Phase analysis of adaptive changes in the circadian rhythm of systolic blood pressure in concomitant severe traumatic brain injury

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Abstract

With the most severe injuries in patients of group 1 (50.2±5.6 points), the severity of the condition and impaired consciousness on the first day after combined severe traumatic brain injury did not differ significantly from those in groups 2 and 3. Changes in the circadian rhythm of systolic blood pressure indicated a better state of compensatory defense mechanisms of hemodynamics at the age of up to 40 years.

Keywords: circadian rhythm, systolic blood pressure, combined severe traumatic brain injury

Relevance. The factors aggravating combined TBI from bone fractures are their shockogenicity, blood loss, increased systemic inflammatory response and coagulopathy, which leads to secondary damage to the brain and lungs, increases the incidence of embolic, pulmonary complications and multiple organ failure. Bone fractures and soft tissue injuries in polytrauma become significant sources of factors of systemic inflammatory response and oxidative stress, which penetrate the blood-brain barrier, modulate immune responses and have a cytotoxic effect in the brain, exacerbating the severity of TBI. Early operations of osteosynthesis, being an operational trauma, can provoke the progression of the inflammatory reaction, the development of systemic complications and multiple organ failure, that is, cause the effect of the "second blow". In polytrauma with TBI, early osteosynthesis for fractures of the femur, unstable fractures of the pelvic bones can reduce the incidence of complications (ARDS, pneumonia, pulmonary embolism, fat embolism syndrome, sepsis and multiple organ failure), reduce mortality and improve anatomical and functional results of treatment. But, being an operational trauma, osteosynthesis can, on the contrary, provoke the development of dangerous systemic complications, that is, cause a "second shock", and excessive intraoperative infusion therapy can induce neuronal apoptosis, ischemic stroke and cerebral edema in patients with TBI concomitant.

aggravate cardiorespiratory disorders and worsen neurological outcome (6). The danger of hypertension for predicting a person's life in a relatively stable state has nothing to do with the significance of the increase in BP in the acute period of severe TBI. In the latter case, the increase in BP is aimed at increasing the perfusion of the affected brain. The authors believe that it is not necessary and unsafe, from the point of view of providing cerebral perfusion, to administer antihypertensive drugs with an increase in BP, no matter what values it reaches. A decrease in systemic BP under the influence of antihypertensive drugs led to a decrease in cerebral perfusion pressure with unchanged ICP values. The decrease in CPP created the risk of hypoperfusion and cerebral ischemia. The researchers did not observe a decrease in ICP with a decrease in blood pressure. This fact makes it possible to exclude such a hypothetical mechanism as a decrease in vasogenic cerebral edema with a decrease in elevated BP (Lund-concept). Cerebral edema with a decrease in BP not only does not decrease, but increases (1-5.7). The ambiguous attitude to changes in blood pressure in STBI stimulated us to study the circadian rhythm of SBP in the acute period of concomitant severe traumatic brain injury (CSTBI).

Purpose of the work: to study and assess the adaptive changes in the phase structures of the circadian rhythm of systolic blood pressure in combined severe craniocerebral trauma.

Material and research methods. The indicators of a comprehensive examination of 30 patients with concomitant severe craniocerebral trauma (STBI) who were admitted to the ICU of the RSCEMA neurosurgical department in the first hours after an accident - 28, catatrauma of 2 patients were studied. In all patients, head injury was accompanied by various associated injuries. In particular, the victims had fractures of the limbs and pelvis, damage to the bones of the facial skeleton, damage to the chest, spine and spinal cord, damage to the abdominal cavity and retroperitoneal organs (ruptures of the spleen, liver, kidney capsules, etc.). According to the testimony of 29 patients, on admission, invasive mechanical respiratory support (MRS) was started. Monitoring was carried out by complex hourly registration of parameters of body temperature, hemodynamics, respiration. Mechanical respiratory support began with mechanical ventilation (ALV) for a short time, followed by transfer to SIMV (tab. 2). The severity of the condition was assessed by 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 (tab. 1). 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, to the extent possible, early correction, syndromic, symptomatic therapy.

Results and discussion

Table 1.

Assessment of the severity of the condition by age

Parameters	Group 1	Group 2	Group 3
age in years	29.5±4.3	51.6±4.8	72.3±9.1
CRAMS, points	4.5±0.6	4.4±0.8	4.8±0.6
ISS, points	50.2±5.6	43.3±7.1	46.2±9.1
GS, points	7.3±0.5	7.9±1.3	7.8±1.5

It was found that with the most severe injuries in patients of group 1 (50.2±5.6 points), the severity of the condition and impaired consciousness did not differ significantly from those in groups 2 and 3.

Table 2.

Respiratory support depending on the severity of the condition

Parameters	Group 1	Group 2	Group 3
age in years	29.5±4.3	51.6±4.8	72.3±9.1
CRAMS, points	4.5±0.6	3.6±1.0	4.8±0.6
ISS, points	50.2±5.6	27.8±20.4	46.2±9.1
GS, points	7.3±0.5	7.3±3.3	7.8±1.5
SIMV, number of days	8.6±3.9	11.8±5.5	17.8±16.9
BIPAP, number of days	4.5±5.3	3.2±1.7	1.4±1.7
CPAP, number of days	5.8±7.1	7.0±0.6	7.6±7.5
Number of mode switching	3.5±2.5	2.5±0.8	3.0±0.8
MRS, duration of respiratory support in days	14.7±8.2	16.5±5.1	26.8±16.5
Number of days in ICU	21.3±11.8	23.0±6.5	36.0±23.0
Total number of days in hospital	25.4±13.1	26.7±9.9	46.5±21.8

The longest duration of intensive care in ICU, prolonged MRS in SIMV mode, the duration of inpatient treatment was observed in group 3 (tab. 2).

Table 3.

Dynamics of the mesor of the circadian rhythm of systolic blood pressure in the acute period of combined severe trauma

days	Group 1	Group 2	Group 3
1	128.2±4.0	119.8±11.9	134.7±9.1
2	125.0±2.4	129.3±7.0	125.9±4.4
3	128.0±2.7	126.0±5.6	126.7±3.4
4	131.5±2.5	134.1±5.6	127.7±3.3

5	124.6±2.6	133.9±5.3	133.4±4.9
6	129.4±2.9	133.4±3.7	133.4±2.9
7	130.7±2.2	134.9±3.2	128.7±2.7
8	127.8±2.6	129.5±4.3	128.6±6.5
9	127.2±2.6	126.6±3.9	128.4±4.7
10	130.2±3.3	124.9±3.4	129.0±5.4
11	124.1±2.1	126.7±3.5	128.2±4.5
12	124.5±2.6	124.4±4.6	129.0±4.3
13	123.6±4.2	121.2±4.6	116.2±4.2*
14	122.5±3.6	117.8±4.0	125.2±4.9
15	121.0±2.4	125.7±4.1	129.2±4.1
16	121.8±3.7	122.6±5.2	126.4±4.3
17	119.8±3.5*	130.5±6.2	131.6±3.4
18	121.0±4.0	130.8±5.3	119.4±4.2*
19	114.1±4.1*	127.4±3.2	121.1±4.4
20	116.5±2.8*	134.7±8.8	123.7±3.6
21	125.6±3.5	139.9±9.0	127.6±5.5
22	127.8±4.7	139.9±9.0	128.5±7.0
23	123.3±5.4	133.8±5.3	123.0±7.8
24	123.7±5.4	137.4±6.1	130.4±6.9
25	117.5±3.9*	134.5±5.5	130.2±6.1

*-deviations are reliable relative to the indicator in 1 day

As presented in tab. 3, on the first day of the combined STBI, age-specific features of the mesor of the circadian rhythm SBP were not revealed. On the following days, in group 1, there was a decrease in the indicator by 19 (6%), 20 (11%), 25 days (8%). In group 2, there were no significant differences due to the large scatter of data on day 1. In group 3, the mesors of the circadian rhythm SBP decreased by 13 (5%), 18 days (11%).

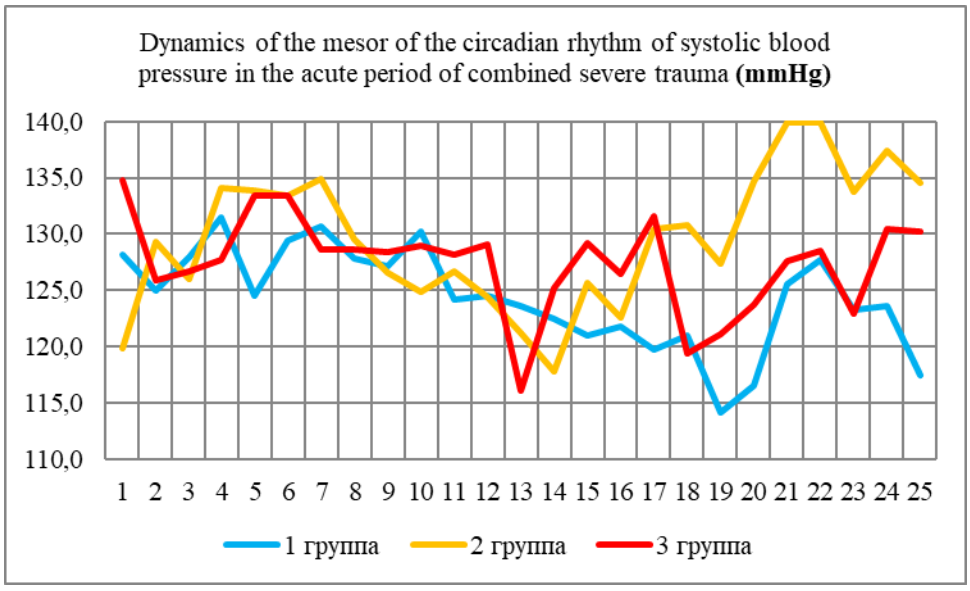

Fig.1

As shown in fig. 1, changes in the SBP circadian rhythm mesor in the acute period of CSTBI occurred in waves with periods of fluctuations in group 1 of 4 days, when the detected decrease in SBP mesor on days 19 and 25 represented the SBP value in the bathyphase of four-day biorhythms. In group 2, the wavelike changes in the SBP mesor fell into two: 14- and 11-day

periods of fluctuations with the acrophase peak on days 7 and 21, which in turn consisted of several four-day phases. In group 3, wave-like fluctuations fit into 5 day periods with a comparatively smaller amplitude of each wave. The minimum values of SBP mesor in bathiphase in group 3 were detected on days 13 and 18, amounting to (116.2±4.2 mmHg and 119.4±4.2 mmHg, respectively).

Thus, the adaptive reaction of hemodynamics in the acute period of CSTBI occurred by changing not only the structure of the circadian rhythm, but also by restructuring the circadian biorhythm in about four to five days with a slightly reduced amplitude of fluctuations in group 3 of patients.

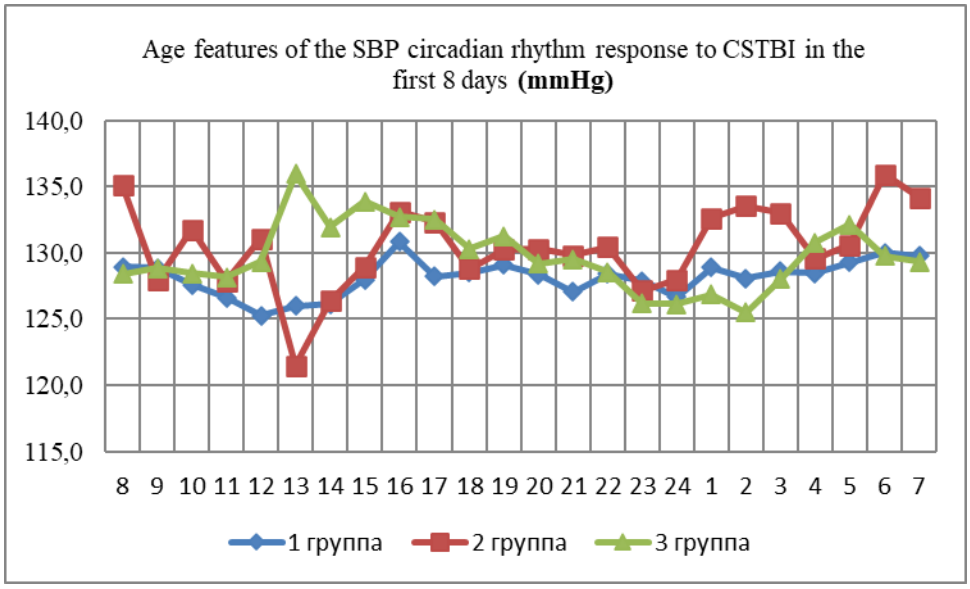

Fig.2

An attempt to identify the distinctive features depending on the time elapsed after the injury made it possible to differentiate the changes in the SBP indicators in the circadian rhythm inherent in each subsequent week after the injury. So, in patients of group 1 in the first 8 days (Fig. 2) during the day, the SBP indicator fluctuated within 125 mmHg at 12 o'clock with a rise to 132 mmHg. at 16 o'clock. On the next day from 9 to 17 (fig. 3), daily changes in SBP were low-amplitude waves within 123 - 127 mmHg. In the third week of observation (from 18 to 25 days), the amplitude of daily fluctuations increased (from 118 mmHg at 14 o'clock to 125 mmHg at 18 o'clock). Thus, the average value of the amplitude of the circadian rhythm SBP in the first 8 days was 6, on days 9-17 - 4, 18-25 days 6 mmHg. In group 2, the mean value of the amplitude of daily SBP changes in the first 8 days was 15 mmHg (121 mmHg at 13 o'clock, 136 mmHg at 6 a.m.). The bathiphase projection at 13 o'clock characterizes the inversion of the circadian rhythm of SBP in group 2 in the first 8 days of treatment. From 9 to 17 days in patients of group 2, daily fluctuations were characterized by low-amplitude wave-like SBP changes with an amplitude of 5 mmHg. In fig. 4, draws attention to the increase in the amplitude (up to 18

mmHg) of daily changes in the SBP index with bathiphase at 11 o'clock and the maximum SBP value at 17 o'clock on the 18-25 day. The revealed changes in the circadian rhythm correspond to the inversion of the circadian rhythm of SBP in age 2 patients at the third week of observation.

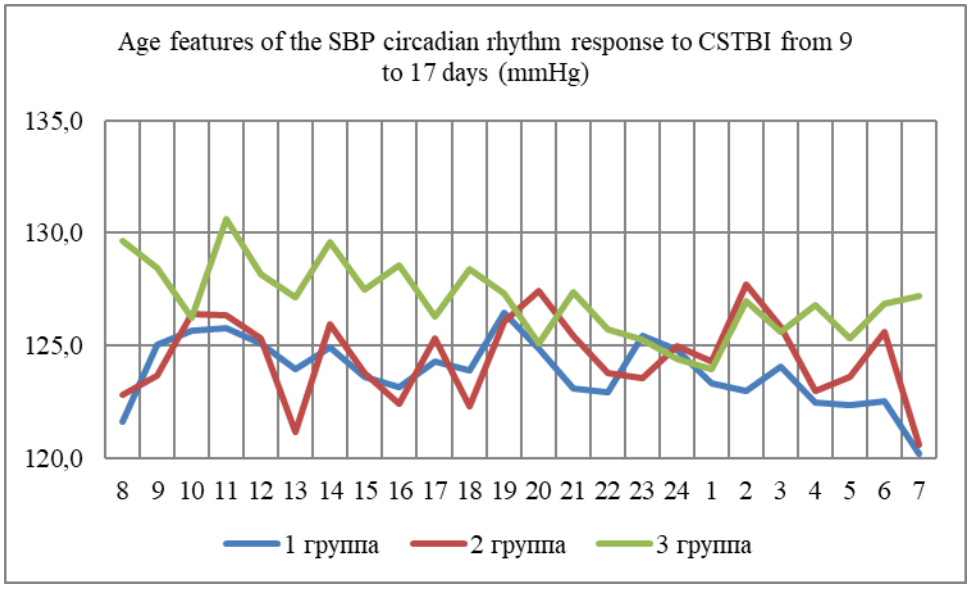

Fig.3

Fluctuations of SBP in group 3 from 1 to 8 days of the acute period of CSTBI were manifested by the projection of acrophase 137 mmHg at 13 o'clock, while the amplitude of daily fluctuations in SBP was 12 mmHg. From 9 to 17 days, the daily fluctuations of patients over 61 years old also differed in low amplitude (4 mmHg), on the next 18-25 days the amplitude of fluctuations increased to 8 mmHg, the acrophase projection was detected at 24 hours, indicating an inversion of the daily rhythm of fluctuations of SBP in group 3 (fig. 4). Thus, the circadian rhythm of SBP in the process of hemodynamic adaptation under conditions of severe stress caused by CSTBI took an active part, being rearranged by a moderate displacement of the acrophase projection in groups 2 and 3 (in the first 8 days and 18-25 days), bathyphase in group 2 on 9- 17 days, decreasing (in all three groups on days 9-17) and increasing the amplitude of daily fluctuations in group 2 on days 17-25 after combined STBI.

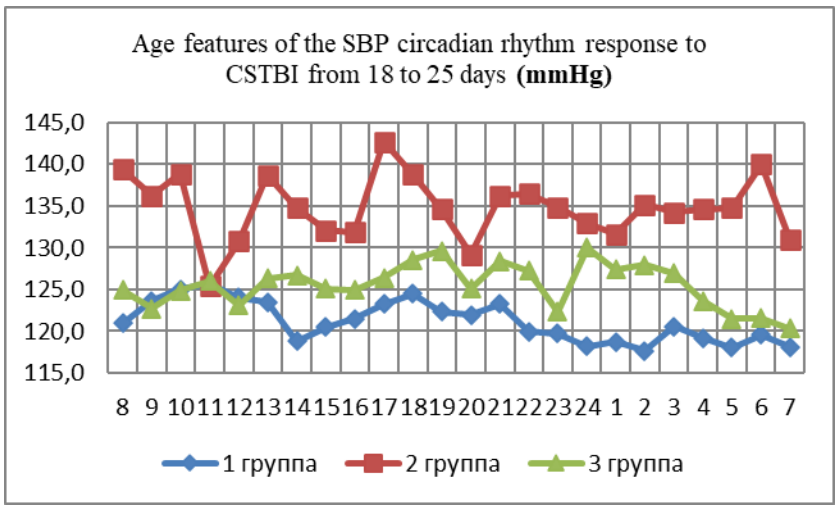

Fig.4

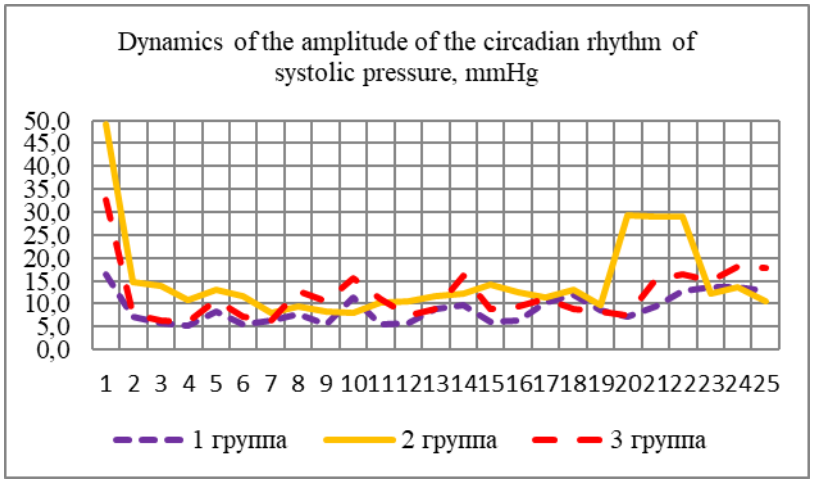

Fig.5

A characteristic common feature was the highest value of the amplitude of the circadian rhythm SBP on day 1, amounting to 16 mmHg in group 1, 50 mmHg in 2, 32 mmHg in group 3. The stress reaction of hemodynamics to CSTBI was manifested by an increase in SBP drops within 1 day, characterizing the instability of myocardial contractility caused not only by stress response to trauma, but primarily by damage to brain structures, an inflammatory reaction not only of brain tissue, but also bone fractures and soft tissue injuries. in polytrauma - sources of factors of systemic inflammatory response and oxidative stress, which penetrated the blood-brain barrier, modulating immune responses and exerting a cytotoxic effect in the brain, aggravating the severity of TBI. The most pronounced changes in cardiac output were found in the 2nd age group (fig. 5). Almost synchronous changes in the dynamics of the amplitude of the daily fluctuation of the circadian rhythm SBP (50 mmHg) with the maximum drop in the indicator in 1 day were revealed in patients of group 2 (85 mmHg) (fig. 6). Attention is drawn to the second

wave of increased daily SBP drops on days 20-22 in group 2 of patients. The repeated SBP stress response may have had a compensatory significance in response to hypoxia caused by repeated attempts to transfer patients to spontaneous breathing. The smallest indicators of the daily SBP fluctuations in 1 day in patients of group 1 with the most pronounced trauma injuries indicate a better state of anti-stress protective mechanisms (in particular, the parasympathetic autonomic system, inhibitory activity of CNS neurotransmitters, and others) at the age of up to 40 years.

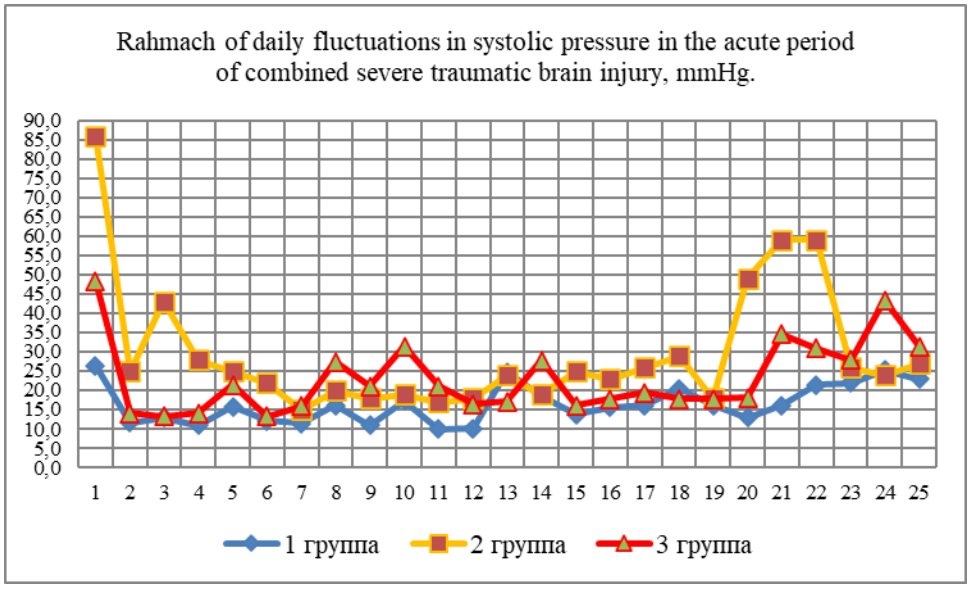

Fig.6

Conclusion. With the most pronounced CSTBI lesions in patients of group 1 relative to age groups 2 and 3, changes in the circadian rhythm of systolic blood pressure indicated a better state of compensatory defense mechanisms of hemodynamics at the age of up to 40 years.

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