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It Is Reported That A Comparative – Correlation Analysis And Prognostic Criteria Of The Main Systemic Mechanisms Of Secondary Brain Damage In Combined Traumatic Brain Injury

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Actuality

Today, there is a constant increase in injuries in all countries of the world. Among the causes of death and disability of the population that occurred as a result of all injuries, damage to the central nervous system comes out on top (30-40%), and in terms of its share in deaths in the most active category of the population (18-45 years old) it is ahead of cardiovascular diseases and cancer (123;129;89;112)

Due to the entire general increase in injuries, the frequency and severity of traumatic brain injuries is steadily increasing (V.V. Lebedev et al., 1981; 1995; 2001, A. Rohrret al., 1979; R.Spence et al., 1990), including up to 50-70% combined with extracranial damage (T.A. Revenko et al,1992; V.Digilio,1995). At the

same time, mortality from combined traumatic brain injury (CBMT) ranges from 12% to 69% (MM). Rozhinsky et al,1976;V.V. Lebedev et al.,1992;2001, S. Bowers et al.,1990., R. Raphely ET al., 1995). According to long-term forecasts (L.B. Lichterman et al, 1979; 1997; 2001, a further increase in the incidence of neurotrauma is expected. This has led in recent decades to the inclusion in the recommendations of various congresses of traumatologists and neurosurgeons as the main task of further studying pathogenesis, developing diagnostic methods and improving the quality of treatment for patients with combined traumatic brain injury.

Research on this problem of combined trauma has been conducted in the CIS countries and abroad for no more than 15-20 years. Extensive factual material has been accumulated, and works containing valuable recommendations for the treatment of combined traumatic brain injuries have been published (G.D. Nikitin, N.K. Mityunin and E.G. Gryaznukhin, 1976; M.G. Grigoriev, N.A. Zvonkov, L.B. Lichterman and A.P. Fraerman,1977;Yu.F. Platonov, 1978; N.E. Kudryavtsev, 1980; V.V. Lebedev, V.P. Okhotsky and N.N. Kanshin, 1980; G.D. Nikitin and E.G. Gryaznukhin, 1983; V.V. Lebedev et al, 1997; 2001; A.N. Konovalov et al., 1994; 1997; 2001, R.Reding et al., 1977 Malisano LP et al. 1994), but many important aspects of the problem remain controversial and unresolved.

Purpose of the study: to study clinical and paraclinical signs of secondary brain damage in acute combined traumatic brain injuries in adults.

After traumatic brain injury, it has been shown by clinical and experimental studies that the physiological mechanisms for maintaining perfusion pressure are damaged. Autoregulation of cerebral blood flow is partially or completely disrupted. Under these conditions, after a traumatic brain injury, the brain is unable to adequately respond to systemic disorders of homeostasis. As a result, various secondary injuries occur in brain tissue, mainly of an ischemic nature, which are found in 80-90% of deceased patients. Currently, among the systemic disorders of homeostasis, most often as secondary damaging mechanisms of the brain, the following are described: hypotension, hypoxemia, sodium metabolism and osmotic homeostasis disorders, hypocapnia and hypercapnia, hyperthermia, extracranial inflammatory complications, etc.

Material and methods:

Arterial hypotension

To determine the prognostic value of arterial hypotension, 187 victims with severe combined traumatic brain injury were selected. The indicators that were observed in patients in terminal condition were excluded from the study. Heart rate and blood pressure were monitored. The dependence of systemic circulation parameters on the kinetics of biogenic amines, data from clinical, neurological and radiological studies were analyzed.

An analysis of sample parameters of systemic hemodynamics on days 1-21 after combined traumatic brain injury revealed that arterial hypotension (at admission and in the first 14 days) was observed by us in 81% of observations *{ early hypotension).* This slightly exceeds the percentage of arterial hypotension occurring based on the results of the analysis of the traumatic coma data bank - 34.6% of observations (156, 157). The number of patients with combined injuries in patients with arterial hypotension was 88%, versus 27% of combined injuries among patients without arterial hypotension. This indicates that hypotension upon admission is associated with combined injuries and is a consequence of blood loss. The most common arterial hypotension was observed among patients with diffuse injuries (51%), and much less often in patients with focal injuries (30%). In patients with early arterial hypotension, the outcomes were worse (85% fatalities and autonomic conditions). In the group of patients who did not experience hypotension, the number of deaths and autonomic conditions was 15% of the observations. According to our data, patients with severe traumatic brain injury have two critical levels of systolic blood pressure drop. The first - with a drop in average blood pressure in the range of 70-89 mmHg - death was observed only in 20% of cases, and the second - with a drop in average blood pressure <70 mmHg - death was observed in 61% of cases.

In addition, in patients with arterial hypotension, despite the fact that the severity of the condition upon admission was approximately the same (the average score on the Glasgow coma scale upon admission did not differ significantly in patients with arterial hypotension - 5.1+0.6 and in patients without it - 5.7±0.4; p>0.05), the average duration of the comatose state was significantly longer in patients who had suffered arterial hypotension (10.2±2.1 days, against 4.9±1.6 days, in patients without hypotension; p<0.05).

In the genesis of early arterial hypotension was an absolute or relative decrease in circulating blood volume due to various causes, observed in 72% of observations, while the decrease in circulating blood volume in 37.2% of observations was pronounced, indicating acute hypovolemia. Hypovolemic disorders in 52% of the observations resulted in a decrease in cardiac stroke volume, which was compensated by cardiac output by increasing the number of cardiac contractions in only 15.1% of the observations. A significant correlation was identified between the cardiac index and the volume of circulating blood (g=0.56; p<0.05) on the SAME side, and between the volume of circulating blood and the shock index on the other (g=0.57; p>0.01).

Regression analysis of the parameters of the cardiac index, stroke index and circulating blood volume in the acute period of severe traumatic brain injury indicated the presence of a strict statistical dependence of changes in heart flow rate on the severity of hypovolemic disorders. A weak linear dependence of systolic blood pressure and the shock index on the number of heartbeats indicated a reduction in the severity of adaptation-compensatory reactions aimed at maintaining the values of systemic blood pressure and heart flow. The data obtained partly coincide with the opinion of Chesnut RM et al., 1993, Gaitur E.I., 1999, Amcheslavsky V.G., 2002, who believe that the outcome of acute hypovolemia in traumatic brain injury does not depend on age, depth of depression consciousness upon admission, combined injuries. The authors believe that the impact of multiple systemic trauma is mediated through systemic hypotension (156). Indeed, the correlation dependence of the volume of circulating blood on the condition of the patient ranked on the Glasgow coma scale is noted as weak HU2, p<0.05).

In the group of patients with hypovolemia, some features of the systems of humoral regulation of blood circulation were identified. They consisted of a short-term activation of the sympatho-adrenal system in the first to third days after the injury (excretion of catecholamines was more than 10 times higher than the normal level: the concentration of adrenaline approached 410.6±92.5 nmol/day, and norepinephrine to 625.4±105.8 nmol/day) followed by its depletion (adrenaline to 40.4±10.2 nmol/day and norepinephrine to 88.4±8.5 nmol/dayday) (respectively t=3.98 and t=5.06; p <0.001).

In a group of patients with fatal outcomes and hemodynamic disorders, significant changes in the mechanisms for maintaining constant fluid volume and electrolyte concentrations were identified, correlating with the identified hypovolemic disorders. In the first three days, in patients with severe traumatic brain injury with hypovolemia and arterial hypotension, antidiuretic hormone secretion values remained within normal values (4.8±0.9 pmol/l), although it is known that a 15% decrease in circulating blood volume should cause a significant increase in antidiuretic hormone secretion. The absence of such a reaction in patients with a decrease in the volume of circulating blood by 20% indicates dysfunction of these nuclear mechanisms in the anterior hypothalamus (Gaitur E.I., 1999).

It should be noted that in response to hypovolemia, activation of the renin-angiotensin-aldosterone system occurred. A feature of this reaction was a significant increase in the concentration of plasma renin activity, which, under conditions of a continuing significant decrease in intravascular blood volume and, consequently, perfusion pressure in the renal vessels, reached five times by 3 days after injury, six times by the fifth, and almost eight times by the seventh day increase. The concentration of aldosterone during the first three days was 1.5-2.0 times higher than normal values, being an adaptation-compensatory reaction aimed at restoring the volume of circulating blood. In addition, activation of the renin-angiotensin-al-desterone system was reflected in the identified correlation dependencies: plasma renin activity - circulating blood volume (g=-0.54; g><0.05), aldosterone - circulating blood volume (g—0.56; p<0.05), al-dosterone - average blood pressure (g=-

0.57; p<0.05), indicating the activation of this humoral system as hypovolemia increases and pressure decreases. A two-fold increase in the concentration of angiotensin, which is one of the components of the compensatory reaction aimed at increasing the tone of resistive vessels and allowing maintaining subnormal values of systolic blood pressure, was also facilitated by an increase in arteriovenous blood bypass. The stated positions are based on the correlation dependences of plasma renin activity and (av)C>2 (g=0.54; p<0.05), plasma renin activity and systemic peripheral resistance (g=0.76; p<0.01).

In the group of observations with early long-term systemic hypotension (mean AD=81±1.2 mmHg), there was a moderate decrease in the average linear blood flow velocity. By 2 - 3 days in the middle cerebral artery, the linear speed of blood flow decreased to 40.1 ±2.1 cm/sec. At the same time, an advantage was observed in the decrease in diastolic blood flow velocity, with preserved values of systolic velocity. Data from the study indicated an increase in peripheral vascular resistance due to increased intracranial pressure and, as a consequence, a decrease in Cerebral perfusion. One of the mechanisms of increasing intracranial pressure during systemic arterial hypotension in patients with severe traumatic brain injury is an increase in central venous pressure. An increase in central venous pressure usually accompanies resuscitation measures for hypovolemic shock. A trace of the increase in intracranial pressure with a decrease in average blood pressure is a drop in cerebral perfusion pressure and the development of hypoxic brain lesions. According to cerebral oximetry (Fig. 8), patients in this group showed a decrease in oxygen saturation of brain tissue (rSCb). Figure 9 presents data on changes in mean arterial pressure, intracranial pressure, mean linear blood flow velocity and oxygen saturation of brain tissue during an episode of arterial hypotension. Our data corresponded to the data of Konovalov A.N. 2001-2004, Amcheslavsky V.G., 2002.

Episodes of arterial hypotension after 14 days *{delayed hypotension)* were often observed in 17% of observations against the background of purulent-inflammatory complications. At the same time, there was no difference in the number of patients with combined injuries in patients with delayed hypotension and without delayed hypotension. In this group, mortality was 14%, and recovery to a vegetative state was 44% of observations. According to other authors, the mortality or emergence to vegetative status in patients with delayed hypotension is 66%, compared to 17% in patients who did not have episodes of hypotension (28,6,156,157) (Figure!)

The cause of delayed hypotension in patients with severe traumatic brain injury was septic conditions, iatrogenic factors, and damage to the central mechanisms of hemodynamic regulation. In the group of patients with delayed arterial hypotension or episodes of arterial hypotension, periods of changes in systemic blood pressure (A±25.5 ttimHg) were accompanied by decreases in the average linear blood flow velocity in the middle cerebral artery (A±15.5±2.1 cm/sec, p<0.05) and a decrease oxygen saturation of brain tissue, indicating gross disorders of autoregulation of vascular tone accompanied by hypoxic disorders.

Thus, early and delayed arterial hypotension is one of the most significant independent adverse prognostic factors in severe combined traumatic brain injury. It is obvious that hypotension is a common determinant of secondary ischemic brain damage. The protocol for management in intensive care of patients with severe combined traumatic brain injury should include measures aimed at the mandatory prevention and rapid elimination of arterial hypotension of any origin.

4.2. Arterial hypoxemia

To determine the effect of arterial hypoxemia on course and outcomes

193 victims with combined traumatic brain injury were selected for traumatic brain disease. The victims were divided into three groups: *Group 1* - patients who did not have episodes of hypotension and hypoxemia before admission and during the first 14 days after injury; // *Group* - victims who experienced episodes of hypoxemia during this period (PaO_2<60 mmHg); /// *Group* - patients who experienced episodes of hypoxemia and hypotension during this period (systole). AD<90 mmHg). The indicators of hypotension and hypoxemia,

which were observed in patients in terminal condition, were excluded from the study.

Most often arterial hypoxemia and hypotension in the acute period. In patients with severe traumatic brain injury, it was observed with combined traumatic brain injuries. Among patients in the third group, the most frequently observed were patients with diffuse brain damage (53 patients) and sheath hematomas (21 patients).

As can be seen from the presented table 1, patients of groups 2 and 3 received more severe injuries than patients of group 1. This is evidenced by the low score of the Glasgow Coma Scale at admission in these patients. Patients of groups 2 and 3 had a significantly longer unconscious state.

Table 20

Relationship between the main clinical indicators, outcomes and study groups

indicators	**1group**	**2group**	**3group**
Number of patients	121	25	78
Average age	28,3±1,7	28,6±3,2	29,9±1,8
Average score for SHKG	6,1±0,2	4,9±0,2**	4,9±0,1**
Average duration of coma (day)	6,2±0,7	10,6±1,5**	11,5±1,0**
Outputs (in %)			
Good recovery	35	14	8**
Moderate disability	24	15**	10**
Glub.disability, vegetative state and death	20	60**	70**

Reliability of the difference between group 1 and the rest:*-r<0.05; **-r<0.01

The presence of hypoxemia led to a significant increase in twice as many patients with «unfavorable » outcomes (lethal outcome, vegetative state, deep disability). The combination of these two factors in one patient (group 3) 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 the injury did not increase the number of deaths see Table. 2).

Table 21.

Number of fatal patients (in %) in patients depending on the type of brain damage

Type of brain damage	**1group**	**2group**	**3group**

Diffuse damage	8%	9%	20%
Intracerebral hematomas	10%	28%	41%
Shell hematomas	18%	60%	62%
For everyone	**12%**	**32%**	**32%**

Reliability of the difference between group 1 and the rest: *-r<0.05; **-r<0.01

However, the combination of arterial hypotension and hypoxemia led to an almost significant increase in the number of patients with fatal outcomes among these patients. For focal injuries, another dependence was identified - both hypoxemia alone and its combination with arterial hypotension

Increased mortality. And finally, the greatest mortality was observed when two systemic damaging factors were combined in patients with shell hematomas. All of the above indicates that these systemic damaging factors have the most unfavorable effect on the outcomes of traumatic brain injury during brain compression.

Upon separate consideration of the influence of the studied damaging factors on the outcomes in patients, it was revealed that in the absence of these damaging factors, the outcomes are not statistically different. If hypotension or hypoxemia is observed, then the number of patients with «unsatisfactory» outcomes in adults increases by almost 1.5 times, and in children by almost three times (28). The combination of these factors increases the number of patients with unsatisfactory outcomes by almost 2 times among adults and almost 4 times among children ($p<0.05$)(28).

Thus, systemic hypoxia is one of the underlying causes of mortality and worsening outcomes in severe combined traumatic brain injury. The most sensitive victims to these factors are patients with acute shell hematomas. Children are more sensitive to both hypoxemia and its combination with hypotension (28).

4.3. Hypocapnia and hypercapnia in mechanical ventilation

A study of carbon dioxide tension in arterial blood was conducted in 145 patients with combined traumatic brain injury. All patients, after admission according to indications, were transferred to artificial ventilation due to inadequate spontaneous breathing. At the same time, the gas composition of the blood was constantly measured up to 4-5 times a day, and the necessary correction was carried out depending on the clinical situation. The study of the gas composition continued for 10 days after the injury. Data related to the terminal state and in patients who experienced brain death within the first 7 days after injury were excluded from the study. All patients, depending on the level of carbon dioxide in the arterial blood, were divided into the following groups:

Group I - 28 patients who experienced normal carbon dioxide levels (moderate hypocapnia RaCO2>ZZ mmHg) or moderate hypercapnia (RaCO2<50 mmHg) within 10 days after injury. This group included patients who had the highest Glasgow Coma Scale at admission, that is, with a «relative to» less severe injury. This is evidenced by the shortest period of unconsciousness. In this group of patients, hypocapnia was not induced using hyperventilation, since there were no indications. Accordingly, the outcomes were the best. The duration of the comatose state was the shortest, and the mortality rate was only 7%.

Group // - 45 patients in whom, in order to combat intracranial hypertension (as evidenced by computed tomography data, and in some cases, the results of measuring intracranial pressure) induced short-term deeper hypocapnia with figures of PaCO2 from 33 to 25 mmHg. Despite the same severity of the injury received as in the first group, these patients presented a longer period of unconsciousness and a greater number of patients with fatal outcome. In this group, 24% of patients died.

Group /// - 43 patients were characterized by a significantly more severe injury than in the first group. In these patients, deep short-term hyocapnia (RACO2<25 mmHg) was artificially induced to combat intracranial hypertension, as indirectly evidenced by computed tomography data (according to which these patients experienced severe swelling of one or both hemispheres in 74% of cases), and sometimes data direct measurement of intracranial pressure. Among them, twice as many patients died than in the second group and 6 times more than in the first — 42%. The duration of the comatose state was greatest.

Group IV - it was characterized by episodes of hypercapnia (PaCO2 over 50 mmHg) in the arterial blood due to the development of pneumonia in these patients, as well as due to combined chest injuries. This led to the fact that their hypercapnia was accompanied by severe hypoxemia (77%). In these patients, compared to the first group, the severity of the injury received, as well as the duration of the comatose state, were not significantly different, but the mortality rate was three times higher.

Thus, based on the analysis of these data, it can be argued that there is a certain relationship between PCA2 in arterial blood, the duration of the comatose state, the severity of edema, brain swelling and outcomes. However, it should be noted that artificially induced, short-term hypocapnia was mainly performed on the most severe patients with severe intracranial hypertension. Therefore, data on the influence of the level of PaCO2 in arterial blood on the main indicators must be considered critically, since they do not reflect only this connection. Basically, short-term hypocapnia was used after ineffectiveness in the use of osmodiuretics, ventricular drainage or with ongoing intracranial hypertension in patients after surgical interventions along the line of intracranial hematomas. The role of long-term severe hypocapnia is difficult to assess according to a number of authors, especially with preserved reactivity of cerebral vessels to carbon dioxide (132, 134, 135, 142, 331, 333, 371, 372).

4.4. Sodium balance disorders

This section presents the results of a study of sodium metabolism disorder in 175 patients over time during the period of patients' stay in the intensive care unit in the first 7-10 days after injury. All patients had severe traumatic brain injury. Depending on the range of fluctuations in individual values of sodium concentration in the blood plasma and the dynamics of this indicator, all patients were distributed into 5 groups (/*group* - patients whose sodium plasma concentration during the study was within the normal range of 135-145 mmol/l. *Group II* - included patients who presented moderate disturbances in blood plasma sodium concentrations between 125 and 155 mmol/L. *Group III* - included patients who had hyponatremia less than 125 mmol/L. *Group IV* — patients who had hypernatremia greater than 155 mmol/L. *Group V* - patients who presented a chaotic alternation of hyponatremia (less than 125 mmol/L) and hy-pernatremia (more than 155 mmol/L).

Group I - 73 patients with normal blood plasma sodium values during the first 10 days after injury, characterized by a less severe injury and the shortest duration of comatose condition. In this group of patients, diffuse injuries were observed more often (34 patients). The outcomes were better than in other groups.

Group II - 103 patients who experienced moderate disturbances in the concentration of sodium in the blood plasma, both in the direction of its decrease and upward during the study period. But the sodium concentration figures did not go beyond 125 - 155 mmol/l. These moderate violations were easily corrected. Diffuse injuries also predominated among this group of patients (51 patients). However, the severity of the injury and the duration of the coma were greater in this group than in the group of patients where the sodium concentration was within normal limits. The outcomes were worse than in the first group. 21 patients died.

Group III - victims who experienced hyponatremia in the first 10 days after injury. The plasma sodium concentration in these patients decreased to 125 mmol/L or less. In these patients, there was no trend towards hypernatremia. Among patients in this group, patients with brain compression predominated (5 patients). The average age of patients was greatest in this group. And the average duration of the comatose state was

significantly longer compared to other groups. The mortality rate was greater than among patients with moderate disorders and normal sodium concentrations.

Group IV - 15 patients, was characterized by the development of hypernatremia with an increase in sodium concentration over 155 mmol/l. Among these patients, the majority had diffuse injuries (6 patients) and brain compression (6 patients). The duration of unconsciousness and lethality in this group were greater than in the previous two groups.

Group V - 9 patients who presented promiscuous alternations between hypernatremia and hyponatremia. All patients in this group were young. The average age was 22.8 years. They received the most serious injury. Among them, patients with brain compression with sheath hematomas predominated (4 patients). The average duration of comatose condition was maximum, as among patients of group 3 for more than 2 weeks. The largest number of patients died: 5 out of 9.

Thus, the pattern of sodium balance disorders was correlated with outcomes of severe combined brain injury. Patients with normal sodium values had the lowest mortality in their blood plasma. In the case of moderate sodium balance disorders (group 2), mortality increased to 24%. With severe disorders towards hyponatremia (group 3), as well as towards hypernariemia (group 4), mortality increased to 36% and 53%, respectively. In patients with gross sodium balance disorders, when one patient presented both hypernatremia and hyponatremia, the mortality was greatest and was 64%.

The most common disorders of osmolarity and sodium balance were observed in diffuse lesions and compression of the brain - in almost half of the cases, while in focal lesions, sodium balance disorders were observed in only one third of observations. Consequently, the most significant disruption of the mechanisms of regulation of osmotic homeostasis is observed in diffuse injuries and enveloped hematomas.

It should be noted that patients with hypernatremia and hyperomolarity were more likely to experience fractures of the base of the skull in the area of the middle cranial fossa, alcohol intoxication and hemorrhagic shock ($p<0.05$). According to other authors, with fractures of the base of the skull in the area of the Turkish saddle, electrolyte imbalances also occur more often, which indirectly indicates primary or secondary damage to the hypothalamic-pituitary region (27, 101, 110, 177).

The severity of cerebral edema was determined to be dependent on sodium imbalance during the first week after injury. As can be seen from the above table, the number of patients whose brain swelling spread within one or both hemispheres was the smallest only in the case of normal sodium values. Whereas, with hypernatremia and hyponate-riemia, the number of patients with the spread of cerebral edema per hemisphere or both was twice as large. This can be explained by the fact that with hyponatremia, the amount of intracellular fluid increases in particular in the brain, which leads to an increase in cerebral edema. Clinically, this is manifested by depression of the level of consciousness, lengthening of the comatose state (79,6,28).

Table 22.

Relationship between the degree of prevalence of cerebral edema and the degree of disturbance in sodium balance

Indicators	1group	2group	3group	4group	5group

Plasma sodium	135-145	125-155	<125	>155	<125+>155
Local swelling	30(41%)	35(41%)	2(18%)	2(13%)**	--
Within the brain's lobe	16(22%)	14(14%)	2(18%)	2(13%)	3(33%)
Within 1 or 2 hemispheres of the brain	27(37%)	54(52%)	7(64%)	11(74%)*	6(66%)
Total	73(100%)	103(100%)	11(100%)	15(100%)	9(100%)

Reliability of the difference between 1 group and the rest:* -p<0,05; * * -p<0,01

An increase in cerebral edema with prolonged hypernatremia may be associated with excessive accumulation of sodium and chlorine in the cell, which leads to increased intracellular edema due to the penetration of water into the intracellular space along an osmotic gradient.

Thus, patients with severe traumatic brain injury have changes in sodium balance and osmotic homeostasis, both towards hypernatremia and its concomitant hyperosmolarity, and towards hyponatremia and hypoosmolarity. Most often they are observed in patients with Diffuse injuries and brain compression. These changes have a significant impact on the course of traumatic brain disease and on the outcomes of traumatic brain injury, directly or indirectly affecting the severity of cerebral edema and other processes.

Conclusion: The study showed that primary brain damage is aggravated by the occurrence of secondary systemic mechanisms, brain damage. In severe TBI, damage to the mechanisms of maintaining perfusion pressure is of greatest importance (autoregulation of cerebral blood flow is partially or completely disrupted), since under these conditions, the brain is able to adequately «» respond to systemic disorders of homeostasis. As a result, various secondary injuries occur in brain tissue, mainly of an ischemic nature. The cause of these ischemic disorders is secondary systemic mechanisms of brain damage. That is why their early detection and interruption is the main goal of treatment measures.

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