Dynamics of the circadian rhythm of the minute cardiac output in the acute period of concomitant severe traumatic brain injury

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Abstract

The increase in the CO mesor in group 1 in the second week after injury was associated with the need for maximum mobilization of blood circulation to ensure the necessary oxygenation of the brain under conditions of secondary pathogenetic mechanisms of CNS damage. Changes in the amplitude of the circadian rhythm of cardiac output (CO) occurred in waves with an unstable wavelength, deformation of the phase structure of weekly fluctuations, with the predominance of the highest values of the amplitude of the circadian rhythm in group 1 on days 1, 13, 17, 22, in group 2 - maximum values of the amplitude were at 1,19.21 days. In group 3 - at 1,19,22 days. Significantly lower values of the CO circadian rhythm mesor are apparently due to the limited compensatory capabilities of the circulatory system in the adaptive restructuring of hemodynamics in the acute period of CSTBI in patients over 40 years of age. Strong direct correlations between CO and SV (0.87), CO and PBP (0.79) were found in group 2 of patients (41-60 years old).

Keywords: circadian rhythm, cardiac output, combined severe traumatic brain injury

Relevance. The metabolic processes of the brain are adapted to the conditions of rich delivery of oxygen and glucose (with a brain mass of about 2% of body weight, it receives 15-20% of cardiac output), therefore, the brain is practically incapable of anaerobic compensation for a lack of energy, which, under conditions of hypoxia, entails a rapid and irreversible damage to the central nervous system [1,2,3]. After CSTBI, the researchers noted a significant decrease in the cardiac index on the fourth day in the blood oxygen transport system. As a result, this led to a decrease in oxygen consumption and was clinically manifested in an aggravation of the general condition of the patients. Damage to the apparatus for regulating external respiration and blood circulation in severe concomitant trauma with severe craniocerebral injury caused by direct brain injury, subsequent ischemia and edema, it often leads to decompensation in the blood oxygen transport system on the third-fourth day of the post-shock period [4]. Lack of information on the assessment, timely correction of changes in cardiac output prompted us to study one of the priority tasks of intensive therapy for CSTBI in the acute period.

Purpose of the work. To study the circadian rhythm of cardiac output in the acute period of combined severe traumatic brain injury.

Material and research methods. We studied the indicators of a comprehensive examination of 30 patients with concomitant severe traumatic brain injury (CSTBI) who were admitted to the ICU of the RSCEMA neurosurgical department in the first hours after an accident - 28, catatrauma in 2 patients. Continuous hourly monitoring of cardiac output (CO), systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse blood pressure (PBP), average blood pressure (avBP) blood pressure was performed for 25 days after CSTBI. According to indications, 29 patients underwent invasive mechanical respiratory support (MRS) on admission. Mechanical respiratory support began with short-term artificial lung ventilation (ALV) followed by a switch to SIMV. The assessment of the severity of the condition was carried out using scoring methods according to the scales for assessing the severity of combined injuries - the CRAMS scale, the assessment of the severity of injuries according to 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 care consisted in identifying and timely correction of deviations: MRS, after removing from shock pain-relieving, 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. As shown in Table 1, on the first day after the injury, the mesor of the circadian rhythm CO did not differ from the normative data.

Table 1

Dynamics of the mesor of the circadian rhythm CO depending on age

Days	Group 1	Group 2	Group 3
1	4.4±0.4	4.8±0.5	4.8±0.7
2	4.6±0.2	4.5±0.4	4.2±0.2
3	5.2±0.4	4.4±0.3*	4.5±0.3
4	4.9±0.2	4.4±0.3	4.2±0.4*
5	4.9±0.2	4.9±0.5	4.5±0.3
6	5.3±0.5	4.9±0.3	4.5±0.2*
7	5.2±0.4	4.9±0.3	4.7±0.3
8	4.9±0.3	5.0±0.3	4.6±0.4
9	5.3±0.3**	5.1±0.2	4.5±0.4*
10	5.1±0.2	4.6±0.3	4.3±0.4*
11	5.2±0.4	5.2±0.5	4.4±0.3*
12	5.6±0.3**	4.8±0.3*	4.8±0.4*
13	5.7±0.5**	5.0±0.3	4.4±0.4*
14	4.9±0.3	4.6±0.3	4.5±0.3
15	5.1±0.4	4.8±0.4	4.8±0.4
16	5.1±0.4	4.7±0.3	4.5±0.3
17	4.8±0.5	5.3±0.4	4.6±0.4
18	4.7±0.4	4.9±0.4	4.6±0.3
19	4.4±0.3	5.0±0.4	4.6±0.6
20	4.7±0.4	4.9±0.3	4.1±0.4
21	4.9±0.3	5.8±0.5	4.2±0.5
22	5.5±0.4	5.1±0.4	4.4±0.8
23	5.1±0.2	5.3±0.6	4.6±0.5
24	5.3±0.4	5.4±0.5	4.5±0.3
25	4.8±0.4	5.4±0.6	5.0±0.6

*- the difference is significant relative to the indicator in group 1

**- the difference is significant relative to the indicator on the first day

Anti-shock, timely infusion corrective therapy led to the restoration of the mesor of the circadian rhythm of CO already in the first day. As shown in Table 1, the CO circadian rhythm mesor in all the injured did not differ significantly from the normative data. In group 1, a reliably significant increase in the mesor of the circadian rhythm of CO on days 9, 12, 13 was revealed (by 20%, 27%, 29%, $p < 0.05$, respectively). In groups 2 and 3, there was no significant CO dynamics in the acute period. Comparative analysis showed that in group 2, the circadian rhythm mesors were significantly less than in group 1 on days 3 and 12 (by 15%, 14%, $p<0.05$, respectively). In group 3, on days 4, 6, 9, 10, 11, 12, 13, significantly lower indicators of the mesor of the circadian rhythm CO were found by 14%, 15%, 15%, 15%, 15%, 14%, 22% ($p<0.05$, respectively). Age-related differences in stress-CO reactions, significantly lower values of the mesor of the circadian rhythm of CO in group 3, apparently, are due to the limited compensatory capabilities of the circulatory system in adaptive restructuring of hemodynamics in patients over 40 years of age. The increase in the CO mesor in group 1 in the second week after injury was most likely associated with the need for maximum mobilization of blood

circulation to ensure the necessary oxygenation of the brain under conditions of secondary pathogenetic mechanisms of CNS damage.

Dynamics of the amplitude of daily fluctuations of CO, l per minute

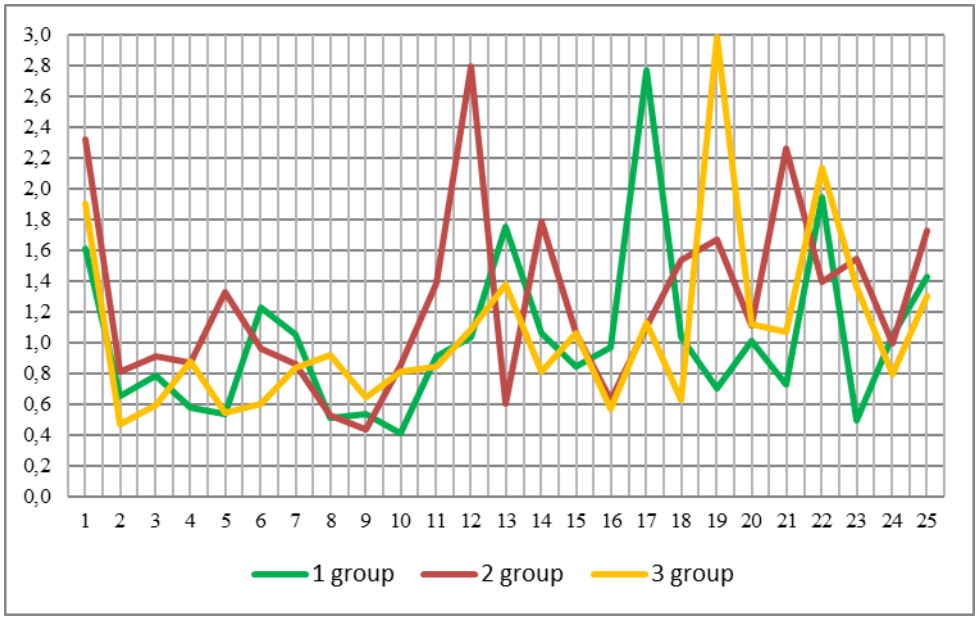

Fig.1

Changes in the amplitude of the circadian rhythm of CO occurred in a wave-like manner with an unstable wavelength, deformation of the phase structure of near-week fluctuations, with the predominance of the highest values of the amplitude of the circadian rhythm in group 1 on days 1, 13, 17, 22, in group 2 - the maximum values of the amplitude were in 1,19,21 days, in group 3 - at 1,19,22 days. Considering that the stress reaction of hemodynamics is manifested by an increase in the amplitude of daily fluctuations, it can be assumed that the ongoing intensive therapy on the indicated days was insufficiently stress-protective, despite the fluctuations in hemodynamic parameters within the permissible normative values during the acute period of CSTBI. That is, the compensatory centralization of blood circulation was preserved, aimed at increasing the oxygen supply to the damaged areas of the brain.

Change in the daily range of CO fluctuations

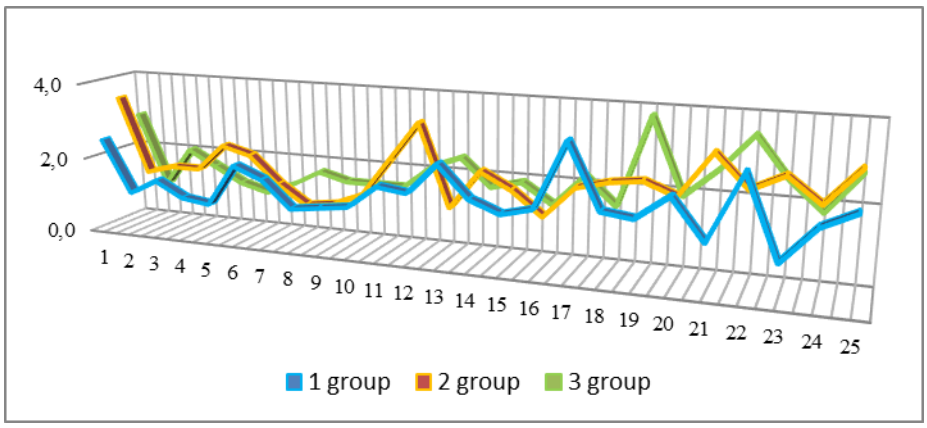

Fig.2

The dynamics of daily changes in the circadian rhythm (fig. 2) revealed a tendency to increase up to 5.25 l/min in group 1 in the morning 9-11 hours. That is, during the first 25 days, only group 1 patients retained a tendency to maintain the physiological projection of the acrophase of the CO circadian rhythm in the morning hours with an oscillation period of 7 hours, while in groups 2 and 3 this sign of the physiological state of the CO circadian rhythm was absent. It is noteworthy that the 5,6,5-hour CO fluctuations occurred at a comparatively lower level of the CO circadian rhythm mesor in group 3 (4.5±0.1 l/min, $p<0.05$). While the total for 25 days, the mesor of the indicator in group 1 was 5±0.1, in group 2 - 5±0.1 l/min.

Acute average hourly CO values in the circadian rhythm

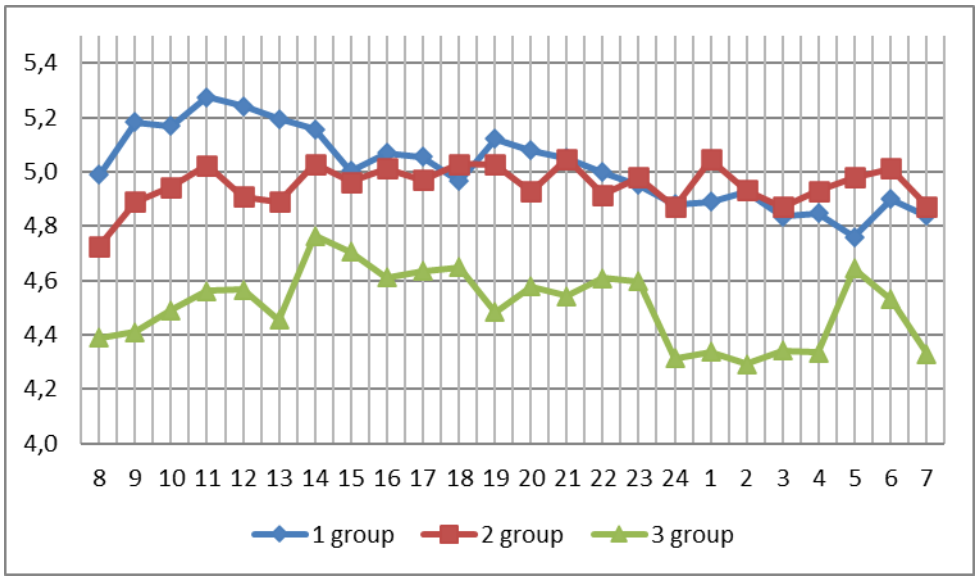

Fig.3

Table 2

Changes in the circadian rhythm of cardiac output in the acute period of CSTBI, l/min

	1-8 days			9-17 days			18-25 days		
Hours	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
8	4.9±0.3	4.5±0.3	4.4±0.2	5.0±0.4	4.6±0.2	4.4±0.3	5.0±0.4	5.0±0.2	4.4±0.6
9	5.2±0.5	4.4±0.6	4.5±0.3	5.3±0.3	5.1±0.8	4.4±0.4	5.0±0.3	5.1±0.3	4.3±0.5
10	5.1±0.1	4.5±0.3*	4.5±0.3*	5.4±0.3	4.9±0.3	4.4±0.4*	4.9±0.4	5.4±0.7	4.6±0.7
11	5.2±0.2	4.8±0.3	4.3±0.3*	5.2±0.5	5.1±0.5	4.8±0.3	5.4±0.7	5.2±0.3	4.6±0.3
12	4.9±0.6	4.5±0.3	4.5±0.3	5.5±0.5	5.1±0.6	4.6±0.3*	5.2±0.6	5.1±0.5	4.6±0.6
13	5.1±0.5	4.7±0.3	4.7±0.5	5.3±0.5	4.8±0.3	4.3±0.4*	5.2±0.5	5.2±0.5	4.4±0.3
14	5.0±0.5	4.8±0.3	4.7±0.6	5.3±0.3	4.7±0.3	4.9±0.5	5.2±0.4	5.6±0.7	4.6±0.6
15	4.9±0.2	5.0±0.7	4.7±0.3	5.2±0.3	4.9±0.2	4.7±0.2	4.9±0.4	5.0±0.7	4.7±0.3
16	5.1±0.4	4.8±0.5	4.8±0.4	5.3±0.4	5.0±0.3	4.5±0.7	4.8±0.3	5.2±0.5	4.5±0.2
17	4.9±0.3	4.6±0.3	4.8±0.5	5.1±0.3	4.9±0.4	4.8±0.4	5.1±0.2	5.4±0.5	4.3±0.3*
18	5.0±0.5	4.7±0.4	4.5±0.4	5.1±0.7	5.0±0.4	4.6±0.5	4.7±0.3	5.4±0.5	4.8±1.1
19	4.8±0.4	4.9±0.3	4.5±0.2	5.5±0.5	4.9±0.4	4.5±0.6	5.0±0.5	5.3±0.4	4.5±0.2
20	4.9±0.3	4.8±0.3	4.8±0.5	5.4±0.6	5.0±0.4	4.3±0.5	4.9±0.6	5.0±0.3	4.7±0.7
21	4.9±0.4	4.8±0.3	4.5±0.2	5.5±0.4	5.0±0.2	4.5±0.4*	4.8±0.5	5.3±0.3	4.6±0.3
22	5.0±0.4	4.7±0.3	4.6±0.3	5.1±0.3	4.9±0.5	4.5±0.3	4.8±0.3	5.2±0.4	4.8±0.5
23	4.9±0.4	4.9±0.3	4.4±0.3	5.1±0.4	4.9±0.4	4.6±0.4	4.8±0.3	5.2±0.5	4.8±0.6
24	4.6±0.2	4.7±0.4	4.3±0.2	5.1±0.4	4.8±0.3	4.4±0.5	4.9±0.4	5.2±0.3	4.2±0.4
1	4.9±0.4	4.6±0.5	4.3±0.2	5.1±0.2	4.9±0.3	4.6±0.3	4.6±0.3	5.6±0.6	4.2±0.6
2	4.9±0.4	4.8±0.6	4.2±0.2*	5.1±0.5	5.1±0.3	4.5±0.3	4.8±0.5	4.9±0.4	4.2±0.4
3	4.9±0.3	4.8±0.5	4.3±0.2*	4.9±0.4	4.8±0.4	4.4±0.4	4.7±0.2	5.0±0.3	4.2±0.4
4	4.8±0.4	4.7±0.5	4.6±0.3	4.8±0.3	5.0±0.5	4.3±0.3	4.9±0.4	5.0±0.5	4.1±0.7
5	4.5±0.2	4.8±0.4	4.5±0.4	5.1±0.4	4.9±0.4	4.6±0.3	4.7±0.5	5.3±0.5	4.9±0.6
6	4.7±0.4	4.8±0.5	4.6±0.3	5.2±0.4	4.9±0.3	4.5±0.1*	4.7±0.4	5.4±0.9	4.6±0.6
7	4.9±0.5	4.8±0.4	4.0±0.3*	5.0±0.3	4.5±0.2	4.6±0.4	4.6±0.4	5.4±0.8	4.4±0.5

*- reliably relative to the indicator in group 1

During the first week of the acute period, hourly analysis of CO in the circadian rhythm did not reveal significant changes (tab. 2). The age-related features of blood circulation adaptation at different times of the acute period of CSTBI were manifested in the fact that in the first week of intensive therapy, a significant difference was revealed in the morning (10.11 hours) in acrophase and at night (at 2.3, 7 hours) - in the bathyphase of the circadian rhythm. CO (fig. 4). Thus, in groups 2 and 3 at 10 a.m. CO was significantly less than in group 1 by 11% (0.6 l/min), $p < 0.05$, respectively. And at 11 o'clock in the morning, only patients of group 3 showed a significantly lower CO by 17% (by 0.9 l/min), $p<0.05$. In group 3, a significantly lower CO indicator was also found at night at 2 am by 14% (0.7 l per minute), at 3 am by 12% (0.6 l/min). At 7 o'clock in the morning, there was significantly less CO than in group 1 by 18% (by 0.9 l /min) in group 3.

During the second week, a significantly significant difference was found between the indicators in groups 1 and 3. Thus, the CO index of patients of group 3 was less than in group 1 at 10, 12, 13, 21, 6 a.m. by 18% (1 l per minute), 19% (0.9 l per minute), 18% (1 l/min), 18% (1 l/min), 13% (0.7 l/min) $p < 0.05$, respectively (fig. 5).

In the third week of the acute period of CSTBI, a significantly significant decrease in CO was revealed only in group 3 at 5 pm by 15% (by 0.8 liters per minute), $p<0.05$ (fig. 6). Thus, the most significant decrease in CO (tendency to hypodynamic type of hemodynamics) was observed in the first week in group 3 and to a lesser extent in group 2, remaining in group 3

during the third week, indicating the most significant tendency to develop circulatory failure in patients over 61 years old.

Circadian rhythms CO for 1-8 days, l/min

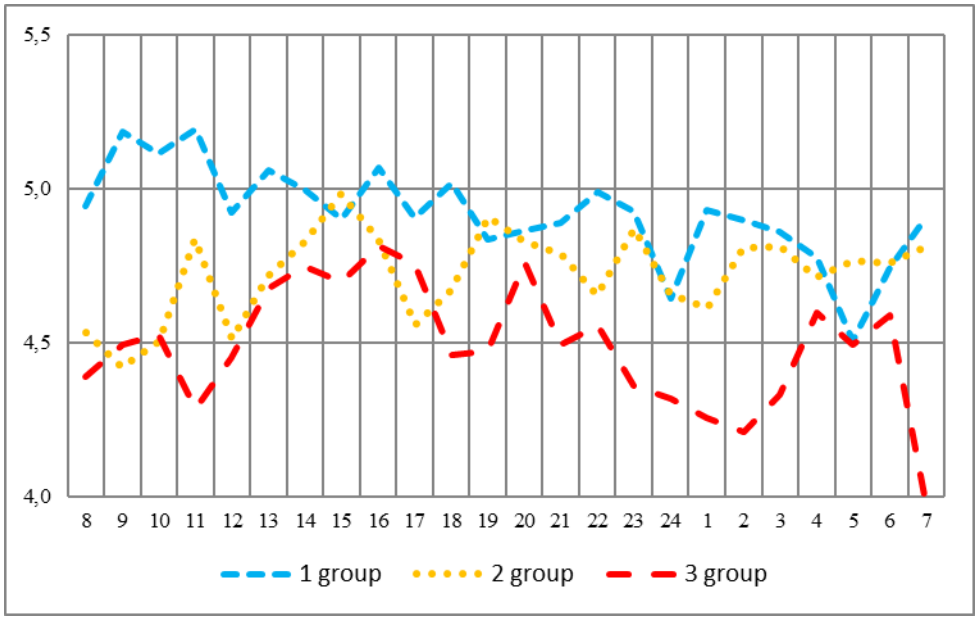

Fig.4

As seen in fig. 4, in the first week of intensive therapy in group 1, the daily CO wave was represented by ultradian low-amplitude 6-5 hour waves with a mesoorium of 4.9±0.1 liters per minute, acrophase at 9-11 hours. In group 2, the amplitude of fluctuations was somewhat higher with a meso-volume of 4.7±0.1 l/min. In group 3, ultradian waves are less constant with a period of fluctuations from 3 to 6 hours (at night), a mesor of 4.5±0.2 liters per minute. Thus, the mesor of the circadian rhythm CO in the first 8 days in group 3 was significantly less than the indicator of group 1 by 7% ($p<0.05$). That is, hemodynamics in group 3 from the very beginning differed in the instability of ultradian rhythms, significantly lower CO. The latter was due to age-related failure of adaptive mechanisms, mitochondrial insufficiency, which determines the energy-deficient state of the myocardium in elderly people.

Fig. 5 shows ultradian rhythms on days 9-17 of the acute period. Attention was drawn to a clearer picture of ultradian oscillations of almost the same amplitude in age groups. The age difference was expressed in the values of the mesor of the circadian rhythm CO on days 9-17. Thus, in group 1, CO mesor was 5.2±0.1 l/min, in group 2 - 4.9±0.1 l/min, in group 3 - 4.5±0.1 l/min. The CO mesor index in group 2 turned out to be less than in the first by 6%, in group 3 it was less than in the first by 13% ($p<0.05$, respectively). That is, throughout the entire acute

period, the index of the mesora of the circadian rhythm CO in group 3 remained lower than in the first two groups.

Circadian rhythms CO on days 9-17, l/min

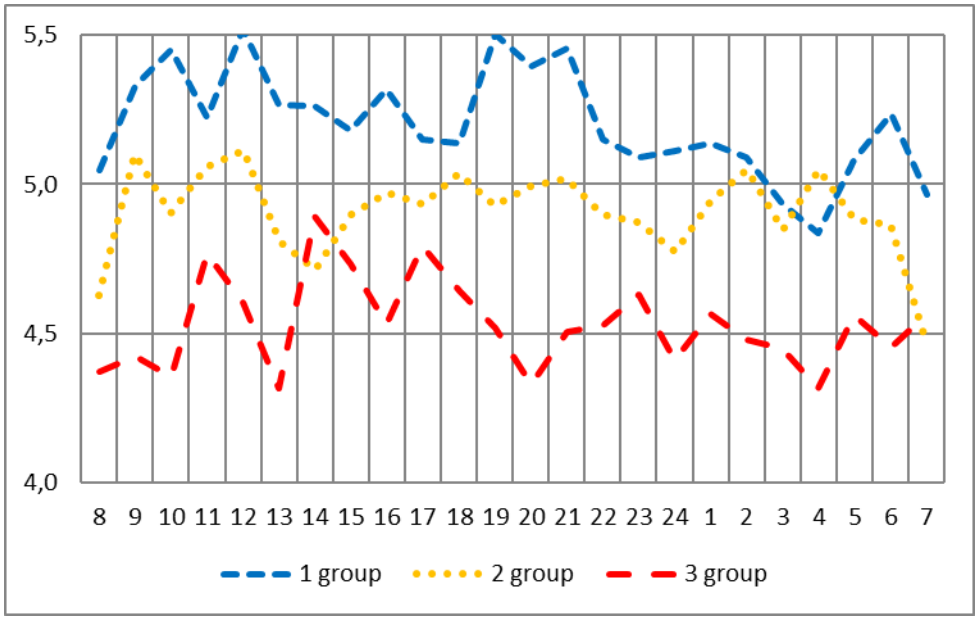

Fig.5

Circadian rhythms CO at 18-25 days l/min

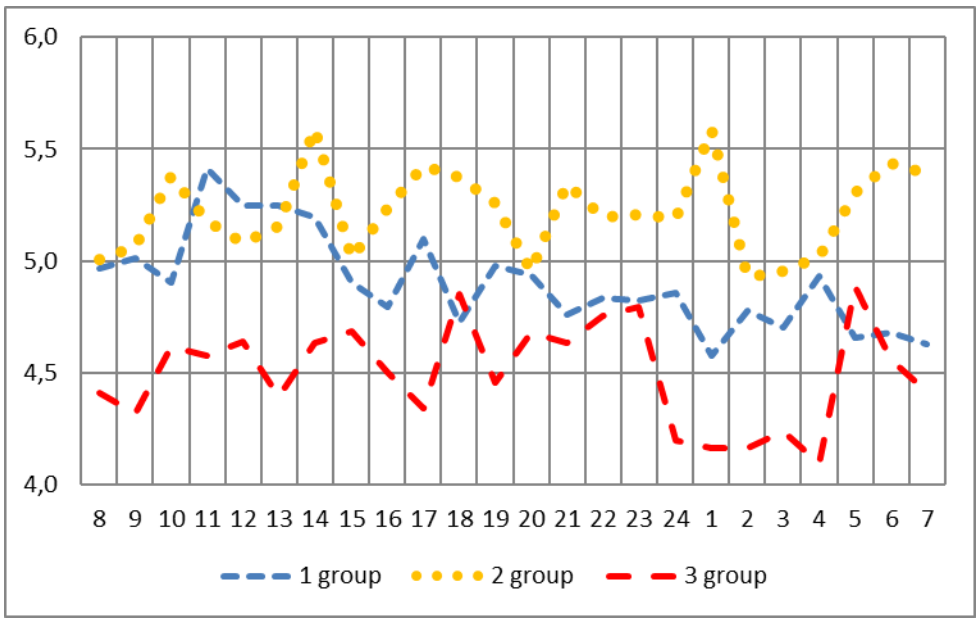

Fig. 6

As shown in Fig. 6, on the 18-25th day of the acute period, the average hourly CO values revealed the oscillatory nature of changes in the circadian birhythm of CO, presented in group 1 by 3-4 hour waves with an acrophase of the circadian rhythm at 11 a.m., a mesor of 4.9 ± 0, 2 l/min. Group 2 was dominated by 4x, 5-hour fluctuations with a relatively large value of the

mesor of the circadian rhythm CO (5.2±0.2 l/min). Ultradian waves in group 3 differed in a significantly lower level of the mesor of the circadian rhythm CO (4.5 ± 0.2 l/min) relative to group 2. That is, on the 18-25th day in persons over 61 years old, a tendency to the formation of a hypodynamic type of blood circulation due to heart failure was revealed.

Correlation links of CO

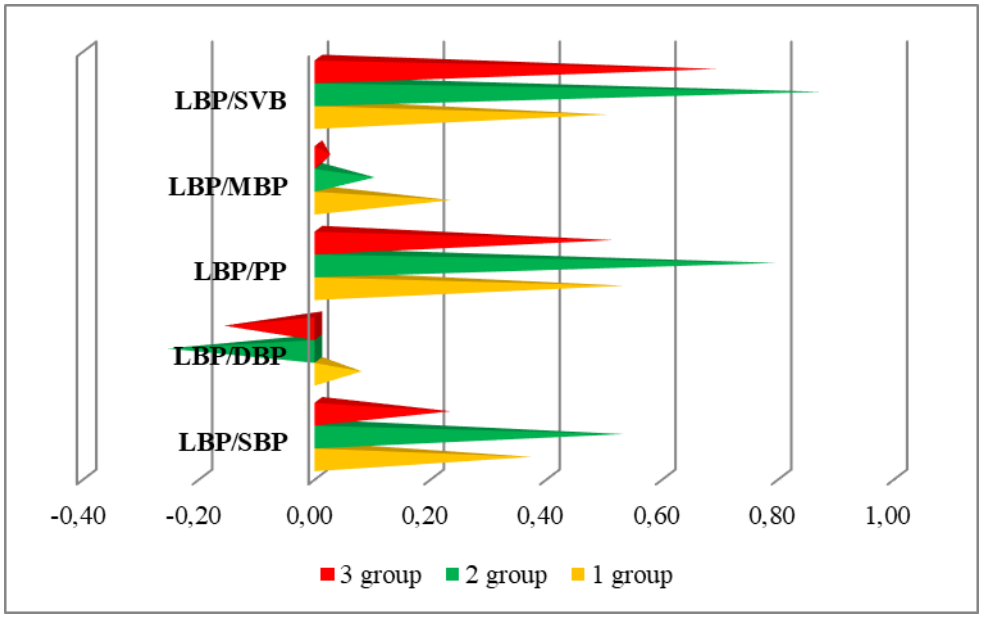

Fig.7

Strong direct correlations of CO and SV (0.87), CO and PBP (0.79) were found in group 2 of patients, a trend was revealed in groups 3 and 1 (fig. 7).

Duration and severity of CO circadian rhythm acrophase shifts in the acute period of CSTBI

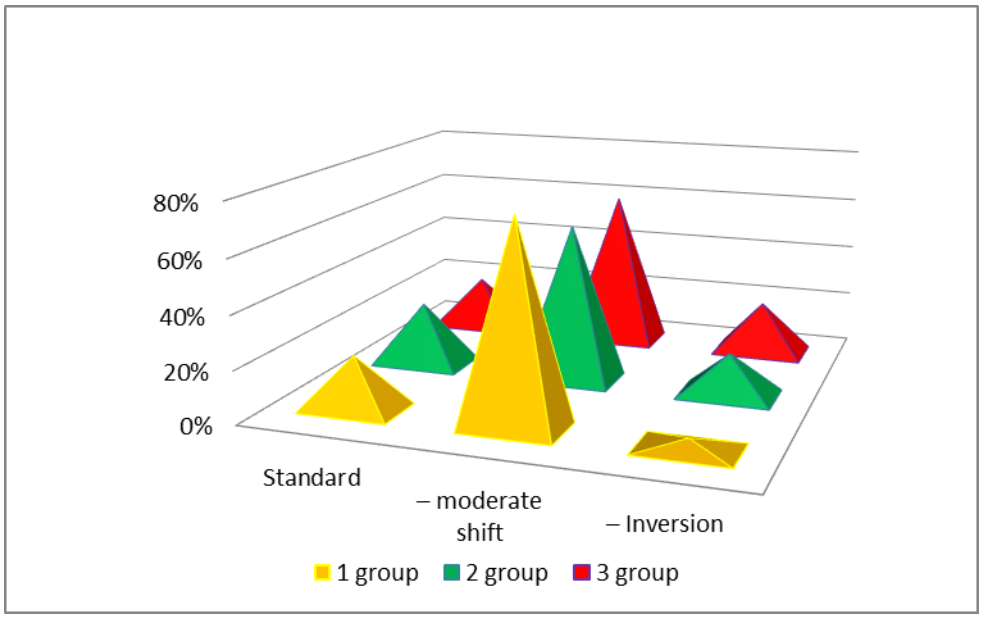

Fig.8

Evaluation of the duration and severity of acrophase shifts made it possible to ascertain the prevalence of a moderate degree of displacement of the CO circadian rhythm acrophase peak in all age groups (fig. 8).

Conclusions. The detected increase in the CO mesor in group 1 in the second week after injury was most likely associated with the need for maximum mobilization of blood circulation to ensure the necessary oxygenation of the brain under conditions of secondary pathogenetic mechanisms of CNS damage. Changes in the amplitude of the CO circadian rhythm occurred in waves with an unstable wavelength, deformation of the phase structure of weekly oscillations. Throughout the entire acute period, the CO mesor of the circadian rhythm in group 3 remained lower than in the first two groups. In patients over 40 years of age, the changes are due to the limited compensatory capabilities of the circulatory system in the adaptive restructuring of hemodynamics in the acute period of CSTBI. Strong direct correlations between CO and SV (0.87), CO and PBP (0.79) were found in group 2 of patients.

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