The Influence of Left Ventricular Stroke Volume on Aortic Root Motion

An Echocardiographic Study

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SUMMARY Aortic root motion was studied in 24 normal volunteers at rest and during the Valsalva maneuver, isometric exercise, and amyl nitrite inhalation. In addition root motion was correlated with the stroke volumes determined at cardiac catheterization in 24 patients. The root has distinct systolic movement, the amplitude and duration of which were easily measured both at rest and during the interventions.

At rest, the mean (±1 se) systolic amplitude of the anterior aortic wall was 11.2 ± 0.5 mm and that of the posterior wall 9.5 ± 0.3 mm. During the strain phase of the Valsalva maneuver anterior wall amplitude fell to 8.2 ± 0.4 mm and the posterior wall to 7.3 ± 0.5 mm (P < 0.001). With release, anterior wall amplitude rebounded to 12.5 ± 0.8 mm and the posterior wall to 10.8 ± 0.5 mm, values greater than control (P < 0.01). With isometric exercise there was no change in amplitude compared to rest; however, amyl nitrite caused an increase in the anterior wall to 13.5 ± 0.8 mm and posterior wall to 11.9 ± 0.6 mm (P < 0.01).

In the patient group the amplitude of posterior wall motion correlated weakly with cardiac index (r = 0.63) and stronger with stroke index (r = 0.78).

This study quantifies the echocardiographic pattern of normal aortic root motion. The findings indicate that the aortic root motion is an index of stroke volume; they further suggest that root motion is acutely sensitive to variations in stroke volume since its amplitude changed in accord with the documented effects of the employed maneuvers on stroke volume.

THERE HAS BEEN CONSIDERABLE INTEREST in using echocardiographic measurements to quantify left ventricular function. In addition to direct systolic and diastolic chamber dimensions, indirect assessments from mitral valve and posterior wall motion have been felt to reflect left ventricular function. Despite the fact that the aortic root receives ventricular outflow directly, little attention has been paid to its motion as a potential index of ventricular performance. The amplitude of aortic root excursions varies considerably in patient populations and it is our impression that this is directly related to ventricular performance (unpublished observations).

To test the sensitivity of aortic root motion to changes in left ventricular outflow, we measured its motion in normal volunteers at rest and during a series of maneuvers that have differing effects on left ventricular hemodynamics. In addition, root motion was correlated to stroke volume in a series of patients undergoing cardiac catheterization.

Methods

The first part of this study was carried out in normal volunteers. Informed consent was obtained from each individual after detailed explanation of the procedures.

Strip chart echocardiograms were made on an Electronics for Medicine multichannel recorder interfaced with a Smith Kline Ekoline 20 echocardiographic instrument as described previously. The electrocardiogram (lead II) was recorded simultaneously. Blood pressure was recorded by the cuff method (Infrasound model 3000 sphygmomanometer). Records were made at a paper speed of 75 mm/sec with time lines every 0.04 sec. The ultrasound transducer (SK1 model C-10), with a frequency of 2.25 MHz, was placed along the left sternal border in the third or fourth intercostal space and directed superiorly and medially to define the aortic root. Care was taken to include elements of aortic leaflets within the root. This served as a reference point for localization in the same manner that mitral elements are used in determining left ventricular dimensions. After initial recognition, the image of the aortic root was magnified to facilitate subsequent measurements. Transducer position and gain utilized at rest were maintained unchanged during each intervention.

The Valsalva maneuver was performed by blowing a mercury column to a height of 40 mm and maintaining it for 20 seconds. The subjects were instructed to perform the maneuver near end expiration to minimize the air interface between the transducer and the heart. Echocardiographic recordings were made at rest, and then continuously from onset of the strain phase of the Valsalva maneuver to 20 seconds after the onset of the release phase. Blood pressure and heart rates were measured at rest and 15 and 40 seconds after the start of the Valsalva maneuver.

Isometric exercise was accomplished by using a hand grip dynamometer as described by Fisher et al. Subjects were asked to maintain 2/3 of their maximum hand grip for 45 seconds. Instruction was given and each subject observed to prevent performance of the Valsalva maneuver during the isometric procedure. Echocardiographic recordings were made at rest, and for 10 second intervals at 15, 40 and 60 seconds after onset of the procedure. Simultaneous blood pressure and heart rates were determined at these times.

The amyl nitrite procedure was performed by having the subject take three deep inhalations from a freshly broken vial of amyl nitrite. Echocardiographic recordings were done at rest and continuously from onset of inhalation until heart rate and BP had returned to resting values. The entire response usually lasted 45 to 90 seconds. Blood pressures were recorded at rest and at 15, 45 and 90 seconds.

A rest period of 5–10 minutes was allowed between in-
tentions. Study sessions were concluded after amyl nitrite inhalation.

The second phase of this study was conducted in a consecutive series of patients undergoing cardiac catheterization for suspected heart disease. Root motion at rest was correlated with the resting cardiac output determined by the Fick technique. All output determinations were confirmed independently by the green dye method (Gilford Model 140 cardiac output system).

Figure 1A shows a magnified recording of the aortic root, and figure 1B shows the parameters measured in schematic form. The amplitude of anterior motion of both walls of the aortic root during systole was measured from initial to peak forward motion (fig. 1B). The duration of the anterior movement was determined using the same points. All measurements were performed for five consecutive cycles during the rest phase and at the specified periods during each intervention. Data were processed according to standard statistical formulae using a Digital PDP-8 computer.

Results

A) Normal Volunteers

Twenty-four subjects were studied, 12 men and 12 women. The age range of the men was 23 to 39 (mean 32 years), and that of the women 22 to 43 (mean 30 years). The mean responses for the hemodynamic variables and aortic root motion during each aspect of the study are compiled in table 1. The effect of each maneuver on aortic root amplitude is further illustrated in figure 2. Figure 3 shows the influence of each intervention on the duration of systolic anterior motion.

The anterior and posterior aortic walls moved in a generally parallel fashion during systole and diastole. The anterior wall, however, had a greater anterior excursion than the posterior wall at rest and in all maneuvers (fig. 4).

Rest: Characteristically, the aortic root moved anteriorly during systole (fig. 1A). This motion began close to the opening and ended near the closing of the aortic valve. The root then moved posteriorly, and at resting heart rates entered a plateau phase which terminated in a presystolic dip, which was noted in all of the study subjects. The downslope of the dip showed a short, abrupt anterior motion in 20 of the 24 subjects, thus rendering it bifid in contour. Its lowest point always occurred after this slight anterior motion. The dip began an average of 95 msec after the P wave and the nadir of its trough occurred 46 msec after the Q wave. With increasing heart rates and shortening of diastole the plateau period became progressively shorter; at rates of 120 or greater the plateau disappeared. The presystolic dip was less well delineated during tachycardia because of the absence of the diastolic plateau. There was no difficulty in obtaining satisfactory recordings of the aortic root during the resting state in any of the participants.

Valsalva Maneuver

Figure 5 shows samples of the aortic root during rest, the strain phase, and the release phase of the Valsalva maneuver. During the strain phase (figs. 2, 5B) there was a consistent decrease in amplitude of both the anterior and posterior walls in all subjects. The mean amplitude of the anterior wall decreased from 11.2 mm at rest to 8.2 mm, and the posterior wall from 9.5 mm at rest to 7.3 mm during the strain phase.

Table 1. Mean Response +1 s.e. to Physiologic and Pharmacologic Interventions in 24 Subjects

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>V8</th>
<th>V8</th>
<th>I10</th>
<th>I10</th>
<th>I10</th>
<th>AN15</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>77 ± 3</td>
<td>95 ± 4</td>
<td>68 ± 3</td>
<td>95 ± 3</td>
<td>98 ± 4</td>
<td>75 ± 3</td>
<td>117 ± 4</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>115 ± 2</td>
<td>119 ± 6</td>
<td>122 ± 3</td>
<td>125 ± 2</td>
<td>138 ± 3</td>
<td>114 ± 2</td>
<td>87 ± 2</td>
</tr>
<tr>
<td>(systolic/diastolic)</td>
<td>65 ± 3</td>
<td>81 ± 2</td>
<td>70 ± 2</td>
<td>80 ± 2</td>
<td>89 ± 2</td>
<td>68 ± 1</td>
<td>44 ± 1</td>
</tr>
<tr>
<td>Anterior aortic wall amplitude (mm)</td>
<td>11.2 ± 0.5</td>
<td>8.2 ± 0.4</td>
<td>12.5 ± 0.8</td>
<td>11.5 ± 0.4</td>
<td>11.6 ± 0.5</td>
<td>11.1 ± 0.4</td>
<td>13.5 ± 0.8</td>
</tr>
<tr>
<td>Posterior aortic wall amplitude (mm)</td>
<td>9.5 ± 0.3</td>
<td>7.3 ± 0.5</td>
<td>10.8 ± 0.5</td>
<td>10.1 ± 0.3</td>
<td>10.3 ± 0.4</td>
<td>10.1 ± 0.4</td>
<td>11.9 ± 0.6</td>
</tr>
<tr>
<td>Anterior duration (msec)</td>
<td>312 ± 5</td>
<td>275 ± 8</td>
<td>291 ± 5</td>
<td>294 ± 6</td>
<td>294 ± 6</td>
<td>304 ± 10</td>
<td>288 ± 4</td>
</tr>
<tr>
<td>Posterior duration (msec)</td>
<td>315 ± 7</td>
<td>267 ± 17</td>
<td>291 ± 5</td>
<td>295 ± 6</td>
<td>295 ± 7</td>
<td>307 ± 8</td>
<td>282 ± 6</td>
</tr>
</tbody>
</table>

Abbreviations: V8 = at 10 seconds of Valsalva strain phase; V8 = immediately after release of Valsalva maneuver; I10 = at 15 seconds of isometric exercise; I10 = at 40 seconds of isometric exercise; I10 = 60 seconds after isometric exercise was begun (15 seconds after cessation of handgrip); AN15 = 15 seconds after amyl nitrite inhalation.
typifies the response in the strain and release phases of the Valsalva maneuver.

**Isometrics**

Figure 7 shows a recording of the aortic root during isometric exercise. While the heart rate and systemic blood pressure increased (table 1), there was essentially no change in the amplitude of the aortic root motion. The duration of anterior motion of both walls shortened, but not as much as it did during the Valsalva maneuver or amyl nitrite inhalation (fig. 3). Satisfactory recordings were made in all subjects. However, in one subject only the posterior wall was adequate for measurement.

**Amyl Nitrite Maneuver**

Figure 8 shows a representative aortic root recording during amyl nitrite inhalation. With this intervention there was a significant increase in heart rate when blood pressure fell. There was also an increase in the amplitude of both the anterior (mean 13.5 mm) and posterior (mean 11.9 mm) wall motion (P < 0.01). All subjects showed this heart rate and amplitude increase along with the fall in blood pressure. The duration of anterior systolic motion shortened to values comparable to those seen during the strain phase of the Valsalva maneuver (fig. 3).

**B) Patients with Heart Disease**

Twenty-four patients, all men, were studied. Their age range was 24 to 62 (mean 49.7). Twelve patients had coronary artery disease, five were normal, three had severe aortic stenosis, two had cardiomyopathy, one had coarctation of
the aorta, and one had mitral stenosis. In each individual the resting cardiac output determined by the Fick method agreed (±10%) with that determined by green dye. The mean heart rate was 68.1 beats/min during the echocardiographic studies and 68.0 at cardiac catheterization. The mean duration between the echocardiographic study and elective cardiac catheterization was 4.8 days.

Since the motion of the posterior aortic wall was most consistently recorded in the first part of this study, its amplitude was correlated with cardiac flow parameters. This is summarized in Table 2. The strongest correlations were between aortic root amplitude and stroke volume and stroke index. The relationship of aortic root amplitude to stroke index in the entire patient population is plotted in Figure 9.

**Discussion**

This study establishes the pattern and range of movement of the aortic root in healthy adults. This consists of a distinct anterior systolic excursion which was readily measured in terms of both its amplitude and its duration at rest and during certain physiologic and pharmacologic interventions. While perhaps less apparent, there is also characteristic diastolic motion.

During systole, the anterior and posterior aortic walls move in a generally parallel fashion; however, the anterior wall consistently has a greater amplitude. A tenable explanation for this difference is that the aortic root expands circumferentially as it is thrust forward in systole. The result is greater anterior movement of the anterior wall since circumferential expansion adds to the net forward motion of the anterior aortic root while it reduces somewhat the anterior travel of the posterior wall.

In diastole at rest there is a triphasic motion composed of (a) an initial posterior movement after the aortic valve closes, followed by (b) a plateau period and finally (c) a presystolic dip. This dip was present in all of our subjects, in twenty of whom it showed a bifid contour. It has been noticeably absent in patients we have examined in atrial fibrillation (unpublished observations). This observation coupled with its relationship to the electrocardiographic P wave leads us to believe that the presystolic posterior motion is produced by atrial systole. The aortic root is thus tugged posteriorly as the atria contract. The explanation of the bifid contour of the dip is not explained by these studies; it may represent the beginning of isovolumic contraction or possibly the separate contributions of right and left atrial systole.

The motion of the aortic root bears a remarkable

![Figure 5](http://circ.ahajournals.org/)

**Figure 5.** Segments from a recording of the aortic root during the Valsalva maneuver. A) Aortic root and left atrium at rest. Arrow illustrates amplitude of anterior motion of posterior aortic wall. B) and C) show the aortic root and left atrium during the strain and release phase of the Valsalva maneuver, respectively. Time lines .04 sec; 1 cm depth lines indicated at left margin.

![Figure 6](http://circ.ahajournals.org/)

**Figure 6.** A continuous recording of the entire Valsalva maneuver at a paper speed of 10 mm/sec. Posterior aortic wall (Ao-PW) amplitude diminishes progressively during strain and overshoots at release. Note the concomitant decrease in left atrial (LA) size. Time lines indicate 1 second.
qualitative similarity to early reports of echoes from the mitral annulus in normal subjects.\textsuperscript{13, 14} In these studies the amplitude of mitral ring excursion tended to vary with stroke volume.\textsuperscript{13} These observations, combined with ours, suggest that the entire base of the heart exhibits comparable motion during the cardiac cycle.

In a study designed primarily to examine aortic valve disease, Gramiak et al. included measurements of posterior aortic root amplitude in 45 normal subjects. Our resting value of 9.5 mm for this measurement is similar to the 10.4 mm in their series. Measurements of anterior aortic root motion, duration of systolic excursions and correlation with cardiac output were not reported in that study.\textsuperscript{6}

A clearly significant relationship of the amplitude of aortic root motion to stroke volume is shown in the measurements obtained from patients undergoing cardiac catheterization. Until further data are obtained, however, it cannot be concluded that aortic root motion is solely dependent on stroke volume. While stroke volume clearly plays an important role, other factors such as the compliance of the aortic root and the initial velocity of left ventricular ejection, may also contribute to root motion.

The maneuvers to which the normal volunteers were subjected also demonstrate that aortic root amplitude is sensitive to interventions which can acutely alter stroke volume. The Valsalva maneuver causes an increase in heart rate and a decrease in both blood pressure and stroke volume during the strain phase.\textsuperscript{15} Sharpey-Schafer has shown that the blood pressure response to the strain phase is essentially a sigmoidal curve; that is, the blood pressure actually rises with the onset of the strain phase, later to fall and then to rise again before the onset of the release phase. After the beginning of the release phase there is a rebound of both blood pressure and stroke volume which exceeds resting values and leads to a reflex bradycardia.\textsuperscript{10} We have demonstrated a similar response in amplitude of the aortic root motion to this maneuver.

Isometric exercise increases the double product, but has little or no effect on stroke volume.\textsuperscript{16, 17} When our subjects performed isometric handgrip exercise, the amplitude of the aortic root was essentially unaffected while, at the same time, there was a considerable increase in both heart rate and blood pressure.

There is some debate as to how much stroke volume is increased by amyl nitrite. Investigators who have studied human subjects during amyl nitrite inhalation have shown a

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**Figure 7.** Segments of recording of aortic root during isometric exercise. Format as in figure 5. A) Shows the aortic root and left atrium at rest and B) these structures after 15 seconds of isometric exercise. C) Shows the same structures at 60 seconds, 15 seconds after completion of the isometric maneuver.

**Figure 8.** Segments of recording of aortic root during amyl nitrite. Format as in figure 5. B and C show the aortic root and left atrium 15 and 90 seconds after amyl nitrite inhalation.
modest (on the order of 10%) increase of stroke volume.18, 19 These studies used techniques for measuring stroke volume that assume a steady state, which is difficult to achieve during acute nitrite administration. Canine studies, in which beat-to-beat changes were followed with intra-aortic flow meters, showed an increase in stroke volume between 5 and 80 seconds after nitrite administration with a maximal increase of 23% at 30 seconds.20 With amyl nitrite inhalation, our subjects all showed an increase in the amplitude of the aortic root motion, while blood pressure fell simultaneously and heart rate increased.

Since systolic amplitude measurements were made at virtually identical points in time (fig. 1), the divergence and convergence of the anterior and posterior walls at peak systole with each of the maneuvers reflects changes in aortic diameter. Thus systolic diameter was smallest during the Valsalva strain phase and largest after Valsalva release and with amyl nitrite (fig. 2). However, it was early recognized that diameter variations were not as extensive as changes in absolute motion (fig. 2), hence these measurements were not pursued in detail.

The above maneuvers also influenced the duration of anterior systolic motion to varying extents. As expected anterior motion became shorter with increasing heart rates, but the degree of shortening was related to the maneuver performed. Thus, it was most abbreviated by the Valsalva maneuver and least affected by isometrics (fig. 3). These findings are consistent with the effects of these interventions on left ventricular ejection time.21, 22 Thus, the ejection period shortens with the acute afterload imposed by isometrics, and considerably more with the decreased peripheral resistance seen with nitrates.23 Since ejection times were not measured simultaneously in these studies a quantitative relationship of the duration of aortic root motion to ejection times cannot be made on the basis of our data.

Aortic root motion offers some advantages as a supplementary index of left ventricular function. The aortic root is easy to image and measure, which is certainly not true of all ultrasonically-defined structures. Since root motion is a response to the action of the entire ventricle, it would not appear to be subject to the errors that local contraction abnormalities produce when assessing stroke volume from left ventricular dimensions. Moreover, root motion is sensitive to acute changes in left ventricular hemodynamics making it potentially useful in following unstable patients.

Acknowledgment

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References

Echocardiographic Studies of Abnormalities Associated with Coarctation of the Aorta

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SUMMARY Echocardiograms were performed in thirty-six patients (aged 4 to 36 years) with proven coarctation of the aorta. Nineteen patients (53%) were found to have marked diastolic eccentricities of their aortic valves (Eccentricity Index > 1.5), indicating the presence of bicuspid aortic valves. One of these patients also had multilayered aortic root echoes in diastole. Five patients had angiographic proof of their aortic valve morphologies which corroborated the echo findings. Five patients with bicuspid aortic valves showed mitral valve diastolic flutter indicative of aortic regurgitation. Idiopathic hypertrophic subaortic stenosis (IHSS) was suspected in four patients (11%) with abnormal systolic anterior motion of the mitral valve; three of these patients also had asymmetric septal hypertrophy. There was catheterization proof of IHSS in one patient. Two patients (5.6%) demonstrated mitral valve prolapse.

COARCTATION OF THE AORTA is commonly associated with other congenital anomalies.1 4 Sometimes these anomalies may not be suspected on the basis of physical examination alone. Echocardiography has become a valuable technique in that it allows the diagnosis of certain cardiac anomalies to be made noninvasively without risk to the patient. This paper describes the echocardiographic features of some cardiac anomalies associated with coarctation of the aorta.

Materials and Methods

Thirty-six patients with coarctation of the aorta were studied by echocardiography. They represent the total number of patients seen in our laboratory with aortic coarctation and echocardiographic examinations. Fourteen patients had preoperative studies only and fifteen had only postoperative echocardiograms. The remaining seven patients had both pre and postoperative echo evaluations. The patients ranged in age from 4 to 36 years; there were 21 males and 15 females.

All echocardiograms were obtained using a commercially available ultrasonoscope (Picker Model 103) and a 2.0 MHz transducer. A Tektronics 565 dual beam oscilloscope was operated as a slave and displayed the ultrasonic data in a B mode on the upper beam. The lower beam was used in multi-trace operation to record the electrocardiogram. Continuous recordings were made on 35 mm film using an oscilloscope record camera. Film transport speed was selected to equal 125 mm/sec as obtained by ordinary strip chart recorders.

Mitrval valve echograms were obtained by placing the transducer in the third or fourth left intercostal space and directing the beam posteriorly and slightly medially. Aortic root echoes were obtained by medial and cephalic rotation of the transducer from the mitral valve position. Echoes from the left ventricular cavity, ventricular septum, and the posterior wall were all obtained by shifting the ultrasonic beam inferiorly and laterally so that it passed just below the mitral valve.

The diagnosis of a bicuspid aortic valve was made using the eccentricity index of the aortic valve cusps in diastole. Our laboratory has previously reported6 the computation of this index as follows: one-half the width of the aortic lumen at the beginning of diastole (L/2) is divided by the minimum distance between the diastolic cusp position and the closest aortic margin (d) (fig. 1). An eccentricity index of 1.5 or more was considered diagnostic of a bicuspid aortic valve. Multilayered echoes in the aortic root, a feature commonly present with bicuspid aortic valves, were also noted. Multilayering is probably due to valve thickening or redundant folds on the valve cusps in the absence of valvular calcification.8

Aortic insufficiency was diagnosed echocardiographically by the presence of typical fine fluttering motion of the mitral valve leaflets in diastole.8

The presence of abnormal systolic anterior movements (SAMs) of the anterior mitral valve leaflet and asymmetric septal hypertrophy (ASH) of the type seen in idiopathic
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