Measurement of Left Ventricular Diameter In the Dog by Cardiac Catheterization

VALIDATION AND PHYSIOLOGIC MEANINGFULNESS OF AN ULTRASONIC TECHNIQUE

By Richard A. Carleton, M.D., and James G. Clark, M.D.

ABSTRACT
A cylindrical ultrasonic transducer on a catheter tip lodged in the cleft between the free wall of the right ventricle and the interventricular septum provides an easily measured echo from the posteroinferior left ventricular epicardial surface. The sonic path represents a diameter of the left ventricle at approximately its largest cross-sectional area. Experiments in six dogs demonstrated that measurements by ultrasound agree within ±6% with simultaneous radiographic measurements of left ventricular diameter. Procedures designed to alter left ventricular volume were employed in 14 dogs to clarify the physiologic meaningfulness of the left ventricular diameter measured by ultrasound. Both end-diastolic and end-systolic diameter increased with anoxia, with sudden injections of saline into the left ventricle, after propranolol, and after inflation of a balloon in the thoracic aorta. These diameters decreased with isoproterenol, with increased heart rate, and after inflation of a balloon in the inferior vena cava. The present technique provides accurate and physiologically meaningful estimates of left ventricular diameter. Ventricular diameter measurements obtained during cardiac catheterization should permit assessment of ventricular distensibility and of ventricular function.

ADDITIONAL KEY WORDS
myocardial force-velocity curves
ventricular preload
ventricular dimensions
ventricular function
ventricular afterload

Although considerable knowledge of cardiac output and pressure performance of normal and abnormal human hearts has been accumulated in recent years, the lack of a method for continuously measuring the dimensions of the heart has seriously limited more detailed study of cardiac physiology in clinical investigation.

Studies performed predominantly in animals have defined the measurements required to characterize myocardial function. The ventricular function curve concepts of Frank and of Starling (1) have been expanded and modified by Sarnoff and his co-workers (2). Later studies, particularly by Sonnenblick (3, 4), have elaborated upon the muscle model proposed by A. V. Hill (5) and have demonstrated its applicability to the myocardium. These pioneering studies have made it clear that techniques which measure myocardial segment length and the velocity of myocardial shortening during ventricular ejection in man during cardiac catheterization would provide important information for the characterization of cardiac function.

Two methods have contributed importantly to information concerning human ventricular function. Each, however, has deficiencies. The indicator dilution technique overestimates ventricular volume (6, 7), and this limits its usefulness for quantitative studies of ventricular function. Angiocardiography has ac-
ceptable precision (8), but the contrast media alter plasma volume (9), heart rate (10), cardiac output (11), and cardiac rhythm (12).

The limitations of these methods for clinical investigation led us to search for other approaches to the measurement of ventricular dimensions. This report describes an ultrasonic technique for measurement of left ventricular diameter in the intact animal and its physical and physiologic validation.

Material and Methods

The initial studies with this technique were performed with hearts excised from dogs killed in other experiments. Subsequent studies were conducted in 22 mongrel dogs (average weight 16.2 kg, range 12.3 to 19.1 kg). Previous experience with barbiturates as the sole anesthetic agent has been unsatisfactory in this and other laboratories (13) because of the associated sinus tachycardia. Accordingly, a mixture containing 42 mg chloral hydrate, 7 mg sodium pentobarbital, and 21 mg magnesium sulfate per milliliter was administered intravenously in a dose of 2 ml/kg. Supplemental anesthesia was given at evidence of arousal. Heart rate was controlled, as indicated in subsequent sections, by an external pacemaker connected to a bipolar pacing catheter in the right atrium. We exposed the femoral vessels in each dog and the right external jugular vein in several. Catheters were placed under fluoroscopic control.

The right femoral vein was used to introduce an 8 French cardiac catheter modified by the addition of a piezoelectric ultrasonic transducer at its tip. This transducer is made by precipitation of lead zirconate crystals with a radial orientation. The resulting transducer is cylindrical with an inner diameter of 2.1 mm, an outer diameter of 3.1 mm, and a length of 8 mm. The inner and outer surfaces of the cylinder are electroplated; wires run from the surfaces through the cardiac catheter and terminate at an electrical connector at the proximal end of the catheter. The transducer is then bonded to a catheter with epoxy resin. The resulting unit is easily introduced through the tricuspid valve and placed into the ventricular cavity. A standard ultrasonic oscillator1 energized the transducer 1000 times/sec (repetition rate); each energy burst lasted 2 microseconds and consisted of oscillations at an ultrasonic frequency of approximately 2.5 megahertz. The transducer serves as a receiver for the remaining 998 of each 1000 microseconds. Ultrasonic energy reflected back to the transducer produces electricity which is amplified and displayed on an oscilloscope. The horizontal sweep of the oscilloscope is triggered by each electrical input to the crystal; the ultrasonic transit time is then represented by distance along the x-axis between the initial energy input and the receipt of the reflected energy.

The cylindrical shape of the transducer provides ultrasonic energy which radiates in a full 360° arc perpendicular to the long axis of the transducer. This shape ensures that the rotational position of the transducer does not modify its function. The ultrasonic-oscillator-osilloscope system is easily calibrated by immersing the transducer in a container of known dimensions and adjusting the horizontal sweep rate of the oscilloscope to provide a visible echo at a point corresponding to the actual distance traversed by the sound through blood or water.

The physical placement of the catheter is similar to that commonly used in diagnostic right heart catheterization and, particularly, in long-term endocardial pacing. Sporadic ventricular premature beats occurred during initial placement of the transducer; when a stable position was established no arrhythmias were observed. The loss of insulation around the transducer was simulated in four dogs by a pacing catheter in the right ventricular clefts that was connected at its external terminals directly to the electrical output of the ultrasonic oscillator. Ventricular tachycardia could be produced at will whenever the repetition rate of the ultrasonic oscillator was below 350/sec. No arrhythmias were encountered with direct endocardial stimulation with the oscillator output at rate exceeding 500/sec. All subsequent studies were conducted with a repetition rate of 1000/sec. The superimposed ultrasonic frequency of 2.5 megahertz had no detectable effect on cardiac rhythm or function.

I. STUDIES IN EXCISED HEARTS

Experience with chronically implanted cardiac pacing catheters suggested that the catheter-tip ultrasonic transducer could be securely lodged in the extreme left portion of the right ventricle where the free right ventricular wall is in contact with the junction of the interventricular septum and the free left ventricular wall. Studies were conducted in ten excised canine hearts to test the hypothesis that an approximately transverse orientation of the transducer in this location would provide ultrasonic energy radiation perpendicular to a cross section of the left ventricle approximately at its largest section and that a nearly continuous record of left ventricular diameter could be obtained. The ultrasonic catheter was easily introduced through the tricuspid
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valve, passed into the cleft between the free right ventricular wall and the interventricular septum, and lodged between the trabecular muscles which form natural crevices in this area. When the catheter was positioned approximately midway between the apical segment of the right ventricle and the pulmonic valve, the transducer position was perpendicular to the long axis of the left ventricle and directly on the interventricular septum.

With the transducer in this position, each heart was suspended and filled with saline solution. No ultrasonic echo could be identified from the interface between the anterior left ventricular endocardium and the left ventricular cavity, nor from that between the left ventricular cavity and the posterior left ventricular endocardium. A uniform intense echo returned from the posterior left ventricular epicardium in each heart. (The degree of amplification of the echo signal necessary to detect the posterior left ventricular wall produced multiple echoes from shorter dimensions within the right ventricle; a reduction in sensitivity permits measurement of the distance between the transducer and the epicardial surface of the right ventricle.) The reflecting surface was precisely identified by locating the area of the left ventricular wall at which the echo was abolished by application of an inverted, saline-filled test tube 1 cm² in cross-sectional area to the wall. In each instance, a single area was identified as the reflecting surface. This location was always in the middle fifth of the posteroinferior left ventricular epicardial longitudinal dimension. The diameter measured by ultrasound was compared with the diameter measured by calipers. The greatest observed difference in the two measurements was 0.5 mm, with ventricular diameters of approximately 60 mm.

Three additional excised hearts were studied after the coronary arteries were ligated, the mitral annulus sealed, and the aorta occluded by a rubber stopper through which a cardiac catheter was passed. The ultrasonic transducer was placed in the right ventricular cleft. Left ventricular cavity volume was varied from 0 to 40 ml by 5-ml increments and decrements. Left ventricular diameter measurements were made at each volume level.

II. VALIDATION EXPERIMENTS IN INTACT DOGS

Comparison of Radiographic and Ultrasonic Diameter.—Dogs 15 through 20 were subjected to a left thoracotomy. A thin polyethylene catheter was introduced into the pericardial sac and sewn tightly to the pericardium to ensure an airtight seal. A platinum disk, 9 mm in diameter, was sewn to the pericardium over the anterior surface of the right ventricle, just anterior to the expected site of ultrasonic catheter placement. The chest wound was then tightly closed after full expansion of the lungs. The ultrasonic catheter was introduced through a femoral vein and advanced into the right ventricle. The transducer was easily lodged in a right ventricular cleft under fluoroscopic control (subsequent post-mortem examination of four animals demonstrated that the transducer was firmly placed between trabecular muscles). Each dog was then rotated on the fluoroscopic table to approximately a 75° left anterior oblique position, to display approximately the maximal transverse left ventricular diameter at the level of the ultrasonic transducer. Two radiopaque markers exactly 5 cm apart were placed on the anterior wall of the chest at the transverse level of the midplane of the left ventricle.

Simultaneous cinefluorography (60 frames/sec) of the heart and motion pictures (64 frames/sec) of the oscilloscope face were made for approximately 15-second periods under each of the various conditions studied in the six dogs. Respiration was suspended after moderate pulmonary inflation during each filming period to lower the diaphragm beneath the cardiac shadow. The initial exposure in three animals was conducted with no air in the pericardium. All subsequent exposures were made after the introduction of 40 ml of air into the pericardial space. Pericardial air did not modify the ultrasonic echo, but did increase the clarity of the radiographic image and therefore the ease with which the largest external transverse left ventricular diameter could be measured from each cine frame. Representative sections of cinefluorographic film and movie film are reproduced in Figure 1. Several procedures were used to modify heart size and the degree of systolic excursion. Propranolol was given to slow heart rate, cardiac pacing was used to speed heart rate, and additional large overdoses of anesthesia were used to produce respiratory arrest followed by eventual anoxic cardiac arrest.

Left ventricular diameter at end-diastole and end-systole, and the magnitude of systolic excursion were measured from successive frames of the motion pictures of the ultrasonic oscilloscope face. The cinefluorographic film was analyzed by adjusting the projected image until the images of both the 9-mm platinum disk and the 5-cm radiopaque ruler achieved actual size. Measurements of the distance between the ultrasonic transducer and the posterior inferior left ventricular wall were made at the times in each cardiac cycle of maximal and minimal values; these were taken to represent end-diastolic and end-systolic diameter respectively. The same five beats were analyzed by each technique. The maximum variation in the measurements within any
Simultaneous frames taken from cinefluorographic film and from film of the ultrasonic echo on the oscilloscopic face obtained in dog 19. The bottom cinefluorographic frame has been enlarged for clarity. The heart shadow is outlined by pericardial air. The elliptical opaque shadow (A) behind the sternum is a platinum disk 9 mm in diameter; the opaque markers (C) anterior to the sternum were 5 cm apart at the transverse level of the left ventricle. The cardiac catheter enters the heart from the inferior vena cava. The ultrasonic transducer at the tip (B) is lodged in the right ventricular cleft. Measurements made from the transducer to the posteroinferior surface of the left ventricle decrease from 63 to 60 mm during these systolic frames.

The oscilloscopic face shows two beams. The bottom calibration beam is adjusted to indicate time equivalent to each 5 mm and 10 mm of distance. The upper beam shows, at the extreme left, the initial electrical input to the transducer and several echoes at distances less than 30 mm. These frames, taken during systole, show a clear echo at a time equivalent to a sonic path of 63 to 60 mm.
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five-beat sequence, by either technique, was 1.0 mm for end-diastole, end-systole, and the change in diameter during systole. The average value for each group of five beats by each technique was used in subsequent comparative analyses.

Relationship of Transducer to Interventricular Groove.—After left thoracotomy in dogs 21 and 22 the pericardium was opened and approximately 4 cm of flexible radiopaque tubing was sewn directly to the interventricular groove on the anterior surface of the heart. After positioning of the ultrasonic catheter, cinefluorographic exposures were made in the lateral and anteroposterior views. The spatial relationship between the tip of the ultrasonic catheter and the fixed marker on the interventricular septum was analyzed in the transverse (X) axis, the superior-inferior (Y) axis, and the anteroposterior (Z) axis. All measurements were made to the same point on the segment of catheter.

III. CHANGES IN LEFT VENTRICULAR DIAMETER

This second series of experiments was designed to study the effects of procedures known to modify left ventricular size on left ventricular diameter as recorded by the ultrasonic technique.

Left Ventricular Volume Load.—A 7 French catheter was passed into the left ventricle through the aortic valve in dogs 11 through 14. Central aortic pressure was recorded through a separate catheter placed in the ascending aorta. All pressures in these experiments were recorded with standard strain gauge transducers and an oscillographic recorder at a paper speed of 50 mm/sec. The ultrasonic transducer was placed in the right ventricular cleft. A constant heart rate was maintained by right atrial pacing. Repetitive injections of 20 ml of saline at 37°C were made into the left ventricle during 1.1 to 1.3 seconds from a power syringe. Each injection encompassed approximately three cardiac cycles. Five minutes elapsed between each injection. Aortic pressure and left ventricular diameter were measured before, during, and after each injection. Data obtained with injections which produced ventricular premature beats were not analyzed.

Effects of Varying Heart Rate and Beta-Adrenergic Receptor Activity.—In dogs 1 through 5 an ultrasound transducer was inserted in the usual fashion, a pacing catheter was placed in the right atrium, and through another catheter in the right atrium isoproterenol was administered at a rate of 4 to 6 μg/min, or propranolol was given as a single dose of 10 mg. Each dog was studied after changes of heart rate of approximately 10 beats per minute. Ventricular diameter became constant after the first several beats after each change in heart rate. Measurements were made during the third minute after each change. Dogs 1 and 2 received only isoproterenol and were studied at only two heart rates during isoproterenol infusion. Dogs 3 through 5 were studied at several heart rates during isoproterenol infusion; propranolol was given 5 minutes after isoproterenol had been stopped. Heart rate was again varied after propranolol. Isoproterenol accelerated the sinoatrial rate and limited the heart rates available for study by right atrial pacing; second degree A-V block occurred in two dogs at faster paced rates after propranolol. Left ventricular diameter at end-diastole and at end-systole, and the systolic excursion were measured at each heart rate in each drug state.

Effects of Increased Aortic Impedance and Decreased Venous Return.—In dogs 6 through 10

![Figure 2](http://circres.ahajournals.org/)
catheters were inserted in the usual way. In addition, a catheter which terminated in a balloon was inserted into the midsdescending thoracic aorta. A separate balloon catheter was placed in the inferior vena cava 4 cm below the diaphragm. Pacing was used to maintain a constant heart rate. After measurements of left ventricular diameter and pressure, the aortic balloon was then inflated with 5 ml of sodium diatrizoate and readings of left ventricular pressure and diameter were repeated for approximately five cardiac cycles. The aortic balloon was deflated and after 5 minutes the measurements were repeated before and 10 seconds after inflation of the inferior vena caval balloon. An infusion of isoproterenol at a rate of 4 to 6 ìg/min was begun, and the above sequence was repeated 5 minutes later. Because of an oversight, the inferior vena caval balloon was not inflated during isoproterenol infusion in dog 10.

Results

I. STUDIES IN EXCISED HEARTS

Results obtained during one of several experiments with the left ventricle from each of three dogs are presented in Figure 2. With a single exception in the second heart, an increase in left ventricular cavity volume by any 5-ml addition produced an increase in left ventricular diameter measured by ultrasound. The studies in the first two hearts suggest the expected slight curvilinear relationship between volume and transverse diameter over this relatively small range of diameter.

The point on the posteroinferior left ventricular wall from which the echo came was identified in each of these hearts by the method described previously. The 1-cm² epicardial region that was the reflecting surface did not change as cavity volume was altered by as much as 40 ml.

II. VALIDATION EXPERIMENTS IN DOGS

The temporal relationship of diameter measurements to left ventricular pressure and the electrocardiogram is shown in Figure 3 from data obtained at a heart rate of 170 beats/min. Diameter measurements were taken at intervals of 15.6 milliseconds.

Comparison of Ultrasonic and Radiographic Diameter.—The ultrasonic echo from the posteroinferior left ventricular wall was slightly

| Table 1: Comparison of Left Ventricular Diameter Measured by Cinefluorography and by Ultrasonic Transit Time |
|-------------|-------------|-------------|-------------|
| Dog | Wt (kg) | No. of observations | End-diastolic diameter (mm) | End-systolic diameter (mm) |
|      |          |                 | Cine | Ultrasound | Cine | Ultrasound |
| 15  | 18.8     | 5               | 59.8 ± 1.22 | 61.0 ± 0.95 | 55.1 ± 1.89 | 56.8 ± 1.68 |
| 16  | 12.3     | 5               | 55.3 ± 0.60 | 55.4 ± 0.58 | 50.0 ± 0.44 | 50.3 ± 0.56 |
| 17  | 18.3     | 10              | 64.0 ± 0.76 | 64.4 ± 0.54 | 57.7 ± 0.49 | 58.0 ± 0.60 |
| 18  | 15.0     | 7               | 59.7 ± 0.98 | 59.9 ± 0.85 | 55.8 ± 0.91 | 55.7 ± 0.63 |
| 19  | 15.9     | 8               | 61.0 ± 0.76 | 61.3 ± 0.63 | 58.3 ± 1.05 | 58.1 ± 0.97 |
| 20  | 17.0     | 7               | 58.4 ± 0.80 | 58.5 ± 0.75 | 54.3 ± 1.30 | 54.3 ± 1.34 |

Values are mean ± SEM.
more intense after air was introduced into the pericardium, but the left ventricular diameter measurement was not altered. The ultrasonic echo present with normal respiration was not altered by the gentle lung inflation used to lower the diaphragms below the cardiac silhouette. The means and standard errors of data obtained by each technique are presented in Table 1; individual comparisons for end-diastolic and end-systolic diameter are presented in Figure 4. The average values for end-diastolic diameter were 60.3 mm by cinefluorography and 60.6 mm by ultrasound. Direct comparison of measurements of end-diastolic or end-systolic diameter obtained by the two techniques demonstrated three discrepancies of as much as 3 mm. The standard deviation of the difference was ±1.6 mm. Assuming the radiographic technique to be without error, the expected variability of left ventricular diameter by the ultrasonic technique would be approximately ±6% with left ventricular diameters of approximately 60 mm.

The average systolic excursion for all dogs was 4.61 mm/cycle by cinefluorography and 4.68 mm/cycle by ultrasound. The systolic excursion measured by the two techniques differed by as much as 0.7 mm in three of 42 comparisons. The standard deviation of the difference between measurements of systolic excursion made by the two techniques was ±0.3 mm. Even if it is assumed that precisely the same diameter was measured by each technique and that the radiographic measurements are error free, the range of likely deviation of the ultrasonic technique would be less than ±15%.

As noted previously, three procedures were used to alter ventricular size during this series of experiments. Neither pacing at widely different heart rates, the administration of propranolol, nor asphyxia altered the comparability of ultrasonic and radiographic measurements. This suggests that the geometric relationship between the transducer and the postero-inferior ventricular surface remains stable through a wide range of ventricular size.

**Relationship of Transducer to Interventricular Groove.**—The results obtained in each of the two dogs were essentially identical. The ultrasonic transducer did not move relative to the interventricular groove marker in either the Y or the X axis in either dog. The transducer did move slightly along its long axis and, therefore, along the X axis. This motion was approximately 0.5 mm to the right during systole and 0.5 mm to the left of its initial position in early diastole. The maximal excursion in the X axis was 1 mm. The stability of the transducer in the Y axis was taken to imply that the crystal was lodged between trabeculae. The stability in the Z axis was taken to imply continued contact with the interventricular septum throughout the cardiac cycle.

**III. Changes in Left Ventricular Diameter With Physiologic Changes**

**Effect of Anoxia.**—Respiratory arrest occurred after administration of an overdose of anesthesia in dogs 15, 19, and 20. Visible cardiac dilation was noted by fluoroscopic observation during the succeeding 3 or 4 minutes in each of the three dogs. The results obtained by ultrasonic measurement of left ventricular diameter are displayed in Figure 5. Both end-diastolic and end-systolic diameter increased;
systolic excursion decreased markedly. Final complete cardiac arrest occurred with a fixed diameter at the level of the end-diastolic diameter displayed in Figure 6.

Left Ventricular Volume Load.—Each rapid injection of warm saline into the left ventricle increased end-diastolic and end-systolic diameter by approximately 3 mm in each dog. Results of a representative injection in each of the four dogs are shown in Figure 6. The increase in diameter occurred in the first diastole after the onset of injection and persisted for three cycles and then returned gradually toward the original diameter during the next three to five cycles. Aortic systolic pressure increased by 10 to 15 mm Hg during the three beats of increased left ventricular diameter. Aortic pressure then fell gradually to, or slightly below, the control levels.

Effects of Varying Heart Rate and Beta-Adrenergic Receptor Activity.—The left ventricular diameters at different heart rates are presented in Table 2. Each increase in heart rate of greater than 20 beats/min decreased left ventricular diameter. End-diastolic diameter decreased slightly more than end-systolic diameter; systolic excursion became smaller with increased heart rate. The data presented in Table 2 were obtained as heart rates were increased; the same inverse relationship between left ventricular diameter and heart rate was seen as rate was progressively decreased back to the intrinsic sinoatrial rate.

Data obtained during isoproterenol infusion and, in dogs 3 through 5, after propranolol administration are also shown in Table 2. Isoproterenol uniformly decreased end-systolic diameter and increased systolic excursion. Changes in end-diastolic diameter were variable. Conversely, propranolol increased end-systolic diameter and markedly reduced systolic excursion. End-diastolic diameter increased above control levels after propranolol in all dogs at faster rates, but was slightly smaller in dog 3 at two lowest rates.

Effects of Increased Aortic Impedance and Decreased Venous Return.—The changes in diameter obtained in this series of experiments are summarized in Figures 7 and 8. Inflation of the aortic balloon uniformly increased left ventricular systolic pressure by $28 \pm 4.0$ (SE) mm Hg from an average of $147 \pm 7.5$ mm Hg. Simultaneously, left ventricular end-diastolic
Effects of Altered Heart Rate and Administration of Isoproterenol or Propranolol on Left Ventricular Diameter

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*ED = end-diastolic; ES = end-systolic; dD = ED – ES.

and end-systolic diameter increased by approximately 1 mm. Administration of isoproterenol to these animals produced changes in diameter similar to those seen in dogs 1 through 5. Inflation of the aortic balloon during isoproterenol infusion increased systolic pressure from a mean of 139 ± 9.0 mm Hg by 52 ± 7.1 mm Hg and end-diastolic and end-systolic diameters by averages of 1.8 and 2.5 mm respectively. Systolic excursion values remained large.

Inflation of the inferior vena caval balloon uniformly reduced left ventricular pressure by an average of 40 ± 7.0 mm Hg. Concomitantly, end-diastolic diameter, end-systolic diameter, and the magnitude of the systolic excursion decreased sharply. The results obtained during isoproterenol infusion were similar. End-systolic diameters decreased even further than with caval balloon inflation alone. Systolic excursion values remained larger than those obtained without isoproterenol.
Diameter measurements returned to their initial levels after each procedure.

**Discussion**

Studies conducted in excised hearts have demonstrated that an ultrasonic transducer on a catheter tip can be securely lodged in the cleft between the free wall of the right ventricle and the interventricular septum. A left ventricular diameter can then be measured in a posteroinferior direction from the transducer. This diameter is approximately at the level of maximal left ventricular cross-sectional area when the transducer is placed at the midposition of the right ventricular cleft. The precision of the method in the excised heart is related only to the precise identification of the place from which the measured echo originates and the physical calibration of the ultrasonic transmission time. Comparisons between radiographic and ultrasonic measurements obtained in anesthetized dogs show close agreement between the left ventricular diameter measured both at end-diastole and at end-systole. The average values for left ventricular diameter measured by ultrasound in dogs 15 through 20 (average weight 16.2 kg) were 60.60 mm and 55.93 mm at end-diastole and end-systole respectively. Rushmer (14) found the average circumference of the left ventricle in a series of dogs to be 15.5 cm; translated into diameter, with the assumption of a circular cross section, this would equal a diameter of 49 mm. Neither the size of the dogs nor the precise location of the circumference along the longitudinal axis of the ventricle was given. Rushmer also noted that left ventricular length measured by a resistance gauge was approximately 5 cm.
and underwent a small change of only 1 mm with each cardiac cycle.

In a similar study, Rushmer and his co-workers (15) sewed barium titanate ultrasonic transducers to either side of the left ventricle in dogs. Recorded left ventricular diameters had a range of 4.5 to 5 cm; the systolic excursion ranged between 4 and 6 mm. The diagrammatic representation of the technique used suggests that a maximal cross-sectional left ventricular diameter was not employed. In addition, the size of the dogs was not given. These reasons may be responsible for the differences in diameter recorded during that study as compared with the slightly larger values obtained in the present study.

Hawthorne (16) measured the left ventricular base-to-apex distance with resistance gauges in dogs after thoracotomy and found a dimension of approximately 7 cm. This dimension changed by only 2 to 3 mm with each cardiac cycle. Total cross-sectional area was estimated in this study by measuring the output of a closed conductive loop placed about the circumference of the left ventricle while the loop was exposed to an alternating magnetic field of homogeneous density. If one assumes a circular cross section, the values of end-diastolic and end-systolic cross-sectional area of 19 and 15.4 cm², respectively, yield an end-diastolic diameter of 4.9 cm and an end-systolic diameter of 4.4 cm. This study, conducted in dogs weighing approximately 14 kg, thus yielded values smaller than those reported in the present study.

A similar study was conducted by Ninomiya and Wilson (17) in dogs weighing 14 to 18
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kg, a range of weights similar to that of the present study. Miniature electromagnetic mutual-inductance gauges were sutured on opposite epicardial surfaces of the left ventricular chamber. These workers found end-diastolic diameters ranging from 44.8 to 66.5 mm (mean = 55.9 mm) and end-systolic diameters ranging from 40.6 to 63.8 mm (mean = 51.0 mm).

Ross and his co-workers (18) studied hearts from dogs with an average weight of 21.3 kg by a technique of rapid fixation of the left ventricle at a known time within the cardiac cycle. An average end-diastolic diameter of 56.7 (range, 51.4 to 61.8) mm and an average end-systolic diameter of 51.8 (range, 49.4 to 53.2) mm was found. These workers also demonstrated that orthogonal diameters at the transverse level of the maximal cross-sectional area of the ventricle were not significantly different within the cavity and therefore that this cross section was statistically indistinguishable from a circle with normal systole and diastole. Acute diastolic dilation produced slightly greater elongation of the vertical cavity diameter than of the corresponding horizontal diameter. The epicardial diameter measured by the present method approximately parallels this vertical diameter.

Each of the studies described above has demonstrated that the transverse left ventricular diameter or circumference does not undergo a uniform decrease and increase within the cardiac cycle. A slight increase in left ventricular diameter occurs during the isovolumic phase of ventricular contraction and a similar, but smaller, decrease in ventricular diameter during isovolumic relaxation. These data suggest that analyses based upon estimates of maximal and minimal diameter will overestimate systolic excursion. The data of Ninomiya and Wilson (17) indicate that the magnitude of this overestimate will be approximately 0.8 mm with systolic excursions of 4.9 mm.

Several of the studies described above emphasize that the predominant change in ventricular dimensions within a cardiac cycle take place in transverse diameter and that only slight changes occur in the left ventricular longitudinal dimension in the closed chest preparation. This indicates that estimates of ventricular size can be obtained from diameter measurements alone. Further evidence of physiologic meaningfulness must come from studies of physiologic procedures.

The present study included observations of the effect of diverse procedures on recorded left ventricular diameter. Increases in preload produced by sudden injections into left ventricles increased measured ventricular diameter. This change is in accord with the effects of increased ventricular volume produced by similar or different means (19). Increased left ventricular after-load has been imposed by inflation of a balloon in the thoracic aorta. This intervention produced easily measured increases in left ventricular diameter. Such increases in ventricular afterload have been shown to increase ventricular size (20). The present studies have shown that left ventricular diameter measured by ultrasound decreased when an inferior vena caval balloon was inflated to diminish left ventricular preload. The resulting decrease in ventricular size is comparable to that encountered in other studies of caval balloon inflation (21). Increased heart rate is known to decrease cardiac size (22); similar decreases were encountered in the present study. Beta-adrenergic agonists increase stroke volume predominantly by decreasing left ventricular end-systolic volume (23). The present study has demonstrated similar changes in left ventricular diameter during the administration of isoproterenol. Conversely, the administration of propranolol has been shown to increase heart size in some human subjects (24) but not to alter heart size significantly in unanesthetized man at rest (25). The present study in anesthetized dogs demonstrated that the administration of propranolol increased left ventricular end-diastolic and end-systolic diameter with a corresponding reduction in systolic excursion. These data demonstrate that interventions known to change left ventricular volume produce directionally appropriate changes in left ventricular diameter as measured by the present ultrasonic technique.
Measurements of left ventricular diastolic diameter have several direct applications to clinical investigation. Firstly, the relationship between ventricular diastolic pressure and diameter will provide estimates of ventricular diastolic distensibility. Secondly, values for diameter at end-diastole and calculated external stroke work values provide data with which ventricular function curves can be plotted (2). Lastly, close approximations of left ventricular myocardial force-velocity relationships may be possible. Ross et al. (18) have demonstrated that the left ventricular cross section approximates a circle. Changes in external diameter, therefore, reflect changes in length of circumferentially oriented myocardial fibers. Repetitive diameter measurements at 64 frames per second (15.6 msec between frames) should then provide directly calculable measures both of instantaneous length and of velocity of shortening of the ventricular circumference. The problem of calculating force generated by the ventricular wall is not directly soluble with data obtained by the present technique, because ventricular wall thickness is not measured. Direct measurements have shown that thickness varies through the cardiac cycle, with increases of approximately 20% (26) to 28% (18) during systole, but that the endocardial and epicardial segments move almost concordantly through the cardiac cycle. Feigl and Fry (27) have shown that myocardial layer-to-layer shear is relatively large during isovolumic contraction, but is small during ejection. Therefore, wall tension calculated from intraventricular pressure and epicardial circumference (28, 29) should provide approximate and directionally appropriate data for use in the assessment of myocardial contractility from force-velocity-length relationships.

The technique has proved to be harmless for application to the intact anesthetized dog. Other experience with cardiac catheterization suggests that an equal degree of benignity is likely during application to man. Preliminary post-mortem studies on human hearts demonstrate that a position of the catheter tip comparable to that used in the dog can be easily accomplished and that left ventricular diameter is easily measurable in the human heart.

The present study indicates that a catheter-tip cylindrical ultrasonic transducer provides precise measurements of left ventricular diameter. Moreover, the appropriate changes of left ventricular diameter with diverse physiologic procedures suggest that the diameter being measured is physiologically meaningful.

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References
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RICHARD A. CARLETON and JAMES G. CLARK

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