Circadian rhythm of pulse blood pressure in the acute period of concomitant severe traumatic brain injury

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Abstract

Changes in PBP in the circadian rhythm during the first week in group 1 (19-40 years old) occurred on average at 41.3±2.3 mmHg, in group 2 (41-60 years old) - 51.3±0.9 mmHg, at 3 (61-85 years) - 50.5±1.3 mmHg, acute period of CSTBI. In the second group, in the first and second near-week periods, the average PBP was found at the level of 50.5±1.2 mmHg, 50.6±1 mmHg, from 18 to 25 days of observation PBP was 55.4±1.4 mmHg, which reflected an upward trend in PBP in the third week of observation. The most pronounced range of daily PBP fluctuations was found in group 2. Revealed significant changes in PBP in the evening and night hours, pathological mixing of the acrophase of the circadian rhythm of PBP at night in patients of group 3.

Keywords: circadian rhythm, pulse arterial pressure, combined severe traumatic brain injury.

Relevance. Brain damage occurs not only at the time of injury, but continues over the next hours and days. The affected brain is very sensitive to: hypotension, hypoxia, increased intracranial pressure. Impaired perfusion of the brain leads to an acute deficiency of macroergs, massive release of excitatory amino acids (glutamate "excitotoxicity"), impaired permeability of cell membranes with the penetration of calcium ions into the cell, and the development of lactic acidosis in ischemic tissue. The starting pathophysiological mechanism in acute cerebral insufficiency, as the final link, is the formation of tissue hypoxia caused by mitochondrial dysfunction. These processes are triggered even with short-term episodes of a drop in cerebral perfusion pressure, develop directly from the moment of injury and, in general, fade away by the end of the first day of ischemia. Further damage to the nervous tissue occurs by the mechanism of an increase in oxidative stress and local inflammation (from 2-3 hours after pathological exposure with a maximum by 12-36 hours) and the progression of apoptosis. Currently, there are

2 areas of urgent measures for acute cerebral insufficiency: restoration of cerebral perfusion and neuroprotective therapy. Prolonged stress-protective therapy (SPT) with ganglion blockers, a- and b-adrenolytics, clonidine and dalargin has a pronounced protective effect on the brain and other functions of the victims. Both early (in the resuscitation phase) and late (in the treatment phase) arterial hypotension are inherently associated with a less favorable outcome. Prevention of hypotension is perhaps more important than medication to maintain blood pressure [1-4].

Pulse blood pressure (PBP) characterizes the dynamic component of the pressor action on target organs, and is also an indirect indicator of increased rigidity of large arterial vessels. Many researchers use PBP as an independent predictor of coronary complications; many have introduced this indicator into the list of standard indicators of daily blood pressure monitoring (DBPM). High PBP is an independent risk factor for coronary atherosclerosis and left ventricular hypertrophy [5]. The lack of data in the literature on the informativeness of the PBP circadian rhythm in assessing the severity of hemodynamic deviations in CSTBI was the reason for studying the indicator in the dynamics of the acute period of CSTBI.

Purpose. To study and evaluate the results of PBP circadian rhythm monitoring in the acute period of combined severe traumatic brain injury.

Material and research methods. The indicators of a comprehensive examination of 30 patients with concomitant severe traumatic brain injury (STBI) who were admitted to the ICU of the RSCEMA neurosurgical department in the first hours after an accident - 28, catatrauma - 2 patients were studied. According to the testimony of 29 patients, on admission, invasive mechanical respiratory support (MRP) was started. Monitoring was carried out by complex hourly registration of parameters of body temperature, hemodynamics, respiration. Mechanical respiratory support was started with artificial lung ventilation (ALV) for a short time, followed by transfer to SIMV. The assessment of the severity of the condition was carried out by scoring methods according to the scales for assessing the severity of combined injuries - the CRAMS scale, the assessment of the severity of injuries on the ISS scale. On admission, impaired consciousness in 29 injured patients was assessed on the Glasgow Coma Scale (GS) 8 points or less. Patients were considered in three age groups: group 1, 19-40 years old (13), group 2 - 41-60 years old (9), 3 - 61-84 years old (8 patients). Complex intensive therapy consisted in identifying and timely correction of deviations: MCI, after removing from shock anesthetic, anti-inflammatory, antibacterial, infusion therapy, correction of protein and water-electrolyte balance disorders, surgical early correction to the extent possible, stress-protective therapy.

Results and discussion.

Table 1.

Circadian rhythm of the mesor of the circadian rhythm of pulse blood pressure in the acute period of combined severe traumatic brain injury (mmHg)

Days	Group 1	Group 2	Group 3
1	50.5±2.7	44.7±4.9	55.8±6.0
2	53.4±1.7	48.6±2.8	51.1±2.7
3	54.8±1.7	49.0±2.7	54.4±1.9
4	56.9±2.3	53.0±3.8	50.3±3.6
5	52.9±2.1	51.8±3.5	54.3±2.4
6	53.6±2.9	52.2±2.8	55.7±2.3
7	55.3±2.8	51.8±2.5	56.2±2.9
8	50.7±2.2	52.8±2.5	55.6±4.6
9	52.0±1.6	52.2±2.3	55.7±4.0
10	52.5±2.1	49.6±2.8	54.1±4.1
11	51.0±2.3	52.3±3.1	55.0±4.1
12	51.7±2.2	50.1±1.8	56.4±3.4
13	53.2±3.9	50.7±2.9	47.6±4.2
14	50.2±2.4	48.1±2.6	50.3±3.4
15	51.0±2.3	49.8±3.1	56.9±5.1
16	50.9±2.9	48.4±2.4	50.0±3.5
17	48.9±2.5	53.9±4.7	55.9±4.1
18	49.0±3.3	52.2±4.1	50.6±2.9
19	45.7±2.4	52.6±3.1	49.9±4.1
20	48.0±2.2	52.7±5.0	49.4±4.3
21	53.2±2.7	61.0±3.6*	50.2±4.6
22	53.9±2.5	55.6±4.3	53.9±7.5
23	51.7±2.5	54.0±4.2	52.8±5.1
24	53.3±2.5	55.1±3.2	55.8±3.7
25	48.9±3.2	60.2±5.9*	54.4±4.9

*-reliably relative to the indicator in 1 day

As shown in Table 1, the mesor of the circadian rhythm of PBP on day 1 did not differ from the generally accepted standard values (30-60 mmHg). During 25 days of the acute period of CSTBI, an increase in the mesor of the circadian rhythm of PBP was revealed only in group 2 by 21 (36%) and 25 days by 34% ($p<0.05$).

Dynamics of the mesor of the circadian rhythm of pulse arterial pressure in CSTBI (mmHg)

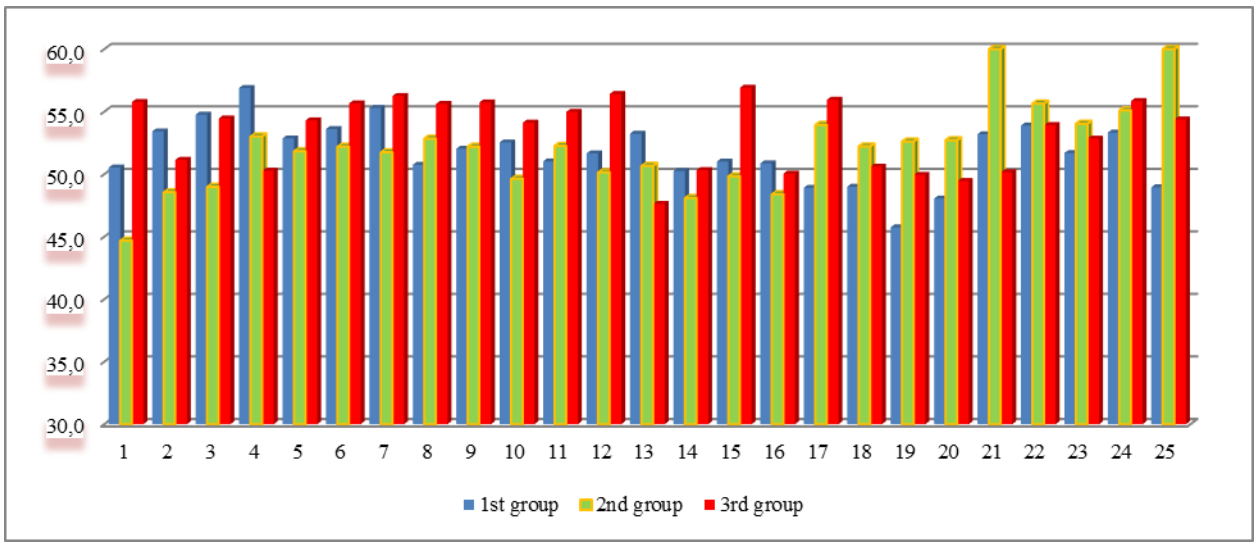

Fig.1

The tendency to an increase in the mesor of the circadian rhythm PBP was also observed in patients of group 3 and, to a lesser extent, group 2 in the first 12 days (fig. 1). While in group 2, there was a more pronounced tendency to increase the mesor of the circadian rhythm of PBP on days 17-25. The revealed hemodynamic feature of group 2 patients characterizes the occurrence of unfavorable changes, possibly requiring a more effective vasodalatory correction. This is confirmed by the tendency to an increase in the DBP mesor of circadian rhythm on days 18-25 of the acute period of CSTBI, presented in the previous article.

Near-weekly mean PBP circadian rhythm in group 1 (mmHg)

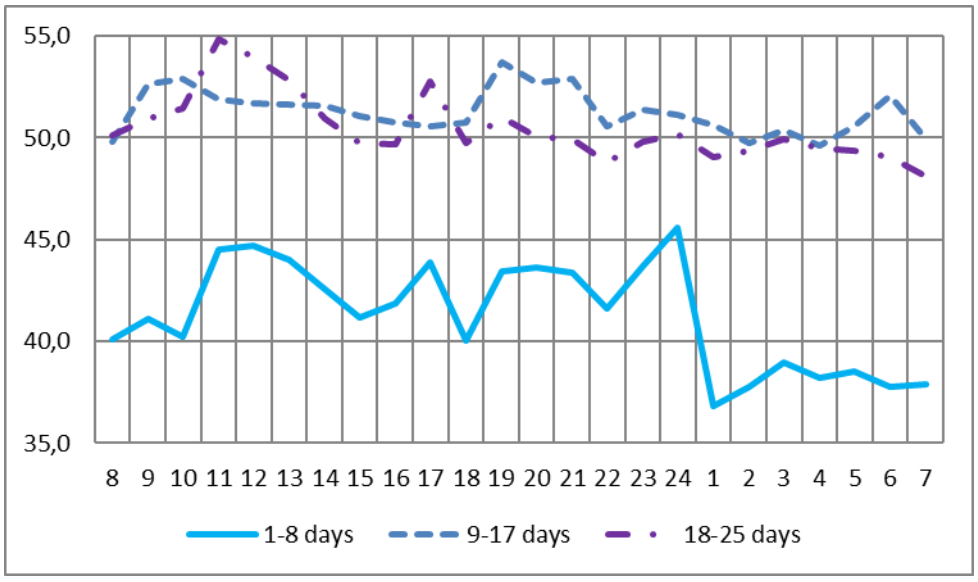

Fig.2

As shown in fig. 2, fluctuations in the PBP index in the circadian rhythm during the first week in group 1 occurred on average at 41.3±2.3 mmHg, in group 2 - 51.3±0.9 mmHg, in group 3 - 50.5±1.3 mmHg, that is, 10 mmHg lower ($p < 0.05$) than in the second week and 9.2 mmHg lower ($p < 0.05$) than in the third week of the acute period of CSTBI. The findings most likely characterize more effective stress-protective therapy during the first 8 days in traumatized patients of group 1.

Circadian rhythms in near-weekly PBP periods in group 2 (mmHg)

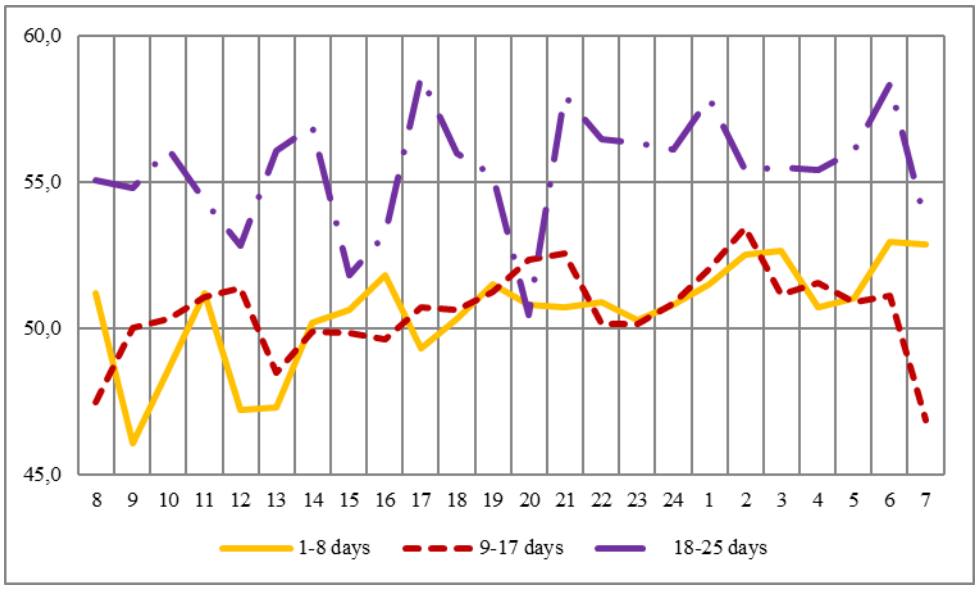

Fig.3

In the second group, in the first and second near-week periods, an average PBP was found at the level of 50.5 ± 1.2 mmHg, 50.6 ± 1 mmHg, from 18 to 25 days of observation PBP was 55.4 ± 1.4 mmHg, which reflected an upward trend in PBP in the third week of observation (fig. 3). The latter can be associated with the limitation of stress-protective therapy in patients of group 2.

Daily fluctuations in average weekly PBP in group 3 (mmHg)

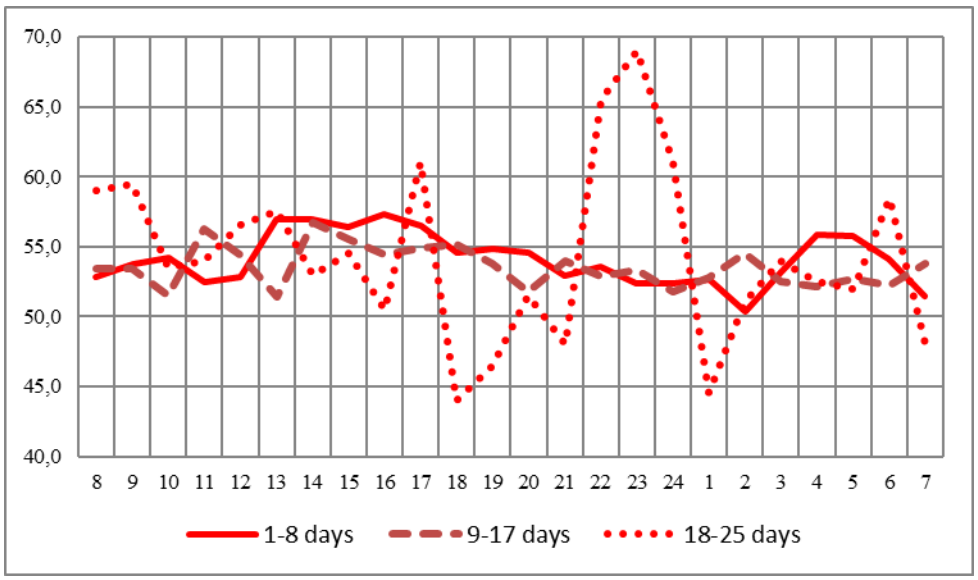

Fig.4

In contrast to the previous first two groups, in persons over 61 years of age throughout the acute period, the average daily indicators in 1, 2.3 weeks did not differ, amounting to 54.1±1.5 in 8 days, from 9 to 17 days – 53.6±1.1, from 18 to 25 days 54.4±4.8 mmHg (fig. 4). Noteworthy are significant changes in PBP in the evening and night hours, which is most likely associated with pituitary-adrenal insufficiency, despite the ongoing hormonal, vasoactive corrective therapy.

Dynamics of the amplitude of daily fluctuations in PBP in the acute period of CSTBI (mmHg)

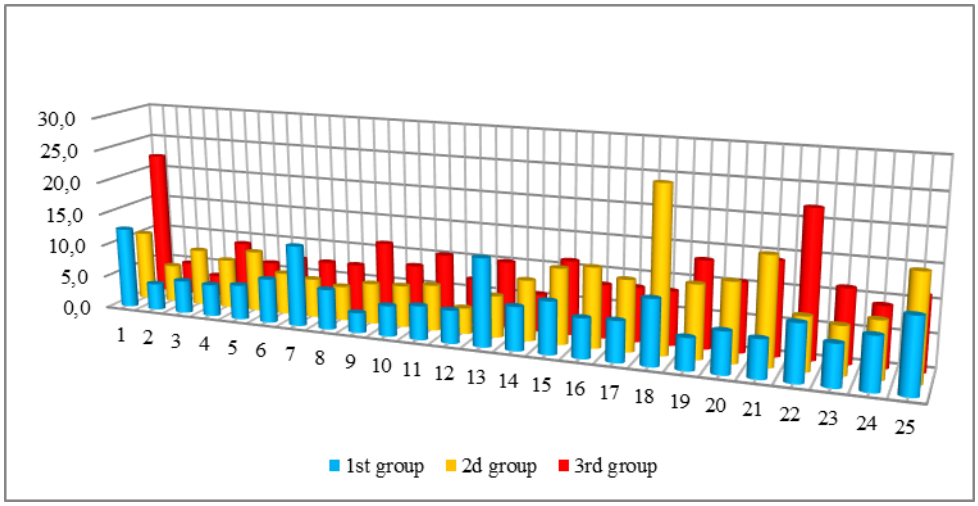

Fig.5

As shown in fig. 5, the least significant changes in the amplitude of circadian rhythm PBP in the dynamics of the acute period were observed in patients of group 1. Changes in the PBP amplitude occurred in waves with periods of fluctuations of 7,6,5,5,4 days. That is, more active stress-protective therapy was accompanied by hemodynamic function (PBP) in the physiological

weekly biorhythm. Thus, active stress-protective therapy contributed to the restoration of the physiological biorhythm of PBP in the acute period of CSTBI as early as the first week after injury. A decrease in the about-week oscillation period in the following days to five days is most likely the result of the restriction of stress-protective therapy. More physiological, I think, should be considered an increase in the duration of active stress-protective therapy up to 25 days in the studied contingent of traumatized patients.

The daily range of changes in pulse blood pressure (in mmHg)

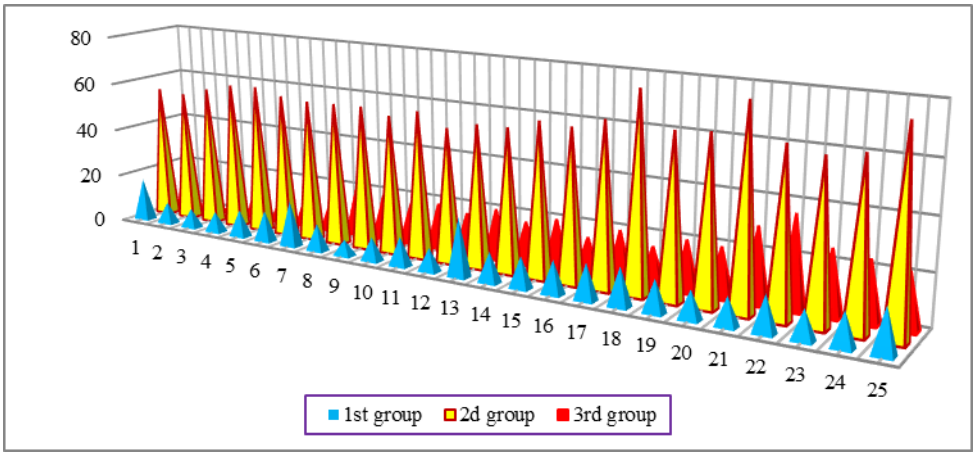

Fig.6

The most pronounced range of daily PBP fluctuations was found in group 2 (fig. 6). Thus, the maximum PBP fluctuations per day were -12.7±2.8 mmHg in group 1, 19.4±4.5 mmHg in group 3, and 61.6±4.7 mmHg in group 2 ($p<0.05$, respectively). The findings confirm the most pronounced hemodynamic instability in patients over 41 years old.

Duration and severity of PBP circadian rhythm acrophase shifts in%.

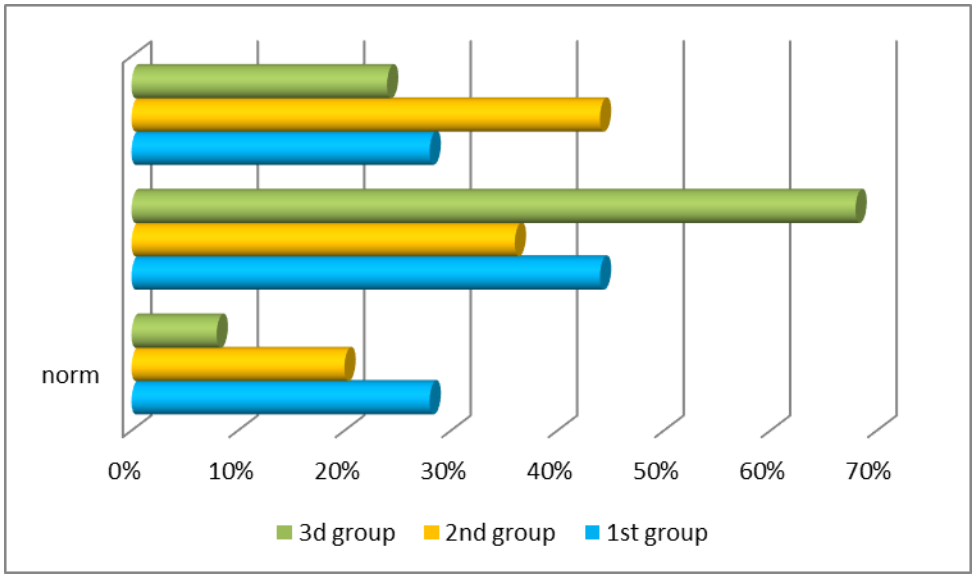

Fig.7

The normal projection of the PBP acrophase peak was 7% in group 3, 18% in group 2, and 27% in group 1. The longest inversion of the PBP circadian rhythm (23%) and the duration of moderate displacements (68% of the time) were found in patients of group 3 (fig. 7).

Correlation Relationships of the PBP mesor with SBP and DBP mesors

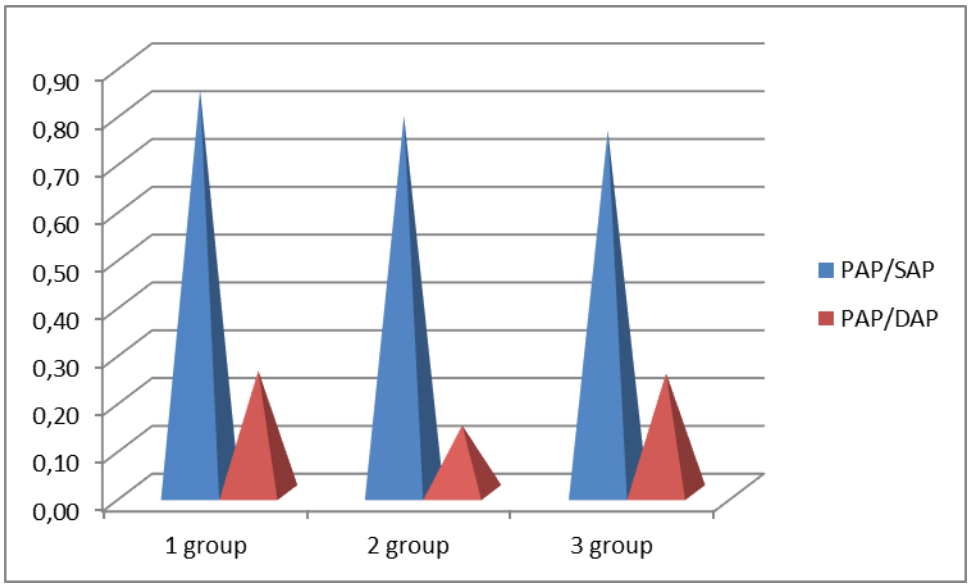

Fig.8

In all age groups, there was a strong direct correlation between changes in SBP and PBP and an insignificant one between DBP and PBP.

Conclusion. Changes in PBP in the circadian rhythm during the first week in group 1 occurred on average at 41.3±2.3 mmHg, in group 2 - 51.3±0.9 mmHg, in group 3 - 50.5±1.3 mmHg, acute period CSTBI. The most pronounced range of daily PBP fluctuations was found in group 2. In the second group, in the first and second near-week periods, the average PBP was found at the level of 50.5±1.2 mmHg, 50.6±1 mmHg, from 18 to 25 days of observation PBP was 55.4±1.4 mmHg, which reflected an upward trend in PBP in the third week of observation. Revealed significant changes in PBP in the evening and night hours, pathological displacement of the acrophase of the circadian rhythm of PBP at night in injured people over 61 years old.

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