tance for 5, 10, 15, 20 and 30 minutes respectively. At the end of the experiment 5 cc. of glucose broth were added to each tube. All the tubes, including the 3 controls, were left at room temperature for several days. Growth of the fungus did not occur in any of the tubes containing the fungus which were exposed to ultraviolet light but luxuriant growth was obtained in all of the controls.

Under the conditions of these experiments it would seem that at least one member of the Gypseum group of ring-worm fungi *(Trichiphyton asteroides)* is markedly susceptible to the action of ultraviolet light. Probably other members of the genus are also susceptible, though such tests as we have employed are more qualitative than quantitative. The rationale of treating this type of infection with ultraviolet light is apparently borne out by the experimental method.

4876

Experimental Aortic Insufficiency. I. Regurgitation Maximum and Mechanisms for Its Accommodation Within Mammalian Ventricle.

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The investigations reported in this paper were designed to determine the greatest magnitude of regurgitation possible in the relaxing mammalian heart under optimal conditions and to study the mechanisms by which such volumes are accommodated. These questions have previously been studied chiefly with the aid of artificial circulation machines. But, as Allan[1] properly concludes, conditions are manifestly so different in the mammalian heart that it would be unwise to transfer such values to the human circulation. A number of physical factors enter into relaxation of the mammalian heart which are difficult to reproduce in an artificial model. Ventricular diastole begins with a phase of *isometric relaxation* during which the intraventricular pressure is reduced from that existing in the aorta to a level below that in the auricle. During this early diastolic phase, averaging 0.07 second in the dog and 0.12 second in man, the ventricular cavity remains in the state of obliteration reached at the end of ejection. Consequently, by far the greatest

[1] Allan, *Heart*, 1926, xii, 200.

backflow and possibly all of it must occur during the subsequent inflow phases. Whether the rapid filling from the auricles hinders aortic regurgitation or, *vice versa,* whether regurgitating blood re-

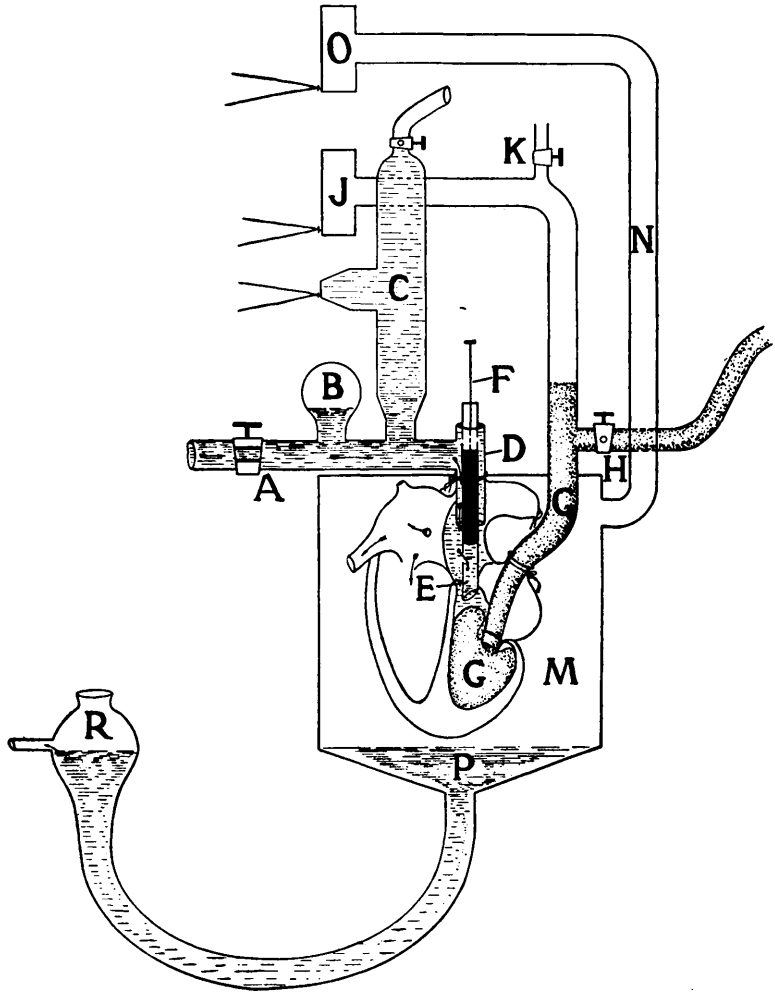

FIG. 1.

Scheme of apparatus. A, arterial system connected to flasks of Tyrode solution under pressure; B, elastic compression chamber; C, optical manometer; D, cannula for perfusing coronaries via ligated aorta; E, trochar cannula allowing regurgitation through side opening when plunger (F) is withdrawn as shown.

G-G, tidal volume inclosed in rubber bag within ventricle and in tube; H, side-cock by which fluid level (G) may be altered; J, segment capsule recording volume of tidal blood; K, side-cock for equalizing pressures.

M, chamber acting as cardiometer for heart and connected by tube (N) to a segment capsule (O) for recording volume changes; P, fluid dripping from right heart after passing through coronary system kept at constant level by compensator (R).

duces the filling from the auricles remains a debatable question. The extent to which a backflow from the aorta is accommodated by replacing inflow from the auricle or by stretching of the ventricle has also not been satisfactorily settled.

Procedure. Our experiments were carried out on cats' hearts arranged to contract isometrically and supplied through the aorta and coronary vessels with nutrient fluid under adjustable pressure. The special arrangements consisted (1) in the introduction of a plunger cannula through the aortic valves for the purpose of producing an aortic insufficiency at will, and (2) in the insertion of a thin rubber balloon into the left ventricle by way of the mitral orifice. In this way, the tidal volume was confined to a closed system and its mixture with the regurgitating fluid was prevented. The changes in aortic pressure, the variations in the tidal volume and, finally, the alterations in the capacity of the heart itself was simultaneously recorded by suitable optical methods. The details of the apparatus should be sufficiently clear from the sketch of Fig. 1. Two technical considerations must, however, be mentioned briefly: Changes in the vigor of contraction due to prolonged lowering of arterial pressure after production of an insufficiency were prevented by limiting our

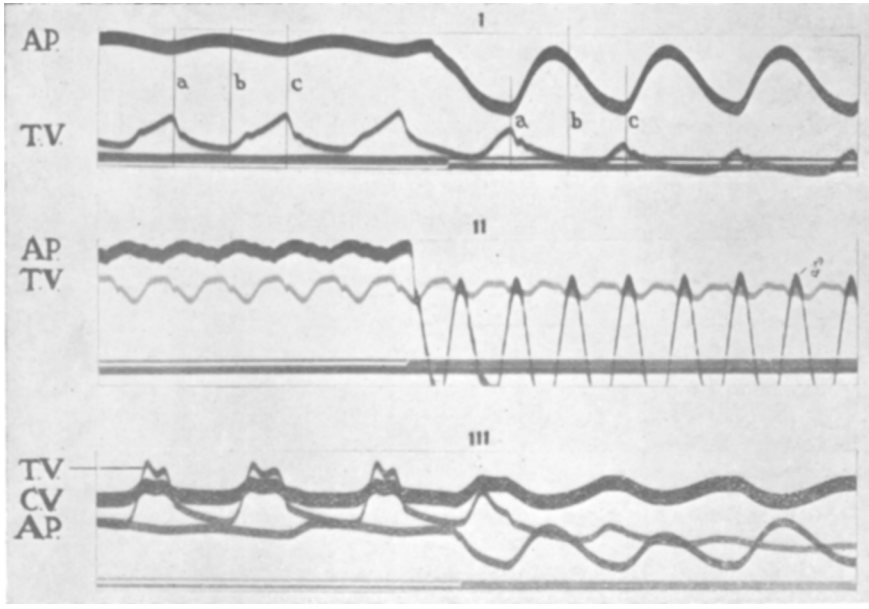

FIG. 2.
Records from 3 experiments showing effects of aortic insufficiency. I, from Exp. 13, X; II, from Exp. 12, VI; III, from Exp. 15, VII.
A.P., arterial pressure; T.V., tidal volume; C.V., cardiometric volume.

observations to 5 or 10 beats after the insidence of a lesion. In order to assure the most favorable conditions for regurgitation—*i. e.,* to recreate the conditions obtaining in the severest clinical instances in which the size of the aortic ring approaches that of the a-v opening—we kept the tubes passing through the aortic and mitral openings identical in size (surface area = 7.1 mm.^{2}).

It should be emphasized that the percentage regurgitation in naturally beating hearts may be considerably less than in our preparation, but that it probably could not be greater.

Results: Many tests were made on 20 different cat hearts. Records from 3 such tests are reproduced in Fig. 2. In each, a portion of the normal records precedes the sudden production of an aortic insufficiency. In the upper set of tracings (I), are shown the changes in aortic pressure (AP), and in tidal volume (TV) entering and leaving the balloon. Previous to the lesion, the aortic pressure variations though recorded with an optical manometer were not very great owing to the fact that the trochar almost entirely filled the aortic ring. The curve of tidal volume changes (TV) presents the gross features of a ventricular volume curve. During systole (ab), the curve at first moves downward rapidly, then, more slowly; during diastolic filling (bc), it first rises with a rapid, then with a slower gradient. When a lesion is produced, the natural diastolic inflow is encroached upon; less fluid enters the balloon and as a result the tidal volume expelled decreases to 60% of its normal volume. These records were taken under conditions highly favorable for regurgitation; the arterial pressure previous to regurgitation was high (mean 95 mm. Hg.) and the venous inflow pressure was very low (10.5 cm. saline). Would the tidal volume be equally reduced if the arterial pressure were lower, or the venous pressure higher? We tested this in many experiments but found no essential difference.

The second set of records (II) in Fig. 2 illustrate the effects obtained when arterial pressures were rather low (mean = 40 mm. Hg.) and the inflow pressure was very high (210 cm. saline). The tidal volume curve (TV) again shows a considerable decrease, owing to an impaired diastolic filling. In this instance diastolic volume and tidal volume were decreased 50%.

Before we may conclude that the difference of 40 or 50% signifies a regurgitation of this magnitude it is necessary to demonstrate that neither the vigor of contraction nor the diastolic size of the whole ventricle altered. The actual regurgitation may actually be larger due to an additional stretching of the ventricles; or it may be

smaller owing to diminished vigor of ventricular contraction. Information on both of these possibilities was obtained in the later experiments by recording, in addition, the volume changes of the entire ventricle (cf. Fig. 1). Such triple records are shown in the third tracing (III) of Fig. 2. The changes in aortic pressure (AP) and in tidal volume (TV) are similar to those shown in the other tracings. The cardiometric curve (CV) of the ventricles indicates that the amplitude of excursion slightly increases; hence, the coincident diminution in the tidal volume cannot be due to less vigorous contractions. On the other hand, we note a rise of the base line equal to about 10% of the normal variation. An additional 10% of regurgitated blood is obviously accommodated by stretching the ventricles. This is approximately the value also found in other tests.

Summary: The maximum aortic regurgitation possible under optimum conditions in the mammalian heart was studied by means of a perfused cat's heart arranged to contract isotonically and in such a manner that the tidal volume entering and leaving the ventricle was kept separate from the regurgitating fluid. The difference between tidal volume before and after a lesion offers an estimate of the volume which regurgitates due to interference with natural filling from the auricles. The increase in the external size of the ventricles indicates the percentage regurgitation made possible by stretching the ventricular myocardium.

Our results show that under optimum conditions the total regurgitation can equal 50-60% of the normal tidal volume in the perfused heart. Of this 40-50% is accommodated by replacing a natural inflow from the auricle and only 10% by additional stretching of the ventricular myocardium.

We believe that the percentage regurgitation could not possibly exceed these values in naturally beating intact hearts; though it may be considerably less.