

Pattern of Changes in Central and Cerebral Hemodynamics in the Basilar Artery during Active Orthostasis in Healthy Individuals

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We studied changes in cerebral and central hemodynamics during active orthostatic load in 26 healthy men aging 20-26 years. Two types of central hemodynamics were revealed: progressive increase in blood pressure throughout the test (type 1) or initial blood pressure increase followed by its decrease with maximum increase in diastolic blood pressure (type 2). The reactions of cerebral hemodynamics to active orthostasis in healthy individuals manifested in reduced cerebral blood flow in the basilar artery. The decrease in blood flow rate in the basilar artery during active orthostasis was associated with vasoconstriction of pial vessels of the vertebrobasilar basin and with the increase in respiratory volume.

Key Words: *active orthostasis; cerebral hemodynamics; central hemodynamics*

Changes in the posture are a reliable way of modulation of the circulatory system [1]. Published data on blood pressure (BP) parameters during the orthostatic load are contradictory. Some authors observed a decrease in the mean BP [6,7,8], while others revealed no changes in BP [6,10] and an increase in diastolic and systolic BP [9] in individuals in upright posture. This controversy can be explained by the use of different orthostatic tests and by differences in the time and methods of evaluation of the analyzed parameters.

There are no common opinion about regularities and mechanisms of changes in cerebral hemodynamics during orthostatic load in healthy individuals. Authors reporting the decrease in the cerebral blood flow rate (CBFR) measured this parameter during the first few seconds of orthostasis [2,3] or during passive orthostasis [8]. Most authors reporting the absence of CBFR changes or its increase applied the procedure of active orthostasis [6] and

calculated the mean values for the whole test [9]. Moreover, the majority of investigators evaluated the postural reactions of cerebral hemodynamics by changes in blood flow parameters in the middle cerebral artery. The data on the effect of postural load on hemodynamics in the basilar artery are scanty.

The aim of the present study was to reveal regularities of changes in basilar blood flow and parameters of central hemodynamics during active orthostatic load in healthy individuals.

MATERIALS AND METHODS

We examined 26 healthy men aging 20-26 years. Evaluation of hemodynamic parameters in the major cerebral vessels, central hemodynamics, and parameters of lung ventilation was performed at rest in supine position for 10 min, then the examinee stood up (active rising) and the parameters were recorded over the next 10 min. We performed simultaneous recording of blood flow parameters in the middle cerebral artery: maximum systolic flow rate (Vs) and end-diastolic rate (Vd) with calculation of the

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mean rate ($V_m = (V_s + 2V_d)/3$, cm/sec) and index of circulatory resistance (Gosling pulsatility index $PI = (V_s - V_d)/V_m$) using a Sonomed 315/M Doppler spectrograph and parameters of systemic hemodynamics: systolic BP (sBP), diastolic BP (dBP), mean (MBP = $dBP + (sBP - dBP)/3$), and HR with subsequent calculation of the hemodynamic profile, including cardiac index and total peripheral resistance (TPR). The recorded parameters of lung ventilation included respiratory volume (recorded using a TYP 44901 volumeter), respiration rate, CO_2 content in the expired air (recorded using a Philips M3046A monitor equipped with a M1460A carbon dioxide sensor).

Statistical analysis was performed using Kruskal—Wallis and Dunnett nonparametric tests. The data were processed using Microsoft Excel and Statistica 5.0 software.

RESULTS

During the orthostatic test, HR in all examinees increased by on average 29.1% per test. In 61.5% cases, BP values gradually increased throughout the test (type 1, group 1, Fig. 1, *a*). In these individuals,

the average increase in systolic, diastolic, and mean BP was 14.8, 31.5, and 22.1%, respectively. Signs of circulatory hyperdynamia with a tendency to vasoconstriction were observed (increase in cardiac index and TPR by 30.8 and 14.6%, respectively, Table 1). In a half of examinees, cardiac index increased throughout the test and by the end of the test this increase was 51.9% from the initial value, while in others, the cardiac index initially increased, but then decreased, though remained above the initial level.

In 38.5% examinees, initial increase in BP parameters followed by their decrease, but not attaining the initial values (type 2 of dynamics, group 2, Fig. 1, *b*). The average increase in systolic, diastolic, and mean BP was 12.4%, 3.1, and 38.8%, respectively. We observed a tendency to circulatory hypodynamia with signs of peripheral vasoconstriction (decrease in cardiac index by 10% and increase in TPR by 59.4%, Table 1). The maximum decrease in cardiac index was 29.2%.

Analysis of cerebral hemodynamics during the orthostatic test revealed a decrease in CBFR in the basilar artery in all examinees despite the increase in BP parameters. In group 1 individuals, the aver-

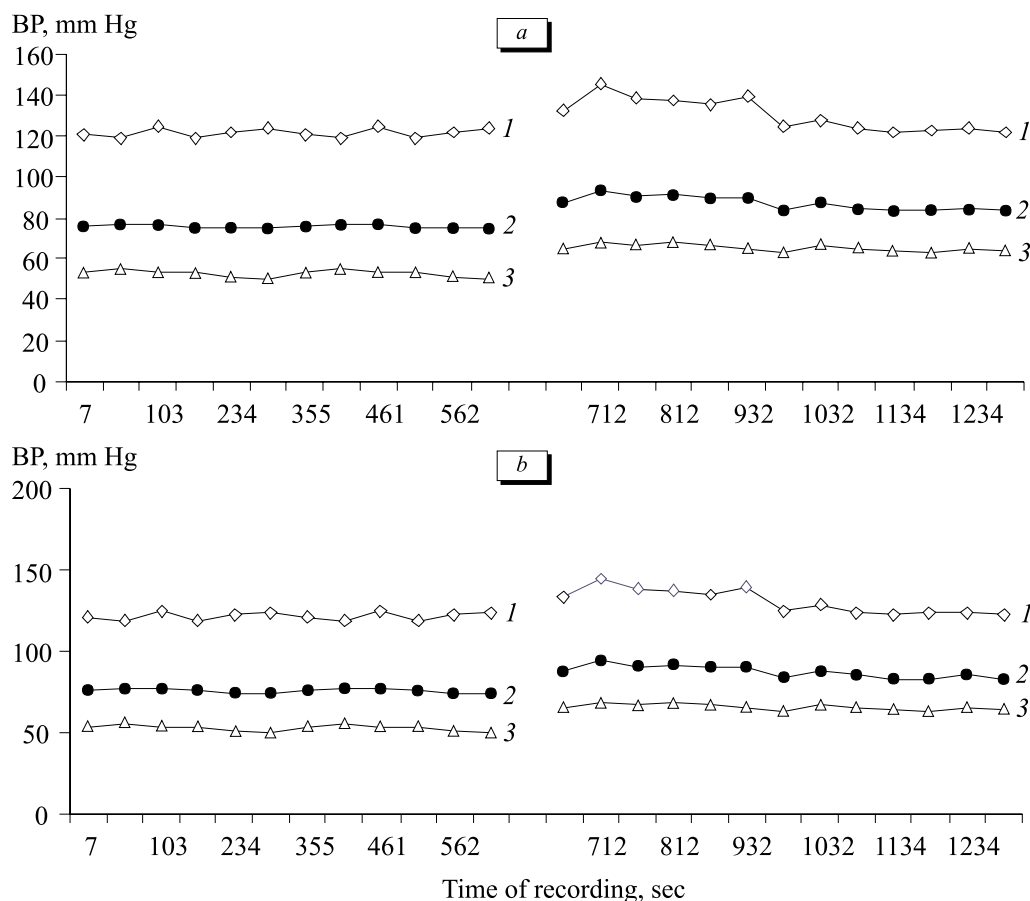


Fig. 1. Dynamics of BP parameters during active orthostasis in healthy individuals. *a*) type 1 of BP dynamics; *b*) type 2 of BP dynamics. 1) systolic BP; 2) mean BP; 3) diastolic BP.

TABLE 1. Parameters of Systemic and Cerebral Hemodynamics (Basilar Artery) at Rest and during Active Orthostasis in Healthy Individuals

Parameter	Group 1 (n=16)		Group 2 (n=16)	
	background	orthostasis	background	orthostasis
LVP, mm Hg	112.7	129.4*	118.2	132.9*
Diastolic BP, mm Hg	44.7	58.8*	44.8	77.9*,**
Mean BP, mm Hg	67.4	82.3*	69.3	96.2*,**
HR, bpm	82.3	105.5*	74.9	97.5*
Cardiac index, liter \times min ⁻¹ \times m ⁻²	3.9	5.1*	4.0	3.6**
TPR, dyne \times sec \times cm ⁻⁵	676	774.9	687.7	1096.2*,**
Blood flow rate, cm/sec				
maximum systolic	69.7	58.9*	71.2	57.9*
end diastolic	35.8	26.8*	37.5	28.3*
mean	47.1	37.5*	46.9	38.2*
Index of circulatory resistance	0.72	0.87*	0.75 (n=5)	0.84
			0.74 (n=5)	0.67**
CO ₂	39.4	35.7	37.1	34.1
Respiration rate	12.8	13.4	15.6**	12.5*
Respiratory volume	493.3	623*	484.5	794.7*

Note. * $p < 0.05$ compared to initial values within the same group; ** $p < 0.05$ between the groups.

age decrease in systolic, diastolic, and mean blood flow rate over the test was 18.3, 33.6, and 25.6%, respectively. A typical feature of CBFR dynamics in these individuals was different degree of the decrease in the rate characteristics by the middle of the test and their increase at the end of orthostasis. In group 2 individuals, the average decrease in systolic, diastolic, and mean blood flow rate over the test was 23, 32.5, and 22.8%, respectively (Table 1). Parameters of CBFR did not return to the initial values; systolic, diastolic, and mean blood flow rate progressively decreased throughout the test (Fig. 2, *a, b*).

In group 1, parameters of cerebral vascular resistance during orthostasis increased by on average 20.8%, while in group 2 nonuniform changes in this parameter were found: in 50% individuals it increased by 12%, in others decreased by 9.5% (Table 1).

In individuals of both groups, the content of CO₂ in the expired air decreased insignificantly, but this decrease did not lead to hypocapnia (Table 1). In group 1 examinees, the respiration rate increased by 1 and the respiratory volume increased by 26%, in group 2 individuals, the respiration rate decreased by 3 and the respiratory rate increased by 64%.

Thus, evaluation of the pattern of changes in central hemodynamics during active orthostasis revealed two types of BP shifts: progressive BP increase throughout the test (type 1) or initial BP

increase followed by its decrease with maximum increase in diastolic blood pressure (type 2).

Type 1 was characterized by signs of circulatory hyperdynamia (increase in cardiac index) and minor increase in TPR within the normal range. These results disagree with most published reports [5,10] where authors observed a decrease in cardiac index and stroke volume and increase in TPR during orthostasis. Increased cardiac output in vertical posture can be explained by mobilization of the blood from depots into venous bed and activation of the musculoskeletal pump, which increased venous return to the heart and, hence, cardiac output, as well as baroreflex-mediated excitation of the vasomotor center, which also determined increased cardiac output in standing posture [11].

In type 2 we observed an increase HR and initial BP increase followed by its decrease with maximum increase in diastolic BP. We observed a tendency to circulatory hypodynamia with sings of peripheral vasoconstriction (increase in TPR and decrease in cardiac index). These results disagree with most published reports [1,6,7,10]. Maximum increase in diastolic BP was related to constriction of collateral peripheral vessels and increase in TPR.

Most authors studying the character of central hemodynamics during orthostatic load observed a decrease in BP parameters [6,7,8] or the absence of BP changes [5,10], which disagree with our findings. BP rise in standing position can be explained

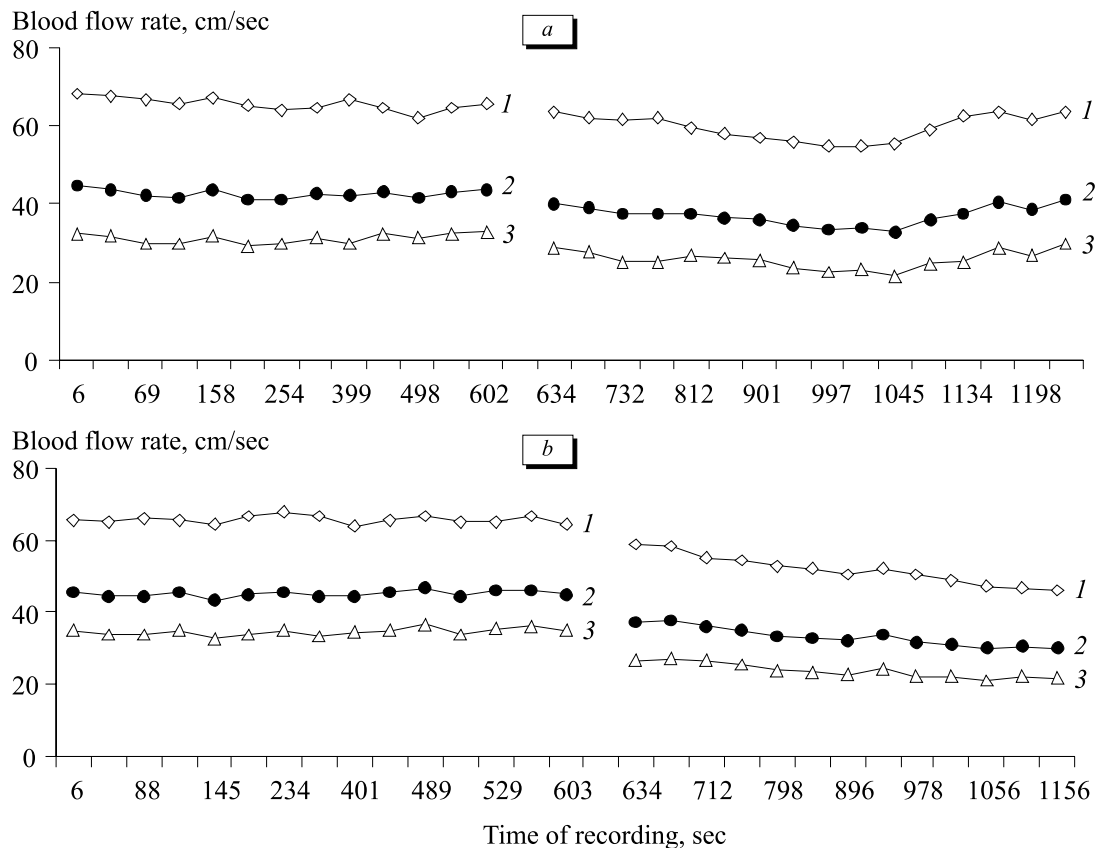


Fig. 2. Dynamics of blood flow rate in the basilar artery during orthostatic test in type 1 (a) and type 2 (b) BP dynamics in healthy individuals. 1) systolic BP; 2) mean BP; 3) diastolic BP.

by increased HR, constriction of peripheral vessels, and increased TPR, which leads to redistribution of the circulating volume and increase in diastolic BP (type 2) and circulatory hyperdynamia (type 1).

Parameters of cerebral hemodynamics during active orthostasis attested to decreased blood flow rate in the basilar artery despite elevated BP. The observed increase in parameters of cerebrovascular resistance in the basin of the basilar artery in examinees of group 1 and 50% examinees of group 2 attested to cerebral vasoconstriction during active orthostasis in healthy individuals, which agrees with published data [2,4,6]. The decrease in cerebrovascular resistance in 50% individuals of group 2 can be explained by autoregulating myogenic vasodilation of pial vessels in the basin of the basilar artery. Autoregulating myogenic vasodilation in the basin of the middle cerebral artery was previously demonstrated [7]. These findings attest to less adequate cerebral autoregulation in the basin of the basilar artery compared to that in the basin of the carotid artery, because signs of vasodilation of pial vessels in the vertebrobasilar basin were detected in only 19.2% examinees.

The decrease in CBFR in the basilar artery during active orthostasis is probably related to blood

redistribution in the standing posture (70% blood is below the heart level [1]), constriction of pial vessels of vertebrobasilar basin, and increase in respiratory volume.

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