Dimensional Changes of the Human Left Ventricle Prior to Aortic Valve Opening

A Cineangiographic Study in Patients with and without Left Heart Disease

By Joel S. Karliner, M.D., Richard J. Bouchard, M.D.,
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SUMMARY

Previous studies of the dynamic geometry of the left ventricle have yielded conflicting results concerning shape changes during the preejection period. Accordingly, left ventricular dimensional changes prior to aortic valve opening in man were analyzed using high-speed biplane cineradiograms exposed in the frontal and lateral projections. In each projection the long axis and three chords perpendicular to it were measured. In six patients without left ventricular disease there was a mean decrease in equatorial diameter of approximately 1 mm before aortic valve opening (P < 0.05), without significant change in the long axis, causing an apparent volume decrease of 4.0 ml or 2.8% of end-diastolic volume (EDV). In five patients with wall motion disorders secondary to coronary artery disease the equatorial diameter decreased by an average of 1.5 mm and volume was diminished by 8.3 ml or 3.9% of EDV. In four of seven patients with primary myocardial disease, an increase in the equatorial diameter and a basal chord occurred, while the apical chord decreased, suggesting nonhomogeneous myocardial involvement. In eight patients with mitral regurgitation, the reduction in equatorial diameter averaged 2.8 mm and volume decreased by 16.7 ml or 8.0% of EDV. In normal patients the occurrence of circumferential fiber shortening prior to aortic valve opening under basal conditions can result in as much as a 9% underestimation of contractile element velocity calculated from dp/dt, whereas in patients with mitral regurgitation this figure may be as high as 31%. These studies indicate that, in man, expansion at the minor equator during the preejection period occurs only under highly abnormal conditions. They further suggest that the reductions in shape and volume prior to aortic valve opening may be significant relative to mechanical analyses of this phase of contraction.

Additional Indexing Words:
Cineangiography  Contractile element velocity  Dimensional changes  dp/dt
Isometric contraction  Isovolumic contraction  Mitral regurgitation
Preejection period  Primary myocardial disease  Shape changes
Wall motion disorder

Previous studies in animals of the dynamic geometry of the left ventricle utilizing external dimension gauges, endocardial markers, and video or sonar methods, have yielded conflicting results concerning shape changes during isovolumic contraction.1-4 In addition, recent attempts to apply the principles of muscle mechanics to the analysis of left ventricular function5,6 have focused attention upon the need for detailed...
Table 1

<table>
<thead>
<tr>
<th></th>
<th>Mean values</th>
<th>Normal patients (6)</th>
<th>Wall motion disorder (5)</th>
<th>Myocardial disease (7)</th>
<th>Mitral regurgitation (9)</th>
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</thead>
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<tr>
<td>Age (yr)</td>
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<td>50.4</td>
<td>38.7</td>
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<td>39–61</td>
<td>17–69</td>
<td>37–68</td>
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</tr>
<tr>
<td>CI (liters/min/m²)</td>
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<td>2.49</td>
<td>2.83</td>
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<tr>
<td>Arterial pressure (S/D) (mmHg)</td>
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<td>145/84</td>
<td>139/76</td>
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<td>16.6</td>
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<td>6–11</td>
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<td>4–30</td>
<td>5–29</td>
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<tr>
<td>SV/EDV (%)</td>
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<td>41</td>
<td>45</td>
<td>47</td>
<td></td>
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<tr>
<td>EDV (ml/m²)</td>
<td>80.4</td>
<td>115.8</td>
<td>84.3</td>
<td>140.8</td>
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<td>Range</td>
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<td>92.6–131.7</td>
<td>52.3–119.8</td>
<td>59.7–290.2</td>
<td></td>
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<tr>
<td>Duration of prejection period (msec)</td>
<td>48</td>
<td>55</td>
<td>61</td>
<td>61</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>35–60</td>
<td>41–85</td>
<td>42–136</td>
<td>41–78</td>
<td></td>
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<tr>
<td>Regurgitant volume (ml)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>10.3–101.4</td>
<td></td>
</tr>
<tr>
<td>Regurgitant fraction (%)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>34.2</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CI = cardiac index; LVEDP = left ventricular end-diastolic pressure; EDV = end-diastolic volume; SV = stroke volume.
information in man concerning ventricular geometry and dimensional changes during contraction, particularly during the period prior to aortic valve opening. With the development of high-speed biplane cineangiography, it has now become possible to obtain precise measurements of ventricular chamber dimensions in man in two projections during a single contraction. This technique was employed in the present study to characterize left ventricular geometry during the isovolumic phase of contraction in patients with normal and abnormal ventricular function.

**Methods**

Twenty-seven patients were studied during diagnostic cardiac catheterization. The first group consisted of six patients who were considered to have normal left ventricular (LV) function on the basis of resting hemodynamic measurements (table 1): LV end-diastolic pressure <12 mm Hg; cardiac index >2.5 liters/min/m²; end-diastolic volume <100 ml/m²; and ejection fraction >0.56. These patients included one with pure mitral stenosis, one with moderate aortic stenosis (peak systolic transvalvular aortic pressure difference 42 mm Hg), two patients with functional cardiac murmurs who were studied to rule out aortic stenosis, one patient with angina pectoris without myocardial infarction, and one patient with atypical chest pain.

The second group included five patients with coronary artery disease and previous myocardial infarction with segmental wall motion abnormalities. No patients were included in this group in whom mitral regurgitation was observed in the angiocardiogram.

A third group consisted of seven patients with left ventricular disease, one of whom had associated severe aortic valve stenosis. Five of these seven subjects had coronary arteriograms, all of which were normal. The remaining two patients, aged 17 and 32 years, respectively, who did not undergo coronary arteriography, were thought clinically to have idiopathic myocardial disease. All the patients in this group had impaired left ventricular performance, evidenced either by a reduced ejection fraction or by an abnormal instantaneous velocity of circumferential fiber shortening determined at maximum intraventricular wall tension. The latter method relies upon frame-by-frame analysis of the cineangiogram for determination of sequential changes in the minor equatorial circumference.9

Finally, eight patients with mitral regurgitation, and one patient with a ventricular septal defect, comprised a fourth group.

Patients were studied in the postabsorptive state after receiving sodium pentobarbital 100 mg intramuscularly. A Courmand needle was placed in the left brachial artery, and left heart catheterization was performed by the retrograde arterial or transseptal technique. The method of study was as follows: the patient was positioned in the biplane cine field and measurements of LV and brachial arterial pressure and cardiac output were made. Biplane cineangiograms were then exposed at 80 frames/sec (22 patients) or 150-200 frames/sec (five patients) in the frontal and lateral projections following intraventricular injection of 75% sodium diatrizoate (Hypaque), 1 ml/kg, over a 2- to 3-sec period. The instant of each cineangiographic exposure was recorded with the ECG and arterial pressure pulse to permit precise correlation of dimensional changes with electrical and pressure events (fig. 1).

End-diastole was identified 0.04-0.06 sec following the onset of the QRS complex, corresponding to an incisura on the LV pressure pulse recorded immediately prior to cineangiography. In most patients the occurrence of aortic valve opening could be readily identified on the cineangiogram by observation of valve leaflet motion. The time required for left ventricular

![Figure 1](http://circ.ahajournals.org/)

The instant of each cineradiographic exposure in the frontal and lateral projections is recorded, in this case at 150 frames/sec. The numbered cine pulses are shown. Simultaneous tracings of the ECG and the brachial arterial pressure pulse permit precise correlation of dimensional changes with electrical and pressure events.

Abbreviations: LAT = lateral projection; AP = anteroposterior projection; ED = end-diastole; AVO = aortic valve opening.
The LV silhouette is outlined at end-diastole in a patient with left ventricular myocardial disease in the frontal and lateral projections. The long axis of the left ventricle (L) is drawn in the frontal projection from the midpoint of the aortic valve plane to the apex, and in the lateral projection from the midpoint of the mitral valve plane to the apex. The lettered chords are perpendicular to and quadrisect the long axis in each plane.

Left ventricular cavity silhouettes were drawn in duplicate in both frontal and lateral projections over two successive cardiac cycles. Only cineangiograms of uniformly high quality were selected for study (fig. 2-4). In instances in which the outer edge of the left ventricular cavity silhouette was irregular due to trabeculations, the border formed by connecting these excrescences defined the left ventricular margin. A long axis of the left ventricle was constructed in the frontal projection.

The dotted lines define the LV cavity borders at end-diastole in a patient with rheumatic mitral regurgitation. The frames depicted occurred just prior to aortic valve opening. The marked shape and volume changes are apparent. The left atrium (LA) has been opacified by contrast material.
from the midpoint of the aortic valve plane to the apex, and in the lateral projection from the midpoint of the mitral valve plane to the apex. Three chords were then constructed perpendicular to the long axis in each plane to quadrisection the long axis (fig. 2). All dimensions were corrected for X-ray magnification and spherical distortion by means of a grid composed of 1 cm wire squares embedded in Lucite. After the patient left the catheterization laboratory, the grid was positioned and filmed in such a way that the X-ray tube-to-grid distance and the distance between the grid and the image-intensifier input phosphor corresponded precisely to the distance between these two pieces of apparatus and the left ventricular midplane in both the frontal and lateral projections. The cineradiograph of this grid was used to correct for magnification and distortion in each projection in each patient (fig. 5).

Generally, the magnification factor was approximately twofold for linear determinations. After correction for magnification linear measurements were reproducible in duplicate determinations within an average of 0.55 mm or 0.89% of total chord length (range 0.44 to 0.61 mm or 0.74 to 1.1%). Volume estimates were made using the area-length method and were reproducible in duplicate determinations within an average of 1.9 ml or 0.97% of control volumes (range 0.5 to 2.5 ml or 0.46 to 1.24%).

The X-ray apparatus consisted of two 3-phase, 1000-ma, 150-kv generators and two 9-inch image intensifiers equipped with 35-mm cine cameras run at 80 frames/sec. The X-ray exposure varied with the size of the patient but generally ranged between 90 and 110 kv and 100 to 150 ma per frame. The X-ray was pulsed, yielding an exposure time of 2–3 msec per frame. The total length of the cineangiograms averaged 10 sec (800 frames), and the total dosage to the skin of the chest of the average patient was 100 rads per study.

**Figure 4**

The beat following a premature ventricular contraction in a patient with a wall motion disorder secondary to coronary artery disease is depicted. The LV cavity margin at end-diastole is outlined just prior to aortic valve opening. No mitral regurgitation is present, and the marked shape change is apparent.

**Figure 5**

Cineradiograph of a grid, comprised of 1 cm wire squares embedded in Lucite, which was used to correct for magnification and distortion in each projection in each patient.
## Table 2

**Dimension Changes during the Preejection Period**

<table>
<thead>
<tr>
<th></th>
<th>Normal patients (6)</th>
<th>Wall motion disorder (5)</th>
<th>Myocardial disease (7)</th>
<th>Mitral regurgitation (9)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean Δ</td>
<td>Range</td>
<td>Mean % Δ</td>
<td>Mean Δ</td>
</tr>
<tr>
<td><strong>Volume (ml)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.98</td>
<td>0.1–8.8</td>
<td>2.77†</td>
<td>8.33</td>
</tr>
<tr>
<td><strong>AP projection (mm)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AB</td>
<td>1.97</td>
<td>0.27–5.19</td>
<td>3.50†</td>
<td>0.89</td>
</tr>
<tr>
<td>CD</td>
<td>0.83 (±)0.82–1.87</td>
<td>1.55†</td>
<td>1.46</td>
<td>0.3–3.0</td>
</tr>
<tr>
<td>EF</td>
<td>0.78 (±)0.14–2.17</td>
<td>2.07†</td>
<td>0.32 (±)0.56–0.9</td>
<td>0.43 (±)0.46</td>
</tr>
<tr>
<td>L</td>
<td>(+)0.38 (±)2.4–2.72</td>
<td>(+)0.66</td>
<td>0.16 (±)2.12–1.96</td>
<td>0.25</td>
</tr>
<tr>
<td><strong>LAT projection (mm)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AB</td>
<td>1.05</td>
<td>0.57–1.76</td>
<td>1.98†</td>
<td>2.53</td>
</tr>
<tr>
<td>CD</td>
<td>1.65</td>
<td>0.68–2.70</td>
<td>3.20†</td>
<td>1.96</td>
</tr>
<tr>
<td>EF</td>
<td>0.89 (±)1.04–3.52</td>
<td>2.10</td>
<td>0.54 (±)2.62–3.39</td>
<td>0.41</td>
</tr>
<tr>
<td>L</td>
<td>(+)0.05 (±)1.55–1.17</td>
<td>0.17 (±)0.64</td>
<td>(+)2.5–0.37</td>
<td>(+)0.84</td>
</tr>
</tbody>
</table>

Abbreviations: Δ = change.
*All numbers are negative unless otherwise noted.
†P = 0.05.
‡P = 0.01.
For the cineangiograms performed at 150–200 frames/sec, the above equipment was utilized, except that a 16-mm high-speed cine camera replaced the 35-mm camera. The ranges for current and voltage were similar to those in the 80 frame/sec studies. The pulse duration was 1.5 msec per frame, yielding a total dose to the skin of the chest of 15 rads per study.

All beats analyzed represented ventricular contractions originating from normal electrical depolarization, and none was preceded by extrasystoles. A separate analysis of contractions immediately following a ventricular extrasystole was made in three patients without LV dysfunction in order to examine the effects of this spontaneously occurring positive inotropic influence.

In patients with mitral regurgitation, the cardiac output and stroke volume were determined by the indicator dilution method immediately prior to the cineangiogram. The total stroke volume, calculated from the cineangiogram as the difference between the end-diastolic and the end-systolic volumes, includes both the volume ejected into the aorta ("forward" stroke volume) as well as the volume which empties into the left atrium. The difference between the "forward" stroke volume, derived from the indicator dilution method, and the total stroke volume, calculated from the cineangiogram, and corrected for any difference in heart rate occurring between the two procedures, is the regurgitant volume. The ratio of the regurgitant volume to the total stroke volume defines the regurgitant fraction.

Results

Left ventricular dimensional changes in the four groups of patients during the interval from the onset of contraction to aortic valve opening are shown in table 2. In patients with normal left ventricular function there was a small, directionally consistent decrement in each of the measured chords in both the frontal and lateral projections, while the long axis was unchanged. These shape changes were accompanied by a calculated volume decrease prior to the onset of ejection averaging 4 ml or 2.8% of end-diastolic volume.

In the five patients with a wall motion disorder due to previous myocardial infarction, similar dimensional changes occurred, producing an apparent mean volume decrement prior to aortic valve opening of 8.33 ml or 3.9% of end-diastolic volume.

Of the seven patients with myocardial disease, an increase in equatorial diameter occurred in two patients in both the frontal and lateral projections, while in two patients disparate changes in the frontal and lateral equatorial diameters occurred, the diameter increasing in the frontal projection and decreasing in the lateral projection. The basal chord increased in these four patients in both projections, while the apical chord diminished in the frontal projection in two of these subjects, and in the lateral projection in the other two patients. These changes were not accompanied by a significant change in ventricular length. In these four patients there was an average volume increment of 2.5 ml or 1.9% of end-diastolic volume. In the remaining three patients with myocardial disease, the chords decreased in the normal fashion prior to aortic valve opening, with an apparent mean volume decrement of 8.8 ml or 5.5% of end-diastolic volume.
In patients with mitral regurgitation the changes in chord lengths were directionally similar to those of the normal patients but of greater magnitude, the equatorial diameter in the frontal plane decreasing by an average of 3.9% (range 2.1 to 7.0%). The mean regurgitant volume in the eight patients with mitral regurgitation was 40.8 ml. Of this amount an average of 32% (range 14 to 63%) of the total regurgitant volume occurred prior to aortic valve opening. A rough correlation ($r = 0.64$) was observed between the amount of regurgitation occurring prior to aortic valve opening (corrected for the apparent volume decrement observed in normal patients without mitral regurgitation) and the total amount of blood regurgitated per beat (regurgitant fraction) (fig. 6).

In order to assess the effect of a positive inotropic event on ventricular geometry during the preejection period, dimensional changes were examined prior to and after spontaneously occurring ventricular premature contractions in three subjects without left ventricular disease (table 3). These changes were similar to, but of greater magnitude than those occurring in normal beats, the equatorial diameter in the frontal projection decreasing by 3.43 mm or 6.0% (range 2.3 to 12.1%), corresponding to an apparent volume decrement of 13.4 ml, or 9.2% of end-diastolic volume.

**Discussion**

Published reports of shape changes during isovolumic contraction contain a variety of conflicting findings. Previous investigations have indicated that during isovolumic systole the canine left ventricle assumes a more spherical shape due to an abrupt expansion of the internal diameter, external circumference, and external length.$^1,^5,^11$ Others have found inconstant changes in measurements of left ventricular dimensions during isovolumic contraction.$^{12,13}$ Using endocardial marker techniques$^2$ and biplane videometry,$^4$ a number of investigators recently have suggested that a decrease in internal dimensions rather than an increment is the major directional change accompanying isovolumic systole in the dog. Continuous measurement of internal left ventricular diameter in awake dogs using sonimicrometer techniques has provided convincing evidence that during isovolumic contraction this dimension decreases.$^6$ Pieper attributed the discrepancy between internal and external diameter measurements during

---

**Table 3**

<table>
<thead>
<tr>
<th>$\Delta AB$</th>
<th>$\Delta CD$</th>
<th>$\Delta EF$</th>
<th>$\Delta L$</th>
</tr>
</thead>
<tbody>
<tr>
<td>mm</td>
<td>% $\Delta$</td>
<td>mm</td>
<td>% $\Delta$</td>
</tr>
<tr>
<td>Frontal</td>
<td></td>
<td></td>
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<tr>
<td>N</td>
<td>1.75</td>
<td>3.23</td>
<td>0.90</td>
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<tr>
<td>Post-PVC</td>
<td>2.29</td>
<td>4.23</td>
<td>3.43</td>
</tr>
<tr>
<td>Difference</td>
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<td>1.0</td>
<td>2.53</td>
</tr>
<tr>
<td>Lateral</td>
<td></td>
<td></td>
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<tr>
<td>N</td>
<td>0.98</td>
<td>1.87</td>
<td>1.69</td>
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<tr>
<td>Post-PVC</td>
<td>2.57</td>
<td>4.5</td>
<td>2.46</td>
</tr>
<tr>
<td>Difference</td>
<td>1.59</td>
<td>2.63</td>
<td>0.77</td>
</tr>
</tbody>
</table>

Abbreviations: N = mean of three normal patients between end-diastole and aortic valve opening; Post-PVC = mean of three post-PVC beats occurring in the same three patients between end-diastole and aortic valve opening.

*All numbers are negative unless otherwise indicated.
isovolumic systole to ventricular wall thickening. More recently, McDonald, in a cineangiographic study, has indicated that in man the mean diameter of the left ventricular body shortens before ejection.

The present study demonstrates that in both normal and abnormal human left ventricles there are small but characteristic shape changes which occur prior to aortic valve opening. With the exception of some patients with primary myocardial disease, the three transverse chords measured in both the frontal and lateral projections decrease in length before aortic valve opening. These shape changes are accompanied by an apparent decrease in ventricular volume, which presumably results from alterations in the area and radii used to calculate ventricular volume by the area-length method. We believe this volume “loss” in patients without mitral regurgitation probably can be accounted for by posterior displacement of the mitral valve, which is known to occur prior to aortic valve opening. Bishop et al. also postulated that this factor could account for a decrease in internal transverse diameter before aortic valve opening.

A number of investigators have suggested that with papillary muscle contraction there is an internal translocation of intraventricular volume from the apical region to the basal part of the left ventricular cavity. Except for the patients with myocardial disease, none of the subjects demonstrated an increase in the basal chord AB (see table 2), suggesting that any internal translocation of volume which occurs prior to aortic valve opening is toward the mitral valve rather than toward the area just below the aortic valve.

It was of interest that in patients with mitral regurgitation an average of 32% of the total regurgitant volume occurred before aortic valve opening. In these patients the average prejection period was 61 msec, or only 19% of the total electromechanical time during systole. Although the pressure difference between the left ventricle and left atrium is greater after aortic valve opening, a relatively large proportion of regurgitant volume occurs dur-

ing the short time prior to aortic ejection. Factors which might account for this phenomenon include left ventricular fiber orientation during shortening, which favors propulsion of blood across the aortic valve, and changes in the compliance of the left atrium as regurgitant volume enters this chamber.

Although none of the patients with myocardial disease had electrocardiographic evidence of bundle-branch block, the increase in dimensions prior to aortic valve opening in some of these patients suggests an asynchronous pattern of contraction. The finding of an increase in dimensions did not correlate with cardiac index, left ventricular end-diastolic pressure, and volume or ejection fraction. Performing a similar dimensional analysis in the right anterior oblique projection in ten patients with Bantu cardiomyopathy, Chambers et al. published graphs suggesting that in at least three of their patients increases in one or more chords during isovolumic systole also occurred. In the absence of ECG evidence of an abnormal pattern of depolarization, the abnormal sequence of contraction in some patients with idiopathic myocardial disease may be due to nonuniformity of cardiac muscle involvement, and indicates that this abnormality need not be specific for coronary heart disease. In the present study asynchronous contraction prior to aortic valve opening was not observed in the five patients with a wall motion disorder secondary to coronary artery disease. However, the observations in this small sample do not preclude the existence in other patients with coronary heart disease of nonhomogeneous contraction prior to aortic valve opening, analogous to left ventricular asynergy during ejection, as described by others.

It has been demonstrated in animal studies that complex alterations in cardiac performance occur after injection of contrast material into left heart chambers. These include alterations in left ventricular contractile force as well as changes in systemic vascular resistance. However, it has recently been documented that in man myocardial contractility is little affected during the first few
beats after injection of contrast medium, especially in patients with reduced left ventricular performance. Since early beats were chosen for examination in this study, it is unlikely that the contrast material itself significantly influenced the results obtained.

These observations have theoretical implications relative to estimates of contractile element velocity based on measurements of dp/dt. These estimates have assumed that no fiber shortening occurs during isovolumic contraction, i.e., that contractile element velocity can be assumed equal to the rate of series elastic extension. However, our data demonstrate that even the small degree of circumferential fiber shortening, averaging 1 mm in 50 msec or 2 cm/sec, which occurs in patients with normal left ventricular function, can alter contractile element velocity calculated from dp/dt by at least 9% (see appendix). Although it is well recognized that measurements of contractile element velocity cannot be made in the presence of mitral regurgitation because of the absence of isovolumic contraction, the potential error in the calculation of contractile element velocity derived from dp/dt in such patients has not previously been estimated. In the present study this error averaged 21% and may be as high as 31%. These data suggest that the rate of circumferential fiber shortening prior to aortic valve opening should be included in calculations on contractile element velocity which are derived from the rate of left ventricular pressure change.

Appendix

Contractile element velocity \((V_{CE})\) is the sum of the instantaneous velocity of circumferential fiber shortening \((V_{CF})\) and the velocity of the series elastic element \((V_{SE})\). Urschel et al. have proposed that \((V_{SE})\) can be calculated by the following formula:\(^{25}\)

\[
V_{SE} = \left( R_i + h/2 \right) \times \left[ 0.234 \frac{(dp/dt)}{P} \right] - \frac{Q}{56 R_i^3} - \frac{Q}{56 R_o^2} + \frac{Q}{56 R_o^2}
\]

where \(R_i = \) internal radius; \(R_o = \) external radius; \(h = \) LV wall thickness; \(P = \) pressure at which the aortic valve opens; and \(Q = \) flow.

For normal subjects assuming dp/dt = 1800 mm Hg/sec, \(P = 50 \) mm Hg, \(R_i = 2.5 \) cm, \(h = 0.8 \) cm, and \(Q = 4 \) ml in 50 msec or 80 ml/sec, \(V_{SE} = 22.7 \) cm/sec, and \(V_{CE} = 22.7 + 2 = 24.7 \) cm/sec. For patients with mitral regurgitation, assuming dp/dt = 1500 mm Hg/sec, \(P = 50 \) mm Hg, \(R_i = 3.0 \) cm, \(h = 1.0 \) cm, and \(Q = 16 \) ml in 50 msec or 320 ml/sec, \(V_{SE} = 21.8 \) cm/sec, and \(V_{CE} = 21.8 + 6 = 27.8 \) cm/sec. Hence, in normal patients and in subjects with mitral regurgitation, \(V_{SE}\) differs by less than 1 cm/sec. The difference in \(V_{CE}\) is produced by the difference in magnitude of \(V_{CF}\); i.e., the rate of circumferential shortening prior to aortic valve opening, which makes up a much greater proportion of \(V_{CE}\) in patients with mitral regurgitation than in normal subjects. It must be recognized that the number assigned to \(V_{CF}\) in the above calculations represents a mean value, and that instantaneous \(V_{CF}\) may well be considerably higher.

Acknowledgment

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