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ROLE OF THE CARDIOPULMONARY BLOOD VOLUME IN CHANGES IN LEFT VENTRICULAR OUTPUT DURING STIMULATION OF SOMATIC AFFERENT NERVE FIBERS

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KEY WORDS: left ventricular output; arterial pressure; venous return to the heart; blood flow in the pulmonary artery; somatic afferent nerve fibers.

Vessels of the pulmonary circulation contain about 20%, and the right and left chambers of the heart up to 10% of the circulating blood volume [5]. Investigations on dogs have shown [7] that during the first 5-6 min of muscular work the deficiency of the venous return to the heart is concealed mainly by the pulmonary reserve and the end-systolic volume. The main source responsible for the change in left ventricular output is the functional residual capacity, which amounts to between 46 and 65% of its diastolic volume, and consists of the reserve and residual volumes [4]. The increase in stroke volume during muscular work in the period preceding the increase in the venous return to the heart takes place on account of the reserve volume [7]. During muscular work activation of afferent fibers of groups III-IV (mainly of type C) of somatic nerves, which react to high-voltage and high-frequency stimulation [9], takes place. Muscular contraction is essential for activation of muscle afferents [8]. The writers showed previously [3] that during the pressor response to stimulation of afferent C-fibers of the tibial nerve, the left ventricular output (LVO) exceeded the venous return to the right atrium in one-third of the experiments. However, the degree of participation of the blood volume in the pulmonary vessels and in the right ventricle and right atrium in the formation of the inflow of blood to the left ventricle during coupled reflexes has virtually not been studied. An increase in the blood volume in a lobe of the lung has been demonstrated [1] during stimulation of the central end of the divided tibial nerve.

The aim of this investigation was to study the importance of the capacitive properties of the pulmonary vessels and chambers of the right heart, and also of neurogenic influences on the right ventricle for changes in LVO in response to electrical stimulation of C-fibers of a somatic nerve. For this purpose the character and magnitude of synchronized changes in blood flow in the ascending aorta, pulmonary artery (PA), and posterior vena cava (PVC) were compared.

EXPERIMENTAL METHODS

Acute experiments were carried out on cats with a closed chest and artifically ventilated and anesthetized with chloralose (40 mg/kg) and pentobarbital (10 mg/kg), intraperitoneally. Artificial respiration was applied by the DP-8 apparatus. The LVO was estimated as volume velocity of the blood flow in the ascending aorta, measured with a vascular transducer (diameter 7 mm) of an RKÉ-1 electromagnetic blood flowmeter, and the blood flow in PA and PVC also was recorded by vascular transducers of an RKÉ-2 electromagnetic blood flowmeter

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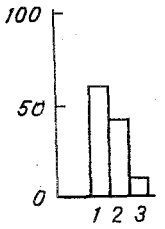

Fig. 1.

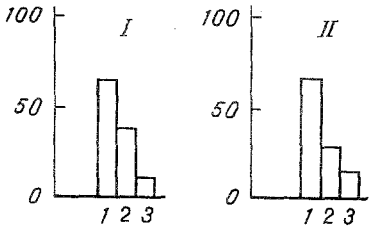

Fig. 2.

Fig. 1. Relationships of different types of changes in blood flow in PA in response to stimulation of C-fibers of tibial nerve (in % of total number of cases). Here and in Fig. 2: 1) increase, 2) decrease, 3) no change.

Fig. 2. Relationships between different types of changes in blood flow along PVC (in % of total number of observations) during stimulation of C-fibers of tibial nerve in group with decrease (I) and increase (II) in blood flow in PA.

(diameters of the transducers were 6-8 and 6 mm respectively). We know [2] that changes in the return of blood along PVC correspond in direction to changes in the total venous return. Blood pressure (BP) was measured in the femoral artery by the transducer of a PDP-400 electromanometer. All parameters were recorded on a USChV-8 automatic ink writer. The tibial nerve was stimulated with bipolar electrodes from an ÉSL-1 stimulator (10 V, 20-40 Hz, 5 msec), and the duration of stimulation was 30 sec. Propranolol (Obsidan, 1 mg/kg) was injected into the femoral vein in a volume of 1 ml of physiological saline.

RESULTS

In 12 experiments stimulation of the tibial nerve caused an increase in LVO in 19 (42%) of 54 tests, a decrease in 17 (35%), an increase followed by a decrease in four (9%), and a decrease followed by an increase in five cases (10%), against the background of a pressor reaction. To determine the role of the capacity of the pulmonary vascular bed in changes in LVO, in the next series of experiments they were compared with synchronized changes in blood flow in PA. It was found that in those cases when LVO was increased, at the time of its maximal value the blood flow in PA was increased in 11 of 19 cases, reduced in seven, and unchanged in one (Fig. 1, Table 1). The increase in the blood flow in PA was 41 ± 18 ml/min and it did not differ significantly from the increase in LVO (24 ± 7 ml/min, $P > 0.05$). In cases when LVO showed a decrease, in most of them (13 of 17) changes in the blood flow in LA also were in the same direction, and only in three tests were they opposite to the changes in LVO or not significant (Table 1). Thus in most cases changes in blood flow in PA coincided with the trend of the changes in LVO.

Extensibility of vessels of the pulmonary circulation creates conditions for the accumulation of a considerable volume of blood in this region, if the increase in the return of blood to the right atrium is not accompanied by a simultaneous change of equal magnitude in LVO. The disparity observed by the writers previously [3] between the venous return to the right atrium and LVO during electrical stimulation of a somatic nerve could be due to an increase in the blood volume in the lungs. However, the similarity of direction of changes in the blood flow in PA and in LVO, discovered in the present investigations, in most cases does not support the view that changes in the latter are connected with a change in capacity on the pulmonary vascular bed.

Furthermore, the increase in LVO under these circumstances is connected with neurogenic influences on the myocardium [3]. Changes in output of the right ventricle could also be the result of manifestation of direct neurotropic influences during the somatic reflex. In 14 experiments, therefore, changes in the blood flow in PA in response to tibial nerve stimulation before and during β-adrenoreceptor blockade were compared. Under blockade conditions the ratio between the number of cases with an increase and with a decrease in the blood flow in

TABLE 1. Changes in Blood Flow in PA and in LVO in Response to Tibial Nerve Stimulation in Cases with Increase (A) and Decrease (B) in LVO

Parameter	A		B	
	n	Δ	*n*	Δ
CO, ml/min	19	27±7	17	27±5
PA (+), »	11	41±18	3	41±31
PA (—), »	7	96±26	13	36±9

TABLE 2. Changes in BP and in Blood Flow in PVC and PA in Cases with an Increase in Blood Flow in PA during Tibial Nerve Stimulation before (A) and during (B) β-Adrenoreceptor Blockade

Parameter	A		B	
	n	Δ	*n*	Δ
BP, mm Hg	24	17±5	11	23±7
PA, ml/min	24	52±7	11	37±8
PVC (+), "	15	16±2	4	28±11
PVC (-), "	5	51±22	6	25±6

TABLE 3. Changes in BP and in Blood Flow along PVC and PA in Group with Decrease of Blood Flow in PA in Response to Tibial Nerve Stimulation before (A) and during (B) β-Adrenoreceptor Blockade

Parameter	A		B	
	n	Δ	*n*	Δ
BP, mm Hg	29	21±4	26	20±6
PA, ml/min	29	69±10	26	49±13
PVC (+), "	18	66±34	5	24±8
PVC (—), "	9	37±10	7	12±4

PA did not differ from the distribution of the tests before blockade. The increase in the blood flow in PA likewise did not differ in value from its increase before the blockade (Table 2). No difference likewise was observed in the magnitude of the changes in blood flow in PA before and during the blockade and in cases when the pulmonary blood flow was reduced (Table 3). No changes in the venous return to the right atrium were found before and after administration of propranolol in experiments with both an increase and a decrease in the blood flow in PA (Tables 2 and 3). Thus propranolol did not affect changes in the blood flow in PA in response to tibial nerve stimulation. Previously [3] the essential role of the neurogenic factor in the changes in LVO was discovered. The absence of differences in the observed changes in right ventricular output before and during β-adrenoreceptor blockade could be connected with differences in the density of the adrenergic innervation of the left and right ventricles.

The decrease in LVO discovered in these experiments could be linked with changes in right ventricular function as a result of a change of pressure in PA. The right ventricle is known to be more sensitive to a rise of pressure in PA than the left ventricle is sensitive to changes of pressure in the aorta [10]. In the next series (19 experiments) agreement between the character of synchronized changes in the return of blood to the right atrium and right ventricular output was therefore studied. In cases in which the pulmonary blood flow was reduced, in 18 of 29 tests the blood flow along the posterior vena cava was increased, in nine it was reduced, and in two it was unchanged (Fig. 2, I). Thus, in more than 60% of cases the decrease in the blood flow in PA could take place against the background of an increased return to the heart. The decrease in the blood flow in PA could be connected with reflex constriction of the vessels of the pulmonary circulation [6] in much the same way as that caused

by reduction of stimulation of the baroreceptors of the carotid sinus [2]. Furthermore, the reduction in the blood flow in PA in most cases in the present experiments took place against the background of an increased return of blood to the right atrium. It is more probable, therefore, that the decrease in blood flow in PA was due to constriction of the pulmonary vessels.

Among factors determining the trend of changes in LVO, besides changes in the venous return to the heart, the intra-aortic pressure, and inotropic influences on the heart [4], the reserve volume in the ventricles must also be mentioned [7]. The reduction in the blood flow in PA against the background of an increased return of blood to the right atrium and right ventricle, which was observed in more than 60% of tests, evidently takes place on account of an increase in the residual volume of the right ventricle and a change in capacity of the right atrium and venae cavae as a result of the increased load thrown on the right ventricle by constriction of the pulmonary vessels. In experiments in which the blood flow in PA was increased, in 15 of 24 tests the changes in PVC were in the same direction, in five tests the blood flow along PVC was reduced, and in three it was unchanged (Fig. 2, II; Table 2).

When the blood flow in PA was increased, in most cases there was thus an increase in the blood flow along PVC. It must be pointed out that in most tests an increase in the blood flow in PA and also an increase in the venous return corresponded to an increase in LVO.

Thus neither the capacitive nor the resistive properties of the pulmonary circulation determine the increase in LVO during tibial nerve stimulation, evidently due entirely to neurogenic influences on the myocardium [3] and active constrictor reactions of the systemic venous bed [11]. Meanwhile the decrease in LVO may be a result of an increase in the load on the right ventricle, associated with constrictor reactions of the pulmonary vessels.

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