DOI 10.26724/2079-8334-2022-2-80-162-168
UDC 616.857: [616.831-073.432.19:616.711.1-073.7]

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VEGETATIVE SYSTEM PATHOGENETIC ROLE IN CHRONIC BRAIN ISCHEMIA, CEREBRAL HEMODYNAMICS DISORDERS AND AUTONOMOUS DYSREGULATION

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The autonomic system plays a leading pathophysiological role in the majority of pathological syndromes development. Its disturbances have an important role in cerebrovascular disorders both formation and clinical manifestation. The purpose of the present study is to assess the state of the autonomic system, cerebral hemodynamics and cerebrovascular reactivity in patients with chronic cerebral ischemia in the stages of compensation and subcompensation. 81 patients with chronic cerebral ischemia were treated and intensively examined to focus on autonomic dysfunctions evaluation and their clinical expression determination. Cephalalgia was the leading syndrome in patients with chronic cerebral ischemia. Autonomic dysfunctions were registered in all examined persons. Sympathetic skin evoked potentials registration showed central nervous system pathological ischemic processes manifestation worsening, sympathetic skin evoked potentials latency increasing, the second phase of sympathetic skin evoked potentials amplitude decreasing that indicated limbic-reticular complex dysfunction, ergotropic processes depletion and the autonomic functions regulation system instability. The ultrasound data confirm that both autonomic system and vasomotor centers dysfunction is the leading mechanism for cerebral ischemia onset and progression, which resulted in cerebral vessels impaired tone and reactivity, neurovasoactive substances accumulatuion, which contributes to the restructuring of the metabolic circuit of autoregulation with a change in the homeostatic range. The data obtained should contribute to chronic cerebral ischemia treatment optimization taking into account the autonomic system individual characteristics of the patients and their cerebral blood flow reactivity.

Key words: autonomic system, chronic cerebral ischemia, hemodynamic, cerebrovascular reactivity, sympathetic skin evoked potentials

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ПАТОГЕНЕТИЧНА РОЛЬ ВЕГЕТАТИВНОЇ СИСТЕМИ ПРИ ФОРМУВАННІ ХРОНІЧНОЇ ІШЕМІЇ МОЗКУ, ПОРУШЕНЬ ЦЕРЕБРАЛЬНОЇ ГЕМОДИНАМІКИ ТА АВТОНОМНОЇ РЕГУЛЯЦІЇ

Вегетативній системі притаманна провідна патофізіологічна роль у розвитку більшості патологічних синдромів. Її порушення відіграють важливу роль у формуванні та клінічному прояві цереброваскулярних порушень. Метою даного дослідження є оцінка стану вегетативної системи, церебральної гемодинаміки та цереброваскулярної реактивності у хворих на хронічну ішемію головного мозку в стадіях компенсації та субкомпенсації. 81 пацієнт із хронічною ішемією мозку був пролікований та інтенсивно обстежений з метою зосередження уваги на оцінці вегетативних дисфункцій та визначенні їх клінічної вираженості. Цефалалгія була провідним синдромом у пацієнтів з хронічною ішемією головного мозку. У всіх обстежених зареєстровані вегетативні дисфункції. Реєстрація симпатичних викликаних потенціалів шкіри виявила погіршення прояву патологічних ішемічних процесів у центральній нервовій системі, збільшення їх латентних періодів та зменшення їх амплітуди, що вказувало на дисфункцію лімбіко-ретикулярного комплексу, виснаження ерготропних процесів, регуляції функцій системи та вегетативної працездатності. Дані ультразвукового дослідження підтверджують, що дисфункція вегетативної системи та вазомоторних центрів є провідним механізмом виникнення і прогресування ішемії головного мозку, що призвело до порушення тонусу і реактивності судин головного мозку, накопичення нейровазоактивних речовин та спричиняє перебудову метаболічного контуру ауторегуляції. зміну гомеостатичного діапазону. Отримані дані повинні сприяти оптимізації лікування хронічної ішемії головного мозку з урахуванням індивідуальних особливостей вегетативної системи пацієнтів та реактивності їх мозкового кровотоку.

Ключові слова: вегетативна система, хронічна ішемія мозку, гемодинаміка, цереброваскулярна реактивність, симпатичні викликані потенціали шкіри

The study is a fragment of the research project "To investigate the chronic convulsive syndrome pathogenetic mechanisms on the model of pharmacological kindling and to study the efficacy of its complex pathogenetical correction by anticonvulsant mechanisms activation", state registration No 0122U000081

In modern conditions, we see constantly increasing prevalence and incidence of cerebrovascular disorders (CVD), especially chronic progressive forms. It should be noted that many manifestations of CVD quite early form clinical syndromes, which further lead to social maladaptation, disability, etc. [2, 7, 10, 13].

It is known that in almost all pathological syndromes and diseases autonomic disorders are involved in the development or presented in the clinical picture, especially in CVD [4, 6]. Disturbance of the integrative activity of the CNS in autonomic dysfunction are manifested by disorganization of all organ systems [1, 8]. It is of particular importance that the regulation of the vascular tone is a complex neurohumoral and metabolic process. Its violations underlie CVD and other so-called "regulatory diseases" [5].

There is no doubt about the crucial role of the autonomic nervous system (ANS) in the occurrence, mechanisms of development and progression of ischemic lesions of the CNS [3, 5]. Thus, cerebral angiodystonic manifestations are obligate pathology in chronic cerebral ischemia (CCI).

The purpose of the study was to assess the state of the autonomic nervous system, cerebral hemodynamics and cerebrovascular reactivity in patients with chronic cerebral ischemia in the stages of compensation and subcompensation.

Materials and methods. 81 patients with CCI (according to International Disease Classification No10) aged from 40 to 65 years were included into the clinical observation. These patients were randomized in to 2 clinical groups. The group 1 was consisted out of 38 patients with CCI in the stage of compensation; the group 2 was consisted out of 43 patients with CCI in the stage of subcompensation. Among all examined patients we choose two leading CCI etiological factors: cerebral atherosclerosis (CA) in 38 patients and hypertension (HT) in 43 patients. We used an original questionnaire for autonomic dysfunctions detection. Autonomic tone (AT) expression was checked using the original "24 stigmas" table [5].

Headache intensity was investigated with the help of visual analogue scale (VAS). Sympathetic skin evoked potentials (SSEP) were recorded according to the method [12]. The following SSEP parameters which were shown to reflect the AT and CNS ischemic lesion expressions were taken into consideration: the second phase amplitude (A_2) and latency duration [4].

Cerebral hemodynamic using Doppler ultrasonography and Transcranial Doppler (ultrasonic scaner "Ultima-PA", Ukraine) was investigated in all the examined patients with CCI. Vascular regulation metabolic circuit was investigated with the help of hypercapnic load with the calculation of the reactivity index (RI^+), the index of reactivity to hyperventilation load (RI^-) and vasomotor reactivity index (VMRI). Myogenic contour was investigated after nitroglycerin (0.05 mg, sublingual intake) with the help myogenic reactivity coefficient (MRC).

To study the neurogenic mechanism of autoregulation the patients were undergone to an orthostatic test. We used an original orthostatic reactivity coefficient (ORC) to assess the cerebral hemodynamic in these conditions [7].

The data obtained were calculated statistically using one-way variant ANOVA parametric criterion accompanied by a post-hoc Newman-Keuls test. The minimum statistical probability was determined at $p<0.05$.

Results of the study and their discussion. The presence of headaches was noted in 76.5 % of patients. Moreover, regardless of the etiological cause of CCI, they were the leading syndromes: 79.1 % (HT) versus 73.0 % (CA), $P<0.05$.

The maximum frequency of cephalalgia was observed in persons in the compensated stage of CCI (group 1) – 92.1 % versus 62.8 % in the subcompensated stage (group 2; $P<0.05$).

Significantly more often headaches were registered in patients with HT, especially in group 1 – in the structure of the known pseudoneurasthenic syndrome, where the relative distribution was 95.0 % versus 88.9 % (in CA).

The results obtained in relation to the intensity of pain in the studied groups indicate that the average VAS values were slightly higher in group 1 (by 11.5 %) compared to group 2.

In the presence of HT, the cephalgic syndrome developed or intensified during or after a night's sleep, in stressful situations, during intense physical activity. At the same time, the pain syndrome had a sharper, often pulsating character, especially with changes in blood pressure numbers, neurasthenic or depressive experiences, and a change in body position. It should be noted that cephalalgia increased in the presence of degenerative-dystrophic changes in the cervical spine, muscular-tonic reactions in the occipital region, etc. Headaches were predominantly localized in the occipital region, were more often diffuse, dull with a feeling of pressure. Possibly at height of a pain vomiting could develop. In some cases, there was an increase in cephalalgia with a decrease in blood pressure. Often, the pain subsided in the afternoon, after taking caffeine-containing drinks. The frequency and intensity decreased as the ischemia progressed. With subcompensation of the pathological process (in group 2), the pain was predominantly of a long-term diffuse nature without nausea.

With CA, as the leading factor in the development of CCI, the headache increased in the process of transition from compensation to the stage of subcompensation, in which it became constant, without precise localization, often accompanied by tinnitus.

AT was changed in 58 (71.6 %) of all examined patients, in group 1 – 26 (68.4 %), in group 2 – 32 (74.4 %). Vegetative imbalance had a pronounced shift towards sympathicotonia in group 1 19 (50.0 %), the minimum values - with vagotonia 7 (18.4 %). As cerebral ischemia progressed in the stage of subcompensation, the state of AT underwent significant changes. A significant decrease was in sympathetic prevalence by 1.5 times ($P<0.05$), a decrease in cases of eutonia and a significant increase in the number of persons with a vagal orientation of AT by 2.2 times ($P<0.05$, fig. 1, A).

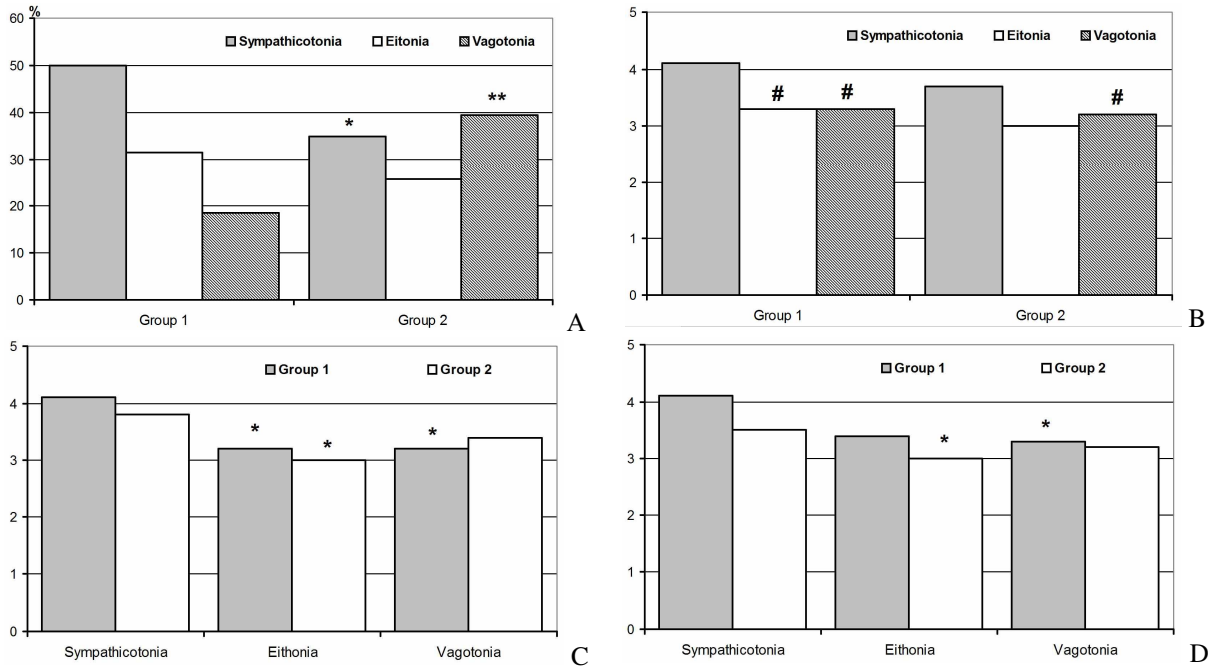

Fig. 1. The clinical manifestation of headache in patients with CCI A – vegetative tone randomization in examined patients; B – headache expression depending from the initial vegetative tone; C – headache intensity in patients with hypertension (points of VAS); D – headache intensity in patients with cerebral atherosclerosis.

Polar deviations of AT values in group 1 gave the maximum frequency of occurrence of cephalalgia: with sympathicotonia – 94.7 %; while maintaining the vagosympathetic balance in this group, the frequency of complaints of cephalalgia was 83.3 %, which was 11.4 % lower in cases of sympathetic orientation of the AT and 16.7 % lower in vagotonia (in both cases P< 0.05, fig. 1, B).

In group 2, patients complained of cephalalgia much less frequently, but their maximum frequency was noted against the background of sympathicotonia (86.7 %). With vagotonia – 64.7 %, with the preservation of the sympathetic -parasympathetic balance, complaints of cephalalgia were minimal – 27.3 % (P<0.05).

According to the VAS, the maximum intensity of cephalgia was against the background of sympathicotonia in both groups: 4.1±0.08 points (group 1) and 3.7±0.06 points (group 2).

Similar patterns persisted in individuals with leading factors for the development of CCI (CA and Headache, fig. 1, C, D).

According to the registration of SSEP, the data obtained regarding changes in the LP of SSEP indicate its elongation with the progression of ischemic brain damage. At the same time, the average delay of the galvanic skin reflex in the group with compensation of the pathological process was 1.43±0.09 sec vs 1.77 ± 0.2 sec in the group of the subcompensated stage of CCI (P<0.05).

A regularity was revealed regarding the change in LP and the severity of cephalgia. In all groups, as well as in individuals with various etiological causes for the development of CCI, with a high intensity of the pain syndrome, a shortening of the LP was noted, which indicates an increase in ergotropic influences with an alleviation of the onset of cephalalgia against the background of sympathicotonia. At the same time, with low-intensity cephalalgia, the LP lengthened, which indicated a trophotropic - more restraining effect on the development of pain syndrome (table 1).

Table 1

Latency randomization depending from headache intensity

Headache	SSEP latencies (sec)					
	Group 1			Group 2		
	Total	CA	Headache	Total	CA	Headache
High intensity (4 or more points)	1.31±0.06	1.32±0.09	1.30±0.12	1.52±0.08	1.67±0.14	1.47±0.08
Low intensity (3 or less points)	1.78±0.04*	1.53±0.12*	2.01±0.21**	2.11±0.13*	2.40±0.22*	1.87±0.16*

Notes: * – p<0.05, ** – p<0.01 – significant differences of the investigated indexes compared with those in patients with high intensity headache (here and further statistical criterion used – one-variant ANOVA + Krushkal-Wallis)

The data obtained indicate the inclusion of the central link and suprasegmental structures of the ANS involved in the implementation of the galvanic skin reflex and the corresponding central delay with possible stimulation of the nociceptive system. In addition, LP elongation can be interpreted as one of the manifestations of the so-called “spring stretch phenomenon” indicating limbic-reticular complex

dysfunction, instability of the system of regulation of autonomic functions in general and accompanying angiodystonic manifestations in brain structures.

In addition, changes in AT clearly confirmed the hypothesis of the depletion of ergotropic processes as the progression of cerebral ischemia with a known increase in parasympathetic influences, which is reflected in Table 2. These patterns are stable and persist regardless of the etiological causes that led to CCI.

Table 2

Changes in SSEP parameters depending from the initial autonomic tone in examined patients

Etiology of CCI	Investigated SSEP parameters			
	1. SSEP latencies (sec) in group 1			
	Total	Sympathicotonia	Eitonia	Vagotonia
CA	1.42±0.07	1.21±0.06	1.46±0.1	1.87±0.16*
Headache	1.43±0.09	1.23±0.07	1.54±0.09	1.85±0.14*
2. SSEP latencies (sec) in group 2				
CA	1.84±0.12	1.35±0.08	1.64±0.13	2.14±0.21*
Headache	1.71±0.11	1.56±0.11	1.80±0.14	1.94±0.16*
3. SSEP A_2 (mV) in group 1				
CA	2.59±0.21*	3.62±0.3	1.86±0.14	1.53±0.12**
Headache	2.72±0.23*	3.36±0.2	2.20±0.23	1.62±0.13**
4. SSEP A_2 (mV) in group 2				
CA	2.32±0.23*	3.75±0.32	2.17±0.21	1.84±0.14**
Headache	2.75±0.25*	3.64±0.19	2.10±0.19	1.82±0.13**

Notes: * – $p<0.05$, ** – $p<0.01$ – significant differences of the investigated indexes compared with relatively normal values

A decrease in the second phase amplitude (A_2) was registered which generally reflects the work of suprasegmental (hypothalamic) ergotropic centers. At the same time, a decrease in A_2 is characteristic of the inhibition of these centres with inhibition of sweating functions. A clear pattern of decrease in A_2 was revealed depending on the depletion of the sympathetic orientation of AT and the increase in vagal prevalence (see Table 2). The mean values of A_2 in both groups were below normal values ($p<0.05$). The difference in the polar values of AT indicates a decrease in A_2 in both groups by 2 times ($p<0.05$), including in individuals with various etiological factors of CCI.

The revealed trend of A_2 decrease and LP elongation is considered as a manifestation of autonomic dysfunction at the limbic-reticular complex level, the appearance of the aforementioned "spring stretching phenomenon".

The leading cause of the development of CCI in CA and Headache are morphological changes in the arterial trunks, microvasculature. In this case, it becomes necessary to assess the hemodynamic significance of pathogenetic factors of cerebral blood supply disorders, the degree of inferiority of the system of its regulation, as well as the range of functional capabilities of the arterial bed. Cerebrovascular reactivity can be an integral indicator of the adaptive capabilities of the cerebral circulation system, the ability of cerebral vessels to respond to changing operating conditions and optimize blood flow in accordance with these conditions.

Ultrasonography in the group with compensated CCI in 60.5 % of patients showed stenosing changes in the arteries of the brain without hemodynamically significant changes. Linear bloodflow velocity slightly decreased in the internal carotid arteries (ICA) and middle cerebral arteries (MCA), while maintaining normal values in the other large arteries.

In the stage of subcompensation, the number of stenoses significantly increased (83.7 %), and in 28.3 % of cases they were hemodynamically significant. A significant linear bloodflow velocity decrease in both MCA and ICA was revealed compared to the control. Linear bloodflow velocity in the anterior cerebral arteries (ACA) remained practically unchanged, due to which there was a decrease in the MCA/ACA ratio. The blood flow in the arteries of the vertebrobasilar basin in group 2 did not change significantly, along with this, there was a decrease in the MCA/basilar artery (BA) ratio. Linear bloodflow velocity indexes in BA increased in comparison with the same in patients of group 1.

Certain regularities in the state of brain hemodynamics in patients with CA and headache were revealed. Moreover, the linear bloodflow velocity decrease in the carotid pool vessels was almost the same with compensation for CCI (group 1). In the stage of subcompensation (group 2) linear bloodflow velocity indexes for PCA in patients with headache and CA were equalized in VA and BA, linear bloodflow velocity indexes were higher in headache but the difference is still lower than in group 1.

When studying the dynamics of RI^+, a decrease in vasodilator was in both groups, and it intensified with the growth of morphological changes in group 2 (fig. 2, A).

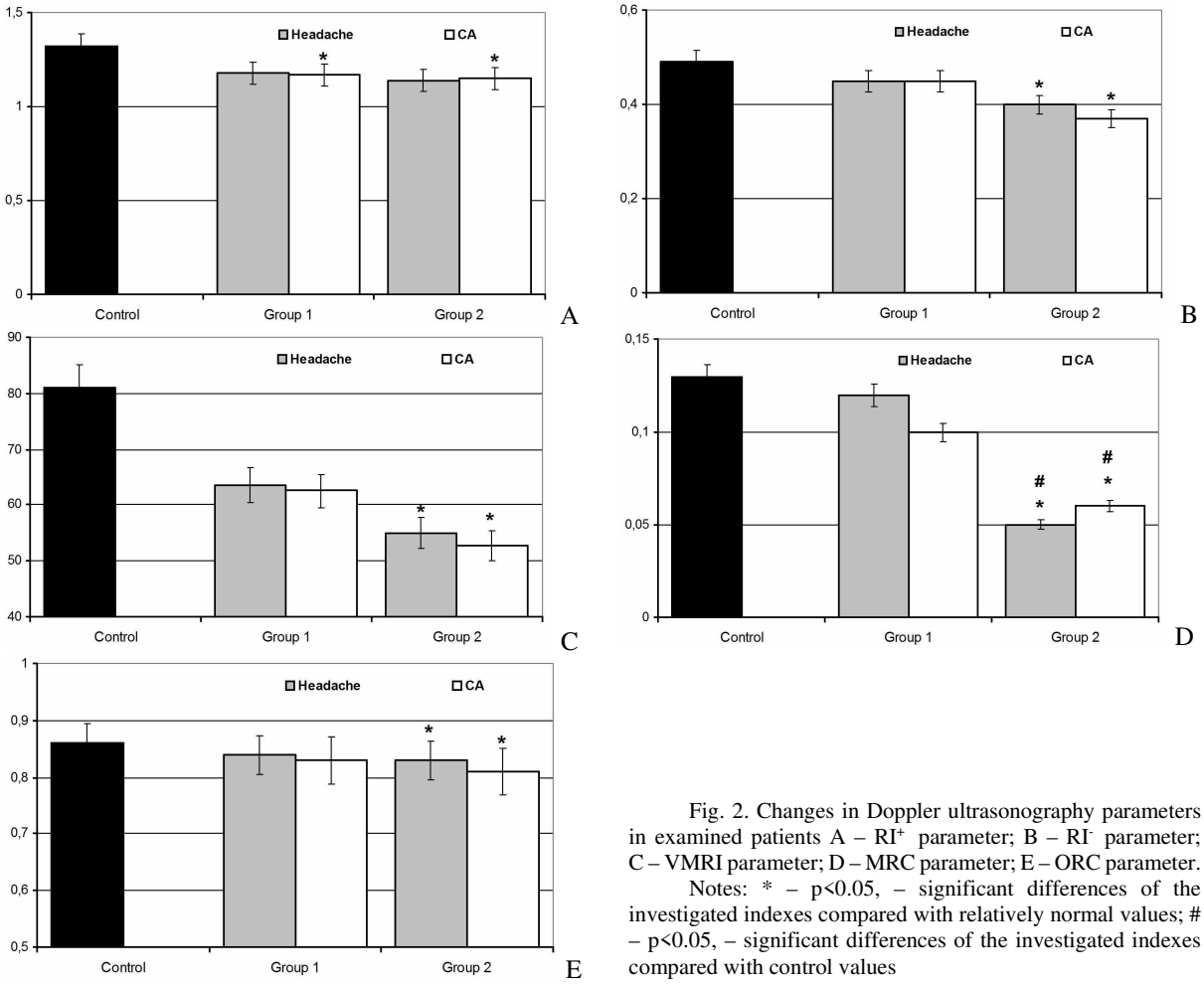

Fig. 2. Changes in Doppler ultrasonography parameters in examined patients A – RI^+ parameter; B – RI^- parameter; C – VMRI parameter; D – MRC parameter; E – ORC parameter.

Notes: * – $p<0.05$, – significant differences of the investigated indexes compared with relatively normal values; # – $p<0.05$, – significant differences of the investigated indexes compared with control values

A downward trend in RI^- depending on the increase in the stage of CCI was observed in both etiological groups, but in contrast to similar data on RI^+, there was no significant difference in indicators in patients with headache and CA (fig. 2, B).

It can be assumed that the depletion of the reserves of the vasoconstrictor component in patients with CVD occurs later than similar changes in the vasodilatory component, does not depend on the etiology of CVD.

The increase in reactivity according to VMRI, as a sign of the search for the optimal sanogenetic variant of cerebral hemodynamics, occurs mainly due to the vasodilator component (Fig. 2, C).

These data indicate the importance of VMRI as an integral factor reflecting the dynamic properties of the homeostatic range of vascular autoregulation with various stages of CCI.

In patients in the subcompensated stage (group 2), the values of MRC significantly decreased compared with the control group, as well as with group 1 (fig. 2, D).

Thus changes in reactivity along the myogenic circuit as CCI progresses are similar to the data of the metabolic circuit of autoregulation and the inversion of the response to nitroglycerin load is a marker of autoregulation failure and correlates with the degree of ischemic brain damage.

The ORC indicators decrease with the progression of the pathological process, and in patients with headache they exceed those in CA (fig. 2, E).

The decrease in the autonomic disturbances prevalence progressed during the transition from the compensated stage of CCI to the subcompensated one . Apparently, autonomic disturbances is mainly realized by the neurogenic circuit of autoregulation, dysfunction of the latter is recorded even in healthy people, however, an increase in the progression to decompensation of vascular pathology indicates its undoubted significance in the processes of impaired tone and reactivity of cerebral vessels.

Therefore, the analysis of the complex clinical observations and electrophysiological registrations allow us to make some preliminary conclusions. One should suppose that headache was the leading syndrome in patients with CCI. It was very often manifested in the compensated stage (92.1 %), in the presence of hypertension (95.0 %), in cases of sympathetic prevalence 94.7 %. We have to state that analogous data were published by [14] who followed also the headache prevalence in patients with transient ischemic attack.

Autonomic dysfunctions were registered in all examined persons, while AT was pathological in 74.4 % of patients. In cases of compensation, sympathicotonia prevailed (50.0 %), in the subcompensated stage of CCI, the AT of the latter decreased by 1.5 times ($p<0.05$), with an increase in vagal orientation - by 2.2 times ($p<0.05$).

An interesting results were received in case of electrophysiological registration. SSEP registration showed CNS pathological ischemic processes manifestation worsening, SSEP latency increasing, the second phase SSEP amplitude decreasing which indicated limbic-reticular complex dysfunction, depletion of ergotropic processes, instability of the autonomic functions regulation system, which is clinically reflected in the form of angiodystonic manifestations. Actually, this method seems to be very perspective on the merge between neurological and concomitant internal diseases that was also proved in clinical investigation used both cold- and heat-evoked potentials for proper and adequate earlier diagnosis [11].

The ultrasound data confirm that both autonomic system and vasomotor centres dysfunction is the leading mechanism for cerebral ischemia onset and progression, which resulted in cerebral vessels impaired tone and reactivity, neurovasoactive substances accumulation, which contributes to the restructuring of the metabolic circuit of autoregulation with a change in the homeostatic range. Our data are in full agreement with the clinical-laboratory observations [9, 15]. Changes in the myogenic conture are similar in direction to the metabolic ones. Such inversions of vascular reactions are most clearly manifested by the response profile to a nitroglycerin load. The latter has a clear increase with the progression of ischemic brain damage, up to complete unresponsiveness. The initial part of these clinical trials was previously published [3].

Resuming we have to stress that our results might contribute to the patients with CCI treatment tactics optimization taking into account the ANS both functional activity and individual characteristics as well as the cerebral bloodflow reactivity.

Conclusions

1. Headache was the leading syndrome in patients with CCI. Autonomic dysfunctions were registered in all examined persons.

2. SSEP registration showed CNS pathological ischemic processes manifestation worsening, SSEP latency increasing, the second phase SSEP amplitude decreasing which indicated limbic-reticular complex dysfunction, depletion of ergotropic processes, instability of the autonomic functions regulation system.

4. The ultrasound data confirm that both autonomic system and vasomotor centres dysfunction is the leading mechanism for cerebral ischemia onset and progression.

5. Our results might contribute to the patients with CCI treatment tactics optimization taking into account the ANS both functional activity and individual characteristics as well as the cerebral bloodflow reactivity.

Prospects for furthers researches include a subsequent comprehensive experimental studies and clinical observations to elucidate the pathophysiological mechanisms of autonomic nervous system dysfunction and their influence on the formation of complex pathogenetic chains of cerebrovascular pathology development. A clear scheme of chronic cerebral ischemia complex pathogenetically substantiated therapy should be a result of these studies.

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Стаття надійшла 30.04.2021 р.

DOI 10.26724/2079-8334-2022-2-80-168-173
UDC 616.728.2-089.28:612.76

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X-RAY CHARACTERISTICS OF THE DYSPLASTIC HIP JOINT IN ADULTS BEFORE AND AFTER ENDOPROSTHETIC SURGERY

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Based on the results of X-ray studies of 49 dysplastic hip joints before and after arthroplasty, it was found that such indices of the dysplastic hip joint in adults as the cervical-diaphyseal angle of the femur, the depth of the acetabulum and the thickness of its bottom before and after the arthroplasty surgery significantly differ from each other. The parameters of the cervical-diaphyseal angle in women had significant differences before and after surgery. The cervical-diaphyseal angle in women after arthroplasty was significantly higher than in men. Abductor strength shoulder indices significantly increase in women after surgery. In men, there was a tendency to reduce the shoulder strength of the abductors after surgery. The parameters of the hip joint, which affect the magnitude of the hip abductors' arm, should be considered as biomechanical criteria for the efficiency of the hip joint muscles. Based on the results of X-ray studies of 49 dysplastic hip joints before and after arthroplasty, it was found that such indices of the dysplastic hip joint in adults as the cervical-diaphyseal angle of the femur, the depth of the acetabulum and the thickness of its bottom before and after the arthroplasty surgery significantly differ from each other. Improving the biomechanics of the dysplastic hip joint can be achieved by restoring the geometric parameters of the hip joint as a result of arthroplasty.

Key words: hip joint, dysplasia, hip arthroplasty, X-ray indices

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РЕНТГЕНОЛОГІЧНА ХАРАКТЕРИСТИКА ДИСПЛАСТИЧНОГО СТЕГНОВОГО СУГЛОБУ У ДОРОСЛИХ ДО ТА ПІСЛЯ ОПЕРАЦІЇ ЕНДОПРОТЕЗУВАННЯ

На підставі результатів рентгенометричних досліджень 49 диспластичних тазостегнових суглобів до і після ендопротезування встановлено, що такі показники диспластичного тазостегнового суглоба у дорослих як шийково-діафізарний кут стегнової кістки, глибина вертлужної западини і товщина її дна до і після операції достовірно відрізняються між собою. Показники шийно-діафізарного кута у жінок мали достовірні відмінності до та після операції. Шийково-діафізарний кут у жінок після ендопротезування був достовірно вищим, ніж у чоловіків. Показники плеча сили абдукторів після операції достовірно зростають у жінок. У чоловіків відзначено тенденцію до зменшення плеча сили абдукторів після операції. Параметри кульшового суглоба, що впливають на величину плеча сили абдукторів стегна, доцільно розглядати як біомеханічні критерії ефективності роботи м'язів кульшового суглоба. Поліпшення біомеханіки диспластичного кульшового суглоба може бути досягнуто шляхом відновлення геометричних параметрів кульшового суглоба в результаті ендопротезування.

Ключові слова: тазостегновий суглоб, дисплазія, ендопротезування, рентгенометричні показники

The study is a part of the research project "To study the biomechanical features of human standing and walking function after hip arthroplasty", state registration No. 0118U006950.

Dysplastic coxarthrosis is one of the most severe manifestations of degenerative-dystrophic lesions of the hip joint. And hip arthroplasty in case of its dysplastic lesion refers to surgery of increased complexity [2, 9].