is of social, economic and defense importance. At the current level of development of medical science, one of the main tasks of treating traumatic brain injury is to preserve not only life, but also the patient's personality and working capacity, since complications that develop after traumatic brain injury lead to disability in patients, which not only reduces the effectiveness of their treatment in the hospital, but also causes great moral and economic damage to the family and society [Nazarov I. P., 2018].

According to Masharipov A.S. and Iskandarov A.I. (2020), traumatic brain injury (TBI) has a general effect on the body, which is manifested not only in the area of direct mechanical damage, but also in various systems of the body, causing a general adaptive reaction, manifested by pathophysiological, biochemical and morphological changes [Masharipov A.S., Iskandarov A.I., 2020].

The main links in the pathogenesis of TBI - hypoperfusion, metabolic acidosis, brain tissue edema, massive release of tissue aggressive factors - are trigger points for the formation of a non-infectious systemic inflammatory response, leading to the development of multiorgan failure. Modern researchers consider microcirculatory dysfunction to be the central mechanism of pathophysiology in the development of multiple organ dysfunction. Possible mechanisms leading to microcirculatory dysfunction in trauma include: tissue hypoxia; endothelial





dysfunction; activation of the coagulation cascade and inhibition of fibrinolysis [ Yuan H. et al., 2024]. The above disorders contribute to the development of the peripheral microcirculatory shunting effect [ Shi B., 2019 ].

Traumatic brain injury is defined as an attack on the brain by an external physical force that can lead to a decrease or alteration of consciousness and, therefore, affect cognitive abilities or physical functions. This directly contributes to death from external causes, the main of which are motor vehicle accidents, falls, physical aggression and pedestrian collisions [Logsdon A.F., 2015].

Acute severe TBI often damages the basal structures of the brain, including the hypothalamic-pituitary system, and causes central reflex and humoral changes throughout the body. The sympathetic nervous system reaction predominates, releasing catecholamines into the general bloodstream. As a result of these centrally determined reactions, microcirculatory disorders occur throughout the body in the first minutes after injury. In severe TBI, these disorders lead to systemic damage to all internal organs, which leads to multiorgan failure. In this case, changes in the lungs, heart, gastrointestinal tract and liver are manifested by the corresponding clinical picture. With obstruction of the upper respiratory tract and impaired central respiratory regulation, the patient's comatose state leads to hypoxia. This further exacerbates the damage to parenchymal organs [Wilfred BS, 2020]. In severe TBI, especially in combination, hypovolemic shock is observed relatively often (in more than 10% of cases). Therefore, the state of shock with a sharp decrease in the perfusion of internal organs, in some cases, can even increase the degree of their damage. If the underlying disease is also complicated by disseminated intravascular coagulation, the process is aggravated. In the lungs, in the first minutes after injury, spasms of small-caliber vessels, increased vascular wall permeability, and edema of the parenchyma occur, which leads to a deterioration in the normal functional parameters of the lungs and is a good environment for the development of infection. In severe TBI, pulmonary blood flow slows down by 2-3 times, shunts open in the lungs, and the ventilation-perfusion ratio is disturbed in proportion to the severity of TBI. This is an additional cause of damage to parenchymal organs due to hypoxia. Changes in microcirculation



disrupt myocardial function. Corresponding dysfunctions appear on the ECG, in more severe cases, heart rhythm disturbances occur, and acute heart failure develops or chronic failure deepens [Meretsky, V.M., 2013].

Acute - this is the period of the disease from the moment of the damaging effect on the brain until the stabilization of the brain functions impaired as a result of injury or death of the victim. In children, the duration of the acute period is shorter than in adults. Depending on the clinical form, its duration can be from two to ten weeks. For concussion - up to two weeks, mild concussion - up to three, moderate concussion - up to five, severe concussion - up to eight, diffuse axonal damage and brain compression - up to ten weeks [Ignatenko V.V., 2008].

In the intermediate period of the disease, there is a complete or partial recovery or compensation of the general body, brain and centralized functions impaired as a result of the injury. For mild concussion, its duration is up to two months, for moderate concussion - up to four months, for severe concussion - up to six months. In childhood, this period can last up to two years [Armstrong R. C. et al., 2024].

In the long-term period of the disease, functional-clinical recovery or the emergence and / or development of new pathological conditions caused by brain damage may occur. The duration of the long-term period with clinical recovery is not limited to two years, with a progressive course of the disease. In children with mild and moderate BMSH, the long-term period can reach 1.5-2.5 years, with severe BMSH - 3-4 years [Kim I. L., 2024].

Traumatic brain injury is one of the most important global problems in the field of health today due to its incidence, epidemiology, mortality and high level of disability, which justifies the need for scientific research in this area.

### **LIST OF REFERENCES**

1. Бывальцев В. А. и др. Черепно-мозговая травма. Учебное пособие.— Иркутск: ИГМУ, 2018. — 154 с.
2. Дралюк М.Г., Дралюк Н.С. / Черепно-мозговая травма – Ростов-наДону.: Феникс; Красноярск: Издательские проекты, 2016. – 192 с.



3. Игнатенко В.В., Чернухин М.Т., Петров Л.В., Структура непосредственных причин смерти при черепно-мозговой травме в различные сроки посттравматического периода // Вестник Самарской гуманитарной академии. Серия «Право» 2008. №1. С.87-91
4. Ким И. Л. Неврологические аспекты посттравматической энцефалопатии //Miasto Przyszłości. – 2024. – Т. 48. – С. 1846-1848.
5. Курсов С.В. Интенсивная терапия у больных с тяжелой черепно-мозговой травмой / С.В. Курсов, Н.В. Лизогуб, С.Н. Скороплет // Медицина неотлож. состояний. — 2017. — №32(15). — С.44–49.
6. Машарипов А.С., Искандаров А.И. Экспертная оценка давности черепно-мозговой травмы по морфологическим изменениям внутренних органов// Сибирский медицинский журнал, 2010, № 2. С. 51-52.
7. Назаров И. П. Интенсивная терапия тяжелой черепно-мозговой травмы (лекция 3) //Сибирское медицинское обозрение. – 2008. – Т. 50. – №. 2. – С. 91-97.
8. Armstrong R. C. et al. White matter damage and degeneration in traumatic brain injury //Trends in neurosciences. – 2024. – Т. 47. – №. 9. – С. 677-692.
9. Anthony DC, Couch Y, Losey P, Evans MC. The systemic response to brain injury and disease. //Brain Behav Immun. 2012; 26:534–540.
10. Dewan M. C., Rattani A., Gupta S., Baticulon R. E., Hung Y.-C., Punchak M., Agrawal A., Adeleye A. O., Shrimel M. G., Rubiano A. M., Rosenfeld J. V., Park K. B. Estimating the global incidence of traumatic brain injury. Journal of Neurosurgery. 2018;1–18.