FURTHER OBSERVATIONS ON EXPERIMENTAL AORTIC INSUFFICIENCY: *II. Cinematographic Studies of Changes in Ventricular Size and in Left Ventricular Discharge*

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FURTHER OBSERVATIONS ON EXPERIMENTAL AORTIC INSUFFICIENCY

II. Cinematographic Studies of Changes in Ventricular Size and in Left Ventricular Discharge

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INTRODUCTION

In 1923, Wiggers (1) ventured the declaration that existing experimental evidence contradicted the view so prevalent among clinicians, viz., that a large proportion of the blood ejected during systole flows back through a leaking aortic valve during diastole. Since that time the question has been re-investigated by other methods both in animals and in man. In a more recent review (2) this work also was critically considered and reasons were given for believing that crucial proof has still not been adduced in favor of the view that the regurgitating volume in aortic insufficiency is very considerable. The radiographic and fluoroscopic researches on heart size in the dog after production of experimental lesions were among the work reviewed. Bazett and Sands made several reports (3, 4 and 5) of their investigations on surviving animals. They found that rupture of an aortic cusp caused an immediate reduction in the area of the x-ray shadow; but they attributed this to a coincident cardiac acceleration. Obviously, no conclusions as to the magnitude of reflux could be drawn from such observations. They found, however, that the heart size gradually returned to normal with the lapse of time, while the cardiac acceleration persisted. This, together with the postmortem findings that the left ventricles had hypertrophied and their cavities had increased in size, inclined these investigators to the belief that considerable regurgitation must have occurred. To us the validity of such deductions appears somewhat questionable. But granting this, the obvious corollary would also appear to follow, viz., that the increase in diastolic size due to

regurgitation could not have been greater than a similar increase occasioned by a change in rate from 133 to 70 beats per minute. According to experimental work on normal hearts this is approximately 10 per cent. Herrmann (6), who made extensive studies upon surviving dogs, also found that rupture of the aortic cusps causes no immediate increase in the areas of x-ray shadows; on the contrary, the area actually decreased in some animals. Enlarged heart shadows were found only in the later stages when hypertrophy was actually present. It is obviously impossible to separate the effects of regurgitation from those due to muscular hypertrophy; Herrmann regarded the latter as the essential cause. Eyster (7), however, obtained earlier evidence of enlargement in similar experiments; the size of the x-ray shadows increased for 3 or 6 days and then gradually returned to normal after another period of about four days.

No attempts have been made to explain these contradictory results, but several reasons suggest themselves to us:

1. The cardiac acceleration may have been of lesser degree in the experiments performed by Eyster. 2. Differences in the method of rendering valves insufficient (e.g., rupture vs. incision) may be accountable for the supervention or absence of secondary circulatory changes. 3. Changes in diastolic volume of the dog's heart may not always be detectable by changes in the outline or area of x-ray shadows, owing to predominant anteroposterior enlargement. Eyster (7) particularly points out that the apex of the left ventricle is most easily stretched. 4. Considerable regurgitation may conceivably occur at the expense of auricular inflow without great additional distention of the ventricle. Experiments recently reported by Wiggers and Green (8) indicate that this mechanism of accommodation obtains under the most favorable conditions that can be created in a perfused heart.

Some of the difficulties encountered in determining and evaluating the changes in cardiac outline by means of x-ray shadows from intact animals can be obviated by recording the changes in size of the exposed heart by the cinematographic method. The following advantages are obvious:

1. The rate of the heart can be controlled artificially, thus eliminating the possible influence of heart rate changes on ventricular size. 2. The outlines of the ventricles can be accurately determined.

3. The size of the ventricular outline can be registered at successive short intervals during systole and diastole rather than during maximum diastolic relaxation only, as in ordinary x-ray exposures.

METHODS

The heart of a dog, anesthetized with sodium barbital, was exposed and supported in a cradle of pericardium made by stitching its cut

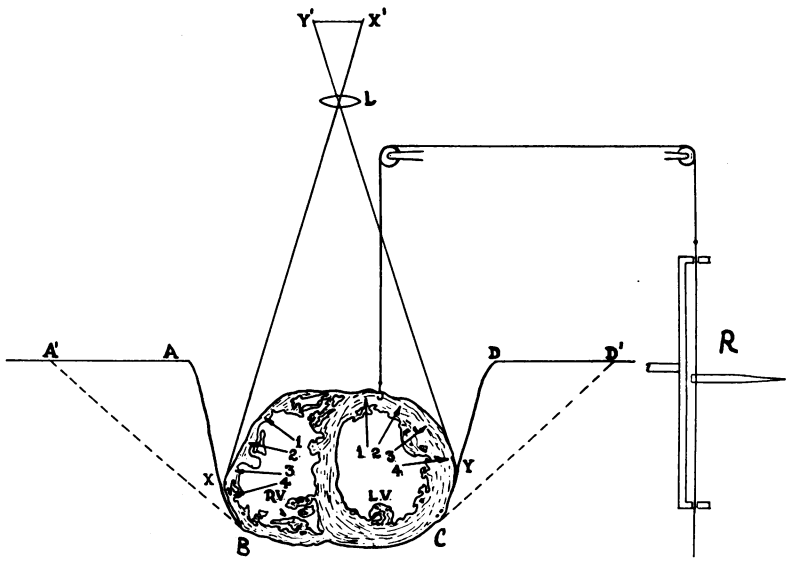

FIG. 1. SCHEMATIC DIAGRAM ILLUSTRATING SUSPENSION OF HEART, REGISTRATION OF ANTEROPOSTERIOR DIAMETERS AND CINEMATOPHOTOGRAPHY

A, *B*, *C*, *D* and A^1, *B*, *C*, D^1, two methods of heart suspension by pericardium; *R*, *V* and *L*, *V*, right and left ventricles respectively; *x*-*y*, plane of frontal projection; *L*, camera lens; x^1-y^1, image on film; *R*, needle recording changes in anteroposterior diameter of left ventricle. Further discussion in text.

edges to the chest wall. Artificial respiration was kept low and regular, so as to prevent the plane of the heart from moving forward more than 3 mm. during inflation. This was actually controlled by registering the anteroposterior movement of the left ventricle on a drum by means of a vertically moving stylus. The principle of this simple expedient is illustrated in figure 1 and actual records of the movements are shown in figure 2.

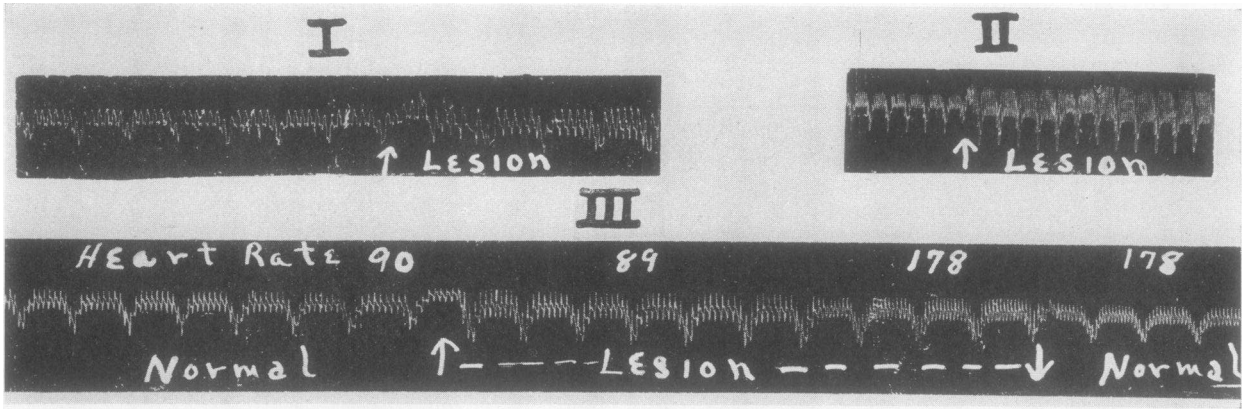

FIG. 2. THREE RECORDS SHOWING EFFECT OF AORTIC INSUFFICIENCY ON ANTEROPOSTERIOR MOVEMENTS OF LEFT VENTRICLE

I, curve showing irregular changes for several beats after a lesion, followed by resumption of normal movements (experiment C-486, II); II, curve showing lesion produced without mechanical disturbance of position (experiment C-472, I); III, curve showing effect of lesion at natural nodal rate of 90 and at artificial rates of 178 (experiment C-487, III, a-d). Further discussion in text.

In these, as in the first experiments on aortic insufficiency reported by one of us (9), an aortic leak was produced by a metal sound with a plunger, the instrument being introduced through the ventricular musculature of the left ventricle and entered into the root of the aorta. When the plunger was withdrawn, communication between the open end of the sound in the aorta and the two side openings of the tube lying within the ventricle was established. The diameter of the tubes varied from 5.5 to 9.3 mm. and created an insufficiency ranging from one-half to seven-eighths the size of the aortic orifice. As a rule areas equal to one-half to three-fourths that of the aortic orifice were employed because they produced no evidence of stenosis when the plunger was in place.

As previously intimated, the rate of the heart was kept constant. To accomplish this and yet maintain a sufficiently slow rate, the S-A node was first clamped in some animals. In a comparatively large percentage of cases, this caused a slow A-V nodal rhythm. By applying somewhat faster rhythmic break shocks to the right auricle, a comparatively slow and constant artificial tempo was maintained.

Synchronous moving pictures and optical aortic pressure tracings were taken before, during and at various intervals after creation of an insufficiency. In other experiments, we studied the changes after discontinuing a lesion by pushing the plunger home.

The method of recording arterial pressure curves has been described in detail by one of us (10). The principle of the photographic arrangements for obtaining moving pictures is obvious from the diagram of figure 1. If a moving picture camera with a suitable lens (L) is placed at a proper distance from the heart, a small picture (x^1–y^1) of the plane x–y is photographed on the film.

Moving pictures were taken on 16 mm. panchromatic film by means of a Bell-Howell camera equipped with a 2.5 cm. F-2.7 Zeiss lens. Sufficient illumination was secured from three 500 watt Mazda lamps equipped with reflectors to permit the use of a diaphragm stop between 2.7 and 4. The moving picture camera was rated to have a speed of 32 exposures per second. Actual tests showed that after the first 3 or 4 pictures and until the fully wound camera had run for 15 seconds the interval of exposure was exactly 0.02 second and the interval of shutter closure was 0.011 second. Individual pictures were thus taken every

0.031 second. The camera was placed at a distance of one foot from the anterior surface of the heart and photographed a picture which approximately filled each frame of the exposed film. The animal board was slightly tilted so that the surface of the heart was parallel with the surface of the film in the camera. After each observation, numbers or data written with chalk on a small black plate were photographed, thus enabling identification of films. Correlation of these pictures with optical pressure pulses was obtained by recording the occasional closing and opening of two signal magnets in the same circuit.

After development and conversion into a positive film, each frame was projected as a stationary picture, the surface area of which was six times that of the actual outline. The successively projected areas were drawn on a roll of semitransparent paper lying flat against a glass pane and so arranged that it could be pulled down from a roll. Space was saved by allowing an overlapping of the drawings of successive heart areas. Subsequently, each area was measured by means of a planimeter and the values expressed in cm.^{2} were plotted on coordinate paper on which the abscissae represented intervals of 0.031 second (cf. figs. 3, 4 and 5).

The physical sources of error introduced by movements of the heart, by changes in respiration, and by the "personal factor" in redrawing and measuring were carefully studied by Strughold (11) in this laboratory and will be reported elsewhere in detail. His studies justified the conclusion that the maximal total error may reach 10 per cent but, by attention to experimental details, this can be greatly reduced. Furthermore, much smaller errors in individual measurements are easily detected by plotting the successive values of surface area measurements for two or more consecutive beats. This may be illustrated by means of the curves of figure 3. A line connecting the successive dots usually gives curves having the general form of volume curves. If an occasional dot falls above or below such a line, as in the case of those labeled *X*, an error is involved. Since we are primarily concerned with the minimal and maximal measurements and their difference, such aberrant values may be disregarded. To guard against the introduction of similar errors in the case of the largest and smallest areas in any cycle, it is important to measure at least two

successive beats. The curves to be acceptable must not show a variation greater than 5 per cent.

After adopting all these technical precautions and also those foreseen by Strughold, we found to our regret that still another source of error remained. Strughold had happily avoided this pitfall by making his chest openings rather large; we, on the contrary, fell into it in some of our experiments by making—as we believed—a slight improvement in the operative technic. We refer to the manner in which the heart is suspended in the pericardium. If, as shown in the cross-section diagram of figure 1, the ventricles rest upon the pericardium stitched to a widely opened thorax, ample room for lateral expansion exists. This is illustrated by the lines A^1 B C D^1 of figure 1. If, however, the chest opening is reduced to A D and the sides of the pericardium A B and C D consequently form a more acute angle with the ventricles, an increase in diastolic ventricular size due to any cause will distribute itself in the directions indicated by arrows 1, 2 and 3, but not in the direction indicated by the arrow 4. Under these circumstances, enlargement of the heart occurs in an anteroposterior direction, but no variation in the lateral diameter x–y and, consequently, in the photographed diameter x^1–y^1 can occur.

The error is more significant when, as in our experiments on aortic insufficiency, the increase in the frontal projection of the two ventricles is effected through an increase in the size of the left ventricle alone. Consequently, we stress the surgical detail essential to successful application of the method; the thoracic opening must be made large and the angle between pericardial and ventricular surfaces must be kept great enough to permit free lateral expansion of the ventricles. The selection of round-chested dogs for experiments of this sort is of great help in accomplishing these aims.

Detailed analysis of representative results

A comprehensive consideration of our results may advantageously be preceded by a detailed discussion of several representative experiments:

The three graphs of figure 3 are introduced partly to show the character of our negative results, partly to demonstrate the effects produced by lung inflation.

Graph A shows the changes in cardiac size previous to an aortic leak. The differences in diastolic size are such as are common when lung inflation comes into play. The first beat was recorded at the end of deflation, the second showing a somewhat greater diastolic size, during beginning of inspiration. They emphasize the importance of selecting beats during comparable respiratory phases, preferably those during the phase of lung deflation.

Graph B represents the 9th and 10th beats after production of an insufficiency. The corresponding changes in the aortic pressure curves reproduced as segments A and B in figure 6 demonstrate clearly that an effective insufficiency had been created.

Graph C represents the outline curves obtained after insufficiency had been maintained for 4 minutes. The corresponding optical pressure curves of segment C in figure 6 show definitely that the vigor of ventricular ejection must have increased.

In spite of the circumstantial evidence of an augmented systolic discharge presented by the pressure pulses (segments B, C, fig. 6),—viz., the larger pulse pressure, coupled with a higher systolic pressure—neither the plots of figure 3 nor actual figures of more extensive measurements give any direct evidence that either the systolic discharge or the diastolic size had increased. Such observations accord with the equally negative results of investigators who have studied the effects of aortic insufficiency in intact animals by x-ray studies. In view of our previous discussion we are not inclined, however, to accept such demonstrations as evidence that the systolic discharge remained unaltered and that no regurgitation occurred. Indeed, graphic curves showing the changes in anteroposterior diameter supply direct evidence to the contrary. As illustrated in figure 2,I, such records unfailingly show an increase in amplitude of the excursions and an elevation of the base line indicating that the diastolic anteroposterior diameter increased and the amplitude of contraction became larger. We must therefore conclude that the changes in cardiac volume mirrored themselves entirely as variations in the anteroposterior diameter of the ventricle. Reference to our experimental notes verifies the suspicion that lateral expansion was prevented by the restricting action of the pericardial support.

The second series of outline curves selected for discussion and repro-

duced in figure 4 were obtained from an animal in which particular care was taken to permit adequate lateral expansion. The chest was widely opened and the pericardial surface made a large acute angle with the surface of the heart. The natural heart rate after clamping the S-A node was 90 per minute. The normal outline curve of the ventricles

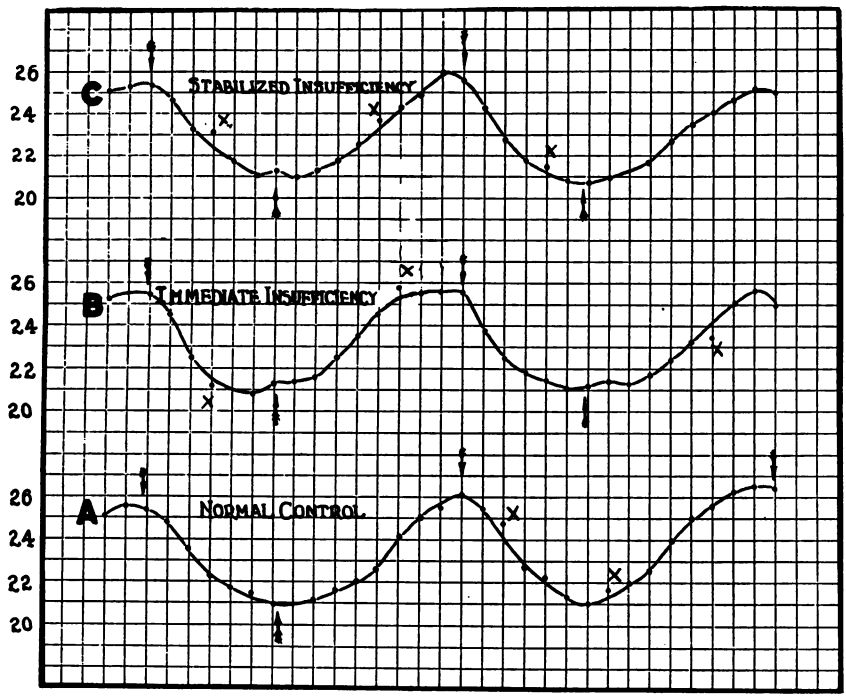

FIG. 3. THREE SETS OF CARDIAC OUTLINE CURVES DURING TWO SUCCESSIVE SYSTOLES AND DIASTOLES

Ordinate figures × 10 = sq. cm. of projected areas; abscissae = 0.031 second; letters *A*, *B*, *C* indicate correspondence with curves of figure 6; arrows indicate beginnings and ends of systole. Discussion in text. (Experiment C-486, II, a-b.)

(D) shows all the characteristics of a normal volume curve, including the long period of diastasis. From moving pictures taken shortly after the production of a leak, the outline curve (E) was plotted. The heart rate remained approximately the same. Curve E shows both an increase in diastolic size and a larger difference between maxi-

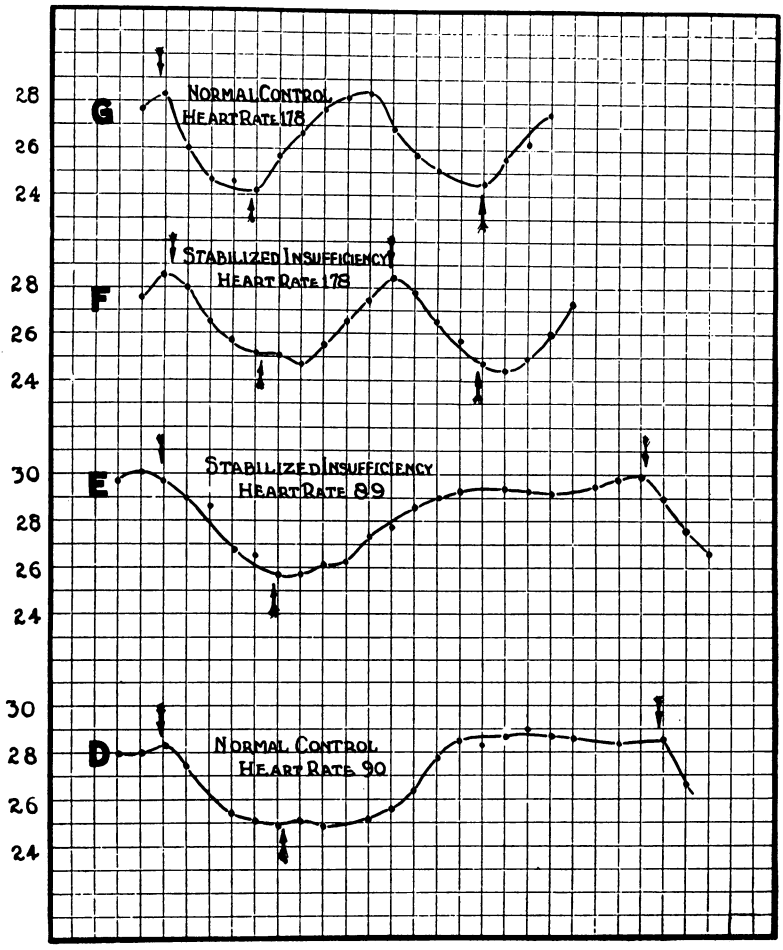

FIG. 4. FOUR GRAPHS SHOWING HEART SIZE CHANGES DURING SYSTOLE AND DIASTOLE WITH NORMAL AND INSUFFICIENT AORTIC VALVES AND AT DIFFERENT HEART RATES

Abscissal and ordinate values as in figure 3; arrows designate beginnings and ends of systole; letters *D*, *E*, *F*, *G* indicate correspondence with curves of figure 6. Discussion in text. (Experiment C- 487, III, a-d.)

mal and minimal size during the heart cycle. The optical pressure curves corresponding to these graphs are reproduced as segments D

and E of figure 6 and the records of anteroposterior changes are reproduced in figure 2, III.

Such curves leave no doubt of the fact that changes in ventricular volume after an aortic insufficiency are correctly reproduced by cinematographic registration when ample room for lateral expansion is provided. The limitations of such a method of support are, however, exemplified by a consideration of the two other graphs of figure 4. Desiring to study the influence of heart rate and duration of diastole on the magnitude of regurgitation, we artificially increased the heart rate to 178, while a constant size of leak was maintained. Finally, while the rapid rate continued, normal valvular conditions were restored by thrusting back the plunger. The changes in the pressure pulses are shown in segments F and G of figure 6 and the alterations in anteroposterior diameters in segment III of figure 2. Graph F in figure 4 shows the effects of increasing the heart rate. We note a decrease in diastolic volume and a moderate diminution in systolic discharge. The contour of the plotted curves are not particularly smooth, however, and the successive beats vary considerably as to minimal sizes. The upper curve showing a curve of successive outlines after restoration of normal valvular action is more regular, but neither the maximal nor minimal sizes differ appreciably from those of the curve below.

This lack of difference occurred in spite of ample room for lateral changes in size, and in spite of the fact that records of anteroposterior diameters (fig. 2,III) showed significant variations. Observations showed, however, that the experimental conditions were unfavorable for accurate filming of the cardiac outline. When the size of the ventricles decreased visibly in consequence of so great an acceleration, the pericardial space available was far too large; the ventricles in consequence flopped about in such a way that changes in focus probably resulted. Under such conditions an accurate comparison of changes in ventricular size is impossible. We enter so fully into these technical details in order to emphasize the reserve and caution that must be exercised in evaluating results.

As a final set of records for detailed analysis, we present in figure 5 the most pronounced effects on ventricular size and output we have succeeded in obtaining. They show in succession (*H*) a normal con-

trol curve, (*I*) a curve during an aortic insufficiency, (*J*) a normal curve after abrogation of the lesion and (*K*) a curve during a second aortic insufficiency. The corresponding optical pressure curves are shown as segments *H*, *I*, *J* and *K* of figure 6. Curves such as these give evidence that creation of a large aortic insufficiency can increase both the diastolic size and the extent of the systolic contraction.

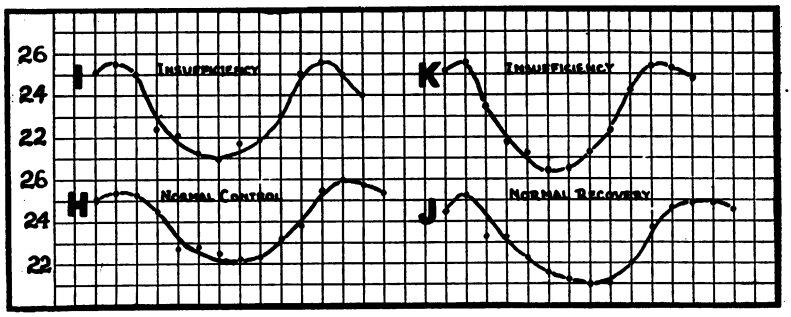

FIG. 5. FOUR GRAPHS SHOWING HEART SIZE CHANGES DURING SYSTOLE AND DIASTOLE WITH NORMAL AND INSUFFICIENT VALVES

Letters *H*, *I*, *J*, *K* indicate correspondence with curves of figure 6. Abscissae and ordinates as in figure 3. Discussion in text. (Experiment C-472, I, a, b, c and II, a.)

The differences in the curves do not, however, permit us to draw direct conclusions as to the magnitudes of the systolic strokes and, by inference, of the degree of regurgitation. In the first place, the increase in size probably occurs entirely in the left ventricle, whereas the curves represent areas in which the right and left ventricles normally have ratios ranging from 10:9 to 10:8. Furthermore, the curves represent changes in the frontal projection of the heart outline rather than true volume curves, regardless of certain resemblances in form. It is not improbable however that the relationships between volume

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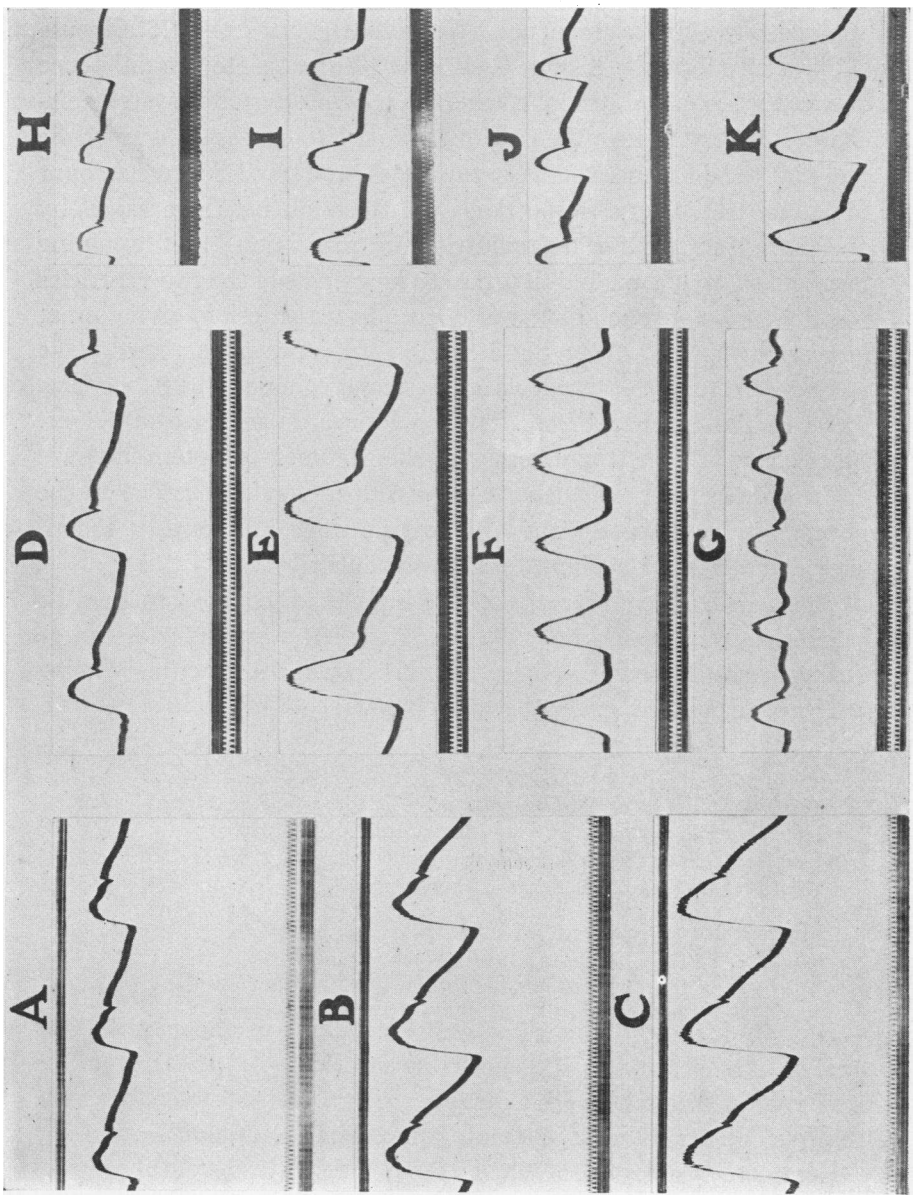
FIG. 6. THREE SERIES OF OPTICAL PRESSURE PULSES FROM AORTA SHOWING DYNAMIC CHANGES PRODUCED BY AORTIC LESIONS

Letters of segments correspond to those on graphs of figures 3, 4 and 5. Distances from base line give relative changes in systolic and diastolic pressure changes correctly. Time 0.02 second. Discussion in text.


A
B
C
D
E
F
G
H
I
J
K

and projected areas established by Bardeen (12) and Skavlem (13) apply also to our measurements. We have attempted, therefore, by use of Skavlem's formula (S.D. = $0.44 \times A^{3/2}$) to translate the difference between systolic and diastolic areas directly into terms of systolic discharge. Doing this, in the case of the first two curves of figure 5, we find that the differences between the largest and smallest areas amount to 34 cm.^{2} and 44 cm.^{2} respectively. After reducing these values by one-sixth, to allow for magnification in projection, and applying Skavlem's formula, we find that the stroke volume of the two ventricles was 5.9 cc. before the lesion and 8.7 cc. after the lesion, giving a net increase of 2.8 cc. Applying a similar procedure in the case of the second set of curves, we calculate the stroke volume to be 8.2 cc. previous to the lesion and 9.6 during a lesion; an increase of 1.4 cc. Since, however, the increases in stroke volumes so determined are entirely due to left-sided effects, these values should be added to the normal stroke volumes of the left ventricle alone, i.e., roughly, to half the calculated stroke volume for the two ventricles.

The percentage increase in left ventricular discharge can then be calculated from these values by taking the left ventricular discharge during an insufficiency as a base. In the case of the experiment being analyzed the following steps become obvious:

	Curves H-I	Curves J-K
	cc.	*cc.*
Stroke volume of two ventricles (normal).....	5.9	8.2
Probable stroke volume of left ventricle (normal).....	3.0	4.1
Increase in stroke volume of left ventricle (insufficiency)	2.8	1.4
Total stroke volume of left ventricle (insufficiency)...	5.8	5.5
Percentile increase in left ventricular discharge.....	48.0	25.0

Comprehensive analysis of results

Having delineated the value and limitations of the method and analyzed the probable and definite information to be derived from the results of individual experiments, we are in a position to consider the results of all experiments in a more comprehensive fashion.

Complete calculations and analyses of changes in the outline curves following aortic insufficiency were made in 23 experiments, selected from a much larger number carried out on 10 different dogs. The heart rate was maintained constant in each set of observations, but

in different experiments ranged from 84 to 170 per minute. In 7 instances the diastolic size was not altered and the systolic discharge either remained unaltered or decreased slightly during existence of an aortic insufficiency.

Four of these negative experiments are satisfactorily explained by improper pericardial suspension; in each, the anteroposterior diameter increased during diastole. The remaining three, in which the minimal systolic area actually diminished during a lesion cannot be definitely accounted for.

The data from the remaining 16 experiments in which the systolic discharge increased are shown in table 1. In most instances, the increase was chiefly or solely due to the increase in maximum diastolic size; minimal systolic size changing but slightly. Experiments 468, V, c and 486 II, however, form two exceptions in which diastolic size diminished and the greater systolic discharge is clearly attributable to a predominant decrease in systolic size. For this reason these results are also questioned. This leaves 14 positive experiments which warrant further deduction. In these, the increases calculated by taking the systolic discharge during insufficiency as a base range from 16 to 31 per cent and from 41 to 58 per cent in two groups of 7 experiments each, the average being 36.4 per cent.

The factors determining the magnitude of the increase could not be satisfactorily analyzed. The increase seemed to bear no definite relation to heart rate, nor to the size of the leak. The heart rates in the two groups listed above were 170–90, 170–84 respectively, i.e., all degrees of increases were found at all heart rates examined. In experiment 478 the percentile increase appeared to be greater at slower rates, but in experiment 472 the reverse seemed to be the case.

DISCUSSION

Several investigators have carefully studied by means of x-ray methods the effects produced by acute aortic lesions on the maximal diastolic size of the ventricles. Our results indicate that the method is not destined to cast much light on the question as to the magnitude of the regurgitation. Even in our most favorable experiments, the increase in area was comparatively small when the heart rate was kept constant, recognizable differences being evident only when the cardiac outlines were magnified at least 6 times.

TABLE 1

Data from 16 experiments showing increased systolic discharge

Experiment	Stage	Heart rate	Maximum diastolic size ×6	Minimum systolic size ×6	Difference in areas ×6	Calculated systolic discharge of two ventricles	Probable systolic discharge of left ventricle	Percentage increase systolic discharge. Left ventricle
		per minute	*cm.²*	*cm.²*	*cm.²*	*cc.*		
468 IIa	Control	162	271	236	35	6.2	3.1	
b	Insufficiency	162	283	235	48	10.0	6.9	+55
468 IVa	Control	162	262	222	40	7.6	3.8	
b	Insufficiency	162	272	228	44	8.7	4.9	+22
468 Va	Control	170	277	247	30	4.9	3.0	
b	Insufficiency immediately	170	293	260	33	5.7	3.8	+21
c	Insufficiency stabilized		262	224	38	7.0	4.9	+39
469 IIa	Normal	152	204	177	27	4.2	2.1	
IIIa	Insufficiency		214	181	33	5.7	3.6	+41
472 Ia	Normal	162	255	221	34	5.9	3.0	
b	Insufficiency		257	213	44	8.7	5.8	+48
c	Normal after	125	252	210	42	8.2	4.1	
IIa	Insufficiency		254	207	47	9.6	5.5	+25
b	Normal after		249	208	41	7.9	4.0	
c	Normal later		249	208	41	7.9	4.0	
474 I	Insufficiency	150	284	220	64	15.2	10.7	+58
	Recovery	150	275	230	45	9.0	4.5	
II	Normal	160	271	221	50	10.6	5.3	
	Insufficiency		282	229	53	11.6	6.3	+19
	Normal after		272	222	50	10.6	5.3	
478 I	Normal	96	338	64	74	19.0	9.5	
	Insufficiency		350	267	83	22.6	13.1	+27
	Normal after		335	261	74	19.0	9.5	
II	Normal	100	336	260	76	19.8	9.9	
	Insufficiency		349	262	87	24.3	14.4	+31
	Normal after		336	256	80	21.4	10.7	
V	Normal	88	361	295	66	16.0	8−	
	Insufficiency		368	285	83	22.6	14.6	+45

TABLE 1—*Concluded*

Experiment	Stage	Heart rate	Maximum diastolic size ×6	Minimum systolic size ×6	Difference in areas ×6	Calculated systolic discharge of two ventricles	Probable systolic discharge of left ventricle	Percentage increase systolic discharge. Left ventricle
		per minute	*cm.²*	*cm.²*	*cm.²*	*cc.*		
VII	Control	84	345	286	59	13.5	6.8	
	Insufficiency		359	280	79	21.4	14.7	+54
	Control after		337	284	53	11.6	5.8	
VIII	Control		350	279	71	18.2	9.1	
	Insufficiency		365	279	86	23.8	14.7	+39
	Control after		348	276	72	18.3	9.1	
486 IIa	Normal	120	262	212	50	10.6	5.3	
b	Insufficiency immediately		258	207	51	10.9	5.6	
c	Insufficiency later		260	207	53	11.6	6.3	+16
487 IIa	Normal	90	288	249	39	7.3	3.6	
b	Insufficiency	89	302	258	44	8.8	5.1	+29

Furthermore, an undoubted demonstration of increase in diastolic size need not necessarily be interpreted as evidence in favor of regurgitation *during each successive diastole.* For example, it can adequately be accounted for by assuming regurgitation *during a single diastole only*, provided the systolic discharge remains the same.

The results of 14 controlled and carefully evaluated experiments have shown that the systolic discharge of the left ventricle beating at a constant rate may be from 16 to 58 per cent or an average of 36.4 per cent greater during aortic insufficiency than when the valves close normally. Such values give by inference a fair estimate of the percentile regurgitation during each diastole. Two reservations may be thought of however, viz.:

1. The percentile regurgitation may be somewhat less than these figures indicate, owing to the fact that compensatory mechanisms are set into operation which in themselves increase the systolic discharge of the left ventricle (e.g., the increased diastolic size and initial tension of the left ventricle).

2. The percentile regurgitation may be somewhat greater than the

percentile increase in discharge because the regurgitating volume may be accommodated both by stretching the ventricles and by reducing the natural inflow from the left auricle (8).

It is obvious that such influences may be concerned during the first few beats after the production of a lesion. But, it is inconceivable that they can play any important rôle after the circulation has become stabilized, a condition which occurs within 5 or 6 beats after production of a lesion. We may therefore conclude that, on the average, about one-third of the systolic volume regurgitates during diastole in aortic insufficiency. The magnitude of the backflow which may be either greater or less appears to be determined by factors other than heart rate or size of leak.

SUMMARY

1. The changes in ventricular areas before and after production of an experimental aortic insufficiency were studied at constant heart rates, for the purpose of evaluating the percentile regurgitation. Successive photographs of the exposed heart, properly suspended in the pericardium, were taken at intervals of 0.031 second by means of a moving picture camera. Each photograph was subsequently projected, its outline drawn and the area measured. The values so obtained were plotted as curves and related to simultaneous records of aortic pressures and anteroposterior movements of the left ventricle. The advantages of the method and the precautions necessary in its employment are analyzed in detail.

2. The curves of outline changes during systole and diastole resemble volume curves in their general characteristics, but do not permit us to draw direct conclusions as to the degree to which the stroke of the left ventricle increases during aortic insufficiency. This is accomplished by applying Skavlem's formula, determining the increase in output and adding this to one-half the output of the two ventricles. By taking the left ventricular discharge, so determined, as a base, the percentile increase in stroke volume is easily calculated.

3. The results show that during aortic insufficiency the diastolic size is larger and the left ventricular discharge is increased from 16 to 58 per cent in different experiments, on an average 36.4 per cent. Factors other than heart rate and size of leak are responsible for the variations.

4. In the discussion it is pointed out (*a*) that changes in heart size without data concerning the stroke volumes of the left ventricle are of no value in determining the percentile regurgitation, but (*b*) that the percentile changes in stroke volume of the left ventricle gives by inference a fair estimate of the percentile regurgitation.

5. The conclusion is reached that approximately one-third of the discharge during systole regurgitates through insufficient valves during diastole.

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