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Hemodynamics of Arteriosclerosis Influence of Elastic Factor on Circulation.

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Using a modified heart-lung preparation of Starling, with the arterial cannula inserted in the brachial cephalic artery in the first experiments, and in the later experiments in the arch of the aorta, the following experiments were done.

In drawing conclusions as to the external work of the heart, we consider that our experiments establish the diastolic volume of the heart as an index of the energy consumption of the heart and therefore a measure of the work of the heart, provided the heart muscle function remains as constant throughout the period under consideration. Only one experiment is given here.

Calibration of elastic coefficient in terms of percentage volume increase of arterial system to mm. Hg. increase pressure or mm. Hg. increase in pressure/% increase in volume. This is the "effective" elastic coefficient or rigidity of arterial system.

Without Air Chamber or Arteriosclerosis. Content of "effective" arterial system = 210 cc. 2 cc. blood injected into system gives a rise of 56 mm. Hg. in pressure.

2/210 = 0.95% increase in volume for 56 mm. Hg., or 1% increase in volume for 59 *mm. Hg.* A. V. Hills normal artery between 80-120 mm. Hg. showed 6.6% increase in volume for 40 mm. Hg., or 1% increase in volume for 6 *mm. Hg.* Ergo in this system without air chambers and representing extremely severe arteriosclerosis, there is a rigidity 9.8 x normal "mean" artery of A. V. Hill at normal pressure.

With Air Chamber or Normal. Content of arterial system = 240 cc. 20 cc. blood injected into system gives a rise of 27 mm. Hg. 20/240 = 8.3% for 27 mm. Hg., or 1% increase for 3.3 mm. Hg. Ergo rigidity is ½ that of A. V. Hill "mean" normal artery. Rigidity without air chamber or in arteriosclerosis = 18 x that with air chambers or in normal.

Calibration of Cardiometer.

20 cc. = 13 mm.
13 mm. } 12.7 mm.
12 mm.

Aortic cannula = 9 mm.

= 8 mm. at narrow point

Without Air Chamber (Arteriosclerosis)		With Air Chamber (Normal)	
Sys. B.P. = 140	2567 mm. Hg.	= 80	38 mm. Hg.
Dias. B.P. = -116		= 42	
Stroke Vol. = 13.0 cc.		= 13.0 cc.	
Minute Vol. = 1014 cc. Heart rate 78.		= 1014 cc.	
Circul. Vol. = 686 cc.		= 692 cc.	
Coronary flow 328 cc.		= 322 cc.	

Diastolic volume is 1 cc. less without air chambers than with air chambers.

Sys. B.P. = 134	250? mm. Hg.	= 80	38 mm. Hg.
Dias. B.P. = -116		= 42	
Stroke Vol. = 14 cc.		= 13.4 cc.	
Minute Vol. = 1064 cc. Heart rate 76.		= 1018 cc.	
Circul. Vol. = 693 cc.		= 706 cc.	
Coronary flow 371 cc.		= 312 cc.	

Diastolic volume is 0.5-1.0 cc. less without air chambers than with air chambers.

Sys. B.P. = 136	238? mm. Hg.	= 90	52 mm. Hg.
Dias. B. P. = -102		= 38	
Stroke Vol. = 14.3 cc. Rate 74.		= 13.8 cc.	
Minute Vol. = 1058 cc.		= 1021 cc.	
Circul. Vol. = 680 cc.		= 686 cc.	
Coronary flow 378 cc.		= 335 cc.	

Diastolic volume in arteriosclerosis is 0.75 cc. less than in normal.

Sys. B.P. = 128	238? mm. Hg.	= 78	34 mm. Hg.
Dias. B.P = -110		= 44	
Stroke Vol. = 14.5 cc. Heart rate 72.		= 14.5 cc.	
Minute Vol. = 1015 cc.		= 1015 cc.	
Circul. Vol. = 680 cc.		= 706 cc.	
Coronary flow 335 cc.		= 309 cc.	

Diastolic heart volume 0.75 cc. less in arteriosclerosis.

In all other experiments, the diastolic pressure remained above the base line.

Throughout the course of the experiment, lasting 2 hours, heart volume in diastole is approximately 3 cc. less at end than at beginning of experiment:

Minute Vol. = Total output of left ventricle calculated from ½ cardiometer change in systole multiplied by heart rate.

Circul. Vol. = Volume of flow as measured with stop watch and calibrated measuring cylinder on blood flowing out of venous system into venous reservoir.

Stroke Vol. = Output of one ventricle. This value is obtained by multiplying the height of the cardiometer curve in millimeters by 20 and dividing by 2 times the increase in height of the cardiometer tracing when 20 cc. are forced into the cardiometer system from a record syringe connected to the system.

Conclusions: 1. The external work of the heart is not increased when the "effective" rigidity of the arterial system is increased 10

times the normal, provided the lumen of the arterial system remains the same and the rigidity of the venous system remains normal and, therefore, much lower than the rigidity of the arterial. 2. The minute volume of blood flow in the systemic arterial system is not decreased more than 5% when the "effective" coefficient of volume elasticity of the arterial system is increased 10 times the normal, provided the lumen of the arterial system remains the same and the rigidity of the venous system remains normal and, therefore, much lower than the rigidity of the normal arterial system. 3. Apparently at times the systolic ejection time is lengthened and the diastolic filling time of the left ventricle decreased when the rigidity of the arterial system is increased greatly, thus causing slightly decreased diastolic volume of the ventricles and decreased stroke volume. The coronary flow is slightly decreased by the increased length of systole. The external work of the heart is slightly decreased also. These conclusions must be confirmed by optical recording and more accurate stroke volume measurements.

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Presence of the Morning Alkaline Tide in a Case of Achlorhydria.

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In previous communications from this clinic it has been stated that the urinary alkaline tide is primarily due to the secretion of hydrochloric acid by the stomach, for the tide was absent in most cases of achlorhydria.[1] This observation has been confirmed by Ackman[2] and Davies.[3] Further study showed that it was necessary to distinguish between alkalinity developing during the day-time and that shown immediately after awakening,[4] for this latter change is frequently seen in patients where hydrochloric acid is not found in the stomach, and is probably due to the respiratory adjustment of the subject to waking conditions.[5] Recently we have studied a patient with achlorhydria who showed a tide exactly resembling

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[3] Davies, D. T., *Brit. J. Exp. Pathol.*, 1929, x, 1.

[4] Hubbard, R. S., *J. Biol. Chem.*, 1929, lxxxiv, 191.

[5] Leathes, J. B., *Brit. Med. J.*, 1919, ii, 165.