The Three-Dimensional Dynamic Geometry of the Left Ventricle in the Conscious Dog

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SUMMARY The dynamic geometry of the left ventricle was assessed with the use of chronically implanted pulse-transit ultrasonic dimension transducers. The orientation of the transducers allowed the measurement of left ventricular minor and major axis diameters and equatorial wall thickness in the conscious dog. The left ventricle was modeled as a three-dimensional, prolate ellipsoidal shell. Left ventricular and pleural pressures were measured with high fidelity micromanometers. Aortic blood flow was obtained with electromagnetic flow probes. To test the assumptions inherent in this technique, left ventricular mass, internal volume, stroke volume, and peak aortic flow were computed from the dimension data and compared to directly measured values. Correlation coefficients of 0.95 or greater were obtained for each of these comparisons. In addition, the calculated left ventricular mass was constant to within ±6% of the mean value throughout the cardiac cycle. We found that the dynamic contraction pattern of the left ventricle was dependent on the physiological state of the dog. Furthermore, in the conscious state, shortening of the minor axis diameter, lengthening of the major axis diameter, and slight thickening or thinning of the wall were noted during isovolumic contraction (isovolumic ellipticalization pattern). In the open-chested, anesthetized state, however, marked rearrangements in geometry were observed during isovolumic contraction manifested by lengthening of the minor axis diameter, shortening of the major axis diameter, and significant thickening of the wall (isovolumic sphericalization pattern). We also observed that left ventricular volume was significantly diminished in the open-chested state. The isovolumic contraction pattern in open-chested dogs could be changed from sphericalization to ellipticalization by increasing end-diastolic volume with the infusion of saline. During a vena caval occlusion in the conscious state, the contraction pattern changed from isovolumic ellipticalization to isovolumic sphericalization as the end-diastolic volume decreased. Thus, the exact pattern of left ventricular contraction was found to be a function of left ventricular volume.

THE DYNAMIC geometry of the left ventricle has been a subject of interest to cardiovascular physiologists since Harvey’s classic observations in the 17th century.1 Woods1 first emphasized the significance of ventricular geometry in the analysis of cardiac function when he applied Laplace’s law to the heart in 1892. As early as 1905, Henderson3 had accurately measured the dynamic volume characteristics of the ventricles, using plethysmographic techniques, and the pioneering experiments of Patterson et al.4 and Wiggers and Katz5 followed thereafter.

During the past two decades numerous investigators have contributed to the understanding of the dynamic geometry of the left ventricle.6 There remain, however, several controversies as to the exact pattern and extent of dimensional changes that occur throughout the cardiac cycle.7 The changes that have been observed in the configuration of the left ventricle during the isovolumic phases of systole have differed considerably from study to study. Several investigators, using direct methods of dimension measurement, have noted expansion of the minor axis and shortening of the major axis during isovolumic contraction.8–11 Other experiments, utilizing equally valid methodology, have demonstrated the opposite geometric pattern.14–15 Although most authors have concluded that major axis shortening contributes minimally to volume displacement during ejection, the exact contribution and the geometry of major axis shortening have varied substantially.9, 11, 16, 17 The pattern of wall thickening during systole likewise has been controversial.15–17 The reported extent of wall thickening in normal hearts has ranged from 9% to 106%. Up to 60% of total wall thickening has been observed during isovolumic contraction in some studies, but isovolumic thinning has been reported in others. Possible explanations for these discrepant observations include methodological peculiarities; differences in the experimental preparations utilized, and the specific effects of anesthetic agents.

The accuracy of geometric measurements assumes greater importance if shell theory is to be used to estimate the stress-strain characteristics of the ventricular wall. Calculated wall mechanics in any given subject could vary appreciably, depending on the method chosen to measure the dynamic geometry. Therefore, as investigators begin to explore the mechanics of ventricular function,23 it would be important to resolve the controversies noted above.

The present study was designed to measure precisely, in conscious and open-chested, anesthetized dogs, the three-dimensional dynamic geometry of the left ventricle, using recently developed ultrasonic methods. A thorough validation of the measurement technique was performed. Data presented in this communication resolve many previously conflicting hypotheses and explain, in part, the varied results that have been obtained in the past.
Methods

EXPERIMENTAL PREPARATION

Eight healthy adult mongrel dogs (23–31 kg) of both sexes were surgically prepared for subsequent chronic studies. Each dog was anesthetized with intravenous sodium pentobarbital (25 mg/kg) and ventilated with a Bennett MA-1 respirator. A left thoracotomy was performed under sterile conditions through the 5th intercostal space for implantation of pulse-transit ultrasonic dimension transducers on the heart (LTZ-5: resonant frequency, 5 MHz). The transducers were positioned so that the dynamic geometry of the left ventricle could be measured in three dimensions simultaneously (Fig. 1). Matched transducers were sutured to the anterior and posterior wall epicardium to obtain the maximum transverse external diameter in the plane of the minor axis circumference. Transducers were also implanted at the base and apex to measure the external major axis diameter. The apical transducer was sutured to the epicardium of the apical dimple; the base transducer was positioned in the groove between the left sinus of Valsalva and the left atrium, overlying the fibrous continuity between the aortic and mitral valves. Left ventricular anterior wall thickness was measured with the technique developed by Franklin et al.28

An 18-gauge needle was passed obliquely across the anterior wall so that the endocardium between the anterior papillary muscle and the septum was punctured as closely as possible to the anterior diameter transducer. An ultrasonic crystal (2 mm in diameter) was passed through the tract to the point where blood had returned from the needle. A slightly larger crystal was then sutured to the overlying epicardium. The ultrasonic signal amplitude was monitored continuously on an oscilloscope to assist in the sonic alignment of the transducers (Fig. 1).

During the procedure, silicone rubber balloon occluders were positioned around both venae cavae, and a 25-cm length of silicone rubber tubing (Dow Corning) was sutured to the pericardium near the epicardial surface of the left ventricle to permit passage of a micromanometer into the pleural cavity. In four of the dogs, Statham TTQ series electromagnetic flow transducers were implanted around the ascending aorta in addition to the usual instrumentation. The connectors from the dimension and flow transducers, the occluder tubing, and the end of the pleural catheter were implanted in a subcutaneous pouch at the dorsal aspect of the incision, and the thoracotomy was repaired. Postoperatively, the dogs were trained to lie quietly on their right sides on the fluoroscopic table. Intramuscular injections of procaine penicillin G (6 × 10⁴ U) and dihydrostreptomycin (0.75 g) were administered to each dog for 5 days after surgery.

INSTRUMENTATION AND DATA ACQUISITION

The sonomicrometer used in these experiments and the principles of pulse-transit sonomicrometry have been presented elsewhere.27 28 This system converts the transit time of a pulse of ultrasound between two piezoelectric transducers into an analog signal. Since the velocity of sound in body tissues and blood is constant, the measured transit time is directly proportional to the distance between the transducers. Calibrations of the in vivo measurements are obtained either by substituting an electronically generated time delay into the circuitry or by “ex vivo” crystal pairs mounted on a micrometer immersed in saline. The sampling rate of the sonomicrometer is 1 kHz, and the analog output signals are electronically filtered at 150 Hz. The individual channels are pulsed sequentially to eliminate cross-interference. The measured electronic drift is less than 0.05 mm/hour; the relationship between crystal separation and voltage output is linear over the ranges encountered in this study. The minimum resolution of the sonomicrometer-transducer system is approximately 0.05 mm.

Ascending aortic blood flow was measured with a Statham M-4001 gated sine-wave flowmeter. The performance characteristics of this flowmeter have been discussed in detail by McDonald.29 The flow transducers were calibrated before and after implantation with a gravity-fed, saline system. The calibration constants for each probe varied by less than ±6% from the mean value throughout these experiments. The calibration curves of the probes were linear to within ±2%.

In six of the eight dogs, dynamic dimension measurements were recorded at the conclusion of the implantation procedure in the open-chested, anesthetized state. These data were subsequently analyzed as described below and compared to measurements obtained during the awake study in each of these animals. In three additional dogs, dimension and pressure data were recorded in the open-chested state before and after the intravenous infusion of 1,500 ml of 0.9% saline.

Seven to 28 days after implantation, each dog was studied in the conscious state. Every dog was healthy and active at the time of study. One intravenous injection of morphine
sulfate (0.5 mg/kg) was administered when necessary for analgesia. Data acquisition was delayed for at least 1 hour when morphine was used. With the dog lying quietly on its right side on the fluoroscopic table, the subcutaneous pouch and the right groin were anesthetized locally with 1% lidocaine (Xylocaine). The dimension transducer connectors were exteriorized and directly coupled to the sonomicrometer; the flow probe leads were connected to the flowmeter. A Millar PC-350 micromanometer was introduced percutaneously into the right femoral artery and advanced into the left ventricle under fluoroscopic control. An identical manometer was passed into the pleural space through the implanted pleural catheter. An air-tight connector on the external end of the pleural catheter prevented pneumothorax. Both manometers had been zeroed to atmospheric pressure and simultaneously balanced and calibrated at 37°C.

The analog measurements of left ventricular minor axis diameter, wall thickness, major axis diameter, intracavitary pressure, pleural pressure, and aortic flow were recorded on magnetic tape with a Hewlett-Packard 3520-B FM recorder. Data were recorded with each dog in the resting state and during the course of three venous caval occlusions. Calibrations were recorded before and after data acquisition to ensure that significant drift had not occurred during the study. The pressure calibrations changed by less than 0.25 mm Hg, and the dimension calibrations changed by less than 0.05 mm in every study.

At the conclusion of the experiments the dogs were killed and autopsies were performed to verify the position of the ultrasonic transducers. The epicardial transducers were found to be properly seated, and the endocardial wall thickness transducer was within 1 mm of the endocardial surface in every dog. The myocardium of the left ventricle was examined for abnormalities. Other than a 0.5-mm capsule of fibrous tissue surrounding the endocardial crystal, the myocardium was normal in all studies. After excision of adjacent right ventricular muscle, valvular tissue, and fat, the left ventricle and septum were weighed. Left ventricular myocardial volume was measured by the displacement of saline in a volumetric cylinder, and myocardial density was calculated.

In four of the preparations, the heart was removed at autopsy with the transducers still in position. The left atrium was opened, and all mitral valvular tissue and chordae tendineae were excised. A Lucite disk with an attached thin-walled Latex balloon was sewn into the mitral annulus; the aortic annulus was occluded with a vascular clamp. After the balloon was filled with 100 ml of saline, a small catheter was inserted transmurally into the ventricle to evacuate any fluid or air that might prevent the balloon from conforming to the endocardial surface. Dimension measurements were then recorded as the balloon volume was decreased by 10-ml increments. These data were obtained within 30 minutes after the dogs were killed.

**DATA ANALYSIS**

The recorded physiological data were digitized at 5-msec intervals and smoothed once with a 100-Hz digital filter using an IBM 1130/System 7 computer. The geometry of the left ventricle was represented as a three-dimensional, prolate ellipsoidal shell. In all calculations, the measured minor and major axis diameters were assumed to represent the external diameters of the shell. The measured anterior wall thickness was used as the dynamic shell thickness at the minor axis circumference. The shell thickness at the base and apex was assumed to be 55% of the equatorial value. The ratio of 55% was based upon measurements made in our laboratory on six postmortem hearts and is similar to the values published by Streeter and Hanna. The dynamic internal volume ($V_i$) of the shell was computed using the formula for a prolate ellipsoid:

$$V_i = \frac{\pi}{6}(b - 2h)(a - 1.1h)$$

where $b$ is the external minor axis diameter, $h$ is the equatorial wall thickness, and $a$ is the external major axis diameter. The external shell volume ($V_e$) was calculated using the equation:

$$V_e = \frac{\pi}{6}(b^2a)$$

Stroke volume was computed as the change in the internal shell volume during ejection. Aortic flow was obtained from a least squares estimate of the first time derivative of the internal shell volume during ejection. Cardiac output (stroke volume x heart rate) and end-diastolic volume were normalized to the body surface area with the use of the equation:

$$\text{Surface area (m}^2) = 0.112 \sqrt{\text{weight, kg}^2}$$

The stroke volume was divided by the internal shell volume at the beginning of ejection to obtain the ejection fraction.

The dynamic volume of the ellipsoidal shell was estimated by comparing the difference between the external and internal volumes, and the shell mass was obtained as the shell volume x 1.06 g/cm³ (the average myocardial density determined in the eight hearts). The dynamic variation in the calculated shell mass throughout the cardiac cycle was computed in every study. The mean shell mass calculated in each of the eight hearts was compared to the left ventricular mass measured at autopsy using a paired t-test. The dynamic eccentricity ($e$) at the midwall of the shell was determined from the equation:

$$e = \sqrt{(a - 0.55h)^2 - (b - h)^2}$$

The transmural pressure of the left ventricle was computed as the difference between the intracavitary pressure and the pleural pressure.

Stroke volume was also calculated by integration of the electromagnetic aortic flow curves in four dogs assuming zero flow at end-diastole. In each dog, the peak flow and stroke volume measured with the flowmeter were compared to the same data calculated from the dimension measurements for several systoles during the course of a venous caval occlusion. The contribution of each dimension to volume displacement during a control ejection was evaluated in every dog by holding one dimension constant at the end-diastolic value and calculating the stroke volume while the other two dimensions varied normally. The change in the stroke volume from control was assumed to represent the contribution of the constant dimension to volume displace-
ment. In four postmortem hearts the static intracavitary balloon volumes were compared to the corresponding volumes calculated from the geometric measurements. Fourier analysis was performed on the dimension data from every study.

Results

RAW DATA

Typical analog recordings of the physiological data obtained during a control period in the conscious dog are shown in Figure 2. The phases of the cardiac cycle were defined from the left ventricular pressure and aortic flow curves. During the rapid-filling phase of diastole, both left ventricular diameters increased and the wall thinned. The rate of dimension change was always the greatest during the rapid-filling phase. Fifty percent of the diastolic dimension change usually occurred before the diastolic pressure minimum, and the major portion of the diastolic change in all three of the dimensions occurred during the rapid-filling period. During atrial systole, increases in the minor axis diameter were small, the wall thinned, and the major axis diameter decreased. During isovolumic contraction, the minor axis diameter shortened, the wall thickened slightly, and the major axis diameter lengthened. During ejection, the external minor axis diameter shortened by 7.3% to 0.3%, the external major axis diameter shortened by 4.7% ± 0.3%, and the wall thickened by 22.7% ± 2.3%. The dimension data for each dog are given in Table 1.

The hemodynamic measurements were very reproducible in the conscious dog (Table 2). The average end-diastolic transmural pressure was 13.7 mm Hg ± 0.8 mm Hg, and the average peak-systolic transmural pressure was 147.5 mm Hg ± 4.1 mm Hg. The mean calculated intracavitary volume at the beginning of ejection, normalized to the body surface area, was 68.2 ml/m² ± 1.7 ml/m². The mean ejection fraction was 42% ± 2% of the volume at the beginning of ejection, and the average cardiac index was 2.84 ± 0.18 liters/min per m².

VALIDATION PROCEDURES

The average variation in the calculated shell mass throughout the cardiac cycle was ±6% (Table 2). The average left ventricular mass measured at autopsy was 94.6 g ± 5.1 g, and the average mean calculated shell mass was 92.4 g ± 5.7 g. The mean calculated mass in each dog was not statistically different from the measured mass (P > 0.3). The average deviation of the mean calculated mass from the measured mass was 5.1% ± 1.6%.

The postmortem volume correlation data are illustrated in Figure 3. The volume calculated from the dimension measurements correlated well with the volume of saline within the intracavitary balloon (r = 0.97). In any given ventricle, the ultrasonic method tended to slightly overestimate the actual volume at low volumes and to underestimate the volume at high volumes. The phasic characteristics of the measured and calculated flow curves were virtually identical (Fig. 4A). The stroke volume measured with the flowmeter correlated closely (r = 0.96) with the stroke volume calculated from the dimension data over a wide range of values in four awake dogs (Fig. 4B). Likewise, the measured and calculated peak aortic flow rates (Fig. 4C) were similar (r = 0.95).

**Figure 2** Analog recordings of the most common geometric pattern observed in these studies.
The volume and mass calculations were most sensitive to errors in the wall thickness dimension (Fig. 5). A measurement error of +3 mm induced an error in the calculated volume of −29% and an error in the mass of +23%. Likewise, the striking sensitivity of the calculated stroke volume and ejection fraction to errors in the measurement of wall thickening is illustrated in Figure 6. Figure 7 is a representative Fourier analysis of the dimension data from one of the awake dogs. All three of the dimensions were composed of low frequency components.

**DYNAMIC GEOMETRIC PATTERNS**

The most common dynamic dimensional pattern that was observed in the resting, awake dogs is illustrated in Figure 2. The external minor axis diameter shortened and the major axis diameter lengthened during isovolumic contraction. In addition, the anterior ventricular wall thickened slightly during this period, and the global eccentricity increased, manifesting an isovolumic ellipticalization pattern. The isovolumic contraction patterns for each dog are given in Table 1. Under control, resting conditions an isovolumic ellipticalization pattern was observed in dogs 1–5. In dogs 6 and 7, the minor axis diameter lengthened, and the major axis diameter shortened during isovolumic contraction (isovolumic spherocylization pattern). In dog 8, both the minor axis and major axis diameters increased slightly during isovolumic contraction, with no change in the eccentricity. In general, the dimensional changes during isovolumic relaxation were opposite in direction from those observed during isovolumic contraction.

The geometry of the wall thickness dimension in the conscious state was more uniform from dog to dog. The anterior wall thickened slightly during isovolumic contraction and thinned during isovolumic relaxation in every dog. The thickening during isovolumic contraction was occasionally biphasic, first thickening and then thinning, but a small

**Table 2 Hemodynamic, Volume, and Mass Data Obtained during Control Conditions in the Conscious Dogs**

<table>
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<tr>
<th>Dog no</th>
<th>Wet kg</th>
<th>HR mm/min</th>
<th>EDP mm Hg</th>
<th>PSP mm Hg</th>
<th>Vae ml</th>
<th>Vae ml/m²</th>
<th>SV ml</th>
<th>EF % Vae</th>
<th>CO ml/min</th>
<th>CI liters/min per m²</th>
<th>Calc. mass g</th>
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Wt = body weight; HR = heart rate; EDP = end-diastolic transmural pressure; PSP = peak-systolic transmural pressure; Vae = internal volume at the beginning of ejection; SV = ejection phase stroke volume; EF = ejection fraction; CO = cardiac output obtained from the dimension data; CI = cardiac index; b, h, and a are the same as in Table 1.
degree of net isovolumic mural thickening (averaging 11.7% ± 3.1% of the total systolic thickening) was noted in every awake dog (Table 1).

The shortening of the major and minor axis diameters was not concentric, and the ventricles became more elliptical during ejection (Table 1). During diastolic filling, the ventricles became more spherical. Since the minor axis diameter increased, the wall thinned, and the major axis diameter decreased during atrial systole, the ventricles further sphericalized during this period.

On the average, wall thickening accounted for 46.9% ± 2.6% of the ejection-phase volume displacement, and external minor axis shortening accounted for 44.1% ± 2.3% under control conditions (Table 2). There was no significant difference between these two calculations (P > 0.5). Measured external major axis shortening contributed only 9.0% ± 0.7% to the volume displacement, and the difference from the other two dimensions was significant (P < 0.001). When the internal dimensions were analyzed, shortening of the minor axis internal diameter was by far the more important, accounting for 87% of the stroke volume.

In contrast to the geometric patterns observed in the awake studies, the dimensional characteristics in the open-chested, anesthetized state were quite different (Fig. 8). Both diameters were consistently smaller, and the wall was thicker during thoracotomy. Whereas the isovolumic geometric rearrangements were small and isovolumic contraction was fairly isometric in the conscious state, the isovolumic changes were large in the open-chested state. A spherencalization pattern during isovolumic contraction was usually present. Isovolumic thickening accounted for up to 50% of the total systolic wall thickening in the open-chested, anesthetized animals. The average left ventricular volume at the beginning of ejection during thoracotomy was 38.4 ml ± 2.6 ml. The average volume at the beginning of ejection calculated in the same dogs during the awake studies was 63.8 ml ± 2.6 ml, and the difference was statistically significant (P < 0.001). In the three open-chested infusion studies, the isovolumic contraction pattern gradually changed from spherencalization to ellipticalization and the degree of isovolumic wall thickening diminished as left ventricular volume increased.

Dimension measurements obtained during the course of a vena caval occlusion in the conscious state are shown in Figure 9. As the end-diastolic volume progressively fell, the isovolumic contraction pattern changed from one of ellipticalization to sphericalization. This phenomenon was noted...
FIGURE 5 Effect of measurement errors in each of the dimensions on calculated left ventricular volume and mass. Each dimension was varied individually by 1-mm increments, and the percentage change in the calculated volume and mass from control was computed.

in every dog that ellipticalized in the control state (dogs 1–5). The conversion was not always in phase, however, and both diameters frequently lengthened during isovolumic contraction at the point of transition (panel B). In the

remaining three ventricles (dogs 6–8), isovolumic sphericization became progressively more prominent as the end-diastolic volume diminished. The degree of isovolumic wall thickening also changed during the vena caval occlusion, becoming more pronounced at smaller ventricular volumes in every dog. In all of the ventricles, ellipticalization during isovolumic contraction was observed immediately after the release of the vena caval occluders when the end-diastolic volume exceeded the control value. After the vena caval release, the degree of isovolumic wall thickening was diminished in six dogs, and slight isovolumic thinning was noted in two. Thus, the tendency toward sphericization and wall thickening during isovolumic contraction seemed to be an inverse function of ventricular volume, to the point that during maximum vena caval occlusion (panel D), these isovolumic rearrangements in geometry were the major changes observed in each of the three dimensions.

Discussion

Many investigators, using a variety of techniques, have measured the dynamic geometry of the left ventricle. Some of the conflicting results that have been obtained in these studies may have been related in part to differences in methodology. There has always been some question about the degree to which direct methods of measurement (usually utilizing strain gauges) inherently modify the observed displacement,8–11,32 In addition, it is well accepted that ventricular function in open-chested animals may not be representative of the intact, physiological state.14–16 On the other hand, indirect methods of assessing dynamic cardiac geometry sacrifice resolution and fidelity.

The technique of measuring left ventricular dimensions used in the present study has several advantages. The implanted dimension transducers are not physically connected to each other and, therefore, do not influence the motion of the ventricular wall. Since the transducers are highly directional and rotational artifacts are minimal, one can be certain that a satisfactory signal represents a linear dimension.32 Accurate, high fidelity measurements are obtainable in the conscious animal for long periods of time after implantation. Drift, temperature sensitivity, and calibration errors are nonexistent, and the semicontinuous ana-
Figure 8. Dimensional characteristics of the left ventricle in the open-chested, anesthetized state (panel A) and in the same dog 1 week later in the conscious state (panel B).

Figure 9. Changes in left ventricular dynamic geometry, pressure, and ascending aortic flow produced by occluding both venae cavae in the conscious state. Panel A represents control conditions, panels B and C represent progressive phases in the venae caval occlusion, and panel D represents maximum venae caval occlusion.

Log data are easily processed. In the initial phases of the present study, the major uncertainty concerning this technique was the validity of using three simultaneously measured dimensions to represent the global geometry of the entire left ventricle. The volume, mass, and flow correlation experiments demonstrate the validity of this approach.

Sphericalization of the left ventricle during isovolumic contraction was first noted by Rushmer and subsequently has been confirmed by several investigators. However, other workers, utilizing different methodology, have observed shortening of the left ventricular minor semi-axis and lengthening of the major semi-axis during isovolumic con-
traction. Most of the former studies were performed in open-chested, anesthetized preparations and the latter on conscious subjects. The data in Figure 8 illustrate that, rather than methodological inconsistencies, these disparate observations actually reflect differences in the dynamic geometry during these two conditions. Thus, not only is ventricular volume diminished during thoracotomy, but the tendency toward spherocentricization of the ventricle during isovolumic contraction is more prominent.

The excellent experiments of Leshin et al. have previously demonstrated that the left ventricular wall is thicker and that the degree of systolic thickening is diminished in the open-chested state. These factors partially explain the smaller percentage of thickening that has been reported in several studies utilizing open-chested preparations. The data in Figure 8 demonstrate that the pattern of mural thickening also changes during thoracotomy. In the conscious state, minimal anterior wall thickening or thickening followed by slight thinning was observed during isovolumic contraction; net isovolumic thickening accounted for 12% of the total systolic thickening, a value similar to that previously obtained in closed-chested dogs. During thoracotomy, however, up to 50% of the total systolic wall thickening occurred during isovolumic contraction. This observation correlates well with previous studies in open-chested animals. Thus, the degree of wall thickening during isovolumic contraction is more pronounced in open-chested anesthetized preparations.

It appears that mural thickening and changes in the minor and major axis diameters during the isovolumic phases of systole are part of the same phenomenon. The pattern and extent of both were progressively altered during the course of a vena caval occlusion in the conscious state (Fig. 9) and by volume loading in the thoracotomized state. Thus, the tendency toward spherocentricization and wall thickening during isovolumic contraction, and toward ellipsoidalization and wall thinning during isovolumic relaxation, was more pronounced in either preparation when ventricular volume or the degree of mural deformation was diminished. These three-dimensional, isovolumic rearrangements in geometry have been attributed previously to asynchronous activation. Although this factor may have contributed somewhat to the rearrangements observed in the present study, it is unlikely that asynchronous activation was the primary cause, because the magnitude of the isovolumic dimensional changes seemed to be related to ventricular volume. This relationship, therefore, more likely reflects differences between the systolic and diastolic structural characteristics of the left ventricular wall, such as fiber orientation or stress distribution.

Three of the ventricles did not manifest ellipsoidalization during isovolumic contraction under control conditions. The fact that these ventricles did ellipsoidalize when the end-diastolic volume was increased after the vena caval release suggests that these dogs may have been slightly hypovolemic or that intrinsic differences existed between the ventricles. During the vena caval occlusion, the transition from isovolumic ellipsoidalization to isovolumic spherocentricization was not clear-cut in most of the dogs, and at the transitional volume both diameters frequently would increase slightly and the wall would thicken during isovolumic contraction (Fig. 9B). One dog manifested this pattern in the control state. Of course, these geometric changes would be impossible in a true prolate ellipsoidal shell, and this pattern probably reflects the limitations of the ellipsoidal model in approximating the geometry of the left ventricle. However, the general hypothesis presented above should be valid despite this slight inconsistency.

In the present study, the degree of ejection phase shortening of the internal minor and major axis diameters averaged 20.3% ± 1.0% and 8.7% ± 0.4%, respectively, in the conscious state. Shortening of the internal minor axis diameter accounted for 87% of the stroke volume. These data are similar to those previously obtained in closed-chested dogs. The degree of ejection-phase mural thickening in the awake dogs averaged 22.7% ± 2.3% and the overall systolic thickening was 29.0% ± 3.4%. These findings compare favorably with previous observations in closed-chested preparations. The eccentricity of the left ventricle in the conscious dog (Table 1) was considerably more spherical than that previously calculated in postmortem hearts.

It is now well accepted that contrast ventriculography overestimates the extent of systolic mural thickening. This phenomenon probably accounts for the larger percentage changes in wall thickness that have been reported with the use of this technique. Unfortunately, this one factor severely limits the precision of data obtained by contrast ventriculography, because the accurate quantification of left ventricular geometry is most sensitive to errors in the wall thickness dimension (Fig. 5). The sensitivity of the calculated ejection fraction to errors in measured wall thickening (Fig. 6) probably explains the generally higher ejection fractions obtained with ventriculography as compared to those determined in the present study or in previous experiments utilizing direct geometric measurements.

Further analysis of the ventricular dimension data showed that the highest frequency dimension was wall thickness. However, all of the dimension data were relatively low frequency signals and could be approximated by the first five harmonics of the basic frequency (Fig. 7). These findings are similar to those obtained by Vayo using data acquired with mercury-in-Silastic strain gauges. High fidelity dimension measurements, therefore, are not essential in the analysis of dynamic left ventricular geometry.

Thus, the seemingly inconsistent results of previous experiments can be explained, at least partially, by differences in the experimental preparations used. When left ventricular volume is diminished either in the open-chested or the conscious dog, geometric rearrangements during isovolumic contraction, manifested by mural thickening and global spherocentricization, are more prominent. These phenomena may reflect transitional stages from a diastolic to a systolic fiber orientation. In addition, it is evident that the most critical dimension for the accurate definition of left ventricular geometry is wall thickness. These findings may be useful in assessing future studies of the dynamic geometry of the left ventricle.
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