The Functional Anatomy of Ventricular Contraction

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Changes in dimensions of the ventricular chambers and the displacement of specific points on the walls of the heart have been studied by the cinefluorographic technic. The contributions of the various myocardial layers to ventricular ejection have been considered in terms of the anatomic architecture of the heart. The mechanical effectiveness of myocardial contraction is closely related to the degree of diastolic distension and the magnitude of the stroke volume. During contraction, the various layers must exert tension on the connections between them. Energy expended in this manner does not contribute to ventricular ejection but could theoretically promote rapid filling during the early diastolic period.

Previous investigations have indicated that the two ventricles have different functional and anatomic characteristics. Ventricular contraction cannot be visualized as though the two ventricles were a single unit. The mechanical effectiveness of the various layers of myocardial fibers depends in part upon their anatomic location and orientation within the ventricular walls. Information of this sort cannot be obtained from cardiometer records which indicate the combined effects of the musculature of the two ventricles. Further, Haycraft has clearly shown that movements of the ventricular walls must be studied with the thorax intact.

This report deals with an analysis of the factors which influence the mechanical effectiveness of myocardial contraction in terms of the anatomic architecture of the heart.

Methods

While cinefluorographic angiocardiography has proved valuable in studying absolute and relative changes in the dimensions of hearts during the cardiac cycle, it had serious limitations for the proposed investigation. For example, the movements of specific points on the ventricular wall could not be observed. The rapid injection of radio-opaque substances produced abnormal conditions within the circulation. The cardiac chambers could be observed only for very brief intervals during which they were outlined by the contrast media. Reliable measurements of the right ventricular cavity were rarely obtained because the septal border of the chamber was rarely visible.

Another method of measuring changes in size and position of the heart has therefore been developed. Stainless steel wire (3 mm. in diameter) was cut into segments approximately 0.5 to 1.0 cm. long. A shallow groove was filed around the midpoint of each pin. Pairs of these pins were connected by short lengths of very fine rubber fibers covered by windings of linen thread. The metal markers were lined up within the lumen of a stainless steel tube fitted with a stylus by which the pins could be individually ejected from the tube. Under aseptic conditions, thoracotomy was performed on anesthetized dogs; the pericardium was incised and the small trochar containing the pins was thrust transversely through the ventricular walls. By advancing the stylus, the leading pin was ejected and flipped into a position at right angles to the long axis of the trochar, which was then withdrawn back through the wall and the second pin released. In this way a pair of pins, connected by resilient elastic, was installed on opposite sides of the ventricular wall or septum (fig. 1A). As many as five pairs of pins have been installed at various sites during a single operation (fig. 1B). The pericardium was repaired and the lungs were reinflated as the thorax was closed in layers. Within three weeks, the pins had become completely covered by a thin layer of tough white connective tissue. The orientation of the pins within the heart was checked by several methods including cinefluorography, angiocardiography, fluoroscopy and postmortem examination. Serial electrocardiograms over several weeks revealed little alteration in the complexes and the recovery of the animals was apparently complete. Several animals,
FIG. 1. Sagittal section through the ventricles of a dog revealing the position of three pairs of metal pins implanted across the right ventricular wall, interventricular septum and left ventricular wall. These three pairs of pins were inserted along the track of a single trochar thrust through the heart from side to side. The atrial cannula (A.C.) was used in experiments not included in this report.

B. A teleroentgenogram indicating the location of the metal markers in the heart. Pins 1 and 2 were on the endocardial surface of the free wall of the right ventricle. Pins 5 and 6 were on the endocardial surface of the left ventricular wall. (The corresponding four pins on the epicardial surface were made of radiolucent plastic.) Pins 3 and 4 were on opposite sides of the interventricular septum. Ring 8 was sutured to the left ventricle at the left interventricular groove. Ring 7 was fastened to the left ventricle at the right interventricular groove. The arrows indicate the direction of displacement of these metal markers as revealed by cinefluorographic studies.

FIG. 2. The metal markers became covered with a thin investment of fibrous connective tissue. The pin on the left was on the left ventricular aspect of the interventricular septum. The pin on the free wall of the left ventricle nestled between trabeculae carneae. The heart was from a dog sacrificed 309 days after the pins were implanted.

allowed to survive for four to six months, remained healthy and active. One animal was sacrificed after 10 months (309 days) and the heart was found to be normal in size and appearance. The thin, transparent coat of connective tissue investing the metal pins is clearly visible (fig. 2).

Postmortem examination of these animals revealed dense pleural and pericardial adhesions along the lines of incision and in the immediate vicinity of the epicardial pins. In other regions, the pleural and pericardial surfaces were either free or lightly bound by adhesions. Microscopic examination of the ventricular walls in three animals indicated that the metal markers and elastic threads were enclosed in a thin layer of fibrous connective tissue but contiguous areas of myocardium had been unaffected by the procedure.

Results

Cinefluorographic films were obtained on 15 unanesthetized dogs after full recovery from the surgical operation, without anesthesia or restraint. The movements of the markers were clearly visualized during projection of the films.
In most cases the distances between markers were also measured on successive frames, projected at eight times the actual size of the heart. The distances between pins were repeatedly measured; test-retest values for the actual pin separation usually varied less than 1 mm. Frontal vectorcardiograms appeared in the corner of each frame of the cinefluorographic films to correlate excitation with ventricular contraction. From a series of exploratory experiments, examples have been chosen to illustrate some of the factors which influence ventricular contraction.

**Changes in the Length and Width of the Ventricular Chambers**

Tracings have been made of the right and left ventricular chambers on successive frames of cinefluorographic films exposed at 15 and 30 frames per second during angiography on 10 dogs, using methods which have been previously described. On each frame the length of the left ventricular chamber was plotted against the width of that chamber. Within the shaded areas on the graph (fig. 3A) appear the data from 4 of the 10 animals. Values from the remainder of the animals had similar distribution. The arrows indicate the general trend of the alterations in length and width during cardiac cycles. The absolute change in length exceeded the alteration in width but the relative change in width was significantly greater. The volume of blood ejected by a unit change in width is greater than a corresponding change in chamber length. If the left ventricular chamber be compared to a cylinder, the change in volume varies as the square of the radius and directly with the length according to the formula $V \propto R^2L$.

In all cases, plotting the changes in width of the left ventricular chamber against the projected area of its silhouette revealed less scatter of the points than the relation between length and width. The values from the animal appearing at the top of figure 3A are presented by guest on April 8, 2017 http://circres.ahajournals.org/ Downloaded from
in figure 3B. If the projected area is related to the volume of the left ventricular chamber, the width at its midpoint is also closely related to the volume.

Judging from the relation between length and width, right ventricular contraction is apparently accomplished primarily by a shortening of the chamber along its longitudinal axis (fig. 3C), the width at the midpoint varying only slightly.

 Movements of Specific Points on the Ventricular Walls

Observations were made on the movement of metal markers implanted at various points on the internal and external surfaces of the ventricles in 15 dogs. Their movements were analyzed by repeated projection of cinefluorographic films and by serial measurements on successive frames. Since the films represent a planar projection of a three-dimensional structure, only pins lying on the borders were selected for this portion of the study. The silhouette of the heart was traced at the diastolic size and the direction and magnitude of the pin motion during systole was indicated on the tracing. There was some variability in the magnitude of the movement from beat to beat, particularly during respiratory activity, but the direction of the pin displacement was very consistent in any particular animal. The results of this study have been consolidated on a schematic diagram of the cardiac silhouette with arrows indicating the amount and direction of systolic movement of the metal pins in the various animals (fig. 4A). The movement of markers located in the free walls of the ventricles may be described as a result of displacement in two directions: (a) toward the apex (parallel with the wall), and (b) toward the interventricular septum. In the right ventricle, longitudinal shortening of the free wall was the predominant movement with little reduction in right ventricular width. Thus, metal markers moved primarily toward the right ventricular apex. In contrast the markers on the free wall of the left ventricle moved obliquely toward the apex and toward the interventricular septum indicating simultaneous reduction in width and length of the chamber. If the apex of the heart remains relatively fixed in position and the free walls shorten, the longitudinal motion of markers at the base of the heart should be greater than at the apex and in general this was true with two conspicuous exceptions. In one animal, a marker in the outflow tract of the right ventricle moved almost directly toward the septum (fig. 4A, dotted arrow). In another animal, a marker in the midportion
of the left ventricular wall moved directly toward the septum (fig. 4A, dotted arrow).

One pair of pins near the vortex of the left ventricle rotated in opposite directions (fig. 4A). In all cases, the pins on the endocardial surface were displaced further than those of the epicardial surface. This was particularly true of pins near the base of the right ventricle. If points on opposite sides of a ventricular wall move different amounts, and in different directions, the connections between the myocardial layers must be put under tension (vide infra). The distance between markers on opposite sides of the ventricular chamber remained large at the end of systole, indicating that large volumes of blood remained within the ventricular cavities at the end of systole.

**Rotation of the Heart**

Movements of the metal markers on the borders of the cardiac silhouette gave no indication of a rotatory motion of the ventricles during contraction. However, rotation of the heart would produce displacement of the pins on the cardiac borders primarily at right angles to the plane of projection. Special markers were implanted in four animals to determine the extent of rotatory movements of the heart. In one dog a segment of stainless steel wire was embedded transversely in the interventricular septum. After recovery, cinefluorographic films were obtained for study. The animal was positioned so that the wire was perpendicular to the fluorescent screen. It appeared as a small circular opacity during diastole. Rotation of the ventricular chambers around any axis would cause the shadow of the wire to lengthen in one direction or another. Slight rotation around the longitudinal axis of the heart was noted but could not have exceeded 10 degrees of angular rotation.

Small metal rings were fastened externally on opposite sides of the interventricular groove. Rotation of the heart along its longitudinal axis must also rotate the septum. Such rotation of the heart should increase the distance between the metal markers. The results of such an experiment are indicated in figure 4B. During systole, the metal rings initially moved slightly apart and then toward each other during the same systolic interval. The initial movement was counterclockwise as viewed from the apex. In no case was there any indication of rotation which exceeded that indicated in figure 4B.

**Movement of the Interventricular Septum**

In a transverse section through the ventricular chambers, the left ventricular cavity is roughly circular, being bounded by the free wall of the left ventricle and the thick interventricular septum (fig. 6). The right ventricular cavity is crescent shaped due to the convexity of the interventricular septum bulging into the right ventricular cavity. If the interventricular septum becomes more rounded during systole, as the left ventricular cavity becomes reduced in size it might bulge further into the right ventricular cavity contributing to right ventricular ejection. There was no evidence of rightward displacement of the pins installed across the center of the interventricular septum in either the routine experiments (fig. 4A) or those particularly designed to elicit such movements (fig. 4B). The only consistent change in the interventricular septum during systole was a reduction in length along its longitudinal axis.

**Movements of Myocardial Bundles in Different Layers**

Since pins attached to the endocardial surface moved greater distances than corresponding markers on the epicardial surface, our attention was directed toward the fact that the different myocardial bundles are oriented in different directions. Since muscle fibers shorten during contraction, the tension developed must be along the longitudinal axis of the fibers. Four sets of markers were installed within the free wall of the left ventricle. Each set consisted of short segments of thin wire of distinctive shape connected by an elastic fiber. At the midpoint of the elastic was a small metallic sphere. The metal sphere assumed a position within the deep constrictor muscles. Several days after their insertion, fluoroscopy and cinefluorography were employed to detect differential movements of these markers. The animal was positioned so that at least two of these sets
were on a line perpendicular to the fluorescent screen. Although the movements of the markers were decidedly limited in this view, the three markers appeared to be displaced in three different directions during systole. The contraction of myocardial bundles in different directions can easily be observed on cut surfaces of a ventricular block removed during fibrillation.

Muscular Architecture of the Ventricles

The changes in configuration of the ventricular chambers and the movements of specific points of the ventricular walls must be considered in terms of the orientation of muscle fibers within the walls. The superficial myocardial fibers spiral from the base of the heart in a clockwise direction (viewed from the apex) toward the apex. At first glance this arrangement should produce rotation of the chambers during systole. However, if sections are removed from the full thickness of the ventricular wall, the trabeculae carneae on the endocardial surface are seen spiraling in the opposite direction (counterclockwise) back toward the base of the heart.

Between the two thin layers which form the epicardial and endocardial investment of the ventricular wall are layers of fibers which are oriented more nearly around the circumference of the two chambers. These intricately interwoven cuffs of muscle, encircling the base of the heart, represent the deep bulbospiral and deep sinospiral muscles. They are illustrated in figure 5B as though they had been lifted out from between the two spiral layers. This schematic drawing illustrates the orientation of the myocardial fibers in the three layers of muscle, and the continuance of the trabeculae carneae and papillary muscles with fibers on the external surface of the heart. If the superficial sheath of muscle could be overt, the myocardial fibers on the internal and external surface of the heart would all spiral in the same direction with a sharp twist occurring at the vertex. Functionally there are but two muscles of the heart: (1) the spiral muscle which forms the internal and external investment, and (2) the deep constrictor muscles. Simultaneous shortening of the internal and external layers of the spiral muscle would not produce rotation since the oblique tensions exerted by each layer would be mutually counteracted. The result of their combined action would act to shorten the long axis of the chambers.

On the other hand, the deep constrictors act to reduce the circumference (and diameter) of the ventricular chambers. Since the deep constrictor muscles are much more powerful in the left ventricle than in the right, the left ventricle tends to be compressed in width to a greater extent than the right.

The spiral muscles and deep constrictor muscles operate under widely divergent mechanical conditions. Although the heart has an asymmetric geometric form, the deep constrictor...
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muscles most closely describe a cylinder while the superficial spiral muscles tend to enclose a sphere. To the extent that this analogy is correct, corresponding degrees of myocardial shortening by the two layers of fibers will tend to eject different volumes of blood (fig. 6). A unit change in length by fibers enclosing a sphere produces a much greater volume change than the same percentage shortening of fibers arranged around the circumference of a cylinder. This is an anomalous situation, because the combined effect of the two groups of fibers is shortening probably produce tension on the muscular and connective tissue connections between the different layers. The interfascicular tension would represent potential energy, wasted so far as ventricular ejection is concerned, and stored during the systolic interval. It would be released during the early diastolic interval when ventricular myocardium relaxed. This effect is superimposed upon the differences in degree of shortening in the inner and outer layers of the deep constrictor muscles previously reported. The release of this potential energy may act to restore the ventricular chambers toward their diastolic dimensions and facilitate diastolic filling (vide infra).

The significance of the large residual volumes at the end of systole may be assessed in terms of the factors illustrated in figure 6. Until recently ventricular contraction has been thought to evacuate the ventricular chambers almost completely (for example, the change from volume 1 to volume 2 and not the extreme systolic emptying represented by volume 3). The changes in volume produced by unit changes in length by fibers encircling a sphere as contrasted with fibers lying along the circumference of a cylinder (see text).

**Fig. 6.** The configuration of the right and left ventricular chambers as they would appear in transverse section. Evidence is presented that the normal stroke volume represents a change from volume 1 to volume 2 and not the extreme systolic emptying represented by volume 3. The changes in volume produced by unit changes in length by fibers encircling a sphere as contrasted with fibers lying along the circumference of a cylinder (see text).
In other words, ventricular systole produces a change similar to that between volume 1 and volume 2 (fig. 6). In this case a smaller degree of fiber shortening is required to eject a particular stroke volume. The heart frequently responds to an increased load by dilation and certain advantages are gained by the increased fiber length. Among these are: (1) the increased energy release described by Starling's law of the heart, (2) reduction in the degree of shortening required to eject a particular stroke volume, (3) reduction in the loss of tension due to rapid shortening (the viscosity effect) described by Lundin,9 (4) reduction in the energy loss due to interfascicular tension. On the other hand, greater tension must be developed by the contractile units as the myocardial fibers describe circles of greater radius in accordance with the Laplace formula: (P = \frac{T}{R} for cylinders, P = \frac{2T}{R} for spheres, where P is the effective internal pressure, T is the tension of the fibers and R is the radius of the circle described by the fibers.10) The optimum diastolic size probably varies in terms of nature of the load on the heart at any particular time, due to the interaction of the various factors which influence the mechanical effectiveness of myocardial contraction.

**Discussion**

By a new technic, it was possible to obtain a more complete picture of the movements of specific points on the ventricular walls than had previously been possible with an intact thorax. The apex of the heart remained remarkably stationary for reasons which are not at all clear. The left ventricle ejects blood primarily by diminishing the diameter of the chamber with some shortening of its long axis. In contrast the primary function of the free wall of the right ventricle is to move the A-V valve ring toward the apex.

One reason for this study was to seek an explanation for the observation that dogs survive severe damage to the right ventricular wall with little or no change in the circulation.11-13 Bulging of the interventricular septum into the right ventricular chamber has been given as an explanation. The present investigation does not support this view. The myocardial fibers which make up the ventricular walls are not confined to a single ventricle. The fibers, damaged by cautery, extend for long distances beyond the right ventricle into undamaged portions of the left ventricle and interventricular septum. Contraction of the undamaged portion of the damaged fibers may act to reduce the capacity of the right ventricular cavity and propel blood into the low resistance pulmonary circuit. Due to the tremendous surface area in relation to volume, relatively small movements of the right ventricular wall will eject large volumes of blood.2

Evidence is presented that during systole, the myocardial fibers in different layers must pull against each other and apply stretch to the connections between them. This represents potential energy which is wasted so far as systolic ejection is concerned. However, as the ventricles begin to relax, this potential energy would be released and could act to return the ventricular chambers toward their diastolic dimensions. This mechanism would be more effective in the thick-walled left ventricle. Under these conditions, the inflowing blood would encounter extremely small resistance to distention. The rapid filling in early diastole may be facilitated by this mechanism. Such an effect should not be termed a "suction" unless the intraventricular pressure dropped below the pressure immediately outside the walls.

**Conclusions**

1. In intact dogs standing quietly, without restraint, ejection of blood by the right ventricle was accomplished primarily by a shortening of the free wall, drawing the tricuspid ring toward the apex of the heart. Changes in the width of the right ventricular chamber are relatively slight but may be of significance due to the large surface area between the interventricular septum and the free wall of the ventricle.

2. During systole, the left ventricle has reduced capacity due to simultaneous reduction in width and length of the chamber. The thick constrictor muscles encircling the left ventricular cavity are probably responsible for a major
portion of the reduction in left ventricular width.

3. Changes in left ventricular width are closely related to the changes in the projected area of the left ventricular chamber as recorded by cinefluorographic angiocardiography. On this basis a measurement of left ventricular diameter is probably closely related to left ventricular volume.

4. Metal markers on the endocardial surface of the heart were displaced greater distances during systole than were markers on opposite sides of the wall.

5. Evidence is presented that the various myocardial layers are oriented in different directions. Since the myocardial fibers can shorten only along their longitudinal axis, the different layers must apply tension to the connections between them. This tension does not contribute to ventricular ejection, but represents potential energy which is released early in the subsequent diastolic period. If the release of this interfascicular tension tends to restore the ventricles to their diastolic dimensions, early diastolic filling would be accelerated.

6. The contributions of the superficial spiral muscles and the deep constrictor muscles are considered in terms of the volume ejected per unit shortening.

7. The significance of residual blood in the ventricular chambers at the end of systole is discussed in terms of the advantages and disadvantages derived from myocardial contraction from a large diastolic size.

ACKNOWLEDGMENTS

The technical assistance of John A. Hendron and Richard M. Ellis is gratefully acknowledged.

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Circ Res. 1953;1:162-170
doi: 10.1161/01.RES.1.2.162

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