CORRELATION BETWEEN SYSTEMIC AND CORONARY FRACTIONS OF LEFT VENTRICULAR EJECTION DURING PRESSOR REFLEXES

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During excitation of the sympathetic nervous system induced by blocking of the carotid sinus baroreceptors or by stimulation of afferent C-fibers of a somatic nerve, leading to pressor reflexes of the arterial blood pressure (BP), various changes are observed in the cardiac output. The writers showed previously that during these pressor reflexes, at the stage of the transition process disparity is observed between the return blood flow into the right atrium and the left ventricular ejection (LVE) [5, 6]. One factor determining this disparity in our experiments may have been the extent of the coronary blood flow (CBF), which was disregarded when the cardiac output was recorded in the ascending aorta. The role of the coronary factor likewise was disregarded by other investigators [10, 14, 15], who also recorded cardiac output (CO) in the ascending aorta. It was thus impossible to estimate the total LVE (CO_{tot}), which is equal to the sum of the systemic and coronary fractions (SF and CF respectively), and this may have been the reason for the contradictory interpretation of changes in the pumping function of the heart during pressor reflexes. The suggestion that CBF is important when these changes are assessed was put forward previously [3, 8]. An increase of pressure in the aorta leads to an increase in the coronary perfusion pressure, and in turn, during the transition process, this leads to a change in CBF and the blood volume in the coronary bed. Autoregulation of CBF is exhibited only in the steady state [9]. Interest in the role of the coronary factor can also be attributed to the fact that it is not only the mechanical activity of the heart and the level of its metabolism in the heart–coronary vessels system that determines the value of CBF, but, as many other investigators have shown [4], the opposite dependence also applies — coronary inotropic influences on the heart, changing its output.

In the investigation described below correlation between CF and SF of LVE was studied during pressor reflexes evoked by blocking the carotid sinus reflexogenic zone and by stimulation of afferent fibers of the tibial nerve. In this connection it was also necessary to analyze changes in both fractions of LVE and CO_{tot} and their dependence on changes in BP observed during the reflex. Previously, changes in CO under the influence of BP have been assessed only with respect to values of SF [5, 6].

EXPERIMENTAL METHOD

Acute experiments were carried out on cats with an open chest, artificially ventilated, and under chloralose–pentobarbital anesthesia (50 mg/kg and 20 mg/kg respectively, intraperitoneally). The DP-8 apparatus was used for artificial ventilation. SM of LVE was judged on the basis of measurements of the volume–velocity of the blood flow in the ascending aorta (distally to the origin of the coronary arteries) by means of a vascular transducer (diameter 7 mm) of the RKÉ-1 electromagnetic flowmeter. CF was determined by measuring the outflow from the coronary sinus and multiplying it by 3/2. The outflow from the coronary sinus is 65% of the total coronary venous return [16]. Although data for the venous outflow from the coronary sinus as a percentage of the total venous outflow from the heart are contradictory [1], verification under our experimental conditions showed that the total CBF, calculated by the above method, amounts to 5% of the total LVE, which is in agreement with data in the literature [1].

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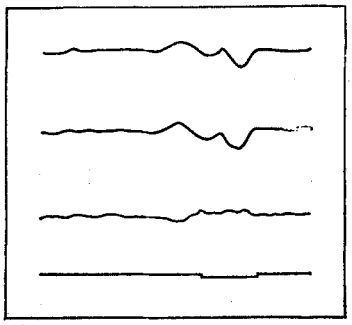

Fig. 1

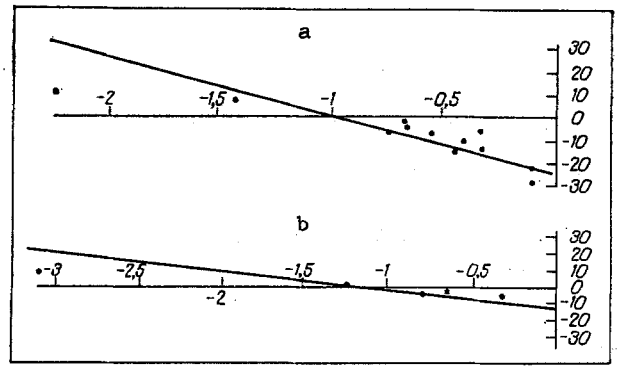

Fig. 2

Fig. 1. Changes in SF of left ventricle, CBF, and BP after occlusion of carotid arteries. From top to bottom: marker of occlusion; time marker 10 sec, velocity of blood flow in ascending aorta (ml/min); BP in femoral artery (in mm Hg); outflow of blood from coronary sinus (in ml/min).

Fig. 2. Dependence of changes in CO_{tot} on ratio of changes in its CF and SF after occlusion of the carotid arteries (a) and tibial nerve stimulation (b) in the group of observations with a decrease in SF. Abscissa, $\Delta CF/\Delta SF$; ordinate, changes in LVE (CO_{tot}, in % of initial value). Regression equation: a) $y = -21.49 \times -22.68$; b) $y = -11.69 \times -12.5$.

The outflow from the coronary sinus was recorded by means of a flow transducer (diameter 3 mm) of the RKÉ-2 electromagnetic flowmeter. A catheter was introduced into the coronary sinus through the auricle of the right atrium, and the outflowing blood passed through the transducer of the electromagnetic flowmeter into a reservoir, located 10 cm below the level of the thorax, from which it was returned by means of an AT-196 pump to the animal through the external jugular vein. The controls of the pump were set to ensure equality of the inflowing and outflowing blood. BP was measured in the femoral artery by the mechanotron transducer of an electromanometer. All parameters were recorded on the N-3021 automatic ink-writer. The carotid sinus baroreceptors were blocked by occlusion of the common carotid arteries. The tibial nerve was stimulated through bipolar electrodes from an ÉSL-1 stimulator (10 V, 20-40 Hz, 5 msec); the duration of stimulation was 30 sec The results were subjected to statistical analysis by Student's t test. The regression line was determined by the method of least squares.

EXPERIMENTAL RESULTS

Correlation between SF and CF of LVE after occlusion of the common carotid arteries was studied in 14 experiments. This correlation was investigated at the time of the maximal rise of BP, for by that time the changes in CBF had reached their maximal values (Fig. 1). The time taken for the maximal changes in CBF and BP to be reached was 22 ± 3 and 25 ± 3 sec respectively.

Occlusion of the carotid arteries at the time of the maximal rise of BP, namely 50-55 mm Hg, caused a decrease of SF in 12 (57%) of 21 observations, and an increase in six (29%) tests. In five tests (24%) SF was unchanged. CF was always increased by occlusion of the carotid arteries.

Changes in BP, SF, CF, CO_{tot}, $\Delta CF/\Delta SF$, and the ratio of ΔSF and ΔCF to the original value of CO_{tot}, in %, are given in Table 1 (series I). It will be clear from Table 1 that significant differences ($p < 0.05$) exist between CO_{tot} and SF in the group with a decrease in SF, due to changes in CF. The ratio of the changes in CF to the changes in SF in this group averaged 0.53 ± 0.17. From this ratio it could be judged which fraction of LVE was the greater. Negative correlation was observed between this ratio and the change in CO_{tot} (Fig. 2a). When the value of $\Delta CF/\Delta SF$ fell below −1.05, the decrease in CO_{tot} changed into an increase.

The role of CF in the change in LVE after occlusion of the carotid arteries in the case of negative values of SF can thus be regarded as significant (Fig. 1). Negative values of CO_{tot} are thus trivial (−2.3%). Consequently, it is impossible to assess the changes in CO_{tot} under these conditions purely on the basis of data for SF.

TABLE 1. Initial Values of BP (mm Hg), CO_{tot} (ml/min), SF (ml/min), CF (ml/min) and Their Changes after Occlusion of the Carotid Arteries (series I) and during Tibial Nerve Stimulation (series II)

Series of observations	Group of observations	BP_{init}	ΔBP	ΔBP, %	SF_{init}	ΔSF	ΔSF, %	ΔCO_{tot} (init)	ΔCO_{tot}	CO_{tot}, %	CF_{init}	ΔCF	ΔCF, %	$\frac{CF_{init}}{CO_{tot}}$ (init)	$\frac{\Delta CF}{\Delta SF}$	$\frac{\Delta CF}{CO_{tot}}$, % (init)	$\frac{\Delta SF}{CO_{tot}}$, % (init)
I	SF + (n = 4)	79±29	54±18	68±17	240±67	29±9	12±6	257±69	37±12	14±7	17±3	7±4	41±25	6,6±1,1	0,31±0,09	2,7±1,4	11,3±6,9
	SF − (n = 12)	99±6	51±7	51±8	313±14	−17±3,1	−5±1,2	332±14	−8,2±2,5*	−2,4±0,7	19±2	9±2	47±10	5,7±0,6	−0,53±0,17	2,7±0,6	−5,1±0,9
	SF = 0 (n = 5)	120±10	21±6	19±6	227±39	0	0	243±39	5±1,7	2,8±1,3	10±1,9	5,1±1,7	31±10	7,6±1,4	—	2,09±0,9	—
II	SF+ (n = 15)	100±5	14±2	14±3	349±16	15±2	4,3±0,5	367±18	17±2	4,8±0,5	18±2	2±0,7	11,1±3,1	5,0±0,3	0,2±0,06	0,54±0,17	4,3±0,5
	SF− (n = 7)	108±10	19±4	17±4	331±31	−6±0,8	−1,8±0,2	346±32	2,2±2	−0,5±0,6	16±2	4±1,6	25±13,7	4,6±0,4	−0,8±0,17	1,1±0,7	−1,6±0,26

Legend. Asterisk indicates significant difference from ΔSF at the $p < 0.05$ level.

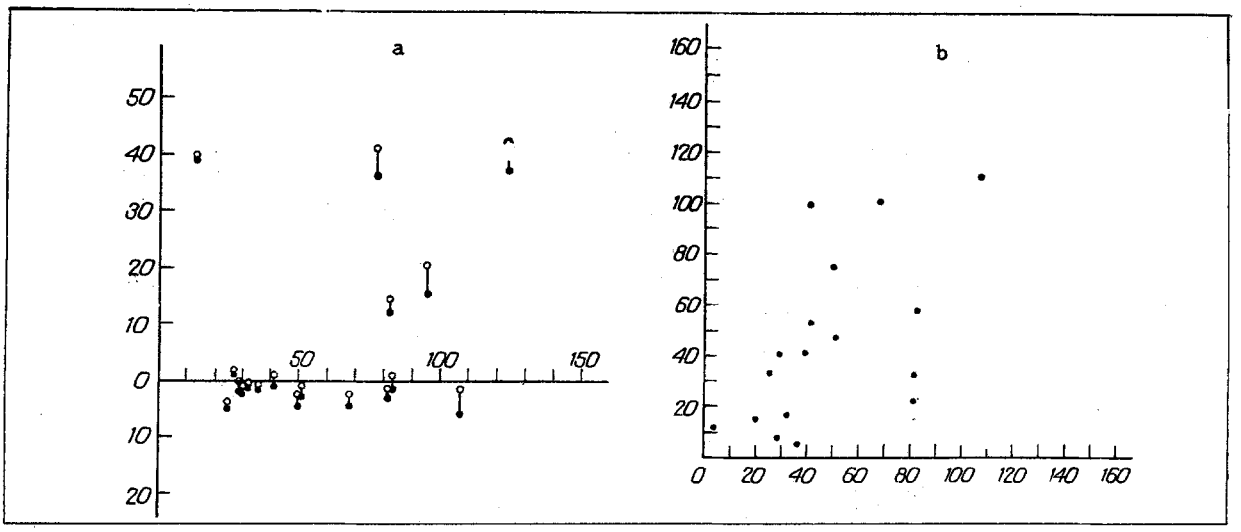

Fig. 3. Dependence of CO_{tot} and SF on changes in BP (a) and correlation between changes in BP and CF (b) after occlusion of carotid arteries. a: Abscissa, changes in BP (in % of initial value); ordinate, changes (in % of initial value) in SF (filled circles) and CO_{tot} (empty circles); b) abscissa, changes in BP (in % of initial value); ordinate, changes in CF (in % of initial value).

After occlusion of the carotid arteries and in the presence of a raised BP, an increase in CBF was observed in a number of investigations [11, 12]. In the present experiments the increase in CBF amounted to 47 ± 10% of the initial level, which was 19 ± 2 ml/min, which is a normal value for cats [13]. An increase of 50% in CBF [3] caused an increase in the amplitude of contractions of LV by 34%. Positive coronary inotropic effects due to an increase in CBF or the coronary perfusion pressure [4] may lead to compensation of the reduced LVE; this probably takes place as a result of the fact that the heart cannot overcome the increased pressure load in the aorta. Accordingly, it may be assumed that coronary inotropic influences have a correcting role.

Comparison of changes in CO_{tot} with changes in BP in the group of animals with a decrease of SF after occlusion of the carotid arteries showed that as BP changed from 20 to 70% of its initial level, CO_{tot} tended toward a constant value. Meanwhile changes in SF under the influence of changes in BP within the same range were negative in value (Fig. 3a). Within this range of BP changes it is evidently possible to envisage a mechanism of homometric regulation [3] for CO_{tot}.

In previous investigations, when decisions regarding LVE were based on measurements of the volume velocity of the blood flow in the ascending aorta (i.e., the value of SF), no conclusion could be drawn regarding the degree of manifestation of homometric regulation of the heart because no allowance was made for the "leaking" of part of the ejection volume into the coronary vessels.

We also found that changes in CBF are directly dependent on changes in BP within this same range (Fig. 3b). Consequently, it may be accepted that homometric regulation of cardiac output is based on changes in CBF.

In the experiments in which SF was unchanged, CO_{tot} was always increased. In animals with an increased SF, changes in SF were always greater in value than those in CF. The $\Delta CF/\Delta SF$ ratio in this case was 0.31. The ratio of SF to CO_{tot} (initial) was higher than the ratio of CF to CO_{tot} (initial). As a result, changes in CO_{tot} did not differ significantly from changes in SF (Table 1, series I).

During electrical stimulation of the tibial nerve, which was used in 12 experiments, the ratio of SF and CF also was studied at the time of the maximal rise of BP. Changes in CBF, just as in the experiments with occlusion of the carotid arteries, reached their peak values at this time. Stimulation of the tibial nerve at the time of maximal increase of BP caused an increase in SF in 15 (68%) of 22 cases and a decrease in SF in 7 (32%) cases; CF was always increased, although its changes were only one-quarter of the magnitude of those after occlusion of the carotid arteries. Sciatic nerve stimulation [2, 7] constricts the coronary

vessels. The response of BP to the same procedure in the present experiments was 2-3 times less than to occlusion of the carotid arteries. Evidently as a result of this, the increase in CBF in our experiments was very small (Table 1, series II). As will be clear from Table 1, CO_{tot} remained virtually unchanged in the group of observations with a decrease in SF. The ratio of the changes $\Delta CF/\Delta SF$ in this group was 0.80 ± 0.17, whereas in the group with an increase in SF it was 0.2 ± 0.06. Thus when this ratio, in absolute values, exceeded 0.5, changes in the coronary fraction were significant for determination of exact values of changes in LVE compared with its assessment purely on the basis of changes in SF. Just as with occlusion of the carotid arteries, if the value of the ratio $\Delta CF/\Delta SF$ is less than -1.07 (Fig. 2b), a change was observed from a decrease to an increase in CO_{tot} in the group with a decrease in SF.

Reduction of the systemic fraction of left ventricular ejection during this procedure, just as with occlusion of the carotid arteries, can thus be explained by shunting of the blood into the coronary system.

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