

# **Age-related features of temperature reaction in the acute period of combined severe traumatic brain injury**

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**Abstract.** The indicator of the mesor of the circadian rhythm of body temperature of the 1st group of patients (19-40 years old) was distinguished not only by the absence of a tendency to hyperthermia, but also by the tendency to normalize the studied indicator by the 30th day. A tendency to a gradual increase in the level of the mesor of the circadian rhythm of temperature on the 18th and subsequent days of intensive therapy was observed in the injured group 2 (41-60 years old). A comparatively lower level of fluctuations in the mesor of the circadian rhythm T°C was revealed in the acute period of CSTBI in patients of group 3 (61-88 years old). The absence of the effect of anti-inflammatory therapy, most likely, indicates the likelihood of a centrogenic nature of hyperthermia, not excluding completely secondary infection in all patients, especially in the late periods (on the 25th day) of combined severe traumatic brain injury in patients of group 3.

Keywords: temperature, combined severe traumatic brain injury.

**Relevance.** The increase in injuries increases not only the frequency, but also the severity of traumatic brain injury (TBI), which is 50–70% associated with extracranial injuries. Mortality from concomitant traumatic brain injury (CTBI) ranges from 12 to 69%. In the general structure of peacetime injuries, the proportion of combined and multiple injuries ranges from 5 to 12%, and among the most severe - up to 40%. An almost constant component of severe associated injuries is TBI, which occurs in such cases with a frequency of 50-72 to 80-82%. The limbs are injured in 22.9% of cases, the chest - in 31%, the abdomen - in 25-29%. Multiple extracranial injuries in combination with TBI occur in 15% of cases. There is strong evidence that hyperthermic response increases the likelihood of death in patients with brain damage [1,2,3,4]. It has been shown that mortality is increased in patients with TBI, stroke, if they have an elevated body temperature in the first 24 hours after admission to the critical care unit. There are

several possible explanations for why hyperthermic conditions increase mortality in patients with brain damage. It is known that the temperature of the brain is not only slightly higher than the internal temperature of the body, but the difference between them also increases as the latter increases. Hyperthermia increases metabolic requirements (an increase in temperature by 1°C leads to an increase in metabolic rate by 13%), which is detrimental to ischemic neurons. An increase in brain temperature is accompanied by an increase in intracranial pressure. Hyperthermia increases edema, inflammation in damaged brain tissue. Other possible mechanisms of brain damage: violation of the integrity of the blood-brain barrier, violation of the stability of protein structures and their functional activity.

Given the effect of elevated temperature on the damaged brain, it is very important to quickly and accurately determine the etiology of the hyperthermic state and begin the correct treatment. Of course, if indicated, the appropriate antibacterial drugs are life-saving agents. However, early and accurate diagnosis of centrogenic hyperthermia can prevent patients from prescribing unnecessary antibiotics and their associated complications.

According to Badjatia N. (2009), 70% of patients with brain damage have an elevated body temperature during their stay in intensive care, and, for example, among patients of general intensive care, only 30–45%. Moreover, only half of the cases reported fever (infectious cause). Among patients of neurosurgical intensive care units (ICU), patients with subarachnoid hemorrhage (SAH) had the highest risk of developing a hyperthermic state, both of fever (infectious genesis) and centrogenic hyperthermic reaction (non-infectious genesis). The authors found that among ICU patients with a neurosurgical profile, only 50% of fever cases have an infectious cause. Other possible non-infectious causes of fever: drugs, venous thromboembolism, non-calculous cholecystitis. Almost any drug can cause fever, but among the most commonly used in ICU settings: antibiotics (especially  $\beta$ -lactams), anticonvulsants (phenytoin), barbiturates. Among TBI patients, patients with diffuse axonal injury (DAI) and damage to the frontal lobes are at risk of developing centrogenic hyperthermia. It is likely that these types of TBI are accompanied by damage to the hypothalamus. For non-infectious fevers, it is typical for the patient to appear in the early stages of hospitalization at the ICU. Blood in the ventricles is a risk factor, since catheterization of the ventricles of the brain often occurs with intraventricular hemorrhage. Centrogenic hyperthermia may not be accompanied by tachycardia and sweating, as is usual with infectious fever, and may be resistant to antipyretics. Although it is desirable to avoid the use of antibiotics without indications due to the development of undesirable side effects, the rejection of antibiotic therapy in patients with sepsis can be fatal [1-5].

Despite the numerous results of studies on the study of the temperature response in brain damage due to the lack of information in the literature on the age-related characteristics of the dynamics of the mesor of the circadian rhythm of body temperature in the acute period of injuries associated with severe traumatic brain injury (STBI), we tried to identify on the basis of a retrospective analysis distinctive characteristics, features in different age groups. The division into groups was dictated by the well-known features inherent in each age group, described in detail in the literature.

**Purpose of the work:** to study and assess the age-related characteristics of the temperature reaction in the acute period of combined severe traumatic brain injury.

**Material and research methods.** The indicators of a comprehensive examination of 27 patients with concomitant severe craniocerebral trauma (CSTBI) who were admitted to the ICU of the neurosurgical department of the RSCRMA in the first hours after an accident - 25, catatrauma of 2 patients were studied. According to the indications, 26 patients on admission started invasive mechanical respiratory support (MRS), 1 patient, due to the lack of direct indications for (mechanical ventilation) MV, intensive therapy was carried out with spontaneous breathing. Monitoring was carried out by complex hourly registration of parameters of body temperature, hemodynamics, respiration. Mechanical respiratory support was initiated by mechanical ventilation (LV) for a short time followed by transfer to SIMV. On admission, impaired consciousness in 26 injured patients was assessed on the Coma Glasgow Scale (GS) 8 points and below. Patients were considered in three age groups: group 1, 19-40 years old (13 patients), 2 - 41-60 years old (7), 3 - 61-84 years old (7 patients). After recovery from shock, anesthetic, anti-inflammatory, antibacterial, infusion therapy, correction of violations of protein, water-electrolyte balance, surgical early correction, as far as possible, syndromic, symptomatic therapy were carried out.

## Results and its discussion.

Table 1

Dynamics of the mesor of the circadian rhythm of body temperature in the acute period of combined severe traumatic brain injury

days	group 1	group 2	group 3
1	37.0±0.2	37.2±0.7	36.8±0.1
2	37.2±0.3	37.4±0.4	37.3±0.3*
3	37.7±0.6	37.3±0.4	37.1±0.2
4	37.4±0.3	37.4±0.4	37.5±0.4
5	37.5±0.4	37.7±0.4	37.3±0.3*
6	37.5±0.3	37.4±0.3	37.4±0.5
7	37.5±0.3	37.5±0.2	37.3±0.4

8	37.5±0.3	37.5±0.4	37.2±0.4
9	37.6±0.3*	37.6±0.1	37.2±0.3
10	37.7±0.4*	37.4±0.2	37.1±0.5
11	37.4±0.5	37.7±0.2	37.1±0.3
12	37.5±0.4	37.6±0.2	37.2±0.3
13	37.4±0.2	37.3±0.2	37.0±0.3
14	37.4±0.3	37.3±0.3	37.2±0.1
15	37.2±0.2	37.4±0.2	37.1±0.2
16	37.2±0.1	37.3±0.4	37.2±0.1
17	37.1±0.2	37.3±0.4	37.1±0.2
18	37.2±0.4	37.4±0.4	37.1±0.3
19	37.3±0.3	37.5±0.4	37.0±0.2
20	37.2±0.4	37.6±0.3	37.0±0.2
21	37.2±0.3	37.5±0.2	37.0±0.2
22	37.4±0.2	37.6±0.1	36.9±0.3
23	37.3±0.3	37.5±0.3	37.3±0.5
24	37.3±0.2	37.7±0.4	36.8±0.2
25	37.3±0.2	37.4±0.3	37.4±0.4*
26	37.3±0.3	37.8±0.2	37.3±0.3
27	37.3±0.2	37.6±0.1	37.0±0.3
28	37.3±0.1	37.8±0.2	37.2±0.4
29	37.3±0.1	37.9±0.03	37.0±0.1
30	36.9±0.4	37.5±0.1	37.2±0.3

\*-reliably relative to the indicator in 1 day

As shown in table 1, on the day of admission to the clinic in the first hours after combined severe traumatic brain injury, the mesor of the circadian rhythm of body temperature in patients of groups 1 and 2 was within subfebrile numbers (from 36.8°C to 37.9°C), in group 3 - 36.8 ± 0.1°C. During the acute period, in group 1 of the injured, a significantly significant increase in the mesor of the circadian rhythm of body temperature was revealed on days 9 and 10 to 37.6 ± 0.3°C and 37.7 ± 0.4°C, respectively. In group 2, there was a tendency to an increase in the temperature reaction throughout the entire period of treatment in the ICU. In group 3, a significantly significant increase in body temperature was detected on days 2, 5, 25 to 37.3 ± 0.3°C, 37.3 ± 0.3°C and 37.4 ± 0.4°C, respectively. That is, despite the ongoing anti-inflammatory complex and antibacterial therapy, on days 9-10 in group 1, on days 2, 5, 25 in group 3, a hyperthermic reaction was revealed, which indicated the insufficient effectiveness of traditional complex therapy in all age groups, which is more likely in total, indicates the likelihood of a centrogenic nature of hyperthermia, especially in the late periods (on the 25th day) of combined severe traumatic brain injury in patients of group 3.

Thus, secondary brain damage remains a problem far from resolving secondary brain damage, there are no effective methods not only for prevention, but also for preserving the structural integrity and functional activity of initially intact brain areas. The existing recommendations are not effective enough in the correction of ischemia, edema, diffuse axonal injuries, cannot serve as methods of preventing an increase in the zone of irreversible changes in the medulla.

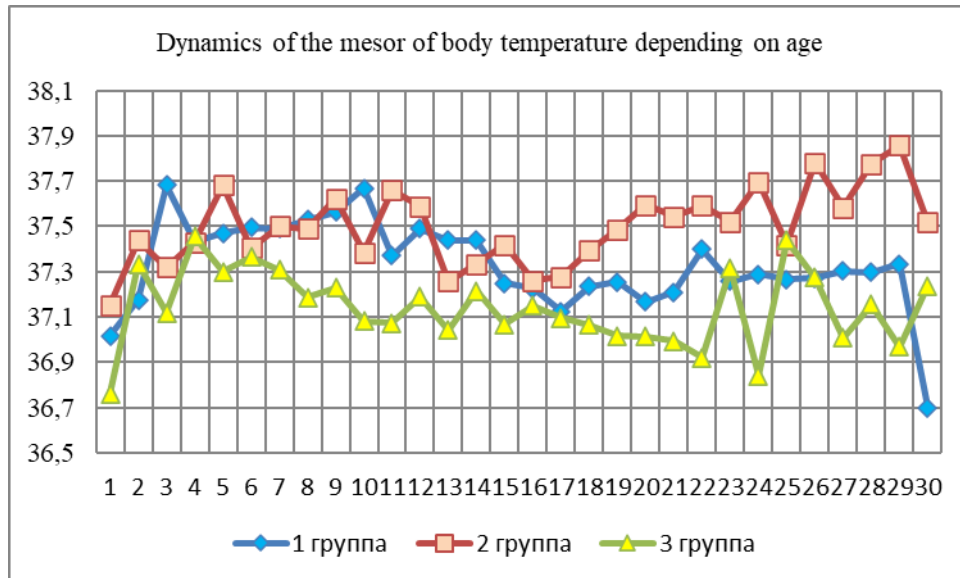


Fig.1

As can be seen from fig. 1, draws attention to the relatively lower level of changes in the mesor of the circadian rhythm  $T^{\circ}\text{C}$  with an increase to  $37.4^{\circ}\text{C}$ ;  $37.3^{\circ}\text{C}$  and  $37.4^{\circ}\text{C}$  on day 4.23.25 of the acute period in patients of group 3. A tendency to a gradual increase in the level of the mesor of the circadian rhythm of temperature on the 18th and subsequent days of intensive therapy was observed in the injured group 2 (up to  $37.9^{\circ}\text{C}$  on the 29th day). The mesor temperature index of the 1st group of patients differed not only by the absence of a tendency to hyperthermia, but also by the tendency to normalize the studied parameter by the 30th day.

Correlations between the dynamics of the temperature response and hemodynamic parameters in age groups are shown in fig. 2.

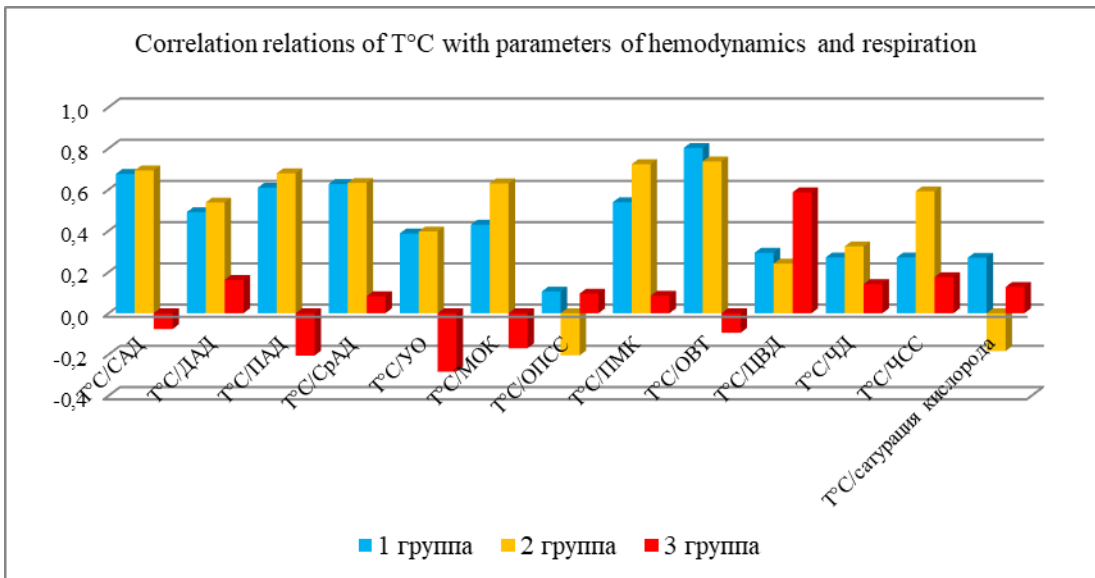


Fig.2

A direct correlation was found between changes in T°C and the dynamics of the mesoor SBP (0.63), PBP (0.62), avBP (0.6), CO (0.6), MVP (0.64), AVT (0, 7) in patients of groups 1 and 2. That is, the systemic inflammatory response to severe trauma was accompanied by a compensatory increase in the functional activity of hemodynamics, which indicated a preserved regulatory mechanism of the central nervous system, despite STBI in patients under 60 years of age. Draws attention to the fact that these compensatory reactions completely disappeared in patients over 61 years of age. Direct correlation between T°C and CVP level (0.58) confirms the likelihood of developing acute heart failure in conditions of an intact systemic inflammatory reaction, the objective indicator of which is hyperthermic syndrome.

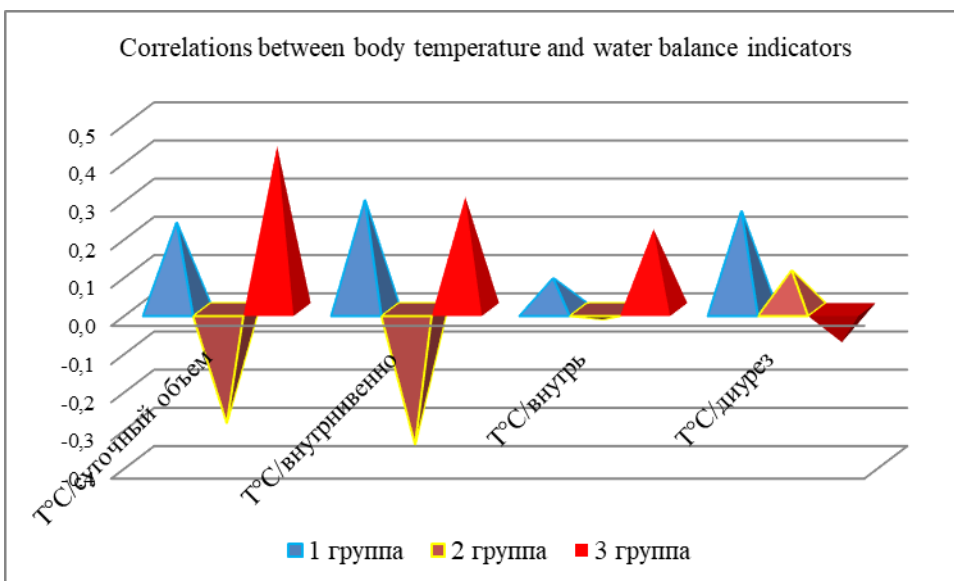


Fig.3

A moderately pronounced direct correlation was found between the dynamics of the mesor of the circadian rhythm of temperature and the total daily volume of the injected fluid (0.38). An insignificant trend towards a decrease in temperature with an increase in the volume of intravenous administration (0.35) was revealed in patients of group 2 (fig. 3).

**Conclusion.** The mesor temperature index of the 1st group of patients differed not only by the absence of a tendency to hyperthermia, but also by the tendency to normalize the studied parameter by the 30th day. A tendency to a gradual increase in the level of the mesor of the circadian rhythm of temperature on the 18th and subsequent days of intensive therapy was observed in the injured group 2. A comparatively lower level of fluctuations in the mesor of the circadian rhythm T°C was revealed in the acute period of CSTBI in patients of group 3. The absence of the effect of anti-inflammatory therapy, most likely, indicates the likelihood of a centrogenic nature of hyperthermia, not excluding completely secondary infection in all patients, especially in the late periods (on the 25th day) of combined severe traumatic brain injury in patients of group 3.

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