THE MYOCARDIUM AND HEMODYNAMICS IN CARDIAC HYPERFUNCTION AND INSUFFICIENCY

COMMUNICATION 1. THE WORK AND MECHANICAL EFFICIENCY OF THE HEART IN EXPERIMENTAL LESIONS OF THE HEART VALVES

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In normal conditions and more especially in disease, the external work of the left ventricle, expressed as the product of the intra-aortic pressure during systole and the minute volume of the heart, reliably reflects the results of the contractile activity of this division of the heart. In valvular defects of the heart, however, the external work is always less than the total work performed by the ventricle. We know, for example, that in mitral incompetence, when the regurgitation of blood into the left atrium may reach 2 liters/min, the expulsion of blood into the aorta and the mean pressure within the aorta may remain normal [4], and consequently the external work of the left ventricle is also normal. In fact, in mitral incompetence, the left ventricle expels an increased volume of blood, consisting of the effective stroke volume and the volume of regurgitated blood, so that the total work of the ventricle and of the heart is undoubtedly increased.

A similar situation arises in marked aortic stenosis, when as a result of the compensatory strengthening of the cardiac contractions and the elevation of the intraventricular pressure to 200-300 mm Hg the minute volume of the heart remains normal while the pressure within the aorta is slightly lowered [5, 6]. The external work of the left ventricle in this case is diminished, although the total work performed by the ventricle is clearly considerably increased.

In order to study the work of the heart objectively in valvular lesions, we have suggested that both the external and the total work of the ventricle should be determined, and the ratio between the external and total work (i.e., a figure indicating what fraction of the total work is converted into external) should be defined as the mechanical efficiency of the ventricle. The present investigation was undertaken to provide an experimental basis for these criteria of the work of the heart, and it utilized the example of experimental aortic stenosis.

EXPERIMENTAL METHOD

Acute experiments were carried out on 22 dogs weighing 12-27 kg. The operation to produce experimental aortic stenosis was performed under morphine–pentothal anesthesia, using controlled respiration with pure oxygen. The right pleural cavity was opened by an incision in the fifth intercostal space from the parasternal to the paravertebral line. The ascending aorta was isolated at its origin and a ligature, consisting of flexible wire covered with PVC with an external diameter of 2 mm, passed beneath it. The ends of the ligature were passed through a hard polythene tube, and after emerging from the tube were clamped so that the ligature fitted closely around the aorta without constricting it. One catheter was introduced through a branch of the right superior pulmonary vein into the left ventricle, and another through the femoral artery into the aorta. The pleural cavity was then dried and sealed by closure in layers. The end of the tube with the clamped ends of the ligature lay on the surface of the chest (above the sutures). A pointed conical rod was introduced under the clamp between the ends of the ligature, and gradually pushed forward parallel to the body surface, thereby drawing the ligature out through the tube and constricting the aorta. In this way a progressive aortic stenosis was produced stage by stage, and the cross sectional area of the stenosed part of the aorta under these circumstances amounted to between 80 and 5% of its initial value.

The pressure inside the left ventricle and aorta was measured in normal conditions and at each stage of stenosis by means of electromanometers of the "Barovar" type, made by the firm of Alvar, and recorded simultaneously with the ECG on an ink-recording "Visograph" apparatus made by the same firm. The minute volume of the heart in normal conditions and in all stages of stenosis was determined by Fick's method, and the oxygen concentration in samples of blood taken from the right ventricle and the femoral artery was measured by means of an "Elema" dish oxymeter, previously calibrated with a Van Slyke's apparatus. The oxygen demand was recorded for a period of 10 min after each stage of stenosis had been induced, using a Prema metabolimeter.

The external work of the left ventricle (W_{ex}) was calculated from the usual formula:

$$W_{ex} = P_a \cdot V_{st} + \frac{mV_2}{2g} .$$

Since $m = \gamma \cdot V_{st}$ and $V = \frac{V_{st}}{t_{syst} \cdot S}$, this formula may be written as follows:

$$W_{ex} = \frac{P_a \cdot V_{st}}{735{,}5 \cdot 10^2} + \frac{\gamma V_{st}}{2g \cdot 10^5} \left(\frac{V_{st}}{t_{syst} \cdot S} \right)^2 , \quad (1)$$

where W_{ex} is the external work of the left ventricle (in kilogram-meters), P_a the mean pressure inside the aorta during systole (in mm Hg), V_{st} the stroke volume of the left ventricle in cubic centimeters, $\gamma = 1.05$ g/cm^3 (the specific gravity of blood), $g = 981$ cm/sec^2 (the acceleration of the force of gravity), t_{syst} the duration of systole (in seconds), and S the cross sectional area of the aorta (in cm^2).

The total work of the ventricle (W_{to}), from our point of view, is measured by the value of the potential energy communicated to the blood by the contracting ventricle from which it is expelled. This energy is given by the product of the mean intraventricular pressure during the period of expulsion and the stroke volume of the blood, and in aortic stenosis it may be calculated from formula (2):

$$W_{to} = \frac{P_{ve} \cdot V_{st}}{735.5 \cdot 10^2} , \quad (2)$$

where W_{to} is the total stroke effort of the left ventricle (in kilogram-meters), P_{ve} the mean stroke pressure in the left ventricle (in mm Hg), and the remaining symbols are as in formula (1). By multiplying the resulting values of the external and total stroke work by the rate of the heart, we obtained the corresponding values of the external and total work of the left ventricle per minute.

The mechanical efficiency of the left ventricle—η—was calculated in percent from formula (3):

$$\eta = \frac{W_{ex}}{W_{to}} \cdot 100. \quad (3)$$

EXPERIMENTAL RESULTS

The results of these observations showed that in "normal" anesthetized dogs the mean value of the total work of the left ventricle in 22 dogs was 5.7 kgm/min, and of the external work 5.4 kgm/min, so that the corresponding mechanical efficiency of the work of the left ventricle was 95.1% (see table).

During moderate compensatory hyperfunction of the heart, caused by closure of 30-50% of the cross sectional area of the aorta, the total work increased very slightly (to 6.1 kgm/min), while the mean stroke pressure within the ventricle and the minute volume of the heart varied around the normal values. The external work was slightly diminished immediately after the fall in the intra-aortic pressure (to 5.1 kgm/min). The corresponding mechanical efficiency also fell to 83.4%.

During more intensive compensatory hyperfunction of the heart, caused by obstruction of 50-70% of the cross sectional area of the aorta, on account of the increased intraventricular pressure the total work of the heart rose to 6.4-6.8 kgm/min. This took place notwithstanding a slight decrease in the minute volume of the heart, for the increase in pressure in the left ventricle was considerable. At this stage of experimental aortic stenosis the external work was either unchanged or diminished depending on the level of the intra-aortic pressure and the minute volume, and its mean value was 5.3-4.4 kgm/min. In all cases the mechanical efficiency was lowered (82.8-64.6%).

Mean Values Showing the Changes in the Minute Volume of the Blood, the Mean Systolic Pressure in the Aorta, and the Mean Stroke Pressure in the Left Ventricle, and the Pattern of the Total and External Work of the Heart and the Mechanical Efficiency in Aortic Stenosis

Indices	Degree of aortic stenosis (% of normal area)							Stenosis discontinued
	0	30-50	50-60	60-70	70-80	80-90	90-95	
Minute volume of blood (ml/min)	4106	3934	3963	3692	3374	3970	3296	3345
Mean systolic pressure in aorta (mm Hg)	94	91	94	84	66	64	46	81
Mean stroke pressure in left ventricle (mm Hg)	106	114	118	136	130	154	199	95
External work (kgm/min)	5.4	5.1	5.3	4.4	3.1	3.6	2.2	3.8
Total work (kgm/min)	5.7	6.1	6.4	6.8	6.1	8.2	8.9	4.1
Mechanical efficiency (%)	95.1	83.4	82.8	64.7	50.8	43.9	24.5	92

With the maximum intensity of compensatory hyperfunction of the heart caused by obstruction to 80.95% of the cross sectional area of the aorta, a decrease took place in the minute volume of blood, as shown by a fall in the intra-aortic pressure and a simultaneous rise in the pressure within the left ventricle (Fig. 1). The total work increased correspondingly to 8.2-8.9 kgm/min, while the external work decreased to 3.6-2.2 kgm/min. As a result, the mechanical efficiency fell to 24.5%, i.e., it was reduced to less than one-quarter the normal value.

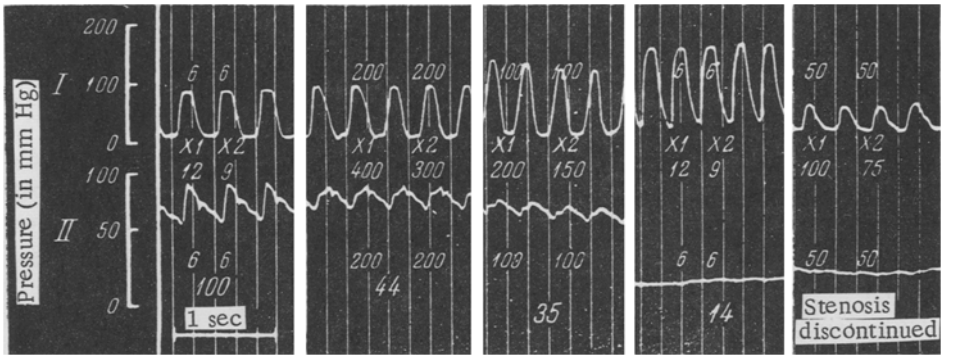

Fig. 1. Changes in the pressure in the left ventricle (I) and aorta (II) during progressive aortic stenosis. 100) Before production of aortic stenosis (initial cross section 100%); pressure in left ventricle 95/10 mm, in aorta 87/55 mm Hg; ventriculo-aortic gradient 8 mm Hg; 44) 5 min after reduction of cross section of aortic orifice to 44%, pressure in left ventricle increased to 103/10 mm Hg, systolic pressure in aorta lowered to 85 mm, but diastolic raised to 65 mm Hg; ventriculo-aortic gradient 18 mm Hg; 35) 5 min after reduction of cross section of aortic orifice to 35%, pressure in left ventricle increased to 133/17 mm, pressure in aorta lowered to 77/55 mm Hg, ventriculo-aortic gradient 56 mm Hg; 14) 5 min after reduction of cross section of aortic orifice to 14%, pressure in left ventricle 150/75 mm, pressure in aorta sharply decreased to 15 mm Hg, pulse waves disappeared; ventriculo-aortic gradient 135 mm Hg. Stenosis discontinued—systolic pressure in left ventricle sharply lowered, to below initial level (70 mm Hg), diastolic pressure increased (20 mm Hg), pressure in aorta not restored (18 mm Hg).

The results of all the determinations of the total and external work, expressed as percentages of the initial values, are given in Fig. 2, from which some idea of the variations may be obtained. These variations do not alter the basic principle, namely, that intensive compensatory hyperfunction of the heart, caused by a valvular defect of the heart of considerable severity, was accompanied in our experiments by an increase in the total work, a decrease

in the external work, and a fall in the mechanical efficiency of the heart in proportion to the degree of severity of the defect (see table).

The method which we used to calculate the total work of the ventricle is applicable to both aortic and pulmonary stenosis, for in these lesions the total work is equal to the product of the stroke pressure inside the ventricle and the minute volume of blood, and the excess of the total over the external work is due entirely to the fact that much of the energy communicated by the ventricle to the blood is lost in overcoming the resistance to the blood flow in the stenosed area. With an increase in the degree of stenosis the resistance to the blood flow increase, as also does the loss of energy in overcoming it; the mechanical efficiency of the ventricle falls in proportion.

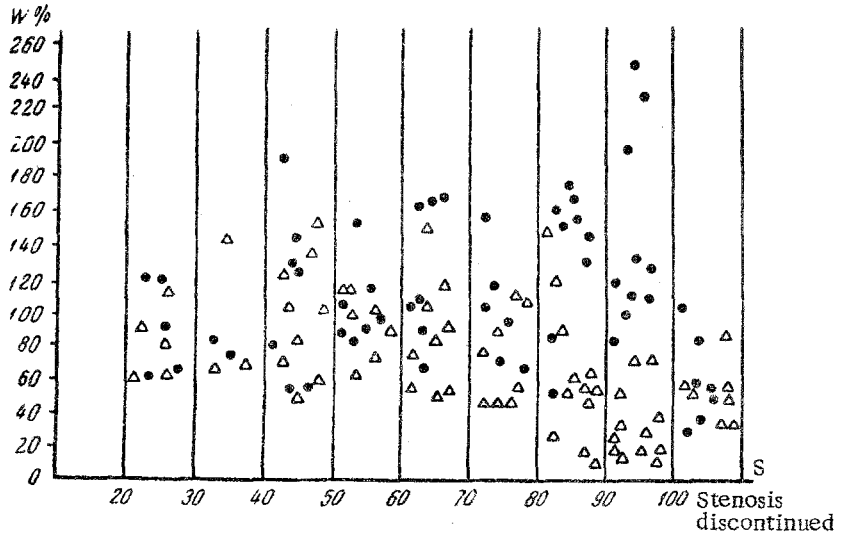

Fig. 2. Changes in the total and external work during progressive stenosis of the aortic orifice. W%) work (as a percentage of initial value); S) degree of aortic stenosis (as a percentage of initial cross section); the dots indicate the total work, the triangles the external work.

In the various forms of valvular incompetence, the excess of the total over the external work is due to the fact that besides the effective stroke volume of blood which is taken into consideration when calculating the external work, there is also the volume of regurgitating blood; although this is not considered, it plays an important part in the calculation of the total work. Hence the total work in aortic incompetence is the product of the stroke pressure inside the ventricle and the sum of the effective and regurgitating stroke volumes of blood. In the case of a severe defect of the semilunar valves the volume of regurgitating blood may be equal to the effective stroke volume, when the total work of the left ventricle will be twice the external work, and the mechanical efficiency will be down to 50%. In mitral incompetence the total work may be calculated in a similar way. In fact, however, the retrograde expulsion of blood from the left ventricle into the left atrium in this condition begins before the semilunar valves have opened, and some of it consequently takes place at a lower pressure than the stroke pressure. This complicates the calculation. The essential fact is that in all valvular defects the normal, subnormal, or low value of the external work is maintained on account of the increased total work of the heart. The latter, in turn, is due to the continuous and fairly intensive compensatory hyperfunction of the heart [1, 2]. Consequently, the characteristic feature of this hyperfunction in valvular lesions is an increase in the loss of energy at the stage of the conversion of total into external work—a lowering of the mechanical efficiency of the heart.

The last factor plays an important role in the development of heart failure in valvular defects, either as a result of disturbances of the compensatory hyperfunction of the heart or as a result of an increase in the degree of injury to the valves. We know that in this form of failure the total efficiency of the heart, expressed as the ratio between its external work and the energy formed by the myocardium on account of oxidation, is always decreased. The results show that the decrease in the total efficiency of the heart in cases of failure due to valvular defects is largely determined by a decrease in mechanical efficiency. We must therefore reexamine the traditional view, according to which the decrease in the total efficiency of the heart in cases of valvular decompensation is determined entirely by a disturbance of the metabolic process in the myocardium [3]. The great value of the modern combined

approach to the treatment of lesions of the heart valves is due to the fact that pharmacological factors are called in to restore the efficiency of the metabolic processes in the myocardium, while the rationale of the surgical correction of the valvular defect lies in the restoration of the mechanical efficiency of the heart.

SUMMARY

In cases with vitium cordis only a part of work done by the ventricle is transformed into its external work. All the work performed by the ventricle may be designated as total work, whereas the ratio of external and total work is designated as mechanical efficiency of the ventricle. The study of the dynamics of external, total work and of the mechanical efficiency of the left ventricle in the process of progressive aortic stenosis development demonstrated that in the given experimental vitium cordis the energy losses for overcoming the blood flow resistance in the area of stenosis lead to a 4-fold reduction in the mechanical efficiency of the left ventricle (as compared to the normal).

Suggestions are presented on determination of total work and of mechanical efficiency in other forms of vitium cordis; it is also suggested that reduction of the efficiency of the heart in cardiac insufficiency depends not only on disturbed myocardial metabolism, but also on the drop of mechanical efficiency of the heart.

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