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(Translated from Byulleten' Experimental'noi Biologii i Meditsiny, Vol. 55, No. 1, pp. 31-35, January 1963)

Original article submitted December 19, 1961

When physical work is performed by a healthy organism, the minute volume of the heart may be increased many times over, not only as a result of an increase in the contractile function of the heart but also by a lowering of the resistance of the vascular field in consequence of a diminution of the tone of the smooth muscle of the arterioles. Because of this fact, a considerable minute volume may be achieved without an undue increase in the strain on the myocardium, and the cardiac hyperfunction mainly follows the lines of the isotonic (economic) variant.

From clinical experience it appears that in diseases of the cardiovascular system a decrease in the resistance of the vascular field plays an important role in the mechanism of economic compensation of the accompanying defects. However, the study of this aspect of compensation of diseases of the circulatory system is incomplete.

In the present research the role of the lowering of the vascular resistance in compensation was investigated in experimental aortic stenosis, producing a gradually progressive cardiac defect.

EXPERIMENTAL METHOD

Using a technique described previously [1], in 22 acute experiments on dogs a gradually progressive stenosis of the aortic orifice was produced, causing a graded constriction of the aortic orifice by 80-95% of its initial cross-section. In all stages of stenosis the pressure in the left ventricle and aorta and also the minute volume of the heart were measured. The pressure was measured by means of a "Barovar" manometer manufactured by the firm "Alvar" and recorded on an ink-recording "Visograph" apparatus made by the same firm. The minute volume was determined by Fick's method. The total peripheral vascular resistance was calculated from the usual formula

\[ R = \frac{1332 \cdot P}{V} \]

where \( R \) is the total peripheral vascular resistance (in dynes \( \cdot \) sec \( \cdot \) cm\(^{-6} \)); 1332 the coefficient of conversion of pressure, expressed in mm Hg, into dynes/cm; and \( P \) is the mean pressure within the aorta (in mm Hg) and \( V \) the volume velocity of the blood flow (in ml/sec).

EXPERIMENTAL RESULTS

It will be apparent from Table 1 that as the aorta was constricted the maximal systolic pressure within the ventricle increased gradually, ultimately reaching 247 mm Hg, i.e., twice the initial value of 122 mm Hg. This change, a direct expression of the increase in the strength of the ventricular contractions, was combined with a gradual lowering of the maximal pressure within the aorta. The intra-aortic pressure fell as a result of the progressive stenosis from 104 to 47 mm Hg, i.e., by more than half. Consequently, the ventriculo-aortic pressure gradient rose from 18 to 200 mm Hg, i.e., more than ten-fold. This ten-fold increase in the ventriculo-aortic pressure gradient was a factor in the maintenance of the normal or subnormal minute volume of the heart, for even when the cross-sectional area of the aorta was reduced by 90-95%, the minute volume fell on the average to not less than 80% of normal.
In this hemodynamic picture, typical of aortic stenosis, particular attention is drawn to the fact that the aortic pressure was halved while only a very small decrease took place in the minute volume of the heart. This change was a definite sign of a lowering of the peripheral vascular resistance.

Calculation of the peripheral vascular resistance showed that in the course of progressive aortic stenosis this index altered variously in different animals. In 16 experiments the resistance fell gradually as the degree of stenosis increased. The mean values characterizing this particular group of experiments are shown in Table 2;

It follows from the figures in Table 2 that with slight and moderate degrees of stenosis the resistance in the animals varied between 87 and 92% of normal; when the cross section of the aorta was closed by 70-80% it fell to 64.44% of normal, and finally, with the maximal degree of stenosis it fell to 23.45%, i.e., to less than one-quarter of the normal value.

The results of all the determinations of the resistance, demonstrating the individual variations, are shown in the graph (Fig. 1).

In the remaining six experiments the total peripheral vascular resistance was not lowered but, on the contrary, it was increased either immediately after the onset of stenosis (2 experiments) or when considerable degrees of stenosis were present (4 experiments).

When comparing these two groups of experiments it must be emphasized that if the lowering of the peripheral vascular resistance was considerable, the increase in the ventriculo-aortic gradient of pressures was largely the result of a fall in the pressure within the aorta, and only to a lesser degree of an increase in the pressure within the ventricle. As a result, the mobilization of the contractile function of the heart was relatively less pronounced; the strain on the myocardium and the total work of the heart did not reach an excessively high level. Compensation was due to a combination of cardiac and extracardiac factors, and was brought about in a sufficiently economic manner.

With an unchanged or increased peripheral vascular resistance, the increase in the ventriculo-aortic gradient resulted, conversely, from an increase mainly in the intraventricular pressure, i.e., from mobilization of the con-