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KUTIKOV Oleksandr Yevgenovych, PhD in Biological Sciences, Associate Professor; Leading Researcher of the Department of Scientific Organization of Neurological and Psychiatric Care of the "Institute of Neurology, Psychiatry and Narcology of the National Academy of Medical Sciences of Ukraine" State Institution ("INPN of the NAMS of Ukraine" SI), Kharkiv; e-mail: akutikov@ukr.net

VOLOSHYN-GAPONOV Ivan Kostyantynovych, MD, PhD; Leading Researcher of the Department of Neuropsychocybernetics of the "INPN of the NAMS of Ukraine" SI, Kharkiv; e-mail: voloshingaponov.ivan@mail.ru

КУТІКОВ Олександр Євгенович, кандидат біологічних наук, старший науковий співробітник; провідний науковий співробітник відділу наукової організації неврологічної та психіатричної допомоги Державної установи «Інститут неврології, психіатрії та наркології Національної академії медичних наук України» (ДУ «ІНПН НАМН України»), м. Харків; e-mail:akutikov@ukr.net

ВОЛОШИН-ГАПОНОВ Іван Костянтинович, кандидат медичних наук; провідний науковий співробітник відділу нейропсихокібернетики ДУ «ІНПН НАМН України», м. Харків; e-mail: voloshingaponov.ivan@mail.ru

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T. A. Litovchenko, V. V. Lebedynets, Iu. V. Iakubenko, A. A. Novikova

THE DYNAMICS OF HEMODYNAMIC INDEXES IN PATIENTS WITH CONSEQUENCES OF MILD TRAUMATIC BRAIN INJURY

Т. А. Літовченко, В. В. Лебединець, Ю. В. Якубенко, Г. А. Новікова

ДИНАМІКА ГЕМОДИНАМІЧНИХ ПОКАЗНИКІВ У ПАЦІЄНТІВ З НАСЛІДКАМИ ЛЕГКОЇ ЧЕРЕПНО-МОЗКОВОЇ ТРАВМИ

Т. А. Литовченко, В. В. Лебединец, Ю. В. Якубенко, А. А. Новикова

ДИНАМИКА ГЕМОДИНАМИЧЕСКИХ ПОКАЗАТЕЛЕЙ У ПАЦИЕНТОВ С ПОСЛЕДСТВИЯМИ ЛЕГКОЙ ЧЕРЕПНО-МОЗГОВОЙ ТРАВМЫ

We examined 59 patients with consequences of mild traumatic brain injuries. The patients were examined by using the method of Doppler ultrasonography of the extracranial brain vessels of the carotid arterial system with the application of hypercapnic and hypocapnic test. During our investigation we detected changes of reactivity of vessels towards the decrease of indicators of linear velocity of blood flow (LVF) under the tests. A paradoxical vascular response was marked in all the patients under the hyper- and hypocapnic load; a test-induced delayed vessels reaction was marked in 37 out of 59 patients.

Key words: consequences of mild traumatic brain injury, cerebral autoregulation, reactivity of vessels.

Під нашим спостереженням перебували 59 пацієнтів з наслідками легкої черепно-мозкової травми. Всім пацієнтам проведено доплерографічне дослідження судин головного мозку екстракраніального сегмента артерій каротидного басейну з застосуванням гіперкапнічного та гіпокапнічного тестів. У ході нашого дослідження було виявлено порушення реактивності судин у вигляді зниження лінійної швидкості кровотоку на тлі проведення проб. У всіх пацієнтів була виявлена парадоксальна реакція судин у відповідь на гіпер- і/або гіпокапнію, а також у 37 пацієнтів з 59 спостерігалася відстрочена реакція судин у відповідь на проведені проби.

Ключові слова: наслідки легкої черепно-мозкової травми, реактивність судин.

Под нашим наблюдением находились 59 пациентов с последствиями легкой черепно-мозговой травмы. Всем пациентам проведено допплерографическое исследование сосудов головного мозга экстракраниального сегмента артерий каротидного бассейна с применением гиперкапнического и гипокапнического тестов. В ходе нашего исследования было выявлено нарушение реактивности сосудов в сторону снижения линейной скорости кровотока на фоне проведения проб. У всех пациентов была выявлена парадоксальная реакция сосудов в ответ на гипер- и/или гипокапнию, а также у 37 пациентов из 59 наблюдалась отсроченная реакция сосудов в ответ на проводимые пробы.

Ключевые слова: последствия легкой черепно-мозговой травмы, реактивность сосудов.

Traumatic brain injury is one of prevailing mechanical central nervous system damages. However, the most important from a medical, social and economical point of view are traumatic brain injury (TBI) consequences, as they can gain chronicity, deteriorating the patients' quality of life, restricting their occupational capacity and often provoking a permanent disability [6, 7].

TBI-induced pathogenesis is multifocal and includes various pathophysiological and pathobiochemical processes: oxidative stress, excitotoxicity, perifocal neutron depolarization (within the first minutes & hours after the trauma), autoimmune inflammation, apoptosis (within further days & weeks) etc. In the long run all these mechanisms lead to the loss of neurons and neural bonds between different parts of CNS.

In a long-term perspective TBI generates «posttraumatic syndrome», marked by a headache, dizziness, tiredness, low mood and memory impairment. Depending on its severity and type, TBI results in primary structural-functional brain damage differing in abundance and degree at the subcellular, cellular, tissular and organ level; besides, central regulation of vital functions may be disturbed. Mild traumatic brain injury is a mild case of a diffuse axonal injury. It is recognized that clinical aspects of mild traumatic brain injury are conditioned by asynapsia, mainly functional, between the cerebral cortex, subcortical structures and brain stem. Morphologic substrate of mild traumatic brain injury includes synaptic apparatus and redistribution of interstitial fluid. Disturbed circulation may also be registered in case of mild traumatic brain injury, arising from small vessels lesions and white matter capillaropathy [4—6].

The involving of brain stems structures provokes both specific and nonspecific brain systems impairment, manifesting as autonomic and neuropsychological disorders. Mild TBI-induced lesion of limbic-reticular complex leads to circulatory disturbance and to cerebral synaptic apparatus disorder. In the following reticulo-cortico-subcortical malfunction is complicated by dyscirculatory alterations, neurohumoral, neurohormonal and metabolic derangements, produced by a "tension mechanism", common to a stressful situation [3, 5, 7].

Mild TBI primary structural changes trigger the initiation of neurometabolic pathway, generally at ultrastructural level. These alterations factor into tissue hypoxia progression and acid intoxication. Brain injury originates cerebral autoregulation disorder, liquor-dynamic disturbances and alterations in hematoencephalic barrier permeability. Cerebral autoregulation is one of the *fundamental properties* of the *cerebral circulation*. It is of fundamental importance for adequate cerebral blood supply and it is defined by cerebral vessels' capacity to hold constant volume velocity of cerebral blood flow on a change of perfusion pressure. Changes in cerebral vessels reactivity may represent a diagnostic character, as well as they may describe cerebral circulation functional status.

The goal of the present research is to revise the cerebral autoregulation disturbances by defining key indicators of cerebral hemodynamics and those of vessels reactivity in mild TBI patients.

We examined 59 patients with consequences of mild traumatic brain injuries, at the age of 25—35, where 49 male and 10 female patients. The duration of the disease was 2—3 years. All the patients were subject to comprehensive clinical neurological exam. Basic syndromes detected in patients were: cephalalgias, asthenic syndrome and dysautonomia, that is consistent with literature data [5, 6]. All the patients present the following problems: headaches (100 %), rapid fatigability (82.2 %), attention and memory impairment (32.8 %), dizziness (10.6 %), acrimony (52.4 %), anxiety (47.8 %), sleep problems (27.4 %). In neurologic status symptomatology was observed in the form of decrease of convergence (68.2 %), asymmetry of face (76.8 %), revivals of tendinous reflexes (78.5 %), asymmetry of tendinous reflexes (63.6 %), paleness of skin cover (37.7 %), red flare (78.3 %), white flare (27.7 %).

The examination didn't cover patients with a concurrent actual somatic pathology.

The control group included 20 apparently healthy subjects (13 men and 7 women), gender and age-coinciding with the survey sample.

To evaluate hemodynamic index of linear velocity of blood flow (LVF) and indicators of reactivity of vessels in the system of the common carotid arteries (common, surface and internal carotid arteries), all the patients were examined by the method of Doppler ultrasonography (the supersonic apparatus E SAOTE Megas GPX 2004, the linear detector 7.5—10 MHz). The assessment was performed by standardized method. To determine cerebral vessels reactivity hypercapnic and hypocapnic tests were applied. Hypercapnic test was exercised by a post-inhalation breath-holding up to 30 seconds with a cerebral blood flow measurement immediately prior to exhalation. Hypocapnic test was exercised after the hypercapnic one, by way of frequent and deep breathing during 30 sec. None of the patients showed clear signs of distress due to the tests. Before conducting the compression tests, routine Doppler ultrasonography of vessels of common, surface and internal carotid arteries was carried in all the patients in order to exclude coiling, occlusion and hemodynamically relevant stenosis.

In our study, in order to estimate hemodynamics and reactivity of vessels in the carotid arterial system, we took into consideration the primary average LVF, systolic linear velocity of blood flow, the pulsation index (PI) and the index of peripheral resistance (RI). We estimated V_0 — the average LVF at a baseline, V_1 — the average LVF during the hypercapnia, V_2 — the average LVF during the hypocapnia, overshoot (vicarious increase of LVF in response to hypercapnia.

We calculated the coefficient of overshoot (CO), the coefficient of reactivity during the hypercapnia (CR+), the coefficient of reactivity during the hypocapnia (CR–), the index of vasomotor reactivity (IVMR).

The coefficient of overshoot was calculated by the formula:

$$CO = V_1/V_0, \quad (1)$$

in which V_0 means the average LVF at a baseline,

V_1 means the average LVF during the hypercapnia.

The coefficient of reactivity during the hypercapnia (CR+) was calculated by the formula:

$$CR+ = (V_1/V_0) - 1, \quad (2)$$

The coefficient of reactivity during the hypocapnia (CR–) was calculated by the formula:

$$CR- = 1 - (V_2/V_0), \quad (3)$$

in which V_2 means the average LVF during the hypocapnia.

The index of vasomotor reactivity was calculated by the formula:

$$IVMR = [(V_1 - V_2)/V_0] \cdot 100\ \%. \quad (4)$$

The data received were mathematically processed by analysis-of-variance method (Student's *t*-test and Fischer test); correlation relationship was estimated using program packages Excel XP build 10.6612.6625-SP3 (Microsoft), Statistica 6.0 (Statsoft Inc).

Spectrographic shapes of the arterial system in question significantly differ. Analysis of Doppler-shifted spectrum allows classifying them as arteries with a low, mean and high peripheral vascular resistance (PVR). PVR level depends on

the region, wherein a targeted artery supplies blood. Thus, the blood flow in the internal carotid artery, ensuring direct cerebral tissues perfusion, has a low peripheral resistance. In the external carotid artery, ensuring principally soft head tissues perfusion, the blood flow is described by a high peripheral resistance. The blood flow in the common carotid artery is characterized by a mean peripheral resistance. The PVR value models dopplerogram, sizes up magnitudes of telediastolic flow velocity and determines the nature of the audible signal. In this regard, it is in internal carotid arteries that hemodynamic parameter is appropriate to evaluate.

In estimating dopplerographic curves for arteries with a low peripheral vascular resistance the following peaks can be distinguished on a pulsed wave curve: 1 — systolic peak: corresponds to a blood peak flow within the ejection period; 2 — catacrotic wave: corresponds to the entry in the period of relaxation; 3 — dicrotic wave: characterizes the closing period of the aortic valve; 4 — diastolic phase: corresponds to ventricular diastole phase [4, 5].

Patients with consequences of traumatic brain injuries demonstrated paradoxical (abnormal) vascular response that can be represented in 2 types:

— type 1 — increase of peak systolic velocity of blood flow up to 10 % on the average in the internal carotid artery in response to hypercapnia, with no decrease of blood flow velocity in the next phase of hypocapnia, with a parallel decrease of peripheral resistance index (RI) (fig.1);

— type 2 — paradoxical vascular response to hypercapnia in terms of decrease of velocity or its delayed increase with a further velocity gain in response to hypocapnia with a parallel increase of peripheral resistance index (RI), presumably related to a malfunction of vasomotor mechanisms of peripheral minute vessels (fig. 2).

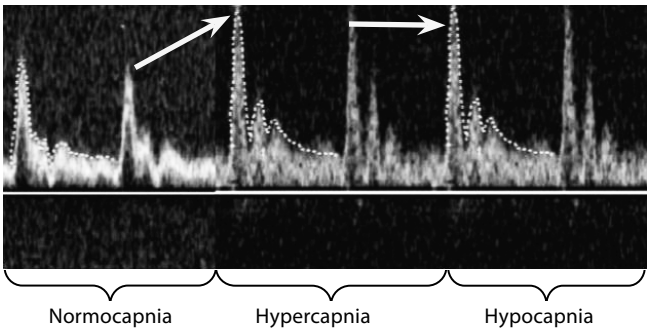

Figure 1. **Variant of cerebral vessels reactivity malfunction in patients with consequences of traumatic brain injuries (type 1)**

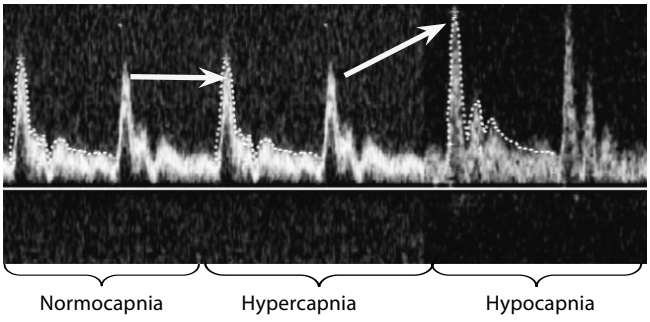

Figure 2. **Variant of cerebral vessels reactivity malfunction in patients with consequences of traumatic brain injuries (type 2)**

After the hypercapnia a transient increase of the blood flow owing to the compensatory vasodilatation was registered in the patients of the control group. The above can be used as an indicator of autoregulation. Temporary hyperemic response in carotid system vessels, manifesting as a short-term LVF increase, allows calculating the index, characterizing the vasodilatation in response to a transient decrease of perfusion pressure.

Whereas comparing to the control group changes were detected in the form of vicarious increase of LVF in response to hypercapnia, and the further LVF decrease in response to hypocapnia, during the hypercapnic and hypocapnic tests (fig. 3).

The figure 3 demonstrates dopplerograms of the blood flow from the internal carotid artery during the hypercapnic and hypocapnic tests in the patients of the control group. The figure makes visible the increase of peak systolic velocity of blood flow in the internal carotid artery up to 29 % in response to hypercapnia. This effect conforms to a sufficient functional (perfusion) reserve of *cerebral circulation. The decrease of* peripheral resistance in the brain under the influence of CO2 manifested as the decrease of peripheral resistance index in the internal carotid artery.

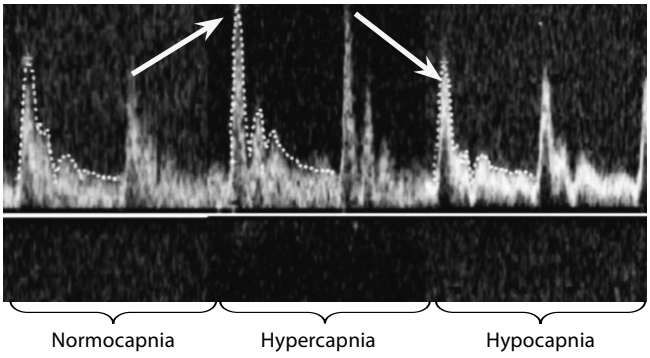

Figure 3. **LVF dynamics during the hypercapnic and hypocapnic tests in the patients of the control group**

During the research by Doppler ultrasonography with the hypercapnic and hypocapnic tests the following changes were identified in the control group: the increase of LVF after the hypercapnic test to 49.2 %, the increase of LVF after the hypocapnic test to 26.8 % (fig.1). The coefficient of overshoot (CO) was 1.31 ± 0.03. The normal meaning of the coefficient of overshoot is 1.24 to 1.54; the reduction under 1.2 indicates hyporesponsiveness. The meaning of the coefficient equaling 1.00 indicates areactivity.

During the research by Doppler ultrasonography with the hypercapnic and hypocapnic tests in patients with consequences of mild traumatic brain injuries the CO of 1.11 ± 0.02 ($p<0.01$) was identified. The increase of LVF after the hypercapnic test to 29.8 % was observed, while after the hypocapnic test, the LVF showed an increase of 38.4 %. Under the hypercapnic load in patients with consequences of mild traumatic brain injuries the coefficient of reactivity was lower than that of the patients in the control group (0.33 ± 0.05 and 0.45 ± 0.05 RVU respectively, $p < 0.0001$).

Under the hypocapnic load in patients with consequences of mild traumatic brain injuries the coefficient of reactivity was lower than that of healthy subjects (0.45 ± 0.07 and 0.55 ± 0.046RVU respectively, $p < 0.0001$). The index of vasomotor reactivity in patients with TBI consequences was lower, than that of the control group (70.1 ± 0.05 and 96.2 ± 0.02 respectively, $p < 0.0001$).

A slight blood flow asymmetry was identified, increasing during hyper- and hypocapnia. The patients with consequences of mild traumatic brain injuries showed an anticipated atherosclerotic cerebral vascular disease, in the form of hemodynamically irrelevant atherosclerotic plaques. A slight blood flow asymmetry was observed, increasing during hyper- and hypocapnic tests. The increase of the contralateral blood flow was shown dependent on the injury in the mild TBI patients. A paradoxical vascular response of the common carotid artery basin was disclosed in terms of poor or delayed vascular response to hypercapnia (the increase of the LVF is naturally present) and the high and/or delayed vascular response to hypercapnia (the decrease of the LVF is naturally present) (fig.2). In addition to the above in 37 out of 59 patients a delayed vascular response to hypercapnic and/or hypocapnic tests has been present (fig.2). The increase of PI up to 1.75 ± 0.02; and the increase of RI up to 0.96 ± 0.04 were present in all the patients in the survey sample with consequences of traumatic brain injuries, which in turn implies liquor hypertension syndrome.

The findings are illustrated in Table 1.

Further, regularity has been revealed, consisting in more significant cerebral vessels reactivity on the opposite side of the injury, eventually coming of a contrecoup.

Table 1

Characteristics of indicators of vascular hemodynamics in the internal carotid artery basin in the patients with consequences of mild traumatic brain injuries

	Patients with consequences of mTBI (n = 59)	Control group (n = 20)
	$M \pm m$	$M \pm m$
Systolic LVF, sm/s	0.72 ± 0.05#	0.95 ± 0.02
Average LVF at a baseline, sm/s	0.33 ± 0.03	0.31 ± 0.008
Average LVF during the hypocapnia	0.37 ± 0.002*	0.41 ± 0.02
Pulsation index	1.75 ± 0.02#	0.83 ± 0.05
Peripheral resistance index	0.96 ± 0.04**	0.75 ± 0.07
Coefficient of overshoot	1.11 ± 0.04#	1.33 ± 0.02
Coefficient of reactivity during the hypercapnia	0.33 ± 0.05#	0.45 ± 0.05
Coefficient of reactivity during the hypocapnia	0.45 ± 0.07**	0.55 ± 0.06
Index of vasomotor reactivity	70.1 ± 0.05#	96.2 ± 0.02

* — $p < 0.05$; ** — $p < 0.01$; # — $p < 0.001$

Malfunction of reactivity of vessels towards the decrease of indicators of the LVF is present in all the patients with consequences of traumatic brain injuries. A paradoxical cerebral vascular response was evident as a delayed response to the tests as carried out. The above response was accompanied by the increase of the LVF after the hypercapnic test to 29.8 % (49.2 % in the control group) and by the increase of the LVF after the hypocapnic test to 38.4 % (26.8 % in the control group). A delayed vascular response to hypercapnic and/or hypocapnic tests results, in our opinion, from reduced vessel wall elasticity on the back of microstructural damage of vessel walls and in the wake of anticipated atherosclerotic vascular disease. Such a response also exhibits a longstanding angiospasm. Liquor hypertension syndrome in such patients is, according to our reckoning, secondary and generates from a longtime vasospasm.

The results of these studies suggest the following conclusions.

Impaired cerebral autoregulation and disturbed reactivity of vessels are evidenced in the patients with consequences of mild traumatic brain injuries in terms of decrease of linear velocity of blood flow and of increase of resistance indexes (PI and RI).

A paradoxical vascular response, arising for TBI patients, is related to reduced vessel wall elasticity on the back of microstructural damage of vessel walls in the wake of anticipated atherosclerotic vascular disease. The above response also exhibits a longstanding angiospasm.

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LITOVCHENKO Tetiana Anatoliivna, Doctor of Medical Sciences, Professor, Head of the Department of Neurology of the Kharkiv Medical Academy of Postgraduate Education (KhMAPE), Kharkiv

LEBEDYNETS Volodymyr Vasylovych, MD, Head of the 1rd Neurology Department of the State Institution for Treatment and Prevention "Central Clinical Hospital Ukrzaliznytsi", Kharkiv

IAKUBENKO Iuliia Valentynivna, Postgraduate Student of the Department of Neurology of the KhMAPE, Kharkiv; e-mail: yakubenko.yulya@mail.ru

NOVIKOVA Hanna Anatoliivna, MD, PhD, Physician of functional diagnostics, of the State Institution for Treatment and Prevention "Central Clinical Hospital Ukrzaliznytsi", Kharkiv

ЛІТОВЧЕНКО Тетяна Анатоліївна, доктор медичних наук, професор, завідуюча кафедрою неврології Харківської медичної академії післядипломної освіти (ХМАПО), м. Харків

ЛЕБЕДИНЕЦЬ Володимир Васильович, завідувач 1-м неврологічним відділенням Державного лікувально-профілактичного закладу «Центральна клінічна лікарня Укрзалізниці», м. Харків

ЯКУБЕНКО Юлія Валентинівна, аспірант кафедри неврології ХМАПО, м. Харків; e-mail: yakubenko.yulya@mail.ru

НОВІКОВА Ганна Анатоліївна, кандидат медичних наук, лікар функціональної діагностики, ДЛПЗ «Центральна клінічна лікарня Укрзалізниці», м. Харків