CORRELATION BETWEEN THE CONTRACTILE FUNCTION OF THE HEART AND THE SYSTEMIC HEMODYNAMICS IN THE EARLY PERIOD OF DISTURBANCE OF THE BLOOD FLOW IN A LARGE CORONARY ARTERY

N. N. Orlova

UDC 616.132.2-005-036.4-07:
[616.12-008.3+616.1-008.1

KEY WORDS: cardiodynamics; contractile function of the heart; coronary insufficiency.

Interest in the study of the consequences of an inadequate blood supply to the heart, not only when the regional coronary blood flow ceases completely, but also when it is disturbed to different degrees, has recently increased considerably [7, 12]. The object of this investigation was to make an experimental study of disturbances of the contractile function of the heart and their role in the formation of the general hemodynamics reaction during partial and considerable (70 and 90%) restriction of the blood flow in the system of the circumflex branch of the left coronary artery.

EXPERIMENTAL METHOD

In acute experiments on mongrel dogs anesthetized with chloralose the blood supply to the heart was restricted without opening the chest, by catheterization and perfusion of the circumflex branch of the left coronary artery with a constant volume of blood. Restriction of the blood flow by an assigned amount [5], controlled by the flow transducer of a flowmeter, was achieved without the use of extravascular approaches to the coronary vessels, attended by their denervation and by considerable trauma. The onset of coronary insufficiency was recorded by the ECG in 15 precardial leads. The contractile function of the heart was tested by catheterization of its chambers with simultaneous recording of the intraventricular pressure and of its first derivative, followed by partly automatized [6] calculation of several indices [8-11]. The cardiac output and systemic hemodynamics were investigated by the thermodilution method and catheterization of the great vessels. Two degrees of restriction of the coronary blood flow were studied — by 70% and 90% of the original adequate level. Manifestations of coronary insufficiency developing during the first 30 min from the time of restriction of the blood flow were studied.

EXPERIMENTAL RESULTS

With a 70% restriction of the blood flow (29 experiments) during the first 30 min of observation a significant decrease in systolic pressure in the left ventricle was observed for the group as a whole, accompanied by very slight and variable changes in the end-diastolic pressure and by a statistically significant decrease in most of the calculated indices of contractile function (Table 1). The data are evidence of depression of the contractile function of the left ventricle. Within the group as a whole some heterogeneity of the responses (mainly quantitative) was observed. In a considerable proportion of the experiments (12 of 29, i.e., 41.4%) most of the indices tested were reduced by 15-20% or more. In some experiments the changes were less marked.

With an increase in the degree of restriction of the coronary blood flow to 90% (23 experiments) the mean values of the decrease in the main indices of contractile function were rather higher (Table 1). Besides an increase in the mean values, the quantitative heterogeneity of the individual responses still remained.

Investigation of the relations between the changes observed in the functional state of the heart and the general hemodynamic reactions accompanying acute disturbance of the cir-

Laboratory of Experimental Cardiology, N. D. Strazhesko Ukrainian Research Institute of Cardiology, Kiev. (Presented by Academician of the Academy of Medical Sciences of the USSR N. N. Gorev.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 92, No. 9, pp. 279-281, September, 1981. Original article submitted January 21, 1981.

 0007-4888/81/9209-1164$07.50

TABLE 1. Changes (in percent of initial level) in Indices of Cardiac Contractile Function 30 min after Restriction of Coronary Blood Flow by 70% (Group 1) and 90% (Group 2)

Index	Group 1		Group 2	
	M ± m	P	M ± m	P
Systolic pressure in left ventricle	−14,1±3,9	<0,01	−19,7±5,9	<0,01
dP/dt_{max}	−21,7±4,4	<0,001	−24,5±3,4	<0,001
$P-dP/dt_{max}$	−18,5±3,6	<0,001	−22,9±3,7	<0,001
$\frac{dP/dt_{max}}{IP}$	−3,8±3,1	>0,2	−1,6±3,7	>0,5
$\frac{dP/dt_{max}}{IIT}$	−7,1±5,3	>0,1	+0,7±6,9	>0,5
V_{CE10}	−8,5±4,0	<0,05	−16,6±4,7	<0,01
V_{CE40}	−17,3±4,1	<0,001	−14,5±3,4	<0,001
V_{pm}	−11,8±3,9	<0,01	−18,1±4,2	<0,001

Legend. − or + sign in front of numbers indicates decrease or increase in corresponding indices.

culation is of considerable interest. Changes in the hemodynamics with 70% restriction of the blood flow consisted of lowering of the systemic arterial pressure (−16.8 ± 3.5%, P < 0.001) and inconsistent changes in cardiac output, as a result of which changes in the minute (−6.8 ± 5.5%, P > 0.2) and stroke volume (+9.7 ± 6.1%, P > 0.1) were not significant.

On a change to 90% restriction of the blood flow the systemic hypotensive reaction increased very slightly (−21.5 ± 3.6%, P < 0.001). Despite the fact that in most cases the cardiac output was reduced (−26.2 ± 5.0%, P < 0.001), in almost one-third of the investigations the opposite changes were found.

The calculated parameters of contractile function thus changed in a more uniform manner under the experimental conditions and the indices of cardiac output. Analysis of each individual experiment revealed two variants of the relations between them: 1) Cardiac output and stroke volume changed in the same way as the change in contractile function, 2) despite a fall in contractility, the cardiac output and stroke volume remained virtually unchanged or actually increased. Further analysis of the changes in cardiac output was carried out with consideration of its principal components. Besides the state of contractility, attention was paid also to changes before and after loading. In some experiments, the end-diastolic volume of the left ventricle was determined by the thermodilution method.

At 70% restriction of the blood flow it was found that the changes in this index could be in opposite directions, as shown by the heterogeneity of the mean effect (+18.9 ± 13.1%, P > 0.05). Characteristically, the decrease in the stroke volume of blood in most cases was combined with a decrease in the end-diastolic volume of the ventricle (−35.8 ± 5.6%, P < 0.01). At the same time the stroke volume increased or remained virtually constant in cases when the end-diastolic volume of the ventricle was increased (+30.4 ± 11.7%, P < 0.05), which, allowing for the reduction in the contractile function of the heart, was evidence of activation of the Frank—Starling mechanism.

During analysis of these results consideration was paid to data in the literature showing that acute injuries to the myocardium may be accompanied by opposite, or sometimes phasic changes in tone of the resistive vessels [1-3]. The possibility of active depressor reactions of the cutaneomuscular vessels of the hind limb during graded restriction of the coronary blood flow was demonstrated under the conditions used in the present experiment by Mudraya [4]. Characteristically, at 70% restriction of the coronary blood flow an increase in the cardiac output was usually accompanied by a distinct fall in total peripheral resistance (−24.1± 7.6%, P < 0.05) whereas a decrease in the stroke ejection was combined in most cases with an increase in peripheral resistance, although it was not statistically significant (P > 0.2). Considering the character of determination of the total peripheral resistance, we interpreted these findings with some degree of caution. Meanwhile the increase in stroke ejection in some cases was combined with the most distinct fall of resistance in the peripheral cutaneomuscular vessels. It can be tentatively suggested that the increase in output at a time when the contractile function of the heart was definitely reduced was the result of a combined effect of activation of the Frank—Starling mechanism and a simultaneous reduction in the after load.

In the experiments with 90% restriction of the blood flow, besides the relationship described above, which were observed in most experiments, another variant of the reaction also was observed, in which a decrease in the stroke volume took place against the background of an increase in the end-diastolic volume of the ventricle; i.e., despite conditions favoring activation of the Frank–Starling mechanism, its manifestation was impeded. It was found under these circumstances that the degree of increase in the end-diastolic volume of the left ventricle was less than the changes which took place during activation of the Frank–Starling mechanism. Consequently, with increasing insufficiency of the blood supply to the heart correlation between the level of its contractile activity and the changing conditions for work of the heart (before and after loading) became extremely complex in character. With increasing severity of ischemia, its action on the myocardium was intensified and the adaptive reactions of the circulatory system as a whole were manifested more clearly or even activated.

With the degrees of restriction of the coronary blood flow tested, in the early period of observation the direct action of the harmful factor on the heart was thus not the only mechanism determining changes in its pumping function. Restriction of the blood flow by 70 and 90% was accompanied not only by a decrease in the indices of cardiac contractile function, but also by indirect changes in the conditions of its functioning in the circulatory system as a whole, which, through a feedback mechanism (on account of changes before and after loading) took part in the formation of the different variants of the hemodynamic supply situation.

LITERATURE CITED

1. N. N. Gorev, M. M. Povzhitkov, S. A. Korol', et al., Kardiologiya, No. 2, 11 (1973).
2. A. V. Dokukin, Byull. Éksp. Biol. Med., No. 4, 29 (1963).
3. A. A. Moibenko, Cardiogenic Reflexes and Their Role in Regulation of the Circulation [in Russian], Kiev (1979).
4. I. S. Mudraya, Fiziol. Zh. (Ukr.).
5. N. N. Orlova, Kardiologiya, No. 6, 85 (1979).
6. M. V. Sin'kov, A. I. Zakidal'skii, A. A. Moibenko, et al., Byull. Vses. Kardiol. Nauch. Tsentra, No. 2, 101 (1978).
7. M. M. Bodenheimer, V. S. Banka, C. Fooshee, et al., Circulation, 58, 789 (1978).
8. J. Cosins, M. Gutierrez-Miranda, R. Reyses, et al., Pflüg. Arch. ges. Physiol., 362, 165 (1976).
9. D. T. Mason, Am. J. Cardiol., 23, 516 (1969).
10. C. W. Urchel, P. S. Vokonas, A. H. Henderson, et al., Cardiology, 65, 4 (1980).
11. U. Veragut and H. Krayenbuhl, Cardiologia, 47, 96 (1965).
12. H. Wyatt, J. S. Forrester, and J. Tyberg, Am. J. Cardiol., 36, 185 (1975).