

PHYSIOLOGY

Contribution of Splanchnic and Musculocutaneous Vascular Compartments to the Formation of Blood Flow Volume in the Vena Cava Posterior during Catecholamine Treatment

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Studies by electromagnetic flowmetry in acute experiments on cats under conditions of the open thoracic cage and artificial ventilation of the lungs showed that 64% of venous return via the vena cava posterior was realized at the expense of the splanchnic and 36% due to the musculocutaneous vessels (abdominal basin of the caudal vein). Epinephrine (20 µg/kg) increased the contribution of the splanchnic venous blood flow to the increase in the blood flow in the vena cava posterior and reduced the contribution of the musculocutaneous veins throughout the entire duration of systemic reactions: 84% of the blood flow increase in the vena cava posterior was due to the splanchnic and just 16% due to the musculocutaneous blood flow. Norepinephrine (10 µg/kg) resulted in a phase-wise involvement of the studied compartments in blood flow increase in the vena cava posterior. During the initial period of systemic reactions (coinciding with the maximum systemic BP rise) the contribution of the musculocutaneous compartment was 13% higher, while later (by the time of the maximum elevation of venous blood flow in the studied compartments) the contribution of splanchnic veins predominated constituting 89% of venous blood flow in the vena cava posterior. These results indicate that venous blood flow increase in the splanchnic vessels largely determined the formation of changes in the vena cava posterior blood flow in response to catecholamines.

Key Words: *vena cava posterior blood flow; splanchnic and musculocutaneous venous blood flow; catecholamines*

The blood flow volume in the vena cava posterior (VCP), the main collector of venous blood outflow from the abdominal organs and hind limbs of animals (splanchnic (SC) and musculocutaneous (MCC) compartments), constitutes $2/3$ of venous return to the

heart via caval veins [2]. These vascular compartments differ from each other by many morphological and functional characteristics, e.g. by the volume of blood in these vessels per unit of tissue. SC contains 7-10 ml blood/100 g tissue, muscular compartment 2.5-4 ml blood. No reliable data on the blood volume in the skin have been reported. A classical review [4] discussing the formation of circulating blood volume in VCP presented mainly the data on changes in the

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capacities of individual vascular compartments of the abdominal cavity or SC in general and in MCC under the effects of various stimuli on the circulatory system. Modern data [2,3,5,8] indicate more pronounced shifts in SC vs. MCC capacity in reflexes from the carotid sinuses, electric stimulation of the sympathetic nerves, and other stimuli. A relationship between the capacity of separate vascular compartments and the duration of cardiovascular reactions has been detected. However, no comparative characterization of the dynamics of SC and MCC vascular capacities and the role of changes in these parameters in the systemic hemodynamics has been carried out. As it remains unclear how changes in the capacity of these vascular compartments contribute to the blood flow shifts in VCP, we studied the quantitative characteristics of shifts in the venous blood flow in SC and MCC and their contribution to the formation of the blood flow volume in VCP over the course of cardiovascular reactions to catecholamines.

MATERIALS AND METHODS

The study was carried out on 8 cats narcotized with urethane and chloralose mixture (1 and 0.01 g/kg, respectively) under conditions of open thoracic cage and artificial respiration (Vita-1 device for artificial ventilation of the lungs), open abdominal cavity, and heparin treatment (1000 U).

Through electromagnetic cuff pickups (Nihon Kohden), the blood flow was recorded in the portal vein (carrying venous blood from the stomach, large and small intestine, pancreas and spleen, *i.e.* from SC), in the abdominal portion of the caudal vein (venous blood outflow from MCC of the hind part of the body in animals and from pelvic organs), and the summary venous SC+MCC outflow to VCP (thoracic portion). Systemic BP in the left subclavian artery was recorded with an EMT-34 electron manometer (Siemens-Elema).

The parameters of the circulatory system were recorded under conditions of catecholamine treatment: epinephrine (20 mg/kg) and norepinephrine (10 mg/kg) were injected into the femoral vein in a volume of 0.1 ml.

The differences in the arithmetic means calculated from the results of measurements were evaluated using Student's *t* test.

RESULTS

Injections of catecholamines (norepinephrine or epinephrine) into the femoral vein increased systemic BP by 22 ± 10 and $21 \pm 8\%$, respectively (the difference between these means being insignificant, $p < 0.01$), paralleled by blood flow increase in the portal and

caudal (the abdominal compartment) veins and of the summary blood flow in these vessels (in the thoracic compartment of VCP). In other words, the direction of changes in the venous flow in the studied vessels was the same: the blood flow increased in them (Fig. 1, *a, b*). The initial values and the degree of changes were different. The initial blood flow intensity in the portal vein was 125 ± 16 ml/min vs. 70 ± 11 ml/min in the caudal vein ($p > 0.99$), these values constituting 64 and 36% of return via VCP, respectively. Epinephrine caused an increase of the blood flow by 12 ± 8 ml/min (9.4% of initial blood flow) in the portal vein and by 5 ± 4 ml/min in the caudal vein (9.0% of the initial blood flow), the means differing at the level of $p > 0.99$. After norepinephrine injection, the maximum increment in the blood flow was 10 ± 4 ml/min (8.4%) in the portal vein and 5 ± 4 ml/min (7.0%) in the caudal vein. These data indicate that, first, blood flow response to catecholamines in SC was somewhat more pronounced than in MCC and, second, changes in venous outflow in each of the studied compartments in response to norepinephrine and epinephrine did not differ much. The maximum increase of the summary blood flow in the studied vascular compartments (blood flow in VCP) was 16 ± 6 ml/min (8.2% vs. the initial 196 ± 25 ml/min) in response to epinephrine and 14 ± 5 ml/min (7.4% vs. the initial 186 ± 14 ml/min) in response to norepinephrine. Despite the fact that changes in venous blood flow in SC were greater ($p > 0.95$) than in MCC, the contribution of shifts in the blood flow in each of the studied vascular compartments to the formation of venous return via VCP was different because of differences in the time course of the unfolding systemic shifts (Fig. 1).

Differences in the dynamics of shifts in the arterial and venous vascular systems were detected. The maximum BP elevation was recorded by the end of the first minute from the beginning of intravenous infusion of the drug (after 56 ± 5 sec for epinephrine and 49 ± 4 sec for norepinephrine; $p > 0.95$), *i.e.* during the initial period of systemic reactions development. The maximum changes in the venous blood flow were recorded significantly later, only by the end of the second minute after intravenous infusion of each drug, during the late period of systemic reactions development. Our previous studies showed that changes in the venous blood flow manifested later than the hemodynamic shifts in arterial circulation (systemic BP, cardiac output, and total peripheral resistance).

The contribution of venous blood flow of each of the studied vascular compartments to the formation of changes in VCP blood flow is presumably determined by differences in the initial values of venous blood flow in SC and MCC and by different dynamics and quantitative characteristics of blood flow shifts

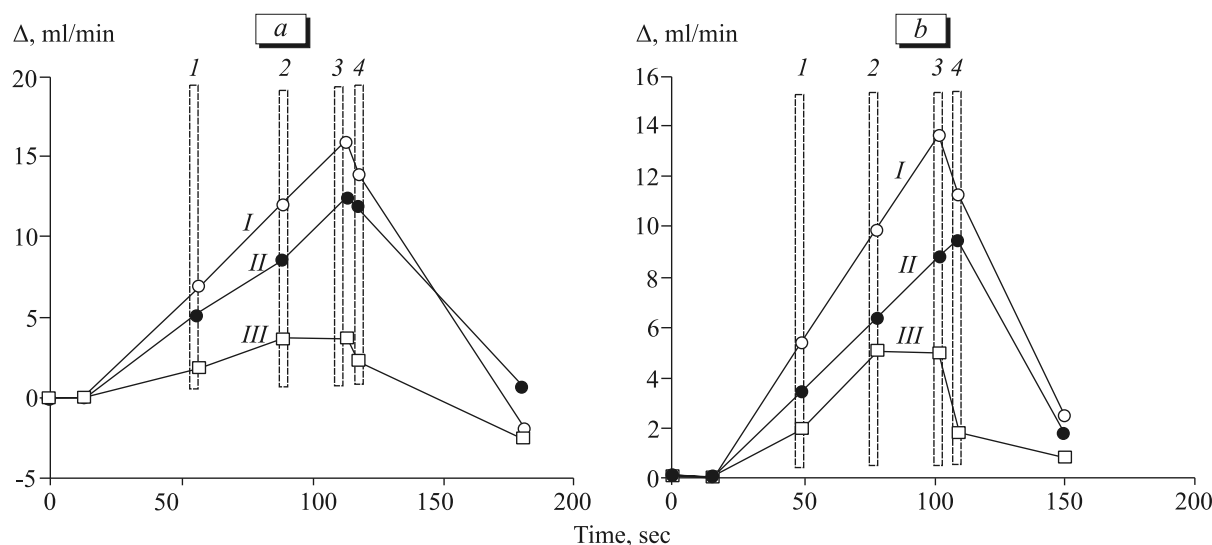


Fig. 1. Changes in the blood flow in the portal, caudal, and posterior caval veins in response to intravenous epinephrine (a) and norepinephrine (b). Blood flow in VCP (I), portal vein (II), and abdominal part of the caudal vein (III). Here and in Fig. 2: interrupted line: time of manifestation of the maximum shifts in the studied hemodynamic values: 1) systemic BP; 2) changes in caudal vein blood flow; 3) changes in VCP blood flow; 4) changes in portal venous blood flow.

in response to infusion of catecholamines. The dynamics of participation of each vascular compartment in the formation of changes in VCP blood flow is presented in Figure 2. The contribution of SC to blood flow increase in VCP in response to epinephrine (Fig. 2, a) slightly decreased (by 3.5%) during the initial period of systemic reactions development, while MCC contribution increased by the same value. Further development of systemic shifts (late period of systemic reactions) was characterized by a significant increase in SC contribution to the formation of changes in VCP blood flow, reaching 84%, while MCC contribution decreased to 16%.

A different picture of contribution of each of the studied vascular compartments was observed in response to norepinephrine (Fig. 2, b). During the initial period of systemic reactions development, the contribution of MCC to shifts in VCP blood flow clearly predominated and was greater in comparison with that in response to epinephrine: a 13% increase in MCC and the same increase in SC. Later (late period of systemic reactions development) the contribution of SC to the formation of changes in VCP blood flow increased significantly, similarly as in response to epinephrine, and reached 89% of the blood flow increase in VCP, MCC venous blood flow contribution being just 11%.

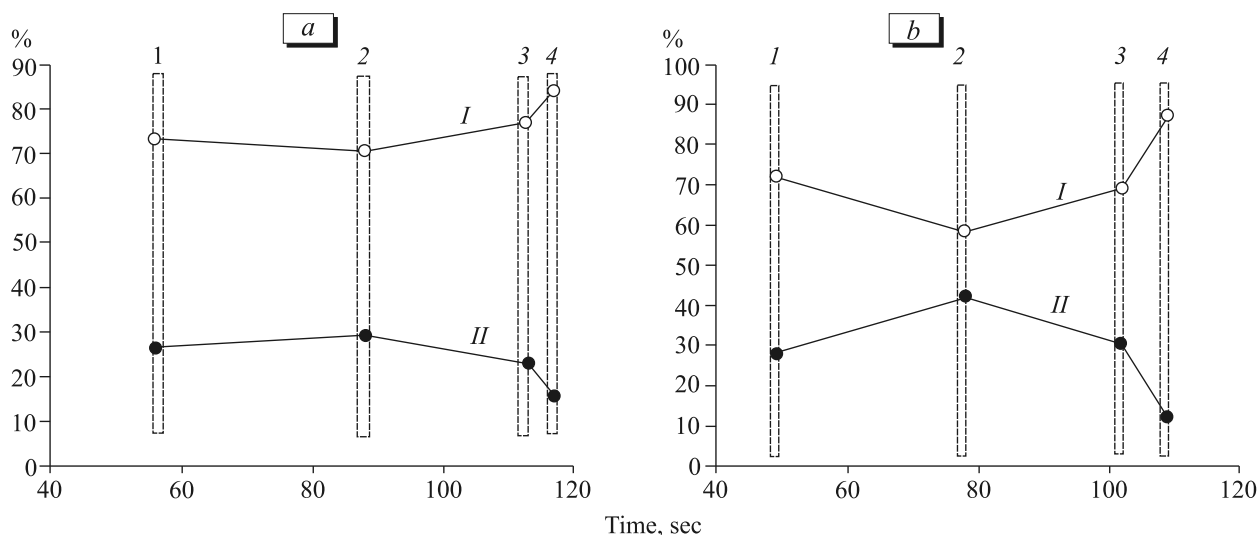


Fig. 2. Contribution of changes in venous blood flow in SC (I) and MCC (II) to shifts in VCP blood flow over the course of systemic reactions in response to epinephrine (a) and norepinephrine (b). Ordinate: contribution of each compartment to shifts in VCP blood flow.

These results indicate differences in the patterns of shifts in the venous blood flow and their contribution to the formation of the greater part of venous return to the heart (VCP blood flow) in response to catecholamines. Changes in the blood flow in the portal vein and caudal fragment of the abdominal vein in response to intravenous infusion of catecholamines manifested later than the maximum shifts in systemic BP. The venous blood flow in each of the studied compartments increased similarly in response to epinephrine and norepinephrine, but blood flow increment in SC was greater than in MCC. On the other hand, the contributions of shifts in these compartments of venous blood flow to the formation of shifts in venous return via VCP were different and depended on the catecholamine injected (presumably, due to differences in their effects on the α - and β -adrenoreactive structures) and time of the systemic reactions development. The degree of shifts in the venous blood flow in VCP in response to epinephrine was determined mainly by shifts in the splanchnic venous blood flow and less so by shifts in the musculocutaneous venous blood flow throughout the entire course of systemic reactions. Norepinephrine promoted a phase-wise involvement of each of these compartments to the formation of shifts in the venous blood flow in VCP. Shifts in the musculocutaneous venous blood flow made a greater contribution to it only during the initial period of the

unfolding systemic reactions, while later, by the time of the maximum shifts in the venous blood flow, the contribution of SC increased and that of MCC decreased. Our results indicated that the formation of changes in VCP blood flow under conditions of intravenous catecholamine treatment was mainly determined by changes in the splanchnic venous blood flow, presumably because of less pronounced than in the musculocutaneous veins mobilization of the blood in this compartment.

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