These biomechanical, biochemical, and morphological studies of varicose veins showed that if they are preserved in Hanks' solution, activity of the enzymes maintaining the basic processes of energy formation in the cell is preserved, evidence that the tissue of the vessel remains viable. In the case of conservation in formaldehyde, enzyme activity lasts only 2 days. Later, destruction of the tissue structures takes place, leading to a stable decrease in enzyme activity, an increase in acidosis and, as a result, loss of viability of the tissues. The mechanical properties of the veins, however, remain at quite a high level.

It can thus be concluded on the basis of the above facts that in the absence of autologous venous material for prosthetic operations on the main lower limb arteries, venous allografts conserved in Hanks' solution and in a 2 solution of neutral formalin can be used. In this case the optimal keeping time is 1 day. Grafts conserved in Hanks' solution also remain viable, as shown by their biomechanical, biomorphological, and biochemical parameters, for up to 7 days whereas conservation in a 2% solution of neutral formalin permits their use for not longer than 2 days.

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CHANGES IN THE SYMPATHOADRENAL SYSTEM DURING STRESS IN RATS WITH HIGH AND LOW RESISTANCE TO ACUTE HYPOXIA

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A leading role in the development of stress, which has many pathogenetic links in common with hypoxia [3], is played by changes in activity of the sympathoadrenal system (SAS) [4], and the possibility therefore cannot be ruled out that the initial level of activity of SAS, which may perhaps be genetically determined, can influence the resistance of animals to hypoxia, and also determine differences in their response to stress. Consequently, in animals differing in their sensitivity to acute hypoxia (AH) differences may be observed in the time course of changes in the sympathetic and adrenal components of the SAS during stress. Meanwhile no morphological investigations of SAS during stress in animals differing in their resistance to AH have been undertaken.

The aim of this investigation was to study the SAS of adult rats with low (LRH) and high (HRH) resistance to AH associated with catecholamine-induced stress.

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TABLE 1. RALT of Ventricles of the Heart of Control Rats of Three Groups (intact, LRH, HRH) (M ± m)

Group of animals	Zone of ventricle							
	apical		middle		basal		ventricle as a whole	
	RV	LV	RV	LV	RV	LV	RV	LV
Intact rats [5]	38,6±4,22	38,4±6,94	36,7±3,04	31,4±2,76	44,4±2,36	33,7±1,81	39,9±1,96	34,5±2,57
NRH (11)	30,1±1,93	27,8±2,9	37,1±2,27	35,2±2,11	43,9±1,31	33,8±1,37	37±1,18	32,2±1,09
HRH (5)	33,5±3,97	34,3±3,31	36,8±2,32	38,8±5,34	50,3±3,95	44,3±5,05	40,2±3,10	39,2±3,3

Legend. Here and in Tables 1-3, number of animals tested shown between parentheses.

TABLE 2. Changes in RALT of Ventricles of the Heart in LRH During Stress

Control and period of stress		Percentage by which M_1 was greater		Part of the heart	
$M_1 \pm m_1$ (n_1)	$M_1 \pm m_2$ (n_2)	M_2	p		
1 h (12)	Control (11)				
37,6±1,82	32,2±1,09	16,8	<0,02	Ventricle as a whole	LV
43±2,56	33,8±1,37	27,2	<0,01	Basal zone	
45,1±3,1	37±1,18	21,9	<0,05	Ventricle as a whole	RV
40,2±4,2	30,1±1,93	33,6	<0,05	Apical zone	
Control (11)	6 h (10)				
32,2±1,09	27,1±1,9	18,8	<0,05	Ventricle as a whole	LV
35,2±2,11	27,3±2,48	28,9	<0,05	Middle zone	
37±1,18	29,5±1,77	25,4	<0,01	Ventricle as a whole	
30,1±1,93	22,5±2,06	33,8	<0,02	Apical zone	RV
37,1±2,27	30,1±1,49	23,3	<0,02	Middle zone	
43,9±1,31	36,3±2,51	20,9	<0,02	Basal zone	
1 h (12)	6 h (10)				
37,6±1,82	27,1±1,9	38,7	<0,01	Ventricle as a whole	
34,1±1,89	22,8±2,56	49,6	<0,01	Apical zone	LV
36,6±2,46	27,3±2,48	34,1	<0,02	Middle zone	
43±2,56	30,7±1,84	40,1	<0,01	Basal zone	
1 h (12)	6 h (10)				
45,1±3,1	29,5±1,77	52,9	<0,01	Ventricle as a whole	
40,2±4,2	22,5±2,06	78,7	<0,01	Apical zone	
42,8±2,46	30,1±1,49	42,2	<0,01	Middle zone	RV
50,8±4,09	36,3±2,51	39,9	<0,01	Basal zone	

TABLE 3. Changes in RALT in Ventricles of the Heart in HRH During Stress

Conrol and period of stress		Percentage by which M_1 was greater		Part of the heart	
$M_1 \pm m_2$ (n_1)	$M_2 \pm m_2$ (n_2)	M_1 in excess of M_2	p		
1 h (20)	6 h (3)				
38,4±2,04	25,7±1,73	49,4	<0,01	Ventricle as a whole	
39,9±4,73	22,1±1,84	80,5	<0,02	Apical zone	LV
40,5±1,68	25,7±3,16	57,6	<0,01	Middle zone	
49,9±4,85	29,7±1,9	68	<0,02	Ventricle as a whole	
46,5±5,93	24,9±2,58	86,7	<0,05	Apical zone	
52,4±7,32	29,3±0,068	78,8	<0,05	Middle zone	RV
50,8±2,14	34,7±3,54	46,4	<0,02	Basal zone	
Control (5)	6 h (3)				
39,2±3,34	25,7±1,73	52,5	<0,02	Ventricle as a whole	
34,3±3,31	22,1±1,84	55,2	<0,02	Apical zone	LV
44,3±5,05	29,3±1,67	51,2	<0,05	Basal zone	
40,2±3,1	29,7±1,9	35,4	<0,05	Ventricle as a whole	
36,8±2,32	29,3±0,068	25,6	<0,02	Middle zone	RV
50,3±3,95	34,7±3,54	45	<0,05	Basal zone	

EXPERIMENTAL METHOD

Male Wistar rats were tested twice for resistance to AH by the method in [1]. The survival time of HRH exceeded 5 min, that of LRH was less than one-third of this amount. Catecholamine-induced stress was created 3 weeks after repeated testing of LRH and HRH weighing 350-400 g by intraperitoneal injection of a 0.2% solution of noradrenalin hydrotartrate in a dose of 2 mg/kg body weight. The heart and left adrenal were removed under thiopental anesthesia (200 mg/kg body weight) after 1 h and 6 h in LRH and HRH. Intact animals, and also LRH and HRH receiving injections of physiological saline, served as the control. Altogether 71 rats were tested. The adrenergic nerves of the heart were revealed by the method in [6] in trans-

TABLE 4. CA Concentration and Area of Complexes of Chromaffin Cells in Adrenal Medulla (M ± m)

Group of animals	Concentration		Area of NA-complexes
	CA	NA	
Intact rats	57,8±1,7 (8)	36±0,9 (13)	16,8±1,5 (13)
LRH: control	55,6±0,7 (19)	35,2±0,5 (20)	15±0,9 (19)
1 h of stress	57,5±0,8 (11)	35,5±0,7 (10)	13,3±1 (10)
6 h of stress	55,5±1,1 (9)	35,7±0,8 (9)	13,1±1,5 (9)
HRH: control	57,7±0,9 (5)	36,4±3,7 (5)	14,9±2,3 (5)
1 h of stress	56,9±1,1 (8)	35,2±1,8 (6)	15,5±1,8 (6)
6 h of stress	58,4±1,5 (5)	36,9±2,2 (5)	17,3±2,4 (5)

verse frozen sections through the heart, 10 μ thick and cut at three levels: apical, middle, and basal. The sections were incubated in a solution of glyoxylic acid for 5-6 min. The relative area of luminescent terminals (RALT) was determined by means of a Stropus grid [5], in the circular parts of the ventricles under a magnification of 280× on a Lyumam-IZ microscope. The mean value for the ventricle, characterizing RALT for the ventricle as a whole, also was calculated. Total catecholamines (CA) of the adrenal chromaffin cells were detected in frozen sections 10 μ thick, stained by Falck's method in the modification of Cottle and Nash [7], whereas noradrenalin (NA) was determined by the method in [6], using the FMEL-IV 9.2 fluorometric attachment with 1.5 μ probe, and 10× objective (CA), and 0 5 μ probe, 20× objective (NA). The relative area of the NA-cells was determined with the aid of a Stropus grid (140×). In each case no fewer than five sections through the adrenals were studied. The results were subjected to statistical analysis by Student's test.

EXPERIMENTAL RESULTS

Analysis of the results of the quantitative histofluorometric study of the myocardial adrenergic innervation of the control rats showed (Table 1) that RALT in the basal zone of the heart was greater in the right (RV) than in the left ventricle (LV) of the intact animals by 31.8%, and in LRH by 29.9% ($p < 0.01$). In LRH, RALT in RV was 14.9% greater than in LV ($p < 0.01$). Thus in intact rats and in LRH, an interventricular gradient (IVG) of RALT was discovered. In HRH no difference was found between the ventricles.

In LV of LRH and HRH, RALT was greater in the basal zone than in the apical by 61.4% ($p < 0.01$) and 50.1% ($p < 0.02$) respectively, i.e., an apicobasal gradient (ABG) was found. In RV of the intact rats and LV of all the control groups RALT in the basal and apical zones was the same. These findings are in agreement with those of [4], which showed a more abundant innervation of RV of the rat heart than of LV, and of the basal zone compared with the apical zone. The present study also showed that in all the control animals RALT of the corresponding parts of the heart was virtually identical. Consequently, testing twice did not affect the number of fluorescent terminals in the ventricles of the heart, but changed only the ratio between RALT of its different parts. It can also be tentatively suggested that the differences in resistance to AH were not due completely to the adrenergic innervation of the heart.

During stress RALT of the parts of the ventricular myocardium studied changed in both LRH and HRH. It follows from Table 2 that RALT in LRH was increased 1 h after injection of NA compared with the control in the basal zone of LV by 27.2% ($p < 0.01$) and in the apical zone of RV by 33.6% ($p < 0.05$). By 6 h, RALT had fallen in all zones of both ventricles compared with 1 h, and was also found to be lower than control values in all zones of RV and in the middle zone of LV (RALT in the apical and basal zones of LV did not exceed the limits of the control values). Unlike LRH, in HRH a tendency was observed after 1 h for RALT to increase in the apical zones of both ventricles and the middle zone of LV. In the basal zone of LV of LRH, RALT was greater than in HRH by 23.2% ($p < 0.05$). Analysis of the data in Table 3 shows that RALT, in HRH just as in LRH, was lower than the control level in the basal zone of both ventricles, and in the apical zone of LV and the middle zone of RV. In all parts of the heart studied RALT in the comparable groups 6 h after injection of NA became virtually identical. Changes in RALT in the parts of the heart studied in LRH and HRH during stress led to changes in its IVG and ABG. The IVG of RALT, present in the control in LRH and absent in HRH, was found 1 h after injection of NA in both groups of animals: RALT in RV was greater than in LV in LRH by 20% ($p < 0.05$) and in HRH by 45.6% ($p < 0.01$) (in the

basal zone of the heart). After 6 h of stress, IVG of RALT disappeared in both LRH and HRH. Thus independently of the control value, IVG of RALT showed identical changes during stress in LRH and HRH; toward 6 h, moreover, in HRH, IVG of RALT was absent, just as in the control, whereas in LRH it had not yet returned to the control level by this time. The ABG observed in the control in RV was not found 1 h after injection of NA in either LRH or HRH, but by 6 h the gradient was restored in LRH (RALT in the basal zone was 61.3% higher than in the apical zone ($p < 0.01$), but it was still absent in HRH. Despite the absence of ABG of RALT in the control in LV of both groups of animals, during stress it appeared in LRH as early as after 1 h (34.6%, $p < 0.02$) and it remained until 6 h (34.6%, $p < 0.05$), whereas in HRH, ABG of RALT did not appear until 6 h (32.6%, $p = 0.05$). Thus in LRH and HRH, RALT in both ventricles changed identically at the different periods of stress: in RV by 1 h and in LV by 6 h; under these circumstances in LRH the gradient in RV was restored to the control level as early as after 6 h, whereas in LV of LRH, and in both ventricles of HRH, it remained altered.

The study of the adrenals (Table 4) revealed no changes in concentration of CA and area of complexes of NA-containing cells in the adrenal medulla 3 weeks after the second testing, and no differences likewise between LRH and HRH in the control and in stress 1 and 6 h after injection of NA.

Thus an increase in RALT during the 1st hour of stress, connected with an increase in the NA concentration in the adrenergic nerves of the heart is due to uptake of exogenous NA which, according to data in [2], is eliminated in the course of a few minutes from the blood stream by the tissues; under these circumstances the increase in the extraneuronal concentration of NA stimulates its neuronal uptake (uptake 1) [8]. The increase in RALT in the heart of the LRH during stress is evidence that in the heart of the control animals there are terminals containing a small amount of mediator (and for that reason invisible during luminescence microscopy), as well as terminals with a high NA concentration. This emphasizes the functional heterogeneity of the terminal network and its reserve in the context of possible NA uptake from outside, and its synthesis in the nerve fiber. In HRH no increase was observed in RALT during CA-induced stress, evidence that the sympathetic system of the heart in HRH is less able to take up the mediator and, consequently, has a weaker protective function than in LRH, for uptake of NA by adrenergic nerves may cause a reduction in the degree of myocardial damage [4]. The present investigation showed a decrease in RALT toward 6 h of the experiment in all zones of the ventricles in both LRH and HRH; moreover, there was no difference in RALT between the corresponding zones of the ventricles in the two groups of animals. In noninbred rats, however, a decrease in the number of luminescent terminals was found in both ventricles during the first minute after injection of NA, and their number remained low until 6 h [4]. These differences are perhaps connected with differences in the mode of function of the SAS in different strains of rats. Reduction of RALT was associated with a decrease in the NA concentration in the terminals, and was largely due to its release from the axoplasm as a result of injury and increased permeability of the axolemma [4]. Thus the chromaffin cells of the adrenal medulla of intact rats and LRH and HRH contained an equal amount of CA. In CA-induced stress no change in the CA concentration was found after 1 h and 6 h in the adrenal medulla of LRH and HRH. In LRH, 1 h after the beginning of CA-induced stress, RALT was increased in the apical zone of RV and the basal zone of LV; in HRH no change in RALT was observed during this period, as a result of which RALT in LRH in the basal zone of LV after 1 h became greater by 23.2% than in HRH. After 6 h RALT in all zones of the heart of both groups of animals was reduced; RALT of the corresponding zones of LV and RV was virtually identical, moreover, in LRH and HRH. In LRH in the control RALT was greater in RV than in LV, i.e., an interventricular gradient (IVG) was present. No difference was found in HRH. In RV of the control LRH and HRH, RALT in the basal zone of the heart was greater than in the apical zone, i.e., an apico-basal gradient (ABG) was present. In LV of the heart of both groups of rats no differences were found. Irrespective of the initial values of IVG and ABG of RALT showed changes identical in direction during stress in LRH and HRH; moreover, the control values recovered more rapidly: of IVG in HRH and of ABG in LRH. Thus in LRH, during the first 6 h of CA-induced stress, a biphasic reaction of the sympathetic system of the heart (SSH) was discovered. In LRH and HRH the sympathetic component of the SAS is more labile during stress than the adrenal component.

In Wistar LRH and HRH, despite sharp differences in resistance to hypoxia, the response of the SSH to stress is similar in type. The small differences in the response of SSH present at the beginning of CA-induced stress (until 1 h) subsequently disappear (by 6 h).

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