EFFECT OF POSTURE ON ELASTIC PROPERTIES OF THE ARTERIES AND HEMODYNAMICS OF HUMAN LIMBS

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Under natural gravitation conditions changes in the position of the body affect the state of the cardiovascular system. Hemodynamic shifts arising in the arterial part of the vascular system have been studied very thoroughly [7, 11], but changes in the elastic properties of the arteries of similar genesis have been studied quite inadequately, possibly because of technical difficulties. Methods used for noninvasive study of the functional properties of arteries, based on determination of the velocity of spread of the pulse wave, do not take into account the thickness of the wall and the lumen of the vessel [8] and, for that reason, they do not allow these properties to be estimated quantitatively in adequate units of measurement. In clinical practice great importance is attached to postural tests, and they are used for the diagnosis of many diseases [2, 7].

The aim of this investigation was to study changes in the elastic properties and hemodynamic shifts which arise in large arteries of the human limbs during changes in the position of the body, by means of a method developed previously [4], whereby a number of important parameters of the functional properties of arteries can be estimated in adequate units of measurement by the use of the same method.

EXPERIMENTAL METHOD

A homogeneous group of healthy male volunteers (15 men) aged 32.9 ± 1.2 years, with a body weight of 77.5 ± 3.2 kg, height 174.3 ± 1.4 cm, and body surface area 1.92 ± 0.04 cm^2, was studied. The subject, lying on his back, had a standard cuff fitted to his arm or the lower third of his leg, and in the phase of smooth compression the blood pressure was measured indirectly (tachyoscillographically and by Korotkov's method), and the pressure in the cuff and its pulse fluctuations were recorded in the phase of stepwise decompression. From the results thus obtained the heart rate (HR) and blood pressure – minimal (P_{min}), mean (P_{mean}), lateral systolic ($P_{l.s.}$), and maximal (P_{max}) – were determined and the pulse pressure (PP), hemodynamic impact (HI), pulse increase in volume of the intact vessel (ΔV_{in}), elastic resistance of the mechanically relaxed (K_0) and intact (K_{in}) vessels, the bulk modulus of elasticity of the intact vessel (E_{in}), the effective internal radius of the vessel (R_{eff}), the degree of mechanical relaxation (ξ), and the exponential index of change in elastic resistance (α) determined by it, were calculated. Full details of the method of investigation and of the calculations used were described previously [4, 5]. The measurements were made on a special tilting table in the following positions: 1) in the initial, horizontal position (angle of tilting of the table 0°); 2) in the passive antiorthostatic position (angle of tilt 20°); 3) in the horizontal position; 4) in the passive orthostatic position (angle of tilt + 70°); 5) in the horizontal position again. The investigations began 15 min after the subject had been placed in one of the above positions, i.e., when the circulation was fully stabilized [3]. Changes in the above-mentioned parameters in response

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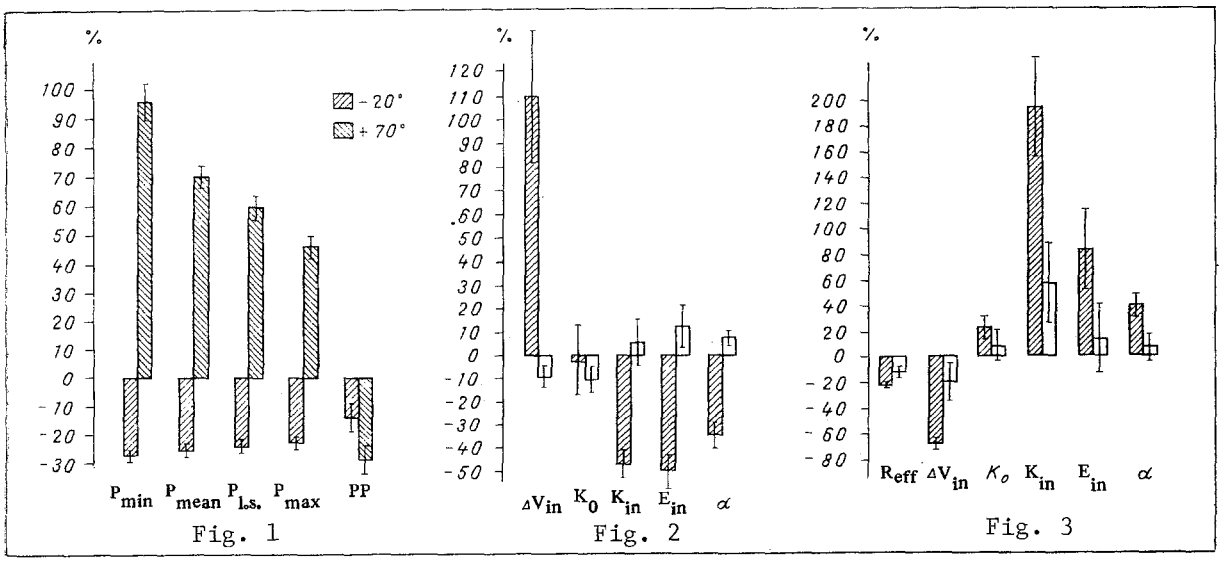

Fig. 1. Degree of response (in percent) of blood pressure in arteries of lower limbs at level of lower third of leg in passive antiorthostatic (—20°) and passive orthostatic (+70°) positions.

Fig. 2. Degree of response (in percent) of elastic properties of arteries in arm and leg in passive antiorthostatic position. Shaded columns — leg; unshaded columns — arm.

Fig. 3. Degree of response (in percent) of elastic properties of arteries of arm and leg in passive orthostatic position. Legend as to Fig. 2.

to a change in position of the body were quantified as the deviation (in percent) from the value in the horizontal position. The results were subjected to statistical analysis by the difference method using Student's t test.

EXPERIMENTAL RESULTS

It will be clear from Fig. 1 and Table 1 that the blood pressure in the lower limbs changed significantly with a change in position of the body: it fell in the passive antiorthostatic position and rose in the passive orthostatic position, due to the difference in hydrostatic pressure gradients in these positions; the relative change of blood pressure was inversely proportional to the pressure itself.

During any change in position of the body the blood pressure in the arms did not change significantly (15 of 18 measurements), which was to be expected because the portion of the limb studied remained virtually at the phlebostatic level and was not exposed to the action of the hydrostatic pressure gradient. The exception was a significant ($P < 0.05$) increase in P_{min} from 77.5 ± 2.4 to 86.1 ± 4.0 mm Hg and a corresponding decrease in PP from 42.6 ± 2.4 to 35.3 ± 3.0 mm Hg in the passive orthostatic position, and also a significant ($P < 0.05$) increase in P_{mean} from 91.8 ± 2.2 to 94.9 ± 1.4 mm Hg in the passive antiorthostatic position. A similar increase in the mean pressure was obtained by other workers previously [10].

The data given above, demonstrating regional differences in responses of the blood pressure, confirm their dependence on the hydrostatic factor [7]. A change in blood pressure and, in particular, a decrease in PP, may indirectly point to changes in the elastic properties of the arteries in response to a change of posture.

It will be clear from Fig. 2 that in the passive antiorthostatic position the pulse filling index ΔV_{in} rose considerably and K_{in}, E_{in}, and α decreased, whereas the parameter K_o, independent of the intravascular pressure and determined by the morphological and functional properties of the vessel [4], did not change significantly. In this same position ΔV_{in} and the elastic properties of the arteries (K_{in} and E_{in}) of the upper limbs did not

TABLE 1. Mean Parameters (M ± m) of Elastic Properties of Arteries and Hemodynamics in Human Lower Limbs during Changes of Posture ($P < 0.001$)

Parameter tested	0°***	−20°	0°***	+70°	0°***
HR, beats/min	63,0±2,6	62,4±2,4***	63,0±3,1	75,6±2,6*	60,6±2,7
P_{min}, mm Hg	74,7±2,7	53,5±2,0	78,9±2,2	153,8±3,1	81,0±2,4
P_{mean}, mm Hg	97,5±2,8	72,5±2,6	99,2±2,2	169,1±3,5	102,5±2,5
$P_{l.s.}$, mm Hg	109,5±2,9	83,3±3,5	110,8±2,5	177,1±4,3	116,0±3,4
P_{max}, mm Hg	128,3±3,6	99,4±3,9	129,4±4,2	189,2±5,2	132,2±3,8
PP, mm Hg	53,7±3,0	45,9±3,1	50,5±3,3	35,6±3,1	51,2±3,5
HI, mm Hg	18,8±2,2	16,1±1,8***	18,5±2,3	12,2±1,9**	16,2±2,1
R_{eff}, cm x 10	1,93±0,06	1,89±0,07*	1,94±0,05	1,51±0,06	1,78±0,06
ΔV_{in}, cm^3 x 10	1,71±0,12	3,44±0,42	1,68±0,20	0,44±0,04	1,30±0,10
K_0, dynes/cm^5 x 10^{-4}	6,36±0,49	6,08±0,55***	6,33±0,58	7,64±1,15**	7,38±0,72
K_{in}, dynes/cm^5 x 10^{-5}	4,68±0,46	2,49±0,48	5,10±0,48	11,92±1,89**	5,79±0,58
E_{in}, dynes/cm^2 x 10^{-6}	1,62±0,16	0,78±0,14*	1,67±0,16	2,64±0,31*	1,61±0,19
	2,01±0,07	1,28±0,11	2,14±0,10	2,72±0,08	2,11±0,11

*$P < 0.01$.
**$P < 0.05$.
***Differences not statistically significant.

change significantly, apart from a small but significant ($P < 0.05$) decrease in K_0 and a corresponding increase in α. The increase in P_{mean} in the antiorthostatic position, observed previously, was evidently connected with this decrease in K_0.

It will be clear from Fig. 3 that the pulse increase in volume of the vessels in the lower limbs in the passive orthostatic position was significantly reduced whereas K_{in}, E_{in}, and α increased significantly, and that K_0 increased, but not significantly. In this same position the elastic properties of the arteries of the upper limbs did not change significantly, but the lumen of the arteries decreased both in the legs (Table 1) and in the arms, where the decrease in R_{eff} from 2.02 ± 0.08 to 1.84 ± 0.07 mm was statistically significant ($P < 0.01$).

During the change into the orthostatic position, both the stroke volume of the heart and the cardiac output are reduced [1, 7, 9, 11]. Since this is so, there is reason to suppose that the decrease in the lumen of the arteries of the arm was evidently connected with the decrease in the stroke volume, and that the increased hydrostatic pressure gradient also exerted an additional influence on the decrease in lumen of the arteries of the leg, possibly through a Bayliss mechanism. This suggestion also was confirmed by the decrease in ΔV_{in}, which itself may affect vascular tone [6]. The increased intravascular pressure in the lower limbs in the passive orthostatic position evidently determines changes in the elastic properties of the arteries of the leg.

It can thus be concluded that the hydrostatic pressure gradient is the cause of the changes observed.

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EFFECT OF VARIABLE HYDROSTATIC PRESSURE ON THE PLATELET–VESSEL WALL SYSTEM

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The important role of disturbances of platelet-vascular hemostasis in the pathogenesis of circulatory disorders can be taken as established [6], although many apsects of regulation of the functional state of the platelet–vessel wall system remain unexplained, and this is an obstacle in the way of the development of methods of adequate correction of its disorders. The mechanism of regulation of the functional state of rabbit platelets has been described [5]: essentially exposure of a platelet suspension to variable hydrostatic pressure (VHP) on an apparatus simulating the conditions which exist in the real blood flow leads to increased ability of the platelets to aggregate and to release ADP. A new approach is thus obtained to the understanding of the mechanisms of development of disorders of hemostasis, especially during fluctuations of the general arterial pressure (AP). It also seemed important to study the problem of whether such a control mechanism also extends to the hemostatic function of the vessel wall, especially in the light of data on the ability of the vascular endothelium to produce substances effectively influencing platelet aggregation and blood coagulation [1, 8].

This paper gives the results of a study of the effect of VHP on various parameters of the functional state of human platelets and the hemostatic function of the vascular wall.

EXPERIMENTAL METHOD

Platelet-rich plasma was obtained from fresh citrate-stabilized blood from clinically healthy blood donors. Platelet aggregation was tested as described previously [2]. Adhesion of platelets to glass was tested by the method in [4]. Blood vessels for investigation were obtained from 15 human cadavers during the first 8 h after death, which was from various causes unconnected with cardiovascular diseases. In the course of the investigations the antiaggregating activity of the vessel wall [7] and the effect of an extract of vascular endothelium on the blood recalcification time, which reflects the blood concentration of thromboplastic factor [1], were estimated. The platelet suspension was exposed to VHP by the method in [5]. During investigation of the effect of VHP on the vessel wall, the same apparatus was used, but the segment of vessel (0.5-0.7 cm) was connected to a syringe, to the plunger of which pressure pulses were applied. The results were subjected to statistical analysis, using the nonparametric Wilcoxon–Mann–Whitney criterion.

EXPERIMENTAL RESULTS

VHP over 100 mm Hg, with a maximum at 180-220 mm Hg, significantly increases the aggregating activity of platelets induced by ADP, serotonin, and adrenalin (Fig. 1). The fact that the effect obtained on human platelets was similar to that obtained on rabbit platelets [5] and that it was independent of the type of aggregant used demonstrates the

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