CORRELATION BETWEEN CONTRACTILITY OF THE LEFT AND RIGHT VENTRICLES OF THE NORMAL AND PATHOLOGICAL HEART

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In recent clincial and experimental investigations [1, 2, 4, 5] the fact has repeatedly been noted that during primary overloading of the left ventricle significant changes take place in a number of parameters of activity of the right ventricle and, conversely, pathology of the right heart has a significant effect on contractility and metabolism of the left heart.

In the investigation described below the character of correlation between the left and right ventricles is analyzed in the normal heart and in the heart with selective pathology of one of its chambers.

EXPERIMENTAL METHOD

Experiments were carried out on 220 mature male chinchilla rabbits weighing 2.5-3.5 kg. In the experiments of series I 160 intact rabbits were used, in which cardiac function was tested 4 times in the course of a year (March 22, June 22, September 22, December 22, 1980), each time every 3 h for 24 h, in order to analyze circadian and seasonal influences on the animal. The animals of series II were divided into four groups, with 15 rabbits in each group. In these rabbits the following pathological processes were stimulated: 1) overloading of the right ventricle by an increased venous return due to formation of an end-to-end anastomoses between the central end of the right jugular vein and the peripheral end of the right carotid arter; 2) overloading of the left ventricle due to increased resistance to cardiac ejection created by stenosis of the ascending aorta by one-third of its cross-section; 3) acute focal ischemia of the left ventricular myocardium, by ligation of the descending branch of the left coronary artery at the junction between its middle and lower thirds; 4) acute focal ischemia of the right ventricular myocardium by ligation of one branch of the right coronary artery at the junction between its middle and lower thirds. These pathological models were formed during operations under superficial hexobarbital anesthesia accompanied by artificial ventilation. The experiments were done in different years, but always in the period from December through March when, according to data obtained previously [3], contractile function of the heart is maximal and is not subjected to any considerable fluctuations. In intact animals, in accordance with the time table given above, the following parameters were determined in animals with coarctation of the aorta and arteriovenous anastomoses 24 and 72 h and 6 days after the time of modeling of the pathological process, and in rabbits with acute focal myocardial ischemia 10, 20, and 40 min after ligation of the coronary artery under actue experimental conditions (superficial hexobarbital anesthesia, controlled respiration, thoracotomy) electromanometrically inside the ventricles, through cannulas tied into them: 1) peak systolic (real) intraventricular pressure in the left and right venticles (IP_pLV and IP_pRV respectively); 2) the peak systolic intraventricular pressure (maximal) recorded during occlusion of the ascending aorta for the left ventricle and pulmonary artery for the right venticle, for 5 sec in each case (IP_mLV and IP_mRV respectively). The numerical data were subjected to statistical analysis by Student's test. Analysis for the presence or absence of correlation between the test parameters was carried out on a TI-58 microcomputer (Texas Instruments). A coefficient of correlation of $r \geqslant \pm 0.7$ was taken to indicate the presence of strong correlation.

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TABLE 1. Character of Correlations between Parameters of Contractility of Left and Right Ventricles in the Normal and Pathological Heart

Pairs of parameters	Coefficient of correlation				
	Normal	Arteriovenous anastomoses	Coarctaction of aorta	Infarct of left ventricle	Infarct of right ventricle
IP_pLV-IP_mLV	+0,99	+0,00	−0,48*	+0,999	+0,92
IP_pLV-IP_mRV	+0,999	+0,90	+0,59*	+0,84	−0,49*
IP_pLV-IP_pRV	+0,74	+0,80	+0,69*	+0,80	−0,20*
IP_pRV-IP_mRV	+0,72	+0,98	+0,68*	+0,95	+0,75
IP_pRV-IP_mLV	+0,94	+0,72	−0,73*	+0,81	+0,14*
IP_mLV-IP_mRV	+0,92	+0,84	0,00*	+0,86	−0,33*

Legend. Asterisk indicates correlations ceasing to be strong or changing to the opposite sign under pathological conditions.

EXPERIMENTAL RESULTS

In the intact heart the parameters of contractility of the left and right ventricles correlated closely (Table 1). This is evidence that the real function of these parts of the heart, as well as the potential contractile capacity of their component muscle fibers, were coordinated with each other sufficiently clearly. This cross-correlation of the characteristics for different parts of the heart enables it to function as a single organ, activity of the parts of which is strictly synchronized. The weakest of these correlations (according to the value of r) are those between IP_pLV-IP_pRV and IP_pRV-IP_mRV. The relative weakness of correlation between the real function of the left and right ventricles can be explained on the grounds that IP_p is a parameter which reflects the volume of blood ejected by one or other ventricle during one systole, and the level of the peripheral resistance to cardiac ejection. Whereas the first component ought to be identical for both ventricles (otherwise pathogenic discoordination in the work of the left and right sides of the heart would arise), the resistance to cardiac ejection of the left and right ventricles of the heart differs, and its variations may not be strictly synchornized. This evidently also accounts for the lower coefficient of correlation than with the other comparisons. As regards the coefficient of correlation for the IP_pRV-IP_mRV pair is concerned, which also is less than the other coefficients, the following explanation may be put forward. Correlation between these two parameters may have a double interpretation for both the left and the right ventricles. First, the value of IP_p may be determined by the potential capacity of the heart muscle, i.e., IP_p may depend on IP_m (the greater the reserves of the myocardium, the more strongly it may contract under real conditions). Second, the value of IP_m may be determined by the real function (the more the reserve capacity has been utilized under real conditions, the smaller the reserve capacity). In both cases the weight of the myocardium may play an essential role, because energy expenditure is undoubtedly connected with the number of cells utilizing this energy. The weight of the right ventricle is much less than the weight of the left ventricle, and it is evidently this which determines the difference between the coefficients of correlation for this pair of parameters in the two ventricles of the heart.

The fact will be noted that the strongest correlation, virtually absolute (r = +0.999) was that between the real function of the left and maximal achievable function of the right ventricle. Since it is difficult to find any logical basis for an influence of the potential capacity of the right ventricle on real function of the left ventricle, a reciprocal relationship is all that can be suggested. Perhaps the cardiac ejection of the left ventricle affects the pulmonary hemodynamics, and this in turn determines the intensity of functioning of the right ventricle and the intensity of expenditure of its reserves. On the basis of the value of the coefficient of correlation, this relationship can be considered to be decisive for relations between the left and right ventricles of the heart.

Analysis of the data in Table 1 shows that during arteriovenous anastomoses and acute focal ischemia of the left ventricle there is no significant change in the character of correlation between the parameters studied. Meanwhile, in coarctation of the aorta all five correlations change, and in acute focal ischemia of the right ventricle four out of six correlations are disturbed.

In our view the explanation of this difference is as follows. In arteriovenous anastomoses and coarctation of the aorta the intact myocardium is overloaded (in the case of anastomoses, by an increased inflow of blood, in the case of coarctation of the aorta, increased resistance to cardiac ejection). However, in arteriovenous anastomoses this over-

loading takes place at the "input," so that adaptive mechanisms can be brought into play successively and coordinated intracardiac hemodynamics ensured when the load on the myocardium is increased. In coarctation of the aorta, this overloading is observed at the "output," which places the intracardiac adaptive mechanisms in an extremely unfavorable situation in which adaptation to function under the new conditions becomes necessary simultaneously and rapidly, and this leads to discoordination of relations between the right and left sides of th e heart.

A different situation arises in acute focal ischemia of the myocardium of the left and right ventricles. The myocardium is damaged in both cases. In acute focal ischemia of the right ventricle, however, the damage arises at the "input" (not overloading of the intact myocardium, but the actual injury), which upsets the whole system of adaptive mechanisms of the heart and leads to desynchronization of activity of its right and left sides. In ischemia of the left ventricle, this "upset" takes place at the "output." Adaptive mechanisms with earlier times of activation remain intact, and reorganization is possible whereby the heart adapts itself (including the left ventricle) to new conditions of functioning, and it maintains the correlations between its left and right sides that are characteristic of the normal situation.

Thus when the load on the intact myocardium of the left ventricle is sharply increased, or in cases of primary injury to the right ventricle, a disturbance of the normal trend of adaptive reactions in the heart takes place, and this leads to a change in the character of correlations between its left and right sides. Disturbance of these correlations may lead, in turn, to disturbance of the activity of the heart as a system.

LITERATURE CITED

1. O. G. Dovgyallo, L. S. Siparova, and E. A. Vitalev, Zdravookhr. Belorussii, No. 2, 18 (1983).
2. N. S. Kuznetsov, S. B. Kabelyuzhenko, Yu. I. Kulagin, and A. F. Prokhoda, Tr. Krym. Med. Inst., 95, 55 (1983).
3. V. A. Frolov, L. V. Efimova, T. A. Kazanskaya, and V. N. Kozyrenko, Dokl. Akad. Nauk SSSR, 249, No. 2, 504 (1979).
4. D. F. Larson, J. R. Womble, J. G. Copeland, and D. H. Russel, J. Thorac. Cardiovasc. Surg., 84, 543 (1982).
5. R. K. H. Wyse, K. C. Welham, M. Jones, et al., Adv. Myocardiol., 4, 97 (1983).

TREND OF THE CARDIAC OUTPUT AND BLOOD RHEOLOGY IN THE EARLY PERIOD OF RESUSCITATION AFTER PREAGONAL AND AGONAL STATES

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The study of the mechanism of formation of postresuscitation circulatory insufficiency after various terminal states occupies an important place in the analysis of the pathogenesis of postresuscitation sickness and prevention and treatment of its complications [3, 6-9, 13]. The aim of this investigation was to study the trend of the cardiac output (CO) and of the flow properties of the blood in the early postresuscitation period after preagonal and agonal states induced by blood loss.

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