An Appropriate Increase in the Proportion of Blood Flow through the Superior Vena Cava to the Total Venous Return during Systemic Cardiovascular Responses

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Systemic pressor and depressor reactions induced by catecholamines, angiotensin II, adrenoblockers, antianginal preparations (corinfar, mesotrin, nitroglycerin), hypoxic hypoxia, and hypothermia in normo- and hypertensive animals are accompanied by an appropriate decrease in the proportion of blood flow through the superior vena cava and an increase in that through the inferior vena cava in the total venous return.

Key Words: *blood flow; cardiovascular reactions; vena cava*

It has been demonstrated that during stable operation of the human and animal cardiovascular system the blood flow through the superior vena cava (SVC) comprises 1/3 and that through the inferior vena cava (IVC) 2/3 of the total venous return (VR) [7,9,10]. In cats, these parameters were found not to differ considerably from the above values, ranging from 29 to 38% and from 62 to 71%, respectively [1,2,4-6]. It has been reported that during transient processes in the circulation and in systemic responses to various endogenous and exogenous stimuli the arterial and venous reactions of certain vascular beds differ in intensity and direction [7,8]; therefore, their contribution to the alterations in VR is different [11]. The relationship between changes in blood flow through the venae cavae and the dynamics of cardiovascular reactions is unclear. Yet it is important for understanding the mechanisms responsible for the formation of the total VR, which determines changes in cardiac output and blood pressure. Our goal was to investigate the relationship between blood flows through the venae cavae during systemic pressor and depressor reactions and to elucidate the hemodynamic mechanisms underlying changes in the ratio between these parameters.

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MATERIALS AND METHODS

Experiments were performed on cats (n=138) under urethane and chloralose anesthesia (0.9 and 0.01 g/kg, respectively). The thorax was opened with an electrocauter, and assisted ventilation was established.

Changes in systemic hemodynamics were studied during pressor and depressor cardiovascular reactions manifested as an increase and decrease in blood pressure, respectively. The pressor reactions were induced by a bolus intravenous injection of catecholamines (epinephrine or norepinephrine) and angiotensin II with and without blockade of α- or β-adrenoreceptors. The depressor reactions were provoked by injection of adrenoblockers (the α-blocker regitin and the β-blocker propranolol), prostaglandin A_1, antianginal preparations (corinfar, mesotrin, nitroglycerin), by hypoxic hypoxia (inhalation of a 10% O_2/90% N_2 mixture), and hypothermia (cool-

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ing of the body to 30°C). The doses of the vasoactive agents are given in Tables 1 and 2. All these stimuli were applied to normotensive animals. Cats with neurogenic or nephrogenic [3] experimental hypertension were injected catecholamines, angiotensin II, and adrenoblockers.

The following parameters were recorded: blood pressure in the left subclavian artery (EMT-34 electromanometer, Elema), cardiac output in the ascending aorta, and blood flow through the SVC and IVC (an MF-5 and MFV-2100 electromagnetic flowmeters, Nihon Kohden). The total peripheral vascular resistance was calculated in a DVK-32 digital complex from blood pressure and cardiac output, and the total venous return was calculated from the blood flow through each vena cava.

A two-channel pump with a constant output (Institute of Experimental Medicine, Russian Academy of Medical Sciences) was used in experiments with a separate perfusion of the vascular beds of the brachiocephalic artery and descending thoracic aorta; blood was collected from the ascending aorta. Hemodynamic parameters were recorded in a Mingograph-81 apparatus (Elema).

RESULTS

Here we present quantitative characteristics of the alterations occurring in blood flows through the venae cavae during systemic reactions. Since the quantitative characteristics of blood pressure, total vascular resistance, and cardiac output require special consideration, they are characterized only qualitatively.

During pressor reactions against the background of increased blood pressure, cardiac output, and total peripheral vascular resistance, VR always increased as well as the blood flows through the SVC and IVC. The depressor systemic shifts accompanied by a decrease in blood pressure and peripheral blood resistance were characterized by a different (compared with pressor reactions) direction of changes in VR (as well as in the cardiac output). For example, the total VR (and cardiac output) and blood flows through the SVC and IVC decreased after administration of α- and β-blockers, mesotrin, and hypothermia and increased in response to hypoxia and corinfar. The decrease in the total VR caused by nitroglycerin and prostaglandin A_1 coincided with a decrease in blood flow through the IVC and an increase in blood flow through the SVC.

Quantitative analysis of changes in the venous circulation has shown that during both pressor and depressor reactions induced by the studied stimuli the ratio between blood flows through the SVC and IVC changed compared with the baseline value. Since the ratio between the blood flows through the venae cavae was calculated relative to the total VR, an increase in the proportion of blood flow through one vena cava was accompanied by a corresponding decrease in the proportion of blood flow through the other. The analysis has shown that the contribution of the blood flow through the SVC to the total VR increased while that of the blood flow through the

TABLE 1. Changes in the Proportion of Blood Flow through the Superior Vena Cava (SVC) Contributing to the Shifts of Venous Return (VR) during Systemic Pressor Reactions

Stimulus	Animals	Mean augmentation of blood flow through SVC relative to total VR, Δ%,* and state of cardiovascular adrenoreceptors		
		intact adrenoreceptors	blocked	
			α-adrenoreceptors	β-adrenoreceptors
Epinephrine, 5, 10 μg/kg	NTA	2.5 (12)	0.2 (12)	7.5 (11)
	NGH	11.1 (4)	0.8 (4)	3.8 (6)
	NPHH	7.6 (11)	-1.1 (11)	10.8 (8)
Norepinephrine, 1.2 μg/kg	NTA	2.0 (13)	1.0 (13)	4.1 (8)
	NGH	11.8 (6)	5.2 (6)	6.0 (8)
	NPHH	6.2 (13)	1.6 (13)	8.9 (10)
Angiotensin II, 0.5 μg/kg	NTA	5.8 (12)	4.6 (12)	8.1 (7)
	NGH	17.5 (4)	5.4 (4)	3.3 (6)
	NPHH	4.9 (11)	3.8 (11)	4.3 (8)

Note. *The errors of the mean were lower than a tenth of the given values. Here and in Table 2: NTA, normotensive animals; animals with neurogenic (NGH) and nephrogenic (NPHH) experimental hypertension. A minus sign indicates a decrease in the blood flow through the SVC. The number of observations is given in parentheses.

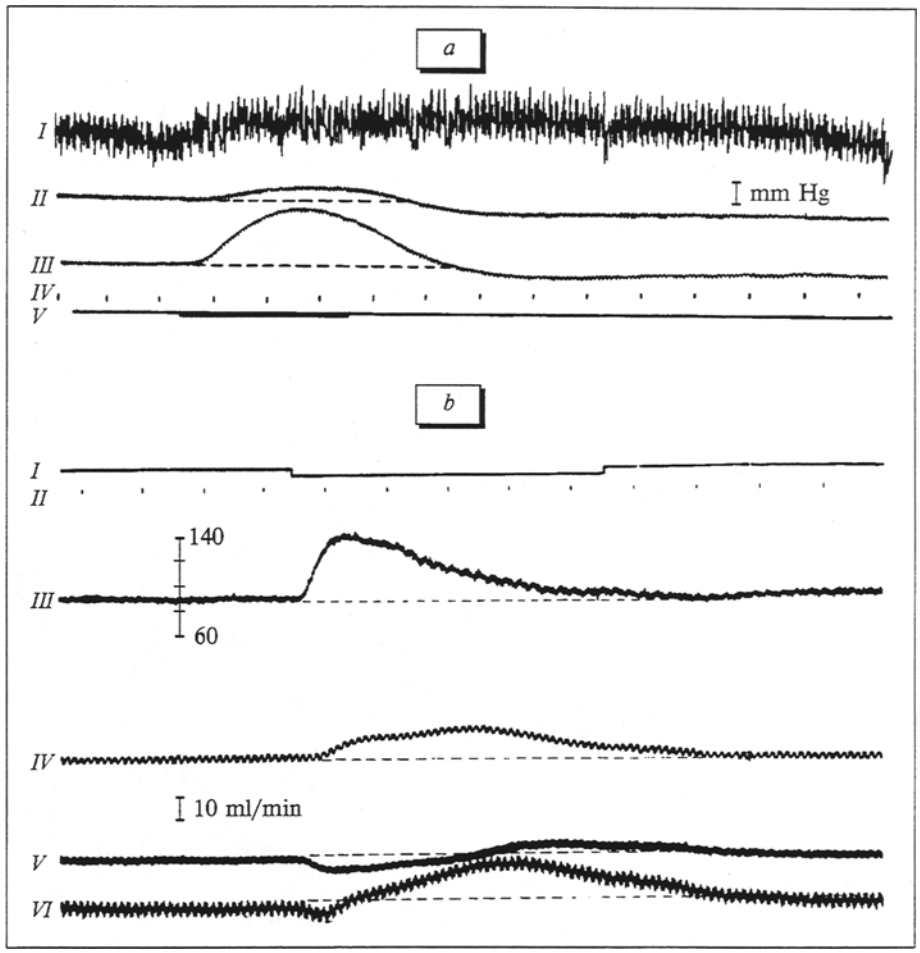

Fig. 1. More pronounced changes in the resistance of the descending aortic vascular bed compared with those in the resistance of the brachiocephalic arterial bed (*a*) and contralateral changes in the blood flow through these vessels (*b*) upon stimulation of sciatic nerve fibers (10 V, 30 pulses/sec, 5 msec). *a*: *I*) aortic pressure, mm Hg; *II*) perfusion pressure in the brachiocephalic arterial bed, mm Hg; *III*) perfusion pressure in the descending aortic bed, mm Hg; *IV*) time marker, 20 sec; *V*) stimulation marker, mm Hg scale. *b*: *I*) stimulation marker; *II*) time marker, 10 sec; *III*) arterial pressure, mm Hg; *IV*) blood flow through the brachiocephalic artery; *V*) blood flow through the descending aorta; *VI*) cardiac output, ml/min.

IVC were observed in the vast majority of experiments: in 26 out of 27 series with pressor stimuli (Table 1) and in 11 out of 12 series with depressor stimuli (Table 2). Only in animals with nephrogenic hypertension did the proportion of blood flow through the SVC decrease and that through the IVC increase in response to blockade of β-receptors and administration of epinephrine against the background of α-receptor blockade (Tables 1 and 2), although in some cases opposite changes were observed. The increase in the proportion of blood flow through the SVC (and the decrease in that through the IVC) varied considerably: from 0.2 to 17.5% during systemic pressor reactions (Table 1) and from 1.2 to 10% during depressor reactions (Table 2).

The greatest increase in the proportion of blood flow through the SVC and decrease in that through the IVC have been recorded at maximal changes in the total peripheral vascular resistance which outstrip the maximum changes in venous blood flow [5,6]. Therefore, it can be assumed that changes in the ratio between blood flows through the venae cavae do not depend on the total vascular resistance but on the resistances of its regional components: the vascular beds of the brachiocephalic artery and of the descending thoracic aorta, which provide venous outflow through the SVC and IVC, respectively.

This hypothesis was tested in a separate series of experiments in which the resistance of these vascular beds during pressor systemic reactions was measured by resistographic methods. In experiments with separate perfusion of the vascular beds of the brachiocephalic artery and descending aorta electrical stimulation of the afferent fibers of the sciatic nerve (10 V, 30 pulses/sec, 5 msec) induced a 4-fold greater increase in the vascular resistance of the descending aortic bed compared with that of the brachiocephalic arterial bed (35.6 and 9% compared with the baseline value, Fig. 1, *a*). Under conditions of natural circulation the different increase in the vascular resistance of these beds may lead to a redistribution of cardiac output between them: an increase in the blood flow through the brachiocephalic artery and a decrease followed by an increase in the blood flow through the descending aorta (Fig. 1, *b*). Therefore, it can be hypothe-

TABLE 2. Increase in the Proportion of Blood Flow through the Superior Vena Cava in Changes in Venous Return during Systemic Depressor Reactions

Stimulus	Animals	Mean values of augmentation in blood flow through SVC relative to total VR, Δ%
Hypoxic hypoxia (inhalation of a 10% O_2/90% N_2 mixture for 20 min)	NTA	3.8 (10)
Hypothermia (cooling of the body to 30°C)	NTA	4.1 (12)
Blockade of adrenoreceptors:		
0.2 and 0.3 mg/kg regitin (α-blockade)	NTA	4.5 (12)
	NGH	2.8 (4)
	NPHH	2.4 (8)
0.3-0.5 mg/kg propranolol (β-blockade)	NTA	6.5 (9)
	NGH	6.5 (6)
	NPHH	-3.4 (6)
Prostaglandin A_1, 1.0 mg/kg	NTA	1.2 (27)
Antianginal preparations:		
2.5 μg/kg corinfar	NTA	8.0 (12)
mesotrin	NTA	10.0 (15)
25.0 μg/kg nitroglycerin	NTA	3.4 (12)

them: an increase in the blood flow through the brachiocephalic artery and a decrease followed by an increase in the blood flow through the descending aorta (Fig. 1, *b*). Therefore, it can be hypothesized that the direction of the changes in the arterial circulation affects changes in the blood flow through the corresponding venae cavae, leading to an increase in the proportion of blood flow through the SVC and a decrease in that through the IVC in the total VR.

Thus, our findings indicate that the contribution of blood flow through the SVC to the total VR increases and that through the IVC decreases during systemic pressor and depressor vascular reactions induced by various stimuli. The redistribution of cardiac output between the brachiocephalic arterial and descending aortic beds due to different changes of vascular resistance could be one of the hemodynamic mechanism responsible for changes in the proportion of blood flows through the venae cavae during systemic reactions.

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