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Comparatively Correlation Analysis And Prognostic Criteriaof The Main System Mechanisms Of Secondary Damage To The Brain With Combined Craniocerebral Injury.

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ABSTRACT

Clinical and experimental studies have been shown,that after the combined craniocerebral injury, physiological mechanisms are damaged We maintain perfusion pressure. Partially or completely disruptedShares Autoregulating the brain blood flow. In these conditions after the craniocerebral injury, the brain is not able to respond to the systemic violator Wear homeostasis. As a result, various secondary damage ariseIn the brain tissue is predominantly corridor character thatWe are curved in 80-90% of the dead patients. Is currently among the systemsviolations of homeostasis, most often as secondary damaging factors of the headthe brain. The following: hypotension, hypoxemia, sodium exchange and osmotoostomy, hypocapnia and hyperDrip, hyperthermia, extra-causal inflammatory complications, etc. [1,2,3,4,10,11].

Keywords: hypertension, craniocerebral trauma, hypovolume, homeostasis, hypoxemia, sodium exchange.

Materials and Methods.

In our study to study the prognostic significance of arterial hypotension187 affected with severe combined craniocerebral trauma were selectedFrom the period from 2017 to 2023. From the study, indicators were observed, which were observed in patients inTerminal condition.The number of heart abbreviations and blood pressure were monitored. Analyzed the dependence of the ROV system circulation from kinetics of biogenic amines, data of aNiko neurological and radiological research.

Analysis of the selective parameters of system hemodynamics in 1-21 days after combined craniocerebral injury was revealed that arterial hypotension (at the receipt and in the first 14 days) was noted at 81% of observations*(early hypotension).*This is somewhat exceeding the percentage of the occurrence of arterial hypotension based on the analysis of the data bank of traumatic coma - 34.6% of observations [15, 17]. The number of patients with combined damage in patients with arterial hypotension was 88%, against 27% of the combined damage in patients without arterial hypotension. This indicates that arterial hypotension with admission is associated with the combined damage and is a consequence of blood loss. The most often arterial hypotension was observed among patients with diffuse damage (51%), and much less often in patients with focal damage (30%). In patients with early arterial hypotension, the outcomes were worse (85% - lethal outcomes and vegetative states). In the group of patients who have not had arterial hypotension, the number of lethal outcomes and vegetative states amounted to 15% of observations. According to our data, patients with severe craniocerebral trauma have two critical levels of the deflection of systolic blood pressure. The first - when the average blood pressure falling in the range of 70-89mmhg- The lethal outcome was observed only in 20% of cases, and the second - when the middle blood pressure falling <70mmhg- The lethal outcome was observed in 61% of cases.

In addition, in patients with arterial hypotension, despite the fact that the severity of the state at the admission was approximately the same, the average duration of the coma-plated state was significantly greater in patients with the arterial hypotension (10.2 ± 2.1 days, against 4.9 ± 1.6 days, in patients without arterial hypotension; $p < 0.05$).

In the genesis of early artimeter hypotension there was an absolute or relative decrease in the volume of circulating blood due to various reasons observed in 72% of observations, while the reduction in the volume of circulating blood in 37.2% of observations has been severe, indicating oral hypovolume. Hypovolemic disorders of 52% of observations led to a decrease in the shock volume of the heart that were compensated by the heart rate by increasing the number of heart abilities only 15.1% of observations. A reliable correlation was found in the heart and the volume of circulating blood ($r = 0.56$; $p < 0.05$) with oneParts, and between the volume of circulating blood and the impact index, on the other ($r = 0.57$; $p > 0.01$).

Regression analysis of the parameters of the heart index, the impact index and the volume of circulating blood in the acute period of severe combined craniocerebral trauma testified about the presence of a strict statistical dependence of the changes in the death of the heart from the severity of hypovolemic disorders. Weak linear dependence of systolic blood pressure and shockoThe index from the number of heart abbreviations testified the reduction of the severity of adaptation and compensatory reactions of the award-winning the values of the systemic blood pressure and the death of the heart. The obtained data partly coincide with the view of some authors[1], believing that the variant of the outcome of acute hypovoilia in the craniocentar degrade injury does not depend on the age, depth of oppression of consciousness when adopted, combined damage. The authors believe that the influence of multiple system trauma is mediated through systemic arterial hypotension [156]. Indeed, the correlation dependence of the volume of circulating blood from the state of the patient ranked by the gloss of the Glasgow compects is as weakHu2, $P < 0.05$).

In the group of patients with hypovolemia, some features of humoral regulation systems for blood circulation were revealed. They were in the short-term activation of the sympatho-adrenal system in the first third day after the injury (in the form of 10 tyolic raising the level of catecholomin).

In the group of patients with lethal outcome and hemodynamic disorders, significant changes in the mechanism of maintaining the constant of liquid volume, and the concentrations of electrolytes correlated

[1]Гайтур Емиль Ильич Повреждения головного мозга при тяжелой ЧМТ: диссертация – доктора медицинских наук: 14.00.28 Москва 1999; Потапов А.А., Коновалов А.Н., Корниенко В.Н., Кравчук А.Д. и др. Современные технологии и фундаментальные исследования в нейрохирургии // Вестн. РАН. 2015. Т. 85, № 4. С. 299-309.

with the identified hypovo-sharpic disorders were revealed. In the first three days in patients with severe craniolast trauma with hypovolemia and arterial hypotension, the awarding of the anti-dihretic hormone was preserved within normal values (4.8 ± 0.9 pmol / l), although it is known that the reduction in the volume of the circulating blood by 15% should cause complications in the form of secretion AGG[2].

In addition, the activation of the Renin-angiotensin-aldosterone system was reflected in the revealed correlation dependences: the activity of the platemin refinence and the volume of circulating blood ($r = -0.5$, $p < 0.05$), aldosterone and the volume of circulating blood ($r = -0.56$; $p < 0.05$), al-dosteron - average blood pressure ($r = -0.57$; $p < 0.05$) indicating the activation of this humoral system as the pressure of hypovolece of the pressure of the pressure is increased. A double-screw increasing concentration of angiotensin, as a rule, leads to an increase in blood flow in arterio-venous shunts of the brain.

In the group of observations with early long systemic hypotension (the medium hell = 81 ± 1,2mmhg), There was a moderate decrease in the average linear rate of blood flow. By 2-3 days in the mid brain artery, the linear blood flow rate was reduced to 40.1 ± 2.1 cm / sec. At the same time, the diamydrical velocity of the blood flow was observed, with the stored values of the systolic speed. The studies were tested for increasing the peripheral vascular resistance due to increased intracranial pressure and as a result of the reduction of cerebral perfusion. One of the mechanisms of increasing intracranial pressure at systemic arterial hypotension, in patientsxWith severe craniocent brain trauma, is the increase in central venous pressure. The increase in central venous pressure is usually accompanied by the resuscitation measures with hypovo-shaomic shock. We follow the increase in intracranial pressure at a decrease in middle blood pressure is the fall of cerebral perfusion pressure and the development of hypoxic brain defects. According to the cerebral oximetry (Fig. 8), in patients of this group, a saturation of orange saturation of the brain tissue (rscb). In Fig. 9 shows the changes in the mean blood pressure, intracranial pressure, the average linear blood flow rate and oxygen saturation of the brain tissue during the episode of arterial hypotension. Our data corresponded to these authors[3].

Episodes of arterial hypotension after 14 days (*delayed hypotension)*Often, against the background of purulent-inflammatory complications, there were 17% of observations. At the same time, there was no difference in the amount of patients with the combined damage in patients with deferred hypotension and without delayed hypotension. In this group, lethality was 14%, and the output in the vegetative state is 44% of observations. According to other authors, lethality or exit in vegetative status in patients with deferred arterial hypotension is 66%, compared with 17% in patients who did not have the episodes of hypotension[8,11, 15, 17].

The reason for the deferred hypotension in patients with severe craniolasty trauma was septic states, natrogenic factors, the defeat of the central mechanisms of regulation of hemodynamics. In a group of patients with deferred arterial hypotension or episodes of arterial hypotension, periods of change in systemic blood pressure (and ± 25,5mmHg) were accompanied by decreases of the average velocity of linear blood flow in the mid brain artery (and ± 16.5 ± 2.2 cm / sec, $p < 0.05$) and a saturation of orange saturation oxide of the brain tissue, testifying to rough violations of the autoregulation of the vascular tone accompanying hypoxic disorders.

Thus, early and deferred arterial hypotension is one of the most significant independent unfavorable prognostic factors in severe combined craniocerebral injury. Obviously, the arterial hypotension is a common determinant of the secondary ischemic brain defeat. The protocol of retention of patients with severe HRC should include measures aimed at mandatory prevention and rapid elimination of the arterial hypotenus of any genesis.

To determine the effect of arterial hypoxemia for the course and outcomesThe traumatic disease of the brain was selected 193 affected with combined craniocerebral trauma. The victims were grouped: 1*Group* -Patients who have not episodes of hypotension and hypoxemia before receipt and during the first 14 days after injury; 2*Group*- The victims who had during the period of episodes of hypoxemia (RAO_2<60mmhg); 3*Group*-patients who have during episodes of hypoxemia and arterial hypotension (SYSTOLA. AD <90mmhg). From the study, the indicators of hypotension and hypoxemia were excluded, which were observed in patients in terminal state.

[2]Гайтур Емиль Ильич Повреждения головного мозга при тяжелой ЧМТ: диссертация – доктора медицинских наук: 14.00.28 Москва 1999

[3]Коновалов А.Н., Потапов А.А. Клиническое руководство по черепно-мозговой травме.Clinical manualofheadinjury . М.: 2019.

The most often arterial hypoxemia and hypotension in the acute period. In patients with severe craniolast trauma was observed in combined craniocent brain damage. Among the patients with third group, most often observed patients with diffuse brain damage (53 patients) and shellmatic hematomas (21 patients).
As can be seen from the table shown in 16, patients 2 and 3 groups have been heavier injury than sick 1 groups. This is evidenced by a low scale of the Glasgow Koma at the admission of these patients. Patients 2 and 3 groups have a significantly longer unconscious state.

Table 1 Main clinical indicators and outcomes in research groups

Indicators	**1группа**	**2группа**	**3группа**
Number of patients	121	30	73
Age	25,3 ± 1,7	25,6 ± 3,2	20,9 ± 1,8
Shrk in patients	7,1 ± 0,2	5,9 ± 0,2 **	3,9 ± 0,1 **
Kite duration (days)	5,2 ± 0,7	9,6 ± 1,5 **	10,5 ± 1,0 **
Outcomes (in%)			
Good restoration	35	14	8 **
Moderate disability	24	15 **	10 **
Deep disabilities, vegetative condition and death	20	60 **	70 **

The reliability of the difference between 1 group and the rest: * - P <0.05;
** - P <0.01

The presence of hypoxemia led to a reliable increase of twice as many patients with "unfavorable" outcomes (death, vegetative state, deep disability). The combination of these two factors in one patient (3 group) was accompanied by an even greater increase in "unsatisfactory" outcomes.
Among patients with diffuse brain damage, the presence of only episodes of arterial hypoxemia in the first 14 days after injury did not increase the numberEstate of lethal outcomes (Table 2).

Table2. **Valley of the deaths during intractic character damage during the CDM**

Kind of brain damage	**1 group**	**2 group**	**3 group**
Diffuse damage	9%	9%	21%
Internal humulate hematomas	11%	23%	42%
Shells of hematomas	18%	60%	63%
For all	**12%**	**32%**	**32%**

The reliability of the difference between 1 group and the rest: * - P <0.05;
** - P <0.01

However, the combination of arterial hypotension and hypoxemia led to an almost reliable increase in the number of patients with deaths. For other focus, another dependence is detected - as separately hypoxemia, and its combination with arterial hypotension increased the lethality. And, finally, the greatest mortality was noted with a combination of two system damaging factors in patients with shellmatic hematomas. All above the above indicates that these systemic damaging factors have the most adverse effect on the outcomes of the craniocerebral trauma in the formation of the brain.
With a separate examination of the influence of the scientists in the outcomes in patients, patients was identified that in the absence of these damaging factors, the outcomes are not statistically distinguished. If hypotension or hypoxemia is observed, the amount of patients with "unsatisfactory" outcomes in adults almost 1.5 times increasing, and in children almost three times (28). The combination of these factors increases the number of patients with unsatisfactory outcomes almost 2 times among adults and almost 4 times among children (p <0.05) (28).
Thus, systemic hypoxia is one of the fundamental causes of lethality and deterioration of outcomes with severe combined craniocerebral trauma. The most sensitive to these factors are patients with acute shell hematomas.

Discussion of results

In the period from 2017 to 2023The spheres of 270 patients with CCM are spread. The open CMT was in 54. Regional lymphotropic therapy on the methodology of the Republican Center for Limphology of the MZ of

the Republic of Uzbekistan was held by 134 patients, without extracranial infectious foci or sepsis. The drugs were introduced once a day into the neckline and subparagate lymph nodes on the side of the wound for 2-5 days. Systemic antibiotic therapy was not used.

The method of lymphotropic antibioticotherapy, developed by jointly andyja and Moscow scientists-lymphologists today is quite widely used in the emergency care system. The essence of the method is to introduce small doses of antibiotics in the tissue and creating conditions for temporary, functionally modified microcirculation into the region of administration of antibiotic, so that the latter is absorbed into lymphotic, and not in blood capillary.

The need for delivery of antibiotics to the lymphatic channel is due to the fact that microbes, first of all, penetrate into the lymphatic system and the main place of the application of antibacterial drugs should be the lymph actuses and lymph nodes. Unfortunately, traditional methods for administering antibiotics, such as intramuscular, intravenous, intraperitoneal, etc., do not provide, as a rule, therapeutic concentrations of antibiotics in the lymphatic ring. In addition, in the blood of tissues, the medical concentrations of drugs are held very highly time-free 4-12 hours. This requires frequent repeated injections of antibiotics, which is far from the body, the limous photographic antibiotic therapy used by us was a single day therapeutic dose at the weight of kg and growth. The intravenous administration of such colloids like plasma, blood, albumin, mannitis, gelatinol and other parallel stimulants of the lymphatic drainage from the affected tissues by introducing tissue lymph modifications in the tissue in the tissue stimulants such as trypsin, lidaz, heparin, "aqueous load" - glucose to the newokain the mixture, leads to a pronounced detoxification effect, both at the level of tissues and individual systems and on the body as a whole.

Analysis of extracranial factors that cause global ischemic violations in a damaged brain showed that the systemic ischemia is a single of the main causes of lethality and deterioration of outcomes in the craniocerebral trauma. Arterial hypoxemia and hypotension are most often observed after combined damage [2, 8]. The most sensitive to these factors are patients with acute shellmatic hematomas, as the results of other researchers also testify. According to these data, the lethality in patients with the inducing of the brain and arterial hypotension can reach 93% [3,9]. In addition, in different reacts, adult and children are honored. Children are more sensitive to these systemic disorders [6,28]. Similar results were obtained by other researchers [15]. There are observations that the combination of arterial hypotension and hypoxemia in the same patient with severe craniolast trauma results in virtually 100% death [13, 17, 21].

The data obtained by us are partly coincided with the opinionChesnutr.M. etc., considering that the variant of the outcome of acute hypovolemia with severe craniocerebral injury is mediated through systemic arterial hypotension [16]. The results of our observations coincide with the data of other researchers showing that lethality or exit in the vegetative status in patients with deferred arterial hypotension is met 3.2 times more often, compared to patients who have not noted hypotensive episodes [15,17]. A certain relationship between changes in raso2 and the duration of the coma, the flow and the outcome of the craniocerebral injury is revealed. However, it should be noted that artificial hypocapnium was mainly used to treat the most severe patients who have most often observed episodes of intracranial hypertension. While hyperpatnia was most often accompanied by hypoxemia and more often observed after the CRM + a thoracic cell, as well as the development of pneumonia. According to the authors, short-term hypocapnia can be used for an extreme reduction in intracranial hypertension before it is clarified. According to other authors, the long use of hypocapnin worsens its outcomes [12, 13,15].

A pronounced relationship between the solids balance, osmotic homeostasis, the course of traumatic disease, weight and hedge of craniocerebral injury was established. At the same time, it was revealed that most often rough sodium exchanges are observed in the defending of the brain and diffuse damage. The prognostic importance for the development of hypernatoriumness had the fractures of the base of the skull in the region of the middle cranial yamk, the expressed lateral displacement, alcohol intoxication to trauma and hemorrhagic shock [2, 10, 11, 17].

The dependence of the severity of the brain's edema from the degree of disorder of the sodium balance and osmotic homeostasis is determined. In violations of sodium balance, patients with a half-scale or diffuse edema were more observed. In this case, both hyperosolume and hypoososologenectity was more often identified in patients with expressed brain swelling. This shows that with hyponatremia, there is an increase in intracellular edema, and with long-term hypernoderemia, the increase in the brain's events can be associated with the accumulation of various organic substances inside the neuron. This contributes to the initial reduction in the volume of the brain cells to strengthen the intracellular edema due to the penetration of water into an intracellular space [8,11, 14].

The study of thermostatic violations revealed that most often hyperthermia is observed in severe forms of diffuse damage, less often in the shell hematomas. A reliable relationship between thermal regulation

disorders and the presence of open PMT, combined damage is found. Virginations of thermoregulation arise reliable more often after the fractures of the base of the skull, intracentrusic hemorrhages, subarachnoid hemorrhages. Hyperthermia as a secondary extrinery damaging factor of the brain depends on the severity of the craniocerebral trauma, extends the comaosetic state and catastrophically worsens the outputs [45, 108].
It was found that in patients with severe PMN, purulent-inflammatory complications are noted in most cases. The analysis showed that the development of extracranial inflammatory complications is extremely unfavorable in the prognostic plan. The source of septicosis was often the exogenous, while the source of pulmonary complications and urinfaction was more often endogenous origin [3,8,12].
The study conducted that the primary damage to the brain is exacerbated by the emergence of secondary systemic mechanisms. Brain damage. The greatest value with severe CMRs has damage to the mechanisms of maintaining perfusion pressure (partially or completely disrupted by autoregulation of cerebral blood flow), since in these conditions, the brain is adequately react "to respond to" systemic violations of homeostasis. As a result, various secondary damage in the brain fabric of predominantly corrinstal occurrence occur. The cause of these ischemic violations and secondary systemic mechanisms for damage to the brain are. That is why their early detection and interruption are the main goal of therapeutic measures with CCMs.

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