ary 28th the general condition was better, but it still refused food. Electrocardiogram on that date (Fig. 5) showed a lesser degree of T wave inversion and less marked left preponderance.

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[1] Rohmer, P., *Z. Exp. Path. u. Therap.*, 1912, xi, 426.
[2] McCulloch, H., *Am. J. Dis. Child.*, 1920, xx, 89.
[3] Smith, S. C., *J. Am. Med. Assn.*, 1921, lxvii, 765.
[4] Marvin, H. M., *Am. J. Dis. Child.*, 1925, xxix, 433.

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Work of the Left Ventricle in Normal, Hypertension and Arteriosclerosis.

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It is very probable that increased work of any chamber of the heart is followed after some time by hypertrophy and dilation of that chamber. It is of interest to know the exact increase in work for the left ventricle of the heart in hypertension and in generalized arteriosclerosis.

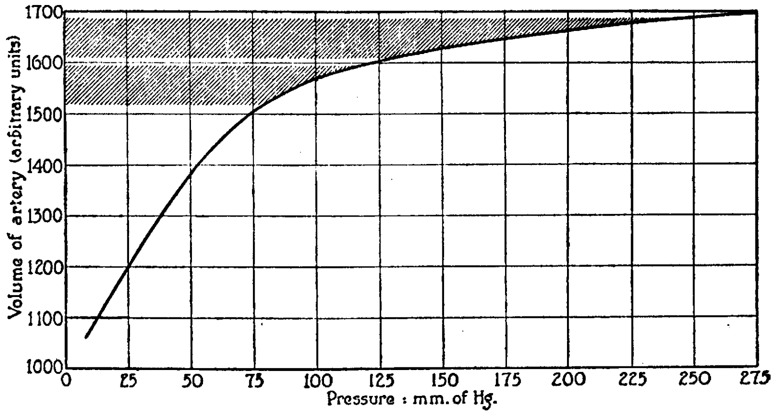
The work of the left ventricle per beat is represented by the equation:

$$\int_{P_1}^{P_2} PdV + \frac{Sv^2}{2g}$$

where P is the pressure of the blood in the aorta, P_1 the diastolic and P_2 the systolic pressures, respectively; V the volume of the arterial system, corresponding to the pressure P, and dV an infinitesimal change in the volume when a small amount of blood is ejected from the left ventricle and the pressure increased by the amount dP. S is the stroke volume, v the velocity of the blood in the aorta and g the constant of gravity. The second term may be neglected in our problem because it makes up only 1 or 2 per cent of the work of the left ventricle and is constant for equal stroke volumes and velocities in the aorta.

If we can find the relation between P and V we can integrate the first term of the work equation and can thus find the work of the left ventricle per beat. Bramwell, Downing and Hill[1] have determined the relation between the volume and pressure in the "mean normal" artery of man. The curve, Fig. 1, is a reproduction of their curve of this relation. If we assume that in the "normal" person the diastolic blood pressure is 80 and the systolic 120, then, ac-

FIG. 1.



Integration of $\int_{P=80}^{P=120} PdV$ and $\int_{P=130}^{P=240} PdV$ from Pressure-Volume relation of Bramwell, Downing and Hill's "mean normal" artery curve. 1 scale division along ordinates equals 100 cc. volume increase. 1 scale division along abscissa equals 25 mm. Hg. pressure.

cording to this curve, the corresponding change in volume when the pressure rises from the diastolic to the systolic is 80 cc. The shaded area between 1515 cc. and 1595 cc. and 80 mm. Hg. and 120 mm. Hg. represents the definite integral

$$\int_{P=80}^{P=120} PdV$$

or the work done in forcing 80 cc. of blood into this arterial system between the pressures 80 and 120. I have determined this area by means of a planimeter, and have calculated the work represented by the shaded area by comparison with the area of one of the small squares, each of which represents 34 gram meters of work. The work represented by this shaded area is 98.6 gram meters of work. In other words, 98.6 gram meters of work are performed when 80 cc. of blood are forced into the "mean normal" arterial system between the pressures 80 mm. Hg. and 120 mm. Hg.

If now 80 cc. of blood are forced into the "mean normal" artery between the diastolic pressure of 130 mm. Hg. and the systolic of 240 mm. Hg., the work represented by the upper shaded area is 192.4 gram meters. In other words, 192.4 gram meters of work are done in forcing 80 cc. of blood into the "mean normal" artery between a diastolic pressure of 130 mm. Hg., and a systolic pressure of 240 mm. Hg. The increase in work necessary to force 80 cc. of blood into the "mean normal" arterial system between the pressures

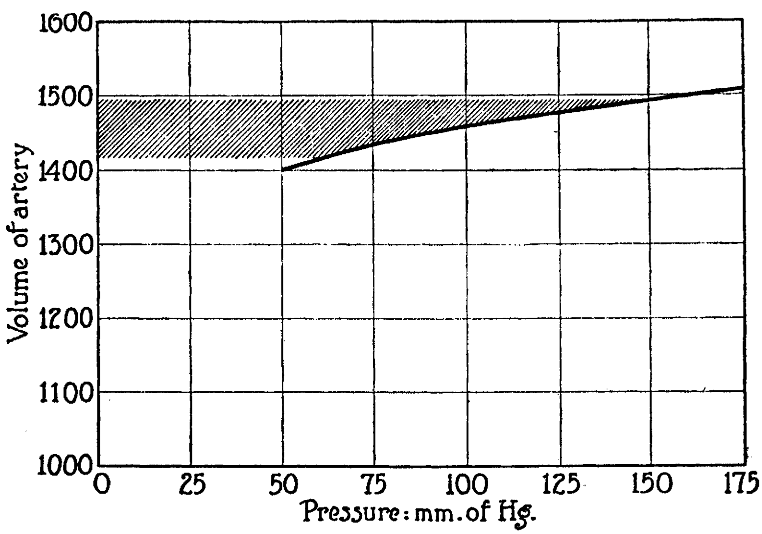
240/130, is 95 per cent greater than the work necessary to force the same amount of blood into this arterial system, between the normal systolic and diastolic pressures 120/80.

In Fig. 2 and in Fig. 3 a piece of the curve of Volume-Pressure relation is drawn for an arterial system in which the rigidity of the system is 2.8 times as great as in the "mean normal" artery of Fig. 1. The rigidity of the arterial systems whose Pressure-Volume relations are shown in Figs. 2 and 3 is 1.7 times as great as the rigidity of the arterial system of a man 80 years old investigated by Bramwell, Hill and McSwiney.[2] We can regard this rigidity as that of advanced arteriosclerosis.

The shaded area in Fig. 2 represents the work done in forcing 80 cc. of blood into this rigid artery between the diastolic pressure 60 mm. Hg. and a systolic of 145 mm. Hg. 105.4 gram meters of work are performed when the stroke volume of 80 cc. is forced into this rigid arterial system between the pressures 60 mm. Hg. and 145 mm. Hg. or 7 per cent more work than is done in forcing the same volume at normal pressures into the "mean normal" artery.

The pressure within the rigid arterial system will rise from 80 mm. Hg. to 185 mm. Hg., if we force 80 cc. of blood into it, when

FIG. 2.



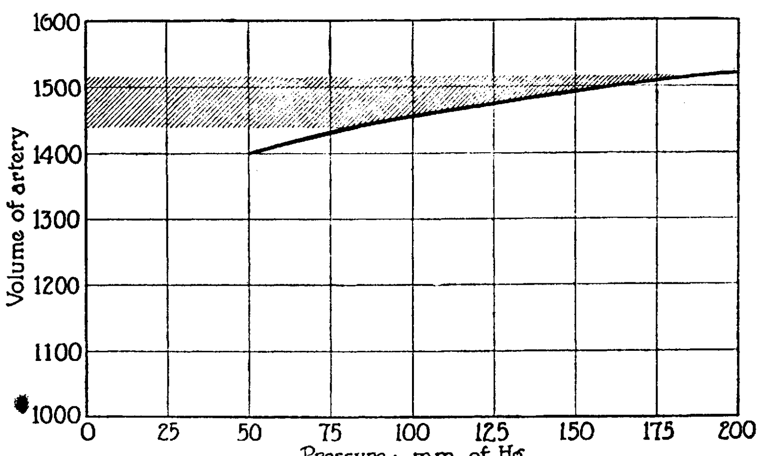
Integration of $\int_{P=60}^{P=145} PdV$ from Pressure Volume relation of an arterial system whose rigidity is 2.8 times that of the "mean normal" artery. The pressure volume curve is the end piece of Bramwell, Downing and Hill's "mean normal" curve shifted into the area of pressures 50-175.

it is already filled to a pressure of 80 mm. Hg. The work done in forcing 80 cc. into this arterial system between a diastolic pressure of 80 mm. Hg. and a systolic of 185 mm. Hg. is represented by the shaded area of Fig. 3, and is equivalent to 136 gram meters of work, or 40 per cent more work than is performed in forcing 80 cc. of blood into the "mean normal" artery at pressures between 80 diastolic and 120 systolic.

Before drawing conclusions from the above determinations, it is necessary to go back and criticise the method by which Bramwell, Downing and Hill obtained the values of the Pressure-Volume relation of their "mean normal" arterial system. In the first place, they determined the data for the coefficient of volume elasticity by measurements of pulse wave velocity at various pressures in isolated carotid arteries obtained soon after death. The volume elasticity of an artery may change after death but Bramwell and Hill[3] have shown that the velocity in an isolated artery compares very well with the velocity measured in a living man. So this objection has little weight insofar as the general form of the curve is concerned.

It is not rigorously correct to apply measurements done on the carotid artery to the pressure-volume relations in the whole arterial system. But insofar as the general problem of calculation of heart

FIG. 3.



Integration of $\int_{P=80}^{P=185} PdV$ from Pressure Volume relation of an arterial system whose rigidity is 2.8 times that of the "mean normal" artery. This pressure volume curve is the end piece of the "mean normal" curve of Fig. 1 shifted into the area of pressures 50-200.

work is concerned under the conditions of this investigation and for the purposes of this study, the determinations of the work of the left ventricle from the Pressure-Volume relations of the "mean normal" artery of Bramwell, Downing and Hill is justifiable.

Bramwell, Downing and Hill constructed their curve of the Pressure-Volume relations in the "mean normal" artery without taking into consideration the "run off" through the capillaries during systole.* The rate of capillary "run off" during cardiac systole increases from a minimum at the beginning of systole to a maximum at the end of cardiac systole. For this reason larger volumes are ejected from the heart in the latter parts of systole when the blood pressures are higher than would be indicated by Bramwell, Downing and Hill's curve.

Our calculation of the work of the heart from Bramwell, Downing and Hill's curve of the Pressure-Volume relation in the "mean normal" artery is somewhat less than the real value of this work, because larger volumes are ejected from the heart at higher pressures toward the end of systole than would be indicated by the Pressure-Volume relations of the curve of the "mean normal" artery. If we correct this curve by the assumption that the systole takes 0.3 seconds and the diastole 0.6 seconds, and that the "run off" from the arteries is proportional to the pressures in the aorta, we calculate an amount of work which is not quite 10 per cent greater than the work calculated from Bramwell, Downing and Hill's curve. The blood pressure in the periphery does not vary as much as it does in the central vessels, and therefore the "run off" is much steadier than that used in the above assumption We are therefore justified in saying that the errors in our calculation are less than 10 per cent, provided the stroke volume remains the same in each calculation. An error of this order vitiates in no way the following general conclusions:

1. In hypertension the work of the left ventricle is enormously increased above the work done at normal pressures, increase in work being very nearly proportional to the increase in the systolic pressure. This increase in work is more nearly proportional to the increase in systolic pressure than it is to the increase in the sum of the systolic and diastolic pressures divided by two, often erroneously called the "mean" pressure. The fact that the "run off" from the

* Bramwell, J. C., Downing, A. C., and Hill, A. V., were cognizant of this, but felt that the amount of blood ejected through the capillaries during cardiac systole would not alter many of the conclusions to be drawn from the curve as constructed by them without consideration of this "run off."

arterial system is greater during the period of systolic pressure makes this conclusion even more secure.

2. Arteriosclerosis increases the work done by the left ventricle only very little if there is as much fall in diastolic pressure as there is rise in systolic pressure. If the diastolic pressure remains constant and the systolic rises, the work of the left ventricle increases, but it is largely this rise in systolic pressure that increases the work of the heart under these circumstances. In other words, arteriosclerosis only increases the work of the heart markedly insofar as it necessitates increased blood pressure. The hypertension is then largely the cause of the increased work of the left ventricle.

This is a complete report.

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[1] Bramwell, J. C., Downing, A. C., and Hill, A. V., *Heart*, 1923, x, 289.

[2] Bramwell, J. C., Hill, A. V., and McSwiney, B. A., *Heart*, 1913, x, 233.

[3] Bramwell, J. C., and Hill, A. V., *Proc. Roy. Soc. London*, 1922, 93 series B, 298.