The Mechanics of Ventricular Contraction

A Cinefluorographic Study

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Linear and planimetric measurements have been made on tracings of the right and left ventricular chambers from successive motion picture frames, obtained by cinefluorographic angiocardiography in anesthetized dogs. At the end of systole, varying amounts of residual blood, some 35 per cent of the diastolic volume, remain in the chamber. Filling occurs more rapidly than ventricular ejection and is virtually completed in early diastole. The left ventricular chamber is reduced in size during systole primarily by a reduction in width rather than a shortening of the longitudinal axis. Increased stroke output was accomplished by three mechanisms: (1) increased emptying of the ventricle by progressive shortening of the ventricular chamber; (2) increased filling in early diastole; (3) increased filling in the presystolic period, presumably because the atrial contraction more effectively distended the ventricle. The mechanisms responsible for the systolic residual blood and the retardation of filling which occurs early in diastole are discussed in terms of the anatomy, geometry and functional characteristics of the ventricular myocardium. A comparison is made of the right and left ventricles with reference to their luminal pressure and fiber tension relationships.

The circulation of opacified blood from the superior vena cava to the descending aorta, studied by means of cinemographic fluorography, has been described in a previous report. Examination and projection of the motion picture films indicated that (1) the ventricles fill more rapidly than they empty; (2) the projected areas of the two ventricles appeared to remain relatively constant after the rapid filling phase; (3) no significant change in ventricular size could be consistently observed during the presystolic period when atrial contraction occurs; (4) during systole, peripheral portions of the concavoconvex right ventricular chamber were almost completely evacuated; (5) the capacity of cylindrical left ventricular chamber was reduced during systole by reduction in diameter while longitudinal shortening was variable and of small degree; (6) the thickness of the ventricular walls increased during systole.

This communication deals with evidence concerning these points derived from linear and planimetric measurements from tracings of the right and left ventricular cavities. The changes in the size and configuration of the ventricular cavities, combined with their anatomic and geometric characteristics, has been related to the function and mechanics of right and left ventricular contraction.

METHODS

Through indwelling catheters or needles, Diodrast* (25 cc. of 70 per cent solution) was rapidly injected into the right jugular veins in 20 dogs, positioned in the left lateral reclining position. Motion pictures recorded the sequence of events for periods of 10 to 14 seconds, using 16 and 35 mm. cinefluorographic equipment. Twelve films were selected for detailed study, of which eight had been exposed at 15 frames per second and four at 30 frames per second. Under the same conditions as obtained during the cinefluorographic angiocardiography, films were also taken of a circular brass plate positioned at the level of the dog's heart. By means of a strip film projector, the image of the brass plate on 35 mm. film was focused on a horizontal surface and the projection distance was varied until the circular image from the film coincided with the original brass plate. In this way, the projected image of the brass plate, and similarly the image of the heart, were accurately restored to natural size. For 16 mm. film, the projection distance was excessive, so smaller images from a standard distance were measured and corrected to natural size.

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size by factors computed from the real and projected dimensions of the brass plate.

The projected images of the right and left ventricular cavities and, whenever possible, the external cardiac silhouettes were traced on plain white paper. The walls of the left ventricular cavity could be drawn with confidence, particularly when the right ventricle had become cleared of contrast media. The locations of the mitral valves were not invariably distinguishable, so the basilar limits of the ventricles

were delineated by straight lines connecting the valve roots, and across the root of the aorta. After establishing the limits of the chamber, the following measurements were made: (1) the left ventricular area using a planimeter, (2) the length of the ventricle from the midpoint of the mitral valve to the apex, (3) the width of the ventricle on a line perpendicular to the longitudinal axis at its midpoint (fig. 6B). The area, length and width of the external cardiac silhouette were also measured when possible.

The anterior or free wall of the right ventricle is concave and the septal wall is convex, so the peripheral extensions of the right ventricular chamber are tapered and difficult to distinguish. The shape of the central portion of the chamber could be measured only during cycles in which concentration of the contrast medium was sufficiently low (fig. 1). Because of the complex shape of the right ventricle and the inherent difficulties involved in producing reliable tracings, the following measurements were performed in only 3 selected cases: (1) the area bounded by the tricuspid valve roots,

![Fig. 1. Contraction of the right ventricle filled with a low concentration of Diodrast, photographed at 30 frames per second. Systole begins in B-2, and is complete in D-2. Filling is virtually complete in E-2.](http://circ.ahajournals.org/)
was complete. This difficulty was circumvented as follows: With the animal on artificial respiration, the superior and inferior venae cavae were clamped through a wide thoracotomy. After a few beats, the heart became virtually empty and the remaining inflow and outflow vessels were also quickly clamped. The heart was excised and washed, and the left ventricle was immediately filled with special dental perfection plaster (D.P.) while the heart continued to contract vigorously. The plaster hardened within one minute and fibrillatory contractions of the myocardium persisted while a similar cast was made of the right ventricle. These casts appeared to reproduce accurately the form of the ventricular chambers observed on cinefluorographic records of the same animals.

**RESULTS**

Photographic prints from sequential motion picture frames illustrate the sequence of events during right ventricular contraction. In one experiment, photographed on 16 mm. film at 30 frames per second, the right ventricle was filled with blood having a low concentration of Diodrast during one cycle (fig. 1). The low density band just below the level of the tricuspid valve suggests that the last increment of blood entered the ventricular chamber from the inferior vena cava (fig. 1A-1, 2, 3). As systole began (fig. 1B-1), the Diodrast became evenly dispersed. The free wall of the right ventricle was drawn toward the convex septal wall, and the lateral portions of the chamber, extending toward the center of the cardiac silhouette (C-1), became progressively cleared of opacified blood, leaving a narrow shadow extending from the valves to the apex (D-1). This narrow shadow represents the form of a sagittal section through the central portion of the right ventricular cavity. The peripheral extent of the right ventricular cavity is more clearly revealed during the succeeding diastolic interval (fig. 1E-1, 2, 3, 4). However, as the entire chamber became completely opaque, the central chamber became obscured. Comparing frames C-1 and E-1, the free wall of the right ventricle obviously increased in thickness during systole.

The anatomic basis for partial or complete obliteration of the tapered peripheral extensions of the right ventricle was provided by reconstructions of the heart from the angiocardiographic studies, supplemented by plaster casts of the ventricular chambers (fig. 2A). A cross-sectional view of the ventricles near the base reveals a large right ventricular inflow tract on the right and the pulmonary conus region of the left. A section approximately halfway from base to apex emphasizes the crescentic shape of the right ventricular chamber. The change in right ventricular configuration from diastole to systole is suggested diagrammatically (fig. 2B). Obliteration of the peripheral portions of the chamber is represented as due to an increased bulging of the interventricular septum coupled with contraction of the free wall. The spiral course of the superficial muscles^1,5,6 and the circumferential arrangement of the deep muscle layers as illustrated by the deep bulbous spiral muscle^4 are indicated in figure 2C.

Left ventricular contraction was associated with a marked reduction in the width, and a relatively insignificant reduction in length. This point is strikingly illustrated in one experiment photographed at 30 frames per second (fig. 3). A more accurate appraisal of the changes in ventricular size was obtained by planimetric measurements of the projected image of the left ventricular chamber (fig. 4). The sequential changes in ventricular area, measured from films taken at 15 and 30 frames per second, were plotted on the same time scale (fig. 5). Three cycles from each of two experiments have been superimposed to indicate the degree of reproducibility of the recorded areas (fig. 5A). The initial rate of reduction in area during systole is rapid, slowing as contraction progresses to completion. However, during the first portion of diastole, the increase in size generally occurred at a more rapid rate than the corresponding phase of systole. In many instances, filling was complete within 0.13 second. A slight increase in area was frequently observed during auricular contraction in the presystolic period. In figure 5B, four cycles from one dog (interrupted lines) are compared with three cycles from another animal in which a progressive increase in the ventricular size occurred during successive presystolic periods (solid lines). This animal had received one injection of contrast media 20 minutes previously and died within 5 minutes after this, the second, injection.
Gross examination of the heart revealed only extreme dilation. In only two other films was there a significant increase in area during the presystolic period.

The changes in ventricular dimensions were measured in one animal with bradycardia of unknown origin (fig. 5C). The cycle length was 1.23 seconds (heart rate 48 per minute). During the diastolic interval, the recorded values were quite variable. The systolic reduction in

the total area of both ventricles was only slightly greater than that of the left ventricle alone. This is consistent with the very small reduction in width of the right ventricle (fig. 5D). The initial reduction in the left ventricular area was associated with a diminished diameter, followed closely by a reduction in length, of the ventricular chamber (fig. 5C). As the width began to increase, the ventricular area continued to diminish due to continued shortening of the ventricle. This temporal relation between the lateral and longitudinal dimensions during contractions was not characteristic of all cases.

In one series of four left ventricular contractions, the stroke volume increased progressively (fig. 5E). The diastolic area increased and the systolic area became progressively reduced. The reduction in systolic area was accomplished almost exclusively by an increased degree of shortening, the width of the ventricle remaining remarkably constant at both systolic and diastolic levels. In several experiments, no progressive increase in the

stroke volume was indicated by changes in ventricular area (fig. 5A).

The left ventricular wall was universally thicker during systole than during diastole (fig. 3). This observation led to a consideration of the geometric characteristics of the left ventricular wall. The left ventricle may be considered as a thick walled cylinder with a short conoid segment at the apex (fig. 6A). An increase in wall thickness during systole suggests that the radius of the inner layer of the ventricular wall was reduced more than the radius of the external surface of the ventricle. The differential changes in the radius and circum-

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**Fig. 2.** A. Reconstruction of the ventricle chambers derived from cinefluorographic films supplemented by casts of freshly excised hearts. B. Changes in configuration of the ventricular chambers during systole (schematic). Note apposition of the peripheral portions of the septum and free wall of right ventricle. C. The superficial bulbospiral and sinuspiral muscles pursue a spiral course to the vortex (after Sabotta⁴). The deep bulbospiral bundle is representative of the deep left ventricle muscles in which the fibers tend to describe a circumferential arrangement in a cylindrical form (from Mall⁴ and Robb and Robb⁴).
The size and shape of the left ventricular chamber and the increase in wall thickness during systole indicated on films photographed at 30 frames per second. Systole begins in A-4 and is complete in C-4. The ventricular area is markedly increased during the next diastolic period (D-1, 2, 3, 4).

The thickness of the ventricular muscle was approximately 30 per cent of the ventricular radius (fig. 3). Therefore, \( r_{D'} = 1.3 \times r_D \) and \( V_{D'} = 1.69 \times V_D \). However, if the wall thickness remained the same during systole, the radius of the outer

* The meaning of these and following symbols is shown in figure 6.

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**FIG. 4.** An enlargement of a single frame of a motion picture which illustrates the configuration of the left ventricle and the external borders of the ventricular musculature.
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The change in volume of the inner cylinder (ventricular chamber) would be 0.64 $V_D$ and the outer cylinder (chamber plus ventricular wall) mass of ventricular muscle is unchanged and that the quantity of blood expressed from the coronary circulation is negligible, the change in total volume of the external and internal cylindrical segments would be identical: $(V_D - V_S) = V_D - V_S$. Under these conditions

![Fig. 5](image-url)

**Fig. 5.** A. The change in left ventricular area during three successive systoles from 2 different dogs. Note the rapid filling immediately after each systole. B. Progressive increase in diastolic filling in the presystolic period in a dog which died five minutes later (solid lines) is compared with four systoles from another experiment (interrupted lines) with a more typical pattern. C. The changes in ventricular dimensions in a dog with bradycardia (heart rate 48 per minute). The measurements were made as indicated in figure 6. D. Changes in the dimensions of the central portion of the right ventricular chamber. E. The changes in left ventricular dimensions during progressive increase in stroke volume (same experiment as D). Note that the increased filling and emptying are accomplished by changes in length, not width.

would be 0.88 $V_D$. Thus the ventricular wall would be reduced in volume by an amount equal to 24 per cent of the initial diastolic volume of the ventricular chamber (0.88 $V_D - 0.64 V_D$). On the contrary, the radius of the external shell is reduced by a smaller amount than the internal cylinder so the ventricular wall thickness increases. Assuming that the $V_S = 1.05 V_D$ and $r_S = 1.03 r_D$. The radius of the external cylinder would diminish 21 per cent while that of the inner cylinder would be reduced 40 per cent during ventricular systole. This explanation clarifies the observed increase in wall thickness, and it also indicates that the amount of shortening of myocardial fibers must vary in different layers of circumferentially
arranged ventricular muscle fibers. Most of the left ventricular wall in the basilar regions is composed of the deep bulbospiral and deep sinuspiral muscle layers\(^4\) in which the fibers tend to be arranged around the circumference of the cylindrical segment (fig. 2C). Since the circumference of a circle is directly proportional to the radius, the percentage shortening of the inner layer of fibers must be greater than that of the outer layers \(C_D - C_A > C_{D'} - C_{A'}\). As a matter of fact, using the values obtained above, the fibers in the outer layer shorten by approximately 20 per cent while those in the inner layer shorten 40 per cent (see below).

If the conoid segment at the apex of the left ventricle were considered as a cone with a radius \(r_D\) and a length equal to one-half the length of the cylindrical portion of the left ventricle (fig. 6A), the apical cone would contain an additional volume equal to only 17 per cent of the diastolic volume of the remainder of the ventricle. If, during systole, the radius of the cone were reduced by the same amount as the cylindrical portion of the ventricle (for example, to 0.6 \(r_D\)) the volume of the cone would be reduced to 0.06 \(V_D\), if no ventricular shortening occurred.

![Figure 6](image)

Fig. 6. A. The configuration of the left ventricle simplified for geometrical analysis (see text). B. The relation of the area, computed as the product of length and width, to the planimetrically measured area in each tracing indicates the reproducibility of the measurements. The sketches indicate the method of measuring length and width of the two ventricles.

Since the data derived from linear and planimetric measurements of the projected left ventricular chambers have been the principle source of information in this investigation, the reliability and reproducibility of the determinations were checked by plotting ventricular areas obtained in two different ways from 270 tracings. The length, width and area of the right and left ventricles were measured on ventricular tracings from 4 different animals in accordance with the methods indicated in the sample...
tracings (fig. 6B). The product of ventricular length and width was plotted against the planimetrically measured area. If the two areas were identical in every case, all points would lie on a line with a 45 degree slope (coefficient of regression = 1.0). Despite the fact that a product of length times width described a rectangular area, the computed area was closely related to the measured area over a wide spread of values obtained by including data from systolic and diastolic intervals in dogs of varying size. As might be expected, the computed areas of the left ventricle were almost invariably greater than the measured areas. Similar measurements of right ventricular areas were plotted on the same graph (fig. 6B). The points fell with remarkable regularity along the line representing a regression coefficient of 1.0. It should be pointed out that a different relationship for both ventricles would have been obtained had the width been measured at any other level. The close relation obtained for the right ventricle may be attributed, in part, to the fact that the width at the midpoint of the ventricle was an average of the widths above and below this point.

**Discussion**

In the course of this investigation, three mechanisms for increasing stroke volume were encountered: (1) the diastolic volume may be augmented by increased filling in early diastole (fig. 5E); (2) more complete systolic emptying may occur with a corresponding reduction in the quantity of residual blood (fig. 5E); (3) the effectiveness of atrial contraction may be augmented by unknown factors to produce progressively increased diastolic filling in the presystolic interval (fig. 5B). The contribution by atrial systole was observed in 3 animals, one of which died spontaneously shortly after the angiocardiography. It should be noted that no progressive change in diastolic and systolic area was demonstrated in some of the experiments (fig. 5A and 5B, interrupted lines).

During systole, the projected area of the ventricular chambers diminished rapidly at first, tending to empty at a slower rate as the contraction neared completion. In every case, considerable quantities of blood remained in the ventricle at the end of systole. Early in diastole, the ventricular area increased, even more rapidly than it had been reduced during systole, and tended to reach a plateau which was sustained until the succeeding systole. However, neither the diastolic size nor the systolic size was fixed; progressive changes occurred at both extremes in successive cycles in certain animals. It seems essential that consideration be given to mechanisms which might determine the diastolic and systolic size of the ventricle.

Lundin7 obtained length-tension curves from small bundles of myocardium isolated from frog hearts and reached the following conclusions: (1) The high viscosity of cardiac muscle facilitates filling of the heart because it permits the diastolic pressure to fall to zero even when the ventricle is dilated. (2) Further, the viscous properties of the myocardium oppose a rapid stretch with a marked increase in tension. Judging by the characteristics of isolated myocardial bundles, it would appear that both initial rapid inflow and abrupt termination of filling may be related to viscosity, a property which is more prominent in cardiac than in skeletal muscle. In contrast with these findings on isolated myocardial strips, Ramsey4 summarized evidence from the intact frog heart as follows: “Normally, the greatest reversible dilation of the heart is reached when the cardiac fibers are at their rest length and exerting maximum tension or return.” The definition of resting length in a ventricle composed of laminated strips of myocardial tissue, arranged circumferentially in a cylinder appears somewhat obscure, since any change in ventricular volume is accompanied by a greater change in length of the inner layers than of the outer layers of muscle. However, if a “resting” size of the ventricle exists, beyond which further distension involves a progressive increase in effective filling pressure, the diastolic plateau could be at least partially explained.

Just as the ventricles are restrained from distending as much as they are capable, ventricular systole fails to empty the chamber completely. Due to the space occupied by the papillary muscles (fig. 2) this residual volume is less than is indicated by the projected areas
of the ventricles. At least three mechanisms may tend to limit the degree of ventricular emptying during systole. Cardiac muscle builds up tension very rapidly and effectively under isometric conditions, but if the muscle is allowed to shorten during contraction, the tension falls off rather sharply. Ejection of blood from the heart requires shortening of the fibers. Thus, the rate of ejection in early systole should be greater than late in systole (fig. 5). Secondly, the pressure in a viscus is related to the tension in the muscular components of the wall as follows: 

\[ P = \frac{2T}{R} \]

where \( P \) is luminal pressure, \( T \) is muscular tension and \( R \) is the radius of the circle described by the muscle fibers. It is obvious that the muscles of the inner shell would contribute more to the luminal pressure than the outer layers, if all fibers exerted the same tension. However, evidence has been presented that, when the volume of the ventricle is reduced to 35 per cent of the diastolic volume, the circumference of the fibers comprising the inner wall of the left ventricle may be reduced by 40 per cent, while the outer fibers shorten about 21 per cent. It is possible that when contraction begins, the inner fibers are most effective in raising intraluminal pressure, but, as shortening progresses, the burden falls progressively more on the outer shells. Indeed, it is conceivable that near the end of contraction, the inner fibers contribute little or nothing and the outer fibers actually expend useless energy producing deformation or wrinkling of the inner laminations. Finally, more than 60 per cent of the blood in the left ventricle is ejected (considering the displacement of papillary muscles) when the radius is reduced only by 40 per cent of the diastolic circumference and further contraction becomes progressively less effective with respect to the volume expelled per unit shortening of the muscle fibers.

The cylindrical shape of the left ventricle is consistent with its function as a high pressure pump. When high pressures develop in the lumen of any hollow viscus, the shape of that viscus tends to approach a cylinder or a sphere. A sphere contains a greater volume per unit surface area than any other geometric configuration. Evidence was presented (fig. 5E) that the change in diameter of the cylindrical segment of the left ventricle was relatively constant and further change in area of the left ventricular silhouette was accomplished by increased shortening. It is tempting to speculate that reduction in width was due primarily to the action of the deep muscle layers with small circumference while the shortening was accomplished by the spirally arranged superficial muscles.

On the contrary, the right ventricle is bounded by a free wall which represents a segment of a sphere with a very large radius. The bulging of the septum into the ventricular chamber produces a configuration which is the antithesis of a high pressure region. With respect to the formula \( P = \frac{2T}{R} \), the tension in the fibers of the right ventricular wall would be extremely high in order to produce pressures corresponding to those in the left ventricle. On the other hand, this architecture is particularly suited to large changes in capacity in the presence of low inflow and outflow pressures. This may explain the following clinical observations: (1) The lower pressure required for diastolic filling of the right ventricle may account for the fact that, normally, the right atrial pressure is lower than the left. (2) The right ventricle is vulnerable to increased outflow pressure. (3) In the fetal heart, where the outflow pressures of the two ventricles are approximately equal, the shape of the right ventricular chamber more closely resembles that of the left ventricle. (4) In the adult, compensation to increased outflow pressures involves distension of the right ventricular chamber to a more spherical configuration.

**Conclusions**

1. The initial filling of the ventricles occurs more rapidly than the ejection during systole. In most cases, filling was completed early in diastole and the ventricular size (area) remained remarkably constant until the succeeding systole. In general, atrial contraction produced little change in the diastolic size of the ventricular cavities.

2. The right ventricular area is bounded by
the concave surface of the free wall and the convex surface of the septum, producing a semilunar configuration in cross section. During contraction, the blood in lateral folds of the ventricle was evacuated, apparently leaving a potential space between the septum and the free wall, in certain areas.

3. The left ventricle most closely resembles a cylinder extended at one end into a conoid segment. Left ventricular systole involves primarily a reduction in the diameter, supplemented by a relatively small reduction in length of the chamber. Judging from the laminated arrangement of myocardial sheets, the reduction in width may be due to the deep muscle components while the shortening is produced by the superficial layers.

4. The wall thickness of both ventricles increases during systole, an observation which is explained on the basis that for any particular change in volume, the circumference (and radius) of the inner layer of myocardial fibers is affected more than that of the outer shell. The fact that the ventricular size remains relatively constant during a major portion of the diastolic period has been discussed in terms of the "viscosity" of the cardiac fibers as described by Lundin.7

5. The presence of residual blood in the ventricles at the end of systole has been discussed in terms of (1) the rapid loss in tension of myocardial fibers during shortening, (2) the changes in the fiber length in the inner and outer layers of muscle and (3) the energy expenditure required to completely evacuate the chamber.

6. The area of the left ventricle, computed as the product of length times width, bears a close relationship to the area measured planimetrically.

7. The right ventricle is architecturally suited to great lability in stroke volume if the outflow pressure remains low. It is correspondingly vulnerable to increased outflow pressures. The left ventricle is adapted for ejection against a high outflow pressure. The relation of the different characteristics of the two ventricles was discussed in terms of common clinical observations.

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