DYNAMICS OF MYOCARDIAL TENSION, THE SIZE AND THICKNESS OF THE LEFT VENTRICULAR WALL IN TWO TYPES OF HYPERFUNCTION

(UDC 616.127+616.124.2]-091-02:616.12-008.61)

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(Presented by Acting Member of the USSR Academy of Medical Sciences V. V. Parin)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 61, No. 6, pp. 41-45, January, 1966
Original article submitted July 13, 1964

The researches of F. Z. Meerson and colleagues [1, 2] have shown that compensatory hyperfunction of the heart, one of the most important compensatory factors in diseases of the blood system, can take place in two different ways. Hyperfunction associated with hypertension and valvular stenosis, and caused by an increase in resistance to the expulsion of the blood, is characterized by a sharp increase in myocardial tension, i.e., it is mainly of an isometric type. By contrast, where there is an insufficiency of the cardiac valves, arteriovenous shunting or anemia, the hyperfunction associated with an increased blood flow to a definite part of the heart, is characterized by an increase in amplitude of the heart's contractions with an insignificant increase in myocardial tension, i.e., it is mainly of an isotonic type.

It has been shown [1], that as disease of the cardiocirculatory system progresses, the two types of cardiac hyperfunction correspond with two types of cardiac insufficiency. In progressive stenoses and hypertony, the predominantly isometric type of hyperfunction is associated with cardiac insufficiency resulting from exhaustion of the functional reserves of the myocardium: in contrast to this the progressive valvular defects with predominantly isotonic hyperfunction are associated with a cardiac insufficiency resulting from a failure to mobilize the functional reserves of the myocardium.

On the basis of this conception, it is possible to calculate myocardial tension accompanying progressive experimental damage to the heart without taking into account the area of cross section of the left ventricular wall and this enables one to devise a method for measuring the thickness of the cardiac wall in a living animal.

In this particular work we have tested the theory described in the previous paragraph under completely controlled experimental conditions: using specially induced aortal stenosis and aortopulmonary shunting we have recorded the thickness of the left ventricular wall, together with other cardiac indices and this has enabled us to obtain a more precise determination of the tension developing in the myocardium of this section of the heart.

EXPERIMENTAL METHOD

The experiments were carried out on male and female dogs weighing 15-21 kg, narcotized with trapanal (35 mg/kg). The thoracic cages of these animals were opened up and controlled respiration was employed throughout the experiments.

Stenosis of the aorta in the supravalvular region was achieved in 4 dogs by means of a device suggested by S. M. Shenderov and A. I. Bartyzel, and consisting of a metal tube with wire which was reinforced at one end with polyvinylchloride insulation. The free end of the wire passed round the aorta and through the tube to the outside, where it was provided with a special attachment piece with a screw thread. Stenosis of the aorta was achieved by rotating the collar of the attachment piece, as a result of which the aorta was constricted by the wire which passed to the exterior. A millimeter scale on the attachment piece enabled us to determine the diminution in external

TABLE 1. Changes in the Size and Thickness of the Left Ventricle and in Its Myocardial Tension, as a Result of Progressive, Experimental Stenosis of the Aorta

Index	Degree of Stenosis (as % of initial size)			
	0	50	63	92
External radius of left ventricle during diastolic phase (in cm)	2.65	2.70	2.82	2.90
Thickness of left ventricular wall during diastole	1.56	1.52	1.50	1.57
Cross-sectional area of left ventricular wall during diastole	18.3	18.5	19.5	20.8
Myocardial tension, calculated without taking into account the cross-sectional area of the ventricular wall (in mgdn)	0.18	0.19	0.45	0.27
Myocardial tension, taking into account the cross-sectional area of the ventricular wall (in mgdn)	0.010	0.011	0.023	0.013

perimeter of the stenosed aorta section quite readily and by calculating the radius of this section we were able to obtain the cross-sectional area of the stenosis at each stage of the experiment.

Aortopulmonary shunts were created in 5 dogs by connecting the ascending aorta to the pulmonary aortic trunk through a Teflon prosthesis, to which was attached a clamp for regulating the blood flow from the aorta to the pulmonary artery.

In the course of the experiments, the pressure in the left and right ventricles were measured electromanometrically using catheters. Similar measurements were made of the pressures in the left atrium, in the aorta and in the pulmonary artery. With the aid of V. S. Sinyakov's ultrasonic equipment we were able to take recordings of the transverse diameter of the left ventricle in an anteroposterior direction simultaneously with the pressure recordings. The thickness of the left ventricular wall in the region of the circular muscle band was measured by a simple device developed and kindly made available to us by V. S. Sinyakov. Myocardial tension in the left ventricle (T) was calculated by a previously described method, but we took into account the changes which occurred in the cross-sectional area of the left ventricular wall, something which has not been done in previous cases. The instantaneous volume of the heart and the size of the blood shunt from aorta to pulmonary artery was determined by the stain (cardiogreen) dilution method, using apparatus supplied by the Cambridge Instrument Company.

RESULTS AND DISCUSSION OF EXPERIMENTS

In all the experiments involving progressive stenosis of the aorta which gave uniform results, it was found that a 50% decrease in the aortic lumen was not accompanied by any significant change in the size of the left ventricle or in the thickness of its wall. In conformity with this, there was also no change in the transverse cross-sectional area of the wall of the left ventricle under these same conditions. The pressure in the left ventricle and the tension developing in its myocardium showed no significant increase (Table 1, Figs. 1 and 2); the instantaneous volume was maintained at what was virtually the normal level. Further decrease in the aortic lumen to a figure of 70-80% of the original value was accompanied by a rapid fall in aortic pressure and a sharp rise in the pressure within the left ventricle, reflecting a 200-250% increase in myocardial tension for this chamber of the heart. The diastolic radius of the left ventricle under such conditions increased by 1-10% and the thickness of its wall decreased by 5-10%; the cross-sectional area of the left ventricular wall only increased by 3-8%.

Stenosis of the aorta, exceeding 90% decrease from the initial size, produced a marked increase in the size of the left ventricle (10-15%) without any sharp decrease in the thickness of its wall, which still remained at 90-97% of the original value. At the same time the amplitude of the left ventricular contraction, measured as the difference between diastolic and systolic dimensions, showed a significant decrease. After a brief rise in the pressure of the left ventricle and a maximum increase in myocardial tension to 350-400% of the initial value, the values of both the indices underwent a considerable reduction. At the same time the dilation of the left ventricle was maintained or even increased slightly.

Insufficiency of the left ventricle, which accompanied maximal aortic compression, was characterized by an increase in final diastolic pressure in this chamber of the heart, by an increase in the left atrial and right ventricular

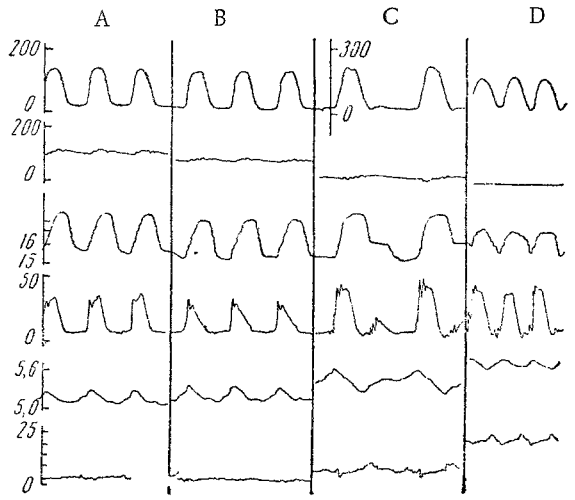

Fig. 1. Changes in the hemodynamics, transverse diameter of the left ventricle and in the thickness of its wall during progressive aortic stenosis. A) Initial recording; B) aortic stenosis of 50% of the initial size; C) constriction of the aortic lumen to 37% of its original size (reverse effect on the pressure change in the left ventricle); D) decrease in transverse section of aorta to 8% of its original size. From top to bottom: pressure in left ventricle; pressure in aorta; thickness of left ventricular wall; pressure in right ventricle; diameter of left ventricle; pressure in left atrium.

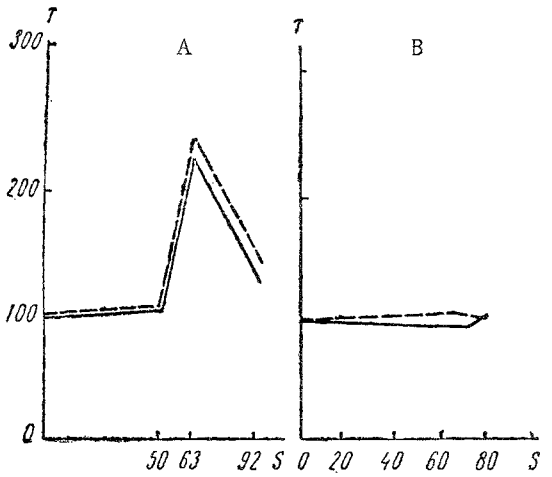

Fig. 2. Dynamics of myocardial tension during aortic stenosis (A) and aortopulmonary shunting (B). The myocardial tension is calculated without taking into account the width and cross-sectional area of the ventricular wall (continuous line) or by including these indices in the calculation (dashes). Along the abscissa axis – degree of aortic stenosis (A) or magnitude of shunting (B); along ordinate axis – myocardial tension (as % of initial value).

pressure and also by a sharp fall in the instantaneous volume, which previously had been maintained at a relatively normal level. The dynamics of certain of the indices associated with progressive aortic stenosis in the experiments, are represented by typical experimental data shown in Fig. 1 and Table 1.

In all 5 experiments with aortopulmonary shunts which gave uniform results, a partial opening of the shunt so as to allow 25-50% of the blood expelled from the left ventricle to make the abbreviated circuit was accompanied by a decrease in the effective instantaneous volume by a mean value of 23%; at the same time the total instantaneous volume was increased by 12%. Further increase in the amount of blood passing through the shunt to 70-75% of the left ventricular output led to an even greater fall in the effective instantaneous volume (mean decrease 54%) and increase in the total instantaneous volume of 30% of the original value.

Progressive increase in the shunting of blood from the large circuit to the smaller pulmonary circuit was accompanied by a brief and inconsiderable fall in left ventricular and aortic pressure; this was followed by a return to the original pressure or even an increase above it in these two sites. The pressure in the right ventricle and the pulmonary artery also increased.

Opening the shunt also evoked a rapid increase in the diastolic dimensions of the left ventricle (8-10%) and an increase in the amplitude of its contractions by a mean value of 32%. In contrast to the picture characteristic of aortic stenosis, progressive increase in aortopulmonary shunting was accompanied by dilation of the left ventricle and a clearly defined decrease in the thickness of the left ventricular wall by 10-15% (Fig. 3). As the progressive aortopulmonary shunting led to some increase in the cross-sectional area of the left ventricular wall, determinations of myocardial tension which took account of this index, were found to be lower than those which ignored the width and cross-sectional area of the wall. The data in Table 2 and Fig. 2 shows that the myocardial tension of the left ventricle did not change or increased to an insignificant extent with progressive increase in aortopulmonary shunting.

Left ventricular insufficiency occasioned by a considerable shunting of blood from the aorta to the pulmonary artery, was characterized by the same hemodynamic features as that resulting from aortic stenosis; however, the former type of insufficiency always developed without any significant increase in myocardial tension.

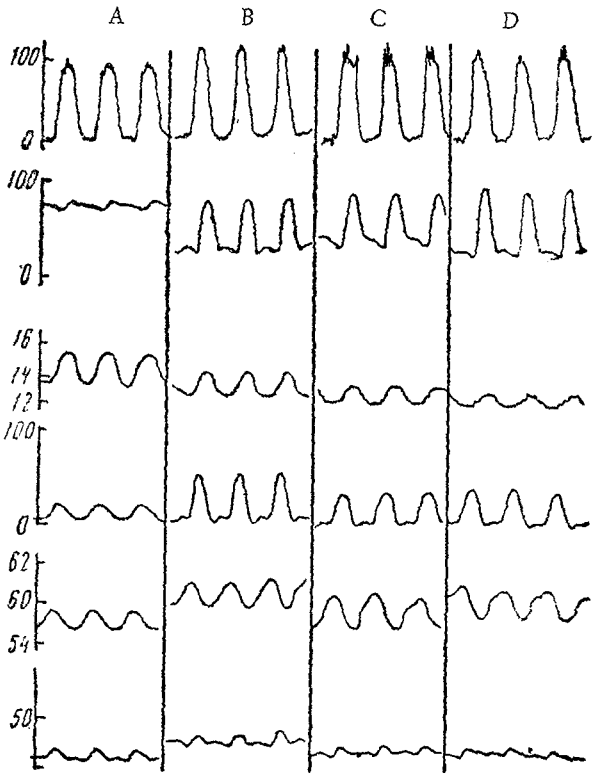

Fig. 3. Changes in hemodynamics, transverse diameter of left ventricle and thickness of its wall during progressive aortopulmonary shunting. A) Initial record; B) 1 min. after opening aortopulmonary shunt to allow 70% diversion of blood; C) 1 min. after increaing shunting to 80%; D) 20 min. after previous recording. From top to bottom: pressure in left ventricular wall; pressure in right ventricle; tranverse diameter of left ventricle; pressure in pulmonary artery.

The facts stated before confirm the view of F. F. Meerson that there are two types of hyperfunction and cardiac insufficiency associated with defects. Moreover, they indicate (when the parameter of ventricular wall thickness is taken into account) that the dynamics of myocardial tension in the left ventricle are essentially the same in both types. The results of our experiments also enable us to recognize two new generalizations which are characteristic of both types of hyperfunction.

TABLE 2. Dynamics of Dimensions and Wall Thickness of Left Ventricle, Together with Tensions Developing in Left Ventricular Myocardium with Progressive Increase in Experimental Aortopulmonary Shunting

Index	Initial data	Magnitude of shunting (as %)		
		70	80	
			1 min	20 min
External radius of left ventricle during diastole (in cm)	2.81	3.03	2.93	3.01
Thickness of left ventricular wall during diastole (in cm)	1.34	1.30	1.24	1.23
Cross-sectional area of left ventricular wall during diastole (in cm^2)	18.0	19.4	18.0	18.3
Myocardial tension, calculated without taking into account cross-sectional area of wall (in mgdn)	0.13	0.145	0.147	0.144
Myocardial tension taking into account cross-sectional area of wall in calculation (in mgdn/cm^2)	0.0074	0.0074	0.0082	0.0079

I. In accordance with Starling's law, it might be expected that the considerable dilation of the left ventricular cavity and the consequent increase in initial length of the fibers prior to contraction, which is found to be associated with arteriopulmonary shunting, ought to bring about a definite increase in the myocardial tension of this chamber. However, as is evident from a study of our results, this does not take place. By contrast, the moderate increase in the dimensions of the left ventricle which accompanies aortic stenosis result in a rather sharp increase in myocardial tension. This can only be explained by assuming that aortic stenosis creates conditions which facilitate a sharp increase in the reactivity of cardiac muscle to stretching of its fibers, whereas in the case of arteriopulmonary shunting these conditions do not exist.

II. The changes in the dimensions and thickness of the left ventricular wall which accompany the two types of defect are different. Whereas with stenosis of the aorta which is characterized by a mainly isometric type of contraction of the left ventricle, the dimensions of this chamber and the thickness of its wall do not change to any extent, in the case of arteriopulmonary shunting, characterized by a mainly isotonic type of contraction, the dimensions of the left ventricle and the amplitude of its contractions show a sharp increase during the earlier stages of compensatory hyperfunction, whereas the thickness of the wall shows a definite decrease.

This suggests that compensatory hyperfunction of the heart in response to the two defects we have described differs not only in the extent to which myocardial tension increases but also in the extent to which different sections of the cardiac muscle are implicated.

SUMMARY

It was shown in acute experiments on dogs that dysfunction of the left ventricle due to progressive stenosis of the aorta or aortopulmonary shunting is characterized by identical hemodynamic indices but in the former case it develops after a sharp increase of tension in the myocardium of the left ventricle, and in the latter case in the presence of an unchanged or slightly increased tension of the myocardium in this heart region.

The dynamics of myocardial tension during the above-mentioned types of dysfunction do not depend substantially on whether the given index is calculated with or without regard to the area of the cross section of the left ventricle wall.

The above findings corroborate the view that during a predominantly isometric hyperfunction of the heart (stenoses of the valve openings, hypertension), cardiac dysfunction results from exhaustion of the functional reserves of the myocardium, and during a predominantly isotonic hyperfunction (defects of the cardiac valves, arteriovenous shunting) , it results from failure to mobilize these reserves.

LITERATURE CITED

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2. F. Z. Meerson, Myocardial Hyperfunction and Heart Insufficiency [in Russian], Moscow (1965).