EXPERIMENTAL METHODS FOR CLINICAL PRACTICE

Correlations of Arterial Hemodynamics with the Severity of Clinical Manifestations of Vertebrobasilar Insufficiency

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A variety of clinical manifestations of vertebrobasilar insufficiency in virtually the same anatomical disorders of the vertebral arteries indicates the multilevel regulation of the cerebral bloodflow aimed at its stabilization and maintenance. The main arteries of the head and peripheral arteries were studied by Doppler ultrasonography in patients with anatomical disorders of vertebral arteries (atherosclerotic, extravasal compressions, congenital abnormalities, *etc.*). A relationship between the variability of clinical picture of vertebrobasilar insufficiency and the diastolic bloodflow in the large aortic arteries was detected.

Key Words: *vertebral arteries pathology; vertebrobasilar insufficiency; ultrasonic dopplerography*

The vertebrobasilar vessels are responsible for blood supply for about $^1/_3$ of the brain. The area supplied by the blood through this system includes the cervical portion of the spinal cord, the brainstem and cerebellum, parts of the thalamus and hypothalamic areas, occipital lobes, posterior and mediobasal compartments of the temporal lobes of the brain [2,5]. Insufficient blood supply to the brain develops if the local bloodflow drops below 40 ml/100 g/min. Compensation for reduced cerebral bloodflow is realized through the aortic arch branches by several routes, one of which is increase of the bloodflow velocity in the contralateral vertebral artery, collateral cerebral circulation at four levels (arterial cerebral circulation (circle of Willis), anastomoses between the surface and deep cerebral arteries, and extracranial level), as well as myogenic, metabolic, neurohumoral, neurogenic autoregulation of cerebral bloodflow [6,8].

Stenosis of arteries delivering blood to the brain leads to disorders in the cerebral circulation. Congenital and acquired deformations of the vertebral arteries (VA), atherosclerosis, extravasal VA compression in diseases of the cervical spine contribute to the pathogenesis of the cerebral circulation disorders [2].

According to published data, the clinical picture of vertebrobasilar insufficiency (VBI) in similar anatomical changes in the vertebral arteries can be different and vary from blurred (asymptomatic) to clinically manifest forms [1,2].

The symptoms of VBI and lesions of the vertebral arteries (hypoplasias, aplasias, deformations,

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extravasal compression) were described, but no relationship between variability of clinical picture of VBI and similar pathological changes in VA was detected. The relationship between the cerebral bloodflow reserve and the status of the large arteries during different periods of the cardiac cycle was never evaluated.

We evaluated the relationship between the severity of VBI and hemodynamic disorders in the basin of vertebral arteries and large aortic vessels.

MATERIALS AND METHODS

The study was carried out in 112 VBI patients aged 45-65 years. Patients with hemodynamically significant stenoses of the carotid vessels, subclavian (SCA) and femoral arteries (FA) of different origin, with abnormal structure of the circle of Willis were excluded from the study. Control group consisted of healthy individuals of similar age (n=30; 17 women and 13 men).

Verification of VBI diagnosis and evaluation of the degree of VA involvement was carried out by standard clinical instrumental methods. The data of Doppler ultrasonography and duplex scanning were used. The main vessels of the head and large aortic vessels were examined, the velocity and direction of the bloodflow during the systolic and diastolic periods of the cardiac cycle were evaluated. Heart work and myocardial function were evaluated by ECG and echocardiography (EchoCG).

All patients were divided into 2 groups: 1) with stenosis of one VA (50-70%) and 2) with bilateral involvement of VA with stenosis of the same degree.

Doppler ultrasonography was carried out on an ANGIODYN device, ECG on a Kardi-2 device (BIOSS). Dulplex scanning and EchoCG were carried out on an IMAGIG SIGMA 5000 SERIES device.

The results were statistically processed using Microsoft Excel and Statistica 6.0 software. The differences were considered significant at $p<0.05$.

RESULTS

The presence, incidence, and severity of symptoms characteristic of VBI were evaluated for each group (Fig. 1).

Both groups included patients with VBI of different severity: from solitary symptoms (blurred forms) to manifest clinical symptoms.

For detailed study of clinical manifestations of VBI, each group was subdivided into two subgroups. The first subgroups (IA and IIA) included patients from both groups with the classical uncomplicated

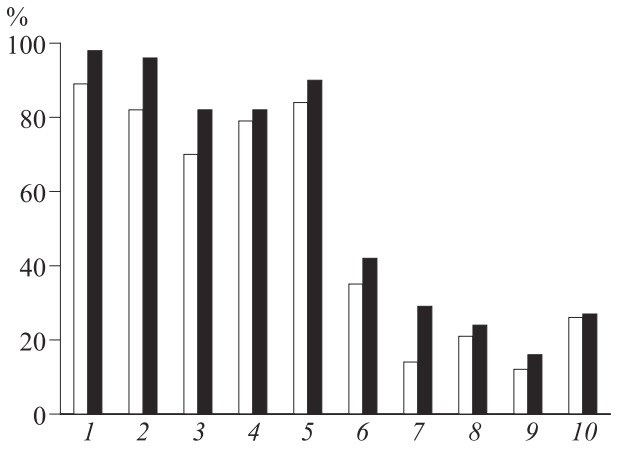

Fig. 1. Incidence of VBI symptoms in patients participating in the study. Light bars: group 1; dark bars: group 2. *1*) giddiness; *2*) ataxia; *3*) vision disorders; *4*) vestibular symptom; *5*) headache; *6*) diplopia; *7*) sensitivity disorders; *8*) photopsia; *9*) psycho-emotional disorders; *10*) autonomic disorders.

picture of VBI (compensated course), while the second subgroups (IB and IIB) included patients with complicated VBI with a history of transitory ischemic attacks or strokes in the vertebrobasilar basin (decompensated course).

Comparison of these groups showed that in group 1 the number of patients with compensated VBI (63%) was 1.7 times higher than the number of patients with decompensated condition (37%). In group 2 the numbers of patients with compensated (55%) and decompensated VBI (45%) were similar. Lesser compensation reserve and more pronounced manifestation of clinical symptoms of VBI were observed in patients of groups IB and IIB in comparison with groups IA and IIA. Presumably, this indicates a higher compensatory reserve (involvement of the collaterals).

The pulse wave and redistribution of blood streams in different anatomical disorders in VA were studied by Doppler ultrasonography [3,4, 7,8].

TABLE 1. Quantitative Evaluation of Bloodflow Parameters for SCA and FA

Parameter	Group	
	IA, IIA	IB, IIB
A=(Ar/As) SCA	-4.06±1.38*	-4.60±1.14*
RB=Ar/As×Tr SCA	-0.59±0.02*	-0.77±0.051*
A=(Ar/As) FA	-3.09±0.088	-3.18±0.051
RB=Ar/As×Tr FA	-0.640±0.033*	-0.800±0.021*

Note. "-": retrograde bloodflow. *$p<0.05$ significant differences between the groups.

The main bloodflow reserve was evaluated using indexes reflecting phase pattern of the bloodflow, duration and intensity of the retrograde bloodflow in the SCA and FA: proportion between the amplitudes (A) of the retrograde wave (Ar) and systolic wave (As; A=Ar/As), duration of the first retrograde wave in SCA and FA (Tr, sec), intensity of retrograde bloodflow (RB=Ar/As×Tr), and number of waves (R).

Comparison of velocities of retrograde bloodstreams in SCA and FA in groups with manifest clinical picture of VBI (compensated and decompensated VBI) showed significant differences. The indexes of retrograde bloodflow intensity and amplitude differed significantly in the studied groups (Table 1).

The findings indicate significant differences between some parameters in groups IA and IIA and IB and IIB in comparison with the control. The index reflecting the intensity of retrograde bloodflow in SCA in patients of groups IA and IIA was lower by 18% than in groups IB and IIB. The retrograde bloodflow index in groups IA and IIA in the femoral arteries was significantly (16%; $p<0.05$) lower than in groups IB and IIB. In patients with decompensated VBI the index of proportion between the first systolic wave (systolic period) and second one (early diastolic period) in SCA was higher by 5.4% than in patients with the compensated condition ($p<0.05$). The retrograde bloodflow index in FA in the group with decompensated course was significantly (9%) higher than in compensated VBI.

The negative phases of SCA and FA were poorly discernible (low amplitude and short retrograde phase) in 36% patients with decompensated VBI with atherosclerotic changes in the vascular wall. According to published data, atherosclerosis impairs elasticity and increase of rigidity of the large vessels, which causes modification of the hemodynamic characteristics and leads to early reflection and reduction of the pulse wave amplitude [6-8]. In our studies, the pressure wave return takes place during the late systole, while normally this happens during the early diastole. This suggests insufficient blood delivery to the brain in patients with decompensated VBI.

Reserves of the extracranial contour of cerebral circulation regulation seem to be activated under conditions of insufficient collateral bloodflow in the brain under conditions of VA involvement. It seems that the compensation of inadequate blood delivery to the brain is due to intensification of the retrograde phases of the aortic large arteries, this indicating a possible relationship and development of compensatory mechanisms between the bloodflow in the vertebral arteries and large aortic arteries in extracranial vessels (SCA and FA).

Presumably, the more pronounced were the retrograde waves (by amplitude and duration) in SCA and FA, the more blood was delivered to the brain. This most likely compensates for inadequate blood supply to ischemic tissues in the cerebral basilar compartments. Hence, clinical manifestations of VBI varied from blurred to manifest VBI forms and depended on the status of the large aortic vessels.

Hence, the variability of clinical picture of VBI depends on the severity of vertebral arteries involvement and on the amplitude and duration of retrograde bloodflow phases in SCA and FA. The suggested indexes of retrograde to systolic wave proportion and intensity of the retrograde bloodflow can serve as the criteria of insufficient compensatory reactions and determine the prognosis of VBI development.

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