

Circadian rhythm of stroke volume of blood circulation in the acute period of concomitant severe traumatic brain injury

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Abstract.

In the acute period of combined severe traumatic brain injury (CSTBI), the stress reaction of hemodynamics manifested itself along with an increase in the amplitude, range of diurnal fluctuations, and displacement of the acrophase, as well as a restructuring of weekly biorhythms into 4-5 diurnal fluctuations in the mesor of the SV circadian rhythm. In all age groups, the SV circadian rhythm was an integral value consisting of 3-4-5 hour waves. In older age groups, cardiac output was significantly lower than in group 1 only in the morning hours of the first 8 days, which was probably due not only to more severe trauma in group 1, but also age-related limitations of adaptive stress restructuring of hemodynamics to severe trauma in patients over 41 years old. The most significant instability of myocardial function was found in injured people over 61 years of age.

Relevance. At present, according to the authors' data, mortality in combined STBI reaches 80%, and among survivors - up to 75% of victims remain with severe neurological defects. The positive dynamics of data on STBI treatment (a decrease in mortality in the United States and other Western countries with STBI to 30-40%), noted in the last decade, is largely associated with an increase in knowledge on the pathophysiology of acute STBI and the improvement of intensive treatment technologies during this period. Brain damage in STBI is determined not only by the primary impact at the moment of injury, but also by the action of various damaging factors during the next hours and days, the so-called secondary brain damage (SBD) factors. Secondary brain damage can be influenced by intracranial (intracranial hypertension, dislocation syndrome, cerebral vasospasm, seizures, intracranial infection) and extracranial factors. An increase in heart rate and a progressive decrease in BP and body temperature are prognostically

unfavorable signs. The most dangerous factors of SBD are arterial hypotension, hypoxia and intracranial hypertension (ICH) [1].

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 CNS [2,3]. In this regard, the study, assessment, timely correction of changes in cardiac output always remains one of the priority tasks of intensive therapy for CSTBI in the acute period.

Purpose of the work. To study the circadian rhythm of the stroke volume of blood circulation 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 craniocerebral trauma (CSTBI) who were admitted to the ICU of the neurosurgical department of RSCEMA in the first hours after an accident - 28, catatrauma of 2 patients were studied. Hourly monitoring of the SV (Stroke volume of blood) indicator was carried out by calculating hemodynamic parameters according to the formula: $SV = PBP \cdot 100 / AvBP$ in ml, where PBP (PP) is the pulse arterial pressure; AvBP (MBP) - average arterial pressure.

According to indications, 29 patients underwent invasive mechanical respiratory support (MRS) on admission. Mechanical respiratory support was started with artificial lung ventilation (ALV) for a short time, followed by transfer to SIMV. The severity of the condition was assessed using scoring methods for assessing the severity of concomitant injuries - the CRAMS scale, the severity of injuries using 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: MRS, 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. As shown in tab. 1, on the first day after injury, the SV mesor of the circadian rhythm did not differ from the normative data. In the first group, on day 3, a significant increase in the mesor of the SV circadian rhythm by 13% ($p < 0.05$) was revealed. In group 2, an increase in the mesor of the SV circadian rhythm was revealed on the 17th and 21st days by

21%, 31% ($p < 0.05$, respectively). In group 3, there were no significant deviations of the SV circadian rhythm mesor in the acute period of CSTBI.

Table1

Dynamics of the mesor of the circadian rhythm SVB in the acute period of CSTBI, ml

Days	Group 1	Group 2	Group 3
1	55.7±3.9	50.3±4.0	57.0±4.3
2	60.9±2.3	50.9±2.9	53.6±2.8
3	63.2±2.1*	52.1±2.2	59.5±2.5
4	59.7±2.8	54.8±4.1	50.9±3.8
5	56.6±2.4	54.8±3.6	54.6±3.3
6	58.1±3.6	54.9±2.6	54.8±2.5
7	59.7±3.8	54.3±3.6	62.0±4.1
8	54.4±3.0	57.3±2.9	57.0±4.1
9	56.9±2.7	57.5±2.5	56.8±4.4
10	55.3±2.4	54.4±3.5	54.1±5.0
11	56.3±3.0	57.3±4.0	51.4±2.9
12	56.8±2.2	57.3±3.2	60.3±5.6
13	61.4±6.3	56.7±2.5	56.4±5.3
14	55.6±2.9	54.8±2.9	55.0±3.4
15	56.3±3.0	55.6±3.2	59.8±3.9
16	55.7±4.0	55.0±2.9	54.3±3.7
17	58.3±6.1	61.0±4.1*	59.7±5.9
18	54.1±3.8	53.7±5.4	60.3±2.9
19	52.7±2.6	56.2±4.3	59.1±8.0
20	55.3±3.0	55.8±4.7	52.0±5.8
21	59.1±3.0	66.1±6.1*	52.9±6.2
22	59.4±3.8	57.2±4.7	58.9±9.5
23	59.1±2.0	58.5±4.8	60.5±6.4
24	60.0±3.1	58.9±4.2	59.0±5.1
25	55.1±3.2	59.7±5.1	57.4±6.4

*-reliably relative to the indicator on the first day

Changes in the mesor of the SV circadian rhythm during the acute period occurred in the form of oscillations with a wavelength of 5-4 days, which were repeated and were more ordered in group 1 (fig. 1). While in group 2, the first weekly fluctuation was replaced by 4-5 day cycles with an increase in amplitude by 17, 21 days. In group 3, more distinct 4-5 daytime fluctuations of the mesor of the SV circadian rhythm were observed. Thus, the stress reaction of hemodynamics noted in the acute period of CSTBI also included the restructuring of about-weekly biorhythms into 4-5 daily fluctuations in the mesor of the SV circadian rhythm.

Dynamics of the mesor of the circadian rhythm SVB in the acute period of CSTBI, ml

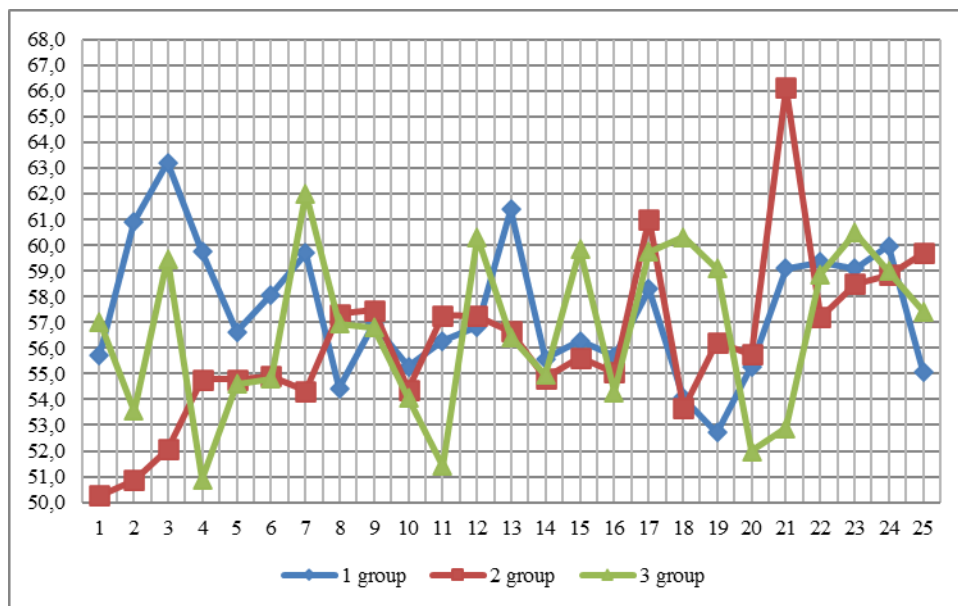


Fig.1

Table 2

Comparative assessment of hourly changes in SV in circadian rhythm at 1, 2, 3 weeks of the acute period of CSTBI, ml

Hours	SVB index 1-8 days after injury			SVB 9-17 days after injury			SVB 18-25 days after injury		
	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
8	59±2	52±4*	54±2*	55±2	54±2	55±3	56±3	56±1	58±8
9	61±3	50±5*	56±3	57±3	56±3	57±4	56±4	56±3	56±8
10	60±3	50±2*	57±5	58±3	55±4	54±6	56±4	58±7	60±6
11	60±3	55±3	53±4	57±3	57±3	57±3	61±7	59±3	60±4
12	58±5	52±1	54±3	57±2	58±5	56±5	60±3	57±5	61±7
13	59±5	53±3	56±5	58±3	56±2	55±5	59±4	60±3	58±4
14	58±5	54±4	58±6	57±2	55±3	58±6	59±2	62±6	60±7
15	57±3	53±4	57±4	56±2	56±2	60±5	58±4	55±7	59±5
16	59±3	54±4	58±5	56±2	56±2	55±9	56±3	58±5	57±4
17	59±4	52±3	57±5	56±3	57±3	58±5	59±2	61±5	55±5
18	60±4	53±5	55±5	56±5	58±3	57±6	55±3	59±4	55±9
19	58±4	55±3	55±3	60±5	57±4	55±5	57±5	59±4	57±5
20	57±3	53±5	58±6	58±5	57±4	54±6	56±6	54±4	55±6
21	58±2	55±3	55±4	59±4	58±1	54±4	56±5	61±3	58±5
22	59±3	54±3	56±4	56±3	56±4	56±4	55±2	58±6	60±6
23	58±5	54±5	55±4	55±3	56±4	58±5	56±3	58±7	62±7
24	56±3	55±4	56±3	57±3	56±3	57±7	57±4	59±3	54±4
1	59±4	55±3	56±5	57±2	58±3	58±5	57±4	62±6	53±8
2	60±5	55±4	55±4	55±3	59±3	58±4	57±4	56±6	52±5
3	60±2	55±3	57±3	55±4	56±4	56±5	56±3	57±4	53±4
4	59±3	54±3	60±5	55±3	58±4	55±5	58±3	57±7	51±8
5	55±4	55±2	59±5	57±3	57±4	57±5	56±5	57±5	61±6
6	58±3	55±4	57±4	58±3	57±3	56±2	55±3	60±7	57±5
7	58±3	55±5	52±5	55±2	54±3	57±6	55±4	58±8	55±7

*-reliably relative to the indicator in group 1

As shown in Table 2, in the first week of intensive care after trauma, the SV indicator in group 2 at 8.9.10 a.m. was lower than in the first by 11%, 9%, 16% ($p < 0.05$, respectively). In group 3 patients SV was less than in group 1 at 8 hours by 8% ($p < 0.05$). Thus, only in the morning hours of the first 8 days in older age groups, cardiac output was significantly lower than in group 1,

which was probably due not only to more severe trauma in group 1, but also age-related limitations of adaptive stress restructuring of hemodynamics to severe trauma. in patients over 41 years old.

Hourly SVB dynamics in circadian rhythm in group 1, ml

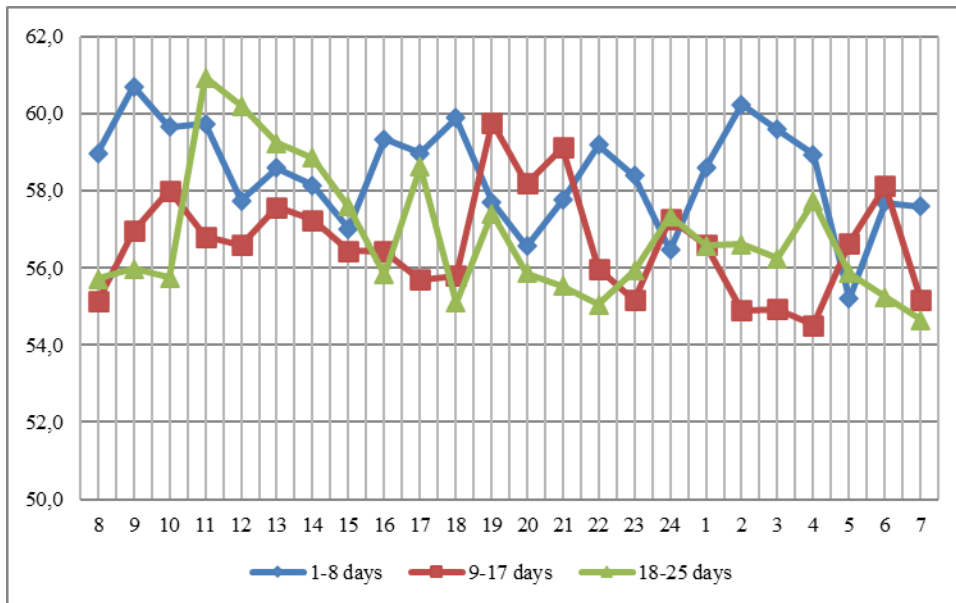


Fig.2

The circadian rhythm of SV in group 1 consisted of three to 4 hour components, and at night, 5 hour periods of fluctuations in the indicator in 1 week of treatment. In the second week, the wavelength increased to 4-6 hours. In the third week of the acute period, three, four, six-hour fluctuations were observed with a maximum amplitude of 11 hours (fig. 2).

In group 2 (fig. 3), in the first week of the acute period of CSTBI, fluctuations were low-amplitude with a tendency to a decrease in SV, on days 9-17 more ordered 4-hour waves prevailed. On days 18-25, with the same oscillation period, there was a tendency to an increase in the amplitude of the four hourly waves.

Hourly SVB dynamics in circadian rhythm in group 2, ml

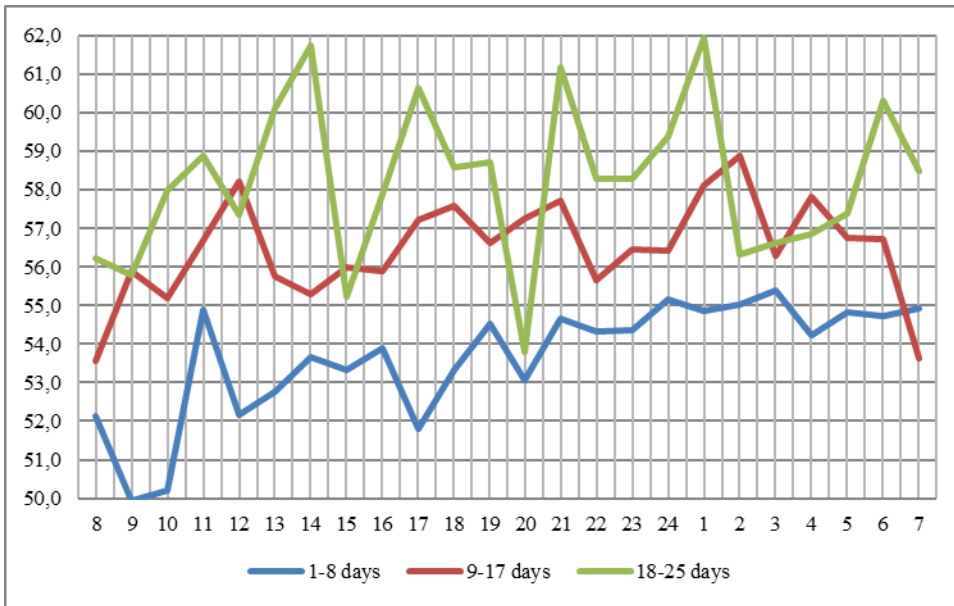


Fig.3

Hourly SVB dynamics in circadian rhythm in group 3, ml

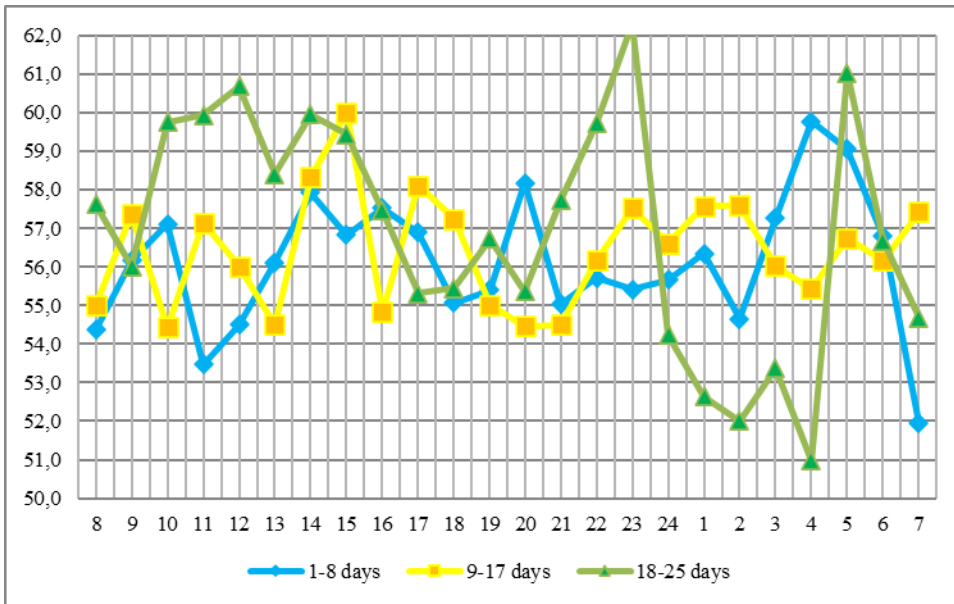


Fig.4

In group 3 (fig. 4) in 1 week, the daily wave consisted of 3-, 4-hour waves, at 2 weeks there was a tendency to an increase in the amplitude. An even greater tendency to increase the amplitude of

5-4 hour waves was noted on the 18-25th day. Thus, in all age groups, the SV circadian rhythm was an integral value, consisting of 3-4-5 hour waves. The amplitudes of these SV ultradianic rhythms, apparently, depend on both the severity of the injury and, perhaps to an even greater extent, on the reserve capacity of the body for the implementation of the stress response in the process of adaptation to new conditions, caused by the severity of the injury and the volume of intensive therapy CSTBI.

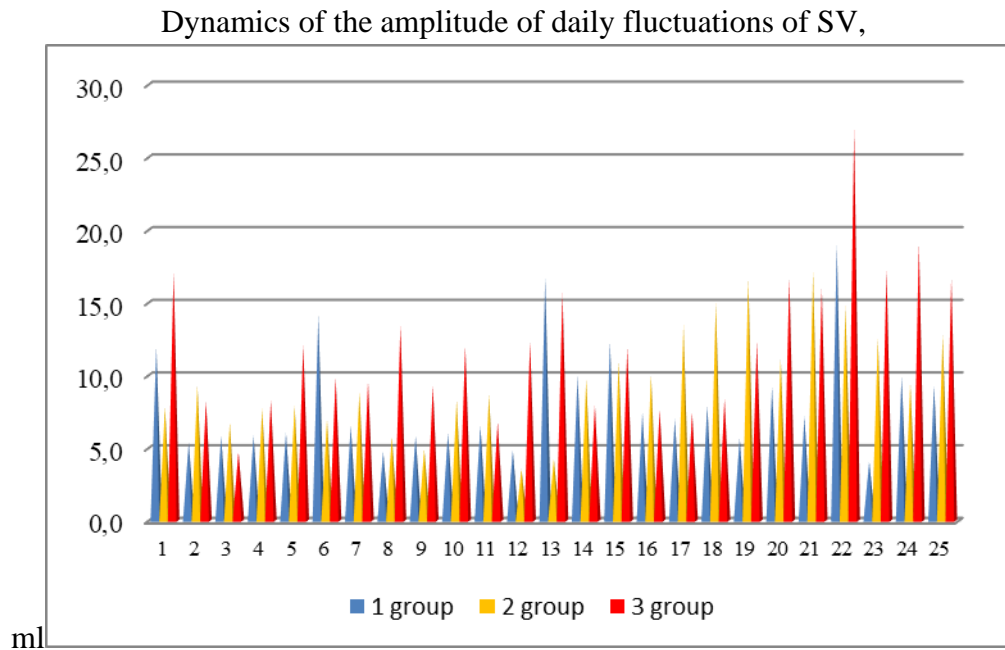


Fig.5

The change in the amplitude of diurnal SV fluctuations occurred in waves with maximum values in group 1 at 1,6,13,22 days. In group 2, the most significant increase in the amplitude of diurnal SV fluctuations was on days 17,18,19,21,22. In group 3, the corresponding changes were recorded at 1,5,8,13,22,23,24,25 days. In groups 2 and 3, an increase in the amplitude of the SV circadian rhythm after 17 days was revealed (fig. 5).

Change in the daily range of SV fluctuations, ml

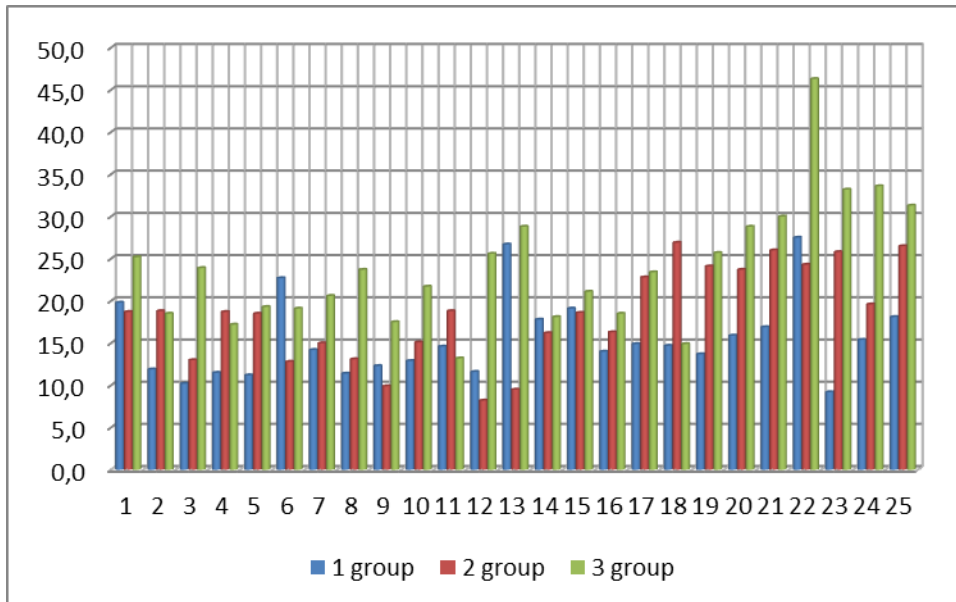


Fig.6

Attention was drawn to the significant prevalence of daily changes in SV in group 3 patients throughout the observation period (fig. 6). The latter characterizes the most significant instability of myocardial function in injured people over 61 years of age.

Correlation links for a period of 25 days

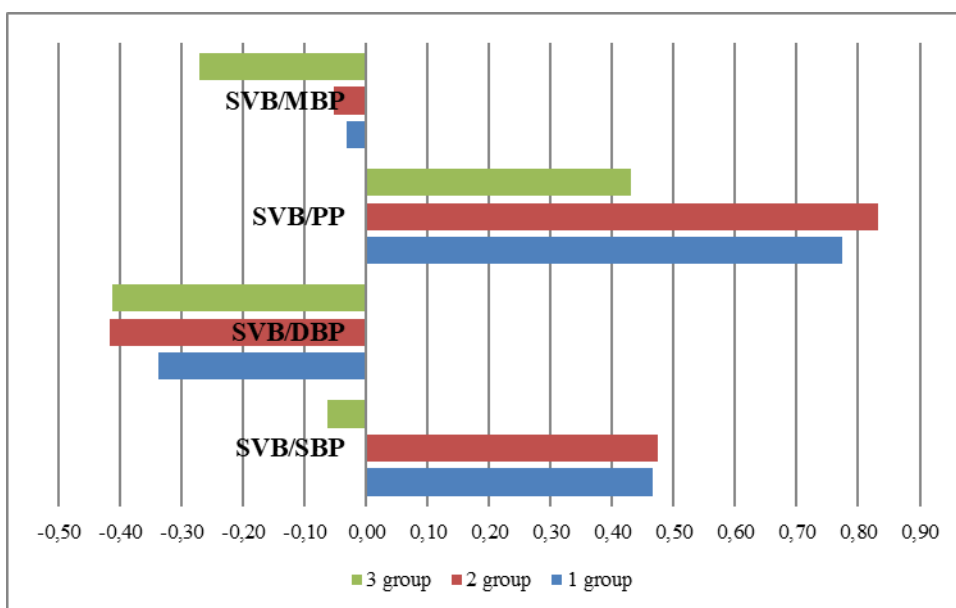


Fig.7

A strong direct relationship between SV and PBP was found in traumatized patients of groups 1 and 2 (0.77; 0.83, respectively). While in group 3, the correlation between SV and PBP turned out to be significantly less (0.43) (fig. 7). The study of SV correlations in the peri-weekly rhythm did not reveal any significant differences depending on the duration of intensive therapy.

Conclusion. In the acute period of CSTBI, the stress reaction of hemodynamics was also manifested by the restructuring of about-weekly biorhythms into 4-5 daily fluctuations in the mesor of the SV circadian rhythm. In all age groups, the SV circadian rhythm was an integral value consisting of 3-4-5 hour waves. In older age groups, cardiac output was significantly lower than in group 1 only in the morning hours of the first 8 days, which was probably due not only to more severe trauma in group 1, but also age-related limitations of adaptive stress restructuring of hemodynamics to severe trauma in older patients. 41 years old. The most significant instability of myocardial function was found in injured people over 61 years of age.

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