Continuous Measurements of Left Ventricular Dimensions in Intact, Unanesthetized Dogs

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Left ventricular dimensions have been directly measured for extended periods of time in intact, unanesthetized dogs under various conditions. The diameter of the left ventricle during diastole is very large in relation to the change in diameter during each cycle. Thus, considerable quantities of blood remain within the chamber at the end of systolic ejection. The stroke output can be increased by either more complete systolic ejection during exercise or by greater diastolic filling during a startle reaction. Accelerated heart rate occurs in both cases. Changes in left ventricular diameter occur very rapidly, often being manifest within the duration of a single cardiac cycle. Mechanisms by which changes in ventricular size can be directly affected by neural and hormonal influences are briefly considered.

The pumping action of the heart depends upon cyclic changes in the dimensions of the cardiac chambers. Direct measurement of the changes in absolute ventricular volume have been limited almost exclusively to cardiomter measurements. The experimental conditions preclude investigation of the cardiac response to stresses encountered during normal activity. Roentgenographic techniques provide some information concerning heart size and configuration without thoracotomy but generally fail to distinguish the various chambers of the heart and are difficult to apply during activity. Indirect measurements of cardiac output permit computation of stroke volume but do not indicate whether the diastolic and systolic ventricular volumes are large or small. Since relatively large volumes of residual blood remain the ventricular cavities at the end of systole, increased stroke volume can be attained by increased diastolic filling or by more complete systolic ejection. Methods for continuously measuring the changes in absolute linear dimensions of the left ventricular cavity have been developed as a first step toward investigating the response of a single cardiac chamber during spontaneous activity in intact, unanesthetized experimental animals.

METHODS

The recording device consisted of a coil (15 by 3 mm.) with two layers of number 39 formvar insulated wire on a nylon form. A steel stylus (15 mm. long) moved freely within the lumen of the coil form and produced variations in the inductance of the coil, which were recorded by a Sanborn strain gage amplifier on a Sanborn Polyviso. It should be emphasized that the stylus was magnetic steel but was not magnetized, so the records indicate the position of the stylus within the coil rather than the rate of change of position.

Under aseptic conditions, the variable inductance gages were installed within the left ventricle, with one end of the stylus fastened to the interventricular septum and the opposite end to the free wall of the left ventricle as indicated in figure 1. Two small metal rods connected by fine elastic threads to the steel stylus were mounted within a specially designed trochar (fig. 1A, 1B). In the free end of the stylus was a small hole through which a long loop of fine silk thread was passed. The loop also passed through the lumen of the coil, which was fastened to another set of pins and mounted on another trochar. The first trochar was then inserted through the free wall of the left ventricle and through the interventricular septum (fig. 1C). Care was exercised to avoid penetrating papillary muscles. The leading metal rod (pin 1) was ejected from the trochar and flipped into a transverse position by the.
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FIG. 1. The method of installing a variable induction diameter gage across the left ventricular cavity is illustrated in an isolated heart. For details, see text.

The trochar was then withdrawn until the second pin could be ejected in the left ventricular cavity (fig. 1E), so the tension of the elastic held both pins firmly against the endocardial surfaces on opposite sides of the interventricular septum. Here the trochar was withdrawn, leaving the loop of thread extending from the free end of the stylus to the outside of the heart.

At this stage, all traction on the heart was released for varying periods to permit recovery from the manual manipulation. The small hole through the ventricular wall sealed itself by muscular contraction; virtually no blood escaped during this period. The second trochar was now brought into position with the loop of thread held taut, and the end of the coil was gently advanced through the ventricular wall, guided by the thread over the free end of the stylus. When the coil was well within the left ventricular chamber, a third small metal rod was ejected (fig. 1F) and was held against the endocardial surface of the left ventricular wall by the elastic connected to pin 4, which remained outside the heart. The second trochar was now withdrawn, leaving the coil supported by pins 3 and 4. The hole in the ventricular wall was frequently distended by the coil so that a suture or a small plug of gelfoam was required to arrest mild bleeding. Finally, the wires extending from the coil were passed through interlobar fissures of the lung and brought individually through the thoracic wall to the outside.

In most experiments, the variable inductance gages were designed for measuring changes in left ventricular diameter, since previously reported data indicated that these changes are more significant than changes in length on two counts: (a) the relative change in diameter is greater, and (b) the quantity of blood ejected by a particular change in diameter greatly exceeds that produced by a corresponding change in length. To confirm this opinion, variable inductance gages were also installed between the region of the apex and the root of the aorta in two dogs (fig. 2B). In these cases, the coil was 3 cm. long and contained a stop so that the stylus could not come out of the coil. This gage was
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Fig. 2. Patterns of records obtained from variable inductance gages installed across the diameter of the left ventricular chamber (A) and from the apex to the root of the aorta (B) are illustrated along with simultaneously recorded electrocardiograms. Gages installed along left ventricular diameters, roughly at right angles to each other (C and D) produced similar patterns. The temporal relations between changes in ventricular diameter (E) and intraventricular pressure (F) indicate that the I wave occurs during isometric contraction.

Variable inductance gages have been installed within the left ventricle of 30 animals. Of these, 20 animals died or were sacrificed after varying periods of time (eight were in good condition when sacrificed more than 10 days after operation and three have been allowed to survive more than a month).

Successful records of left ventricular diameter or length were obtained from 11 of the 20 animals. Records obtained only during the day of operation in four animals failed to reveal the full pattern through the cardiac cycles.

The Changes in Left Ventricular Diameter during the Cardiac Cycle. The recorded curves superficially resemble cardiometric volume curves (fig. 2A). A rapid increase in diameter occurred during the early filling period (EF), followed by a plateau which indicates the duration of slow filling (D), terminated by small upward deflection at the time of atrial contraction (A). Springing from the top of this rounded wave at the end of diastole was a sharp spike, beginning at the onset of the systole (I). After the peak of the I wave, the diameter diminished during the ventricular systole, abruptly at first and slower toward the end of the systolic interval. In most cases, the diameter gage was installed directly across the...
left ventricular chamber (fig. 2, gage C), but a similar pattern was obtained from a gage oriented roughly at right angles to this axis of the chamber (fig. 2, gage D).

Simultaneous electrocardiograms revealed that the P wave terminated before the onset of the A wave and the QRS complex, before the beginning of the I wave (fig. 2A). Therefore, it was concluded that the I wave represents a sudden increase in diameter during the isometric contraction of the ventricle. This was confirmed by simultaneous recordings of the left ventricular diameter and the intraventricular pressure* (fig. 2E, F). Thus, the end-diastolic diameter of the left ventricle is represented by the peak of the A wave.

Most hollow elastic structures tend to assume a more spherical shape as their internal pressure increases, and the abrupt increase in diameter at the onset of ventricular systole might be due to a rounding and shortening of the chamber. In two animals, longitudinal gages were installed within the left ventricular chamber anchored near the apex and near the root of the aorta (fig. 2B). In both cases, this dimension of the heart changed very little during the cardiac cycle. At the apex of the left ventricle there is a region in which the wall is only about 2 to 3 mm. thick. When the coil of one gage was anchored at this point, the distance from the root of the aorta to the apex increased slightly during early systole (fig. 2B). Apparently the thin portion of the ventricular apex bulged as the interventricular pressure became elevated. Another gage was installed with the upper end fastened near the aortic root and the lower end about 1 cm. from the apex toward the interventricular septum. Records from this gage revealed a slight shortening (less than 3 mm.) of this particular longitudinal axis during systole. In other words, the septal wall of the left ventricular cavity apparently shortens very little during systole, which is in line with previously reported data.10 This degree of ventricular shortening does not appear adequate to explain the rather large expansion of ventricular diameter during isometric contraction. Damage to myocardium at the two ends of the coil during its insertion may have allowed local aneurysmal bulging at the onset of ventricular systole.

Response to Startle. Changes in the left ventricular diameter were repeatedly recorded during the response to an unexpected loud noise produced by slapping a table with a meter stick (fig. 3). Little if any change was noted if the animal anticipated or became accustomed to the noise. The most typical response consisted of a transient increase in systolic and diastolic diameter and in heart rate followed by a more sustained increase in diastolic size (fig. 3). On one occasion, an experimental animal was lying relaxed on the floor while records were made at slow paper speed (2.5 mm. per second) for 10 minutes. He appeared to be asleep during the interval marked "somnolent" in figure 3 and the systolic and diastolic diameters diminished to very low levels (the gage was blocked during most of each cycle). At the arrow, a loud noise was produced and within one cardiac cycle, an extremely large increase in ventricular diameter occurred, probably not due to a sudden movement of the animal since artifacts did not appear on the records during running or jumping. In this case, the change in heart rate was insignificant.

Variations in Left Ventricular Diameter during Exercise. Records were obtained during strenuous exercise in two animals. The record illustrated in figure 4 was from an animal which was extremely active from the second to the 25th day after operation. The changes in systolic and diastolic diameters followed patterns which varied somewhat on different occasions. Sample records from four exercise periods by the same dog on one day are shown. The top two records were obtained during two essentially identical exercise periods at 3 m.p.h. on a 2 1/2 per cent grade. At the top appear short segments of a record obtained at a paper speed of 25 mm. per second which indicate the shape of the patterns of individual cycles at times indicated on the second record taken at 2.5 mm. per second. The peaks of the A

* This record was obtained from a specially designed differential-transformer pressure transducer mounted adjacent to the apex of the heart with a short tube extending into the left ventricular chamber.
waves represent the end diastolic diameter and are connected by a continuous line for identification. The third record is a plot of the average heart rate from each successive sequence of five cycles. The initial response to exercise was a marked acceleration of the heart, attaining levels in excess of 240 beats per minute. During this time the diameters during both systole and diastole diminished. As the heart rate slowed, the diameter during diastolic periods increased again and during the remainder of the exertion, it fluctuated above and below the control levels. The heart rate leveled off at about 100 beats per minute. After termination of the exercise, the diastolic diameter increased markedly above the control level for a brief period as the heart rate diminished. In a majority of cases, the record became flattened during systole, indicating that the gage was blocking and that the systolic diameter was being reduced more during the exercise than during the control period.

The response to running 3½ m.p.h. on a 7½ per cent grade is illustrated in the lower three records. In this example, the systolic and diastolic diameters were both smaller than in the upper record. Increased diastolic diameter and tachycardia began to increase just before the treadmill was turned on. Apparently, anticipation of exercise produced a cardiovascular adjustment before the energy output of the skeletal muscles increased. Immediately after exercise began, the diastolic and systolic diameters diminished while the heart rate rose to about 225 beats per minute. As the heart rate diminished, the diameter during diastole increased but remained below the control level during most of the remainder of the exercise period. The gage blocked during systole throughout the remainder of the record indicating greater systolic reduction in diameter than during the control period. Near the end of the exercise period an unusual phenomenon occurred. The diastolic diameter progressively diminished and the heart rate began to increase. The animal sat down on the moving treadmill and refused to run any longer.
EVALUATION OF THE RECORDING TECHNIC

The variable inductance gages were stable and precise. In only one dog did the gage become invested with organizing thrombus. After removal of the heart, the stylus moved freely within the coil with minimal resistance. No changes in the rate of response or in the patterns were observed which could be attributed to this factor. In most animals, the coils were coated with plastic and remained remarkably free of thrombi (fig. 2E).

Utilization of new technics imposes a responsibility for critical analysis of the methods to place the results in proper perspective. It is of great importance to consider the possibility that the recording technic may have changed the function being studied. In this case, the installation of a variable inductance gage within the ventricular cavity unquestionably damaged some myocardium. Gross and microscopic examination a few days or weeks after operation revealed connective tissue reaction along the course of the elastic fibers and lead wires, generally confined to a cross-sectional area 1 to 2 mm. in diameter. The heart illustrated in figure 2 contained both a diameter gage and a pressure gage for 56 days during which the animal remained active and apparently healthy.

Electrocardiograms were taken before each operation and every few days after operation, whether the gage functioned or not. In a ma-
iority of the animals, significant deviation of the S-T segment (2-3 mm.) and some alteration in the configuration of the T-wave were noted immediately after operation. Within a week, this evidence of myocardial damage was largely or totally missing. In some cases the elastic threads connecting the anchoring pins were short and erosion of the internal layers of myocardium under the anchoring pin was caused by the sustained elastic tension. During the first one or two days after operation, ventricular premature contractions occurred with widely varying frequency. The cardiac silhouette on roentgenograms was not detectably greater a few days after operation than immediately before surgery.8

The environment of the heart was altered to varying degrees by the surgery. Dense adhesions between the pericardium and the epicardium were invariably found around the external anchoring pins, lead wires and at the site of the pericardial incision. In most animals, loose pericardial adhesions were found at varying distances from these regions. The interlobar fissures through which the lead wires passed also became tightly fused and the pleura adhered to the thoracic wall along the thoracotomy wound. The extent of pleural adhesions was also quite variable in some cases involving a major portion of the left lung, but the right pleural space was generally free of adhesions. The extent to which the pericardial and pleural adhesions affected cardiopulmonary function is not known.

**Discussion of Results**

The changes in left ventricular diameter during the cardiac cycles were invariably a small fraction of the total diameter of the chamber. However, this does not imply that the stroke volume is a small fraction of the residual blood in the heart for three reasons: (a) changes in volume of an asymmetric cavity cannot be accurately computed from a record of a single dimension; (b) a considerable proportion of the cavity is occupied by papillary muscles and trabeculae carnae (see fig. 2C); and (c) the volume ejected during a particular change in diameter is far greater when the chamber is large than when it is small.9

During the response to a loud noise, the diastolic and systolic diameters promptly increased as the heart rate accelerated, both effects beginning within the interval of a single cardiac cycle.

The initial response to exercise by two animals was a diminution in both systolic and diastolic diameter accompanied by prompt acceleration of the heart. During the remainder of the exercise period the change in diameter during each stroke was apparently increased by greater reduction in diameter during systole rather than by greater expansion during diastole. This may be ascribed to increased “contractility” of the myocardium which can be produced by substances such as epinephrine.1, 9, 11 The chamber was not operating at its maximum diastolic capacity during exercise since both the systolic and diastolic diameters increased immediately after the exertion was over.

The cardiac response to exertion was contrary to expectations since an increase in diastolic diameter was anticipated. Neural controlling mechanisms may affect both the heart rate and left ventricular diameter, judging by the extreme rapidity of the responses and the anticipatory changes which frequently occurred just before the treadmill was started. However, the heart rate and ventricular diameter appear independently variable since the diameter increased during a startle reaction and decreased during exercise while the heart rate usually increased in both situations. The functional importance of the neural controlling mechanisms which influence absolute ventricular dimensions is probably far greater than is indicated by experiments on anesthetized thoracotomized animals. The extent of ventricular ejection is clearly influenced by the degree of myocardial shortening which involves “contractility.” The diastolic distension is determined by the effective filling pressure and the resistance to distension offered by the ventricular walls (distensibility). Both contractility and distensibility are affected by epinephrine,1, 9, 11 which also generally produces tachycardia. However, acceleration of the heart can also result from diminished vagal discharge. Such factors may account for the differences between
the responses to startle and to exertion. The response to exercise has been recorded a total of 30 times on the two animals. More extensive data will be required before these results could be considered characteristic of other animals or man. Nonetheless, the evidence indicates the value of directly measuring important variables in intact, unanesthetized dogs.

CONCLUSIONS

1. Continuous measurements of left ventricular dimensions can be precisely measured by means of variable inductance gages installed within the left ventricular chamber.

2. During systole, changes in diameter are of greater importance in the ejection of blood than shortening along an axis between the apex and the root of the aorta. The contribution to ejection effected by shortening of the free wall of the left ventricle is not yet established.

3. Changes in diameter recorded from gages oriented roughly at right angles are similar in form, which tends to confirm the concept that the left ventricle contracts concentrically, presumably through the action of the circumferentially-arranged deep muscle layers.

4. Startle reactions produce prompt increase in both heart rate and in diastolic and systolic diameters which may be sustained for considerable periods.

5. The typical response to exertion by two dogs was a reduction in systolic diameter of the left ventricle with little change in this dimension during diastole. Thus, increased stroke volume may have been attained by more complete ejection rather than by more extensive diastolic distension.

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