Diagnostic Methods

Doppler Echocardiography

Noninvasive estimation of valve area in patients with aortic stenosis by Doppler ultrasound and two-dimensional echocardiography

Terje Skjaerpe, M.D., Lars Hegrenaes, M.D., and Liv Hatle, M.D.

Abstract

In 30 patients with aortic stenosis, 14 of whom also had significant aortic regurgitation, the velocities in the stenotic jet (V') and below the valve (V) were recorded by Doppler ultrasound. With two-dimensional echocardiography, two subvalvular areas (A) were calculated from leading-to-leading edge ("large") and trailing-to-leading edge ("inner") diameter measurements. The aortic valve area was calculated by the equation of continuity (A' = A × peak V/peak V') and by calculating stroke volume below the valve [A × integral of V(t)] and dividing by the integral of V'(t) (= A''). Based on cardiac output estimations from single-plane angiographic images, Gorlin’s formula was used to calculate invasive valve areas. In patients with no or mild aortic regurgitation a second invasive estimate was based on cardiac output measured by the Fick method. The best correlation was found when A' (with "large" diameter) was compared with invasive results based on cardiac output measured by the Fick method (r = .89, SEE ± 0.12, n = 16); the worst was found when A'' (with "large" diameter) was compared with invasive results based on cardiac output measurements by single-plane angiography (r = .80, SEE ± 0.20, n = 30). The results indicate that valve area in patients with aortic stenosis can be reliably estimated noninvasively, even in those with significant aortic regurgitation.


Because the pressure drop across a stenotic valve depends both on the effective valve area and the flow across the valve, the information it offers about the severity of the obstruction is not complete. The quantitation of valvular stenosis therefore includes an estimation of the valve area.

Ideally a method for the estimation of a valve area should be independent of valve insufficiency, of diseases of other valves, and of ventricular performance. If the valve area varies during the antegrade flow period, the method should give an estimate of the effective area. The method should also be noninvasive to allow frequent follow-up of the patient. So far the various methods described to estimate the valve area in patients with aortic stenosis only partly fulfill these criteria, either because the methods are invasive or semi-invasive, or because they rely on the absence of significant aortic regurgitation or mitral regurgitation.

In this work we evaluated a completely noninvasive method designed to be accurate despite any coexisting regurgitation.

Methods

Theoretical considerations. When there is an obstruction in a flow channel, the product of flow area and flow velocity will be constant at the obstructed and the nonobstructed part of the flow channel, according to the equation of continuity:

\[ A \times V = A' \times V' \]

(1)

where V and A are the spatial mean velocity and the area proximal to the obstruction, and V' and A' are the spatial mean velocity and the area at the obstruction. If V, A, and V' are known, the stenotic area can be calculated by rearranging equation 1 as follows:

\[ A' = A \times V/V' \]

(2)

This equation is valid both in time-steady flow and at each moment during pulsatile flow, provided that the velocity profile does not change from A to A'. It is also assumed that A is constant. If A' is elastic and varies because of the changing pressure drop across the obstruction during pulsatile flow, the size of the valve area calculated by equation 2 will depend on when V and V' are recorded. If peak V and V' are recorded, it can probably be assumed that the maximal A' is calculated, since the pressure drop is highest at this moment, forcing the valve to open maximally.

There are two ways of calculating mean A'. One is to estimate the area at frequent intervals during one flow period and...
calculate a temporal mean area (mean A'). Equation 2 must then be modified to:

\[ \text{Mean } A' = A \times \frac{\text{int}(V(t)/V'(t))}{t} \]  

where \( \text{int}(V(t)/V'(t)) \) is \( V/V' \) integrated over one cycle and \( t \) is the time of one flow period (see Appendix for how equation 3 is developed). This equation involves the laborious procedure of measuring \( V \) and \( V' \) at frequent corresponding intervals on two velocity curves and integrating the quotients.

The other way is to calculate an effective area of \( A'' (A'') \) by estimating volume flow through \( A \) and \( A' \), which must be equal:

\[ A \times \text{int}(V(t)) = A'' \times \text{int}(V'(t)) \]  

where \( \text{int}(V(t)) \) and \( \text{int}(V'(t)) \) are the integrals of the velocity curves recorded at \( A \) and \( A' \). The effective valve area is obtained by rearranging equation 4 into:

\[ A'' = A \times \frac{\text{int}(V(t))}{\text{int}(V'(t))} \]  

The effective valve area is equivalent to a constant area that lets the same volume of flow pass through it when the pressure drop and the duration of flow are identical. The integrals of the velocity curves are easily obtained with a computer and a digitizing tablet or with a mechanical planimeter. The left side of equation 4, when applied to the left ventricular outflow tract (LVOT), defines the stroke volume of the heart, which is an important advantage.

When the velocities proximal to the obstruction are recorded, it must be realized that they are influenced by the obstruction. According to fluid mechanics, dead water zones are formed around a central, tapering core of flow with increasing velocities (figure 1). Because of the corresponding peaking of the velocity profile, recordings representative of the cross-sectional mean velocity should be made proximal to this level. In our experience, the increase in velocities in the LVOT, caused by the valvular stenosis, starts about 0.5 to 1.5 cm proximal to the valve. This is in agreement with the results of another report. Recent experience has shown that the velocity curve of the jet in aortic stenosis can be recorded in almost all patients and that cardiac output can be measured with echocardiography and Doppler ultrasound by recording the diameter and the velocities in the upper LVOT in patients with normal aortic valves. This was the background for the present attempt to apply equations 2 and 5 to estimate the valve area in patients with aortic stenosis.

Patients. Thirty-six consecutive patients were studied. Informed consent was obtained from all. Six were excluded, four because the cardiac output or the pressure drop was not obtained as a result of problems experienced during catheterization, and two because they had significant aortic and mitral regurgitation, precluding calculation of valve area from Gorlin's formula. None were excluded because of failure in obtaining the echocardiographic and Doppler measurements. Of the remaining 30 patients, 10 were women and 20 were men, ranging in age from 38 to 76 years (mean 63.1).

Twenty-one patients were examined with echocardiography/Doppler within 2 days of the catheterization, and three within 4 days. Six patients were examined just before replacement of the valve, and the interval from the time of catheterization ranged from 27 to 87 days (mean 60). None had clinical signs indicating increasing severity of the aortic stenosis during this period.

By angiographic criteria for regurgitation, nine patients had mild (+) aortic regurgitation, nine had moderate (+ +), two had moderate to severe (+ + +), and three had severe (+ + + +). Eight patients had mild (+) mitral regurgitation. Three patients had atrial fibrillation.

Diameter measurements. For the subvalvular diameter measurements we used an Irex III B (Irex Corp.) 3.5 MHz phased-array or an ATL mark III (Advanced Technology Laboratories) 3 MHz mechanical sector scanner. The LVOT and the aortic root were visualized in the parasternal long-axis view, using the intercostal space from which the clearest image of these structures was obtained (figure 2).

At least five copies of the two-dimensional image were recorded. The transducer was repositioned between each recording. The diameter was measured preferably in systole, but sometimes measurements were made in late diastole because the upper part of the LVOT was clearly visualized only in this part of the cardiac cycle. Despite careful adjustment of gain, the presence of calcium deposits in the wall of the LVOT might cause some blooming of the echoes. Therefore, on each image two diameters were measured, one “large,” obtained from leading edge measurements, and one “inner” diameter measured from the trailing edge of the anterior echo to the leading edge of the posterior echo. Because the leading edge of the anterior echo was often difficult to define due to the calcium deposits, the “large” diameter was not allowed to exceed the “inner” diameter by more than 2 mm. If the diameters measured on different recordings varied in size, the largest obtained in at least two

![FIGURE 1. When there is an obstruction of a flow channel, an increase in velocities proximal to the obstruction (level B) will be observed. If, as in the LVOT, the flow proximal to the obstruction is accelerated, the velocity profile at level A is flattened. Velocities representing the spatial mean velocity should therefore be recorded at this level.](http://circ.ahajournals.org/content/images/1.17.png)

![FIGURE 2. The diameter is measured just below the aortic valve to avoid any influence of the systolic thickening of the septum and movement of the anterior mitral valve leaflet. LV = left ventricle; Ao = aorta.](http://circ.ahajournals.org/content/images/2.17.png)
recordings was chosen for the subsequent calculations. The cross-sectional area was calculated by assuming a circular shape of the distal part of the LVOT.

**Velocity recordings.** The velocity recordings were obtained with the Irex III B, which includes a 2 MHz Doppler instrument (Pedof, Vingmed), allowing pulsed- or continuous-wave Doppler measurements to be made simultaneously with imaging. Velocity recordings with a separate, optimized Doppler transducer were also possible.

The subvalvular velocities were recorded from the apex with pulsed-wave Doppler. The image and the audio signal were used to align the ultrasound beam to the direction of flow. The positioning of the sample volume was made as follows: When the sample volume was moved out of the LVOT, gradually increasing velocities were recorded until a much steeper rise occurred 0.5 to 1.5 cm proximal to the valve (figure 3). The corresponding increase in pitch of the audio signal was easily heard. The sample volume was placed just proximal to this level.

The velocities in the stenotic jet were recorded with continuous-wave Doppler. A meticulous search for the highest velocities was made by recording from the apex, along the right sternal border, from the suprasternal notch, and sometimes also from the right and left supraclavicular regions. The separate Doppler transducer was used because the combined echocardiographic/Doppler transducer — even in Doppler-only mode — may fail to display the highest velocities because of suboptimal sensitivity. Typical recordings from the LVOT and the stenotic jet are shown in figure 4.

The peak and the mean pressure drops were calculated as described by Hegrenaes and Hatle.54 Integration of the velocity curves was made with a digitizing tablet and a computer (Cardio 80, Kontron AG) or with a mechanical planimeter (Corasenior, G. Coradi AG). The subvalvular integrals and peak velocities were averaged over 5 beats in sinus rhythm and over 10 beats in atrial fibrillation. In some patients it was difficult to obtain optimal velocity recordings from the stenotic jet in more than a few beats, and the integrals and peak velocities of the recordings from the jet were averaged over only 3 beats. Beats giving the largest integrals were selected, provided that the heart rate was stable. In three patients with atrial fibrillation it was possible to average the integral over 10 consecutive beats. The opening and closing of the aortic valve was identified to ensure that diastolic velocities were not included.

The valve area was calculated from both equations 2 and 5.

Two estimates were obtained from each equation by the two diameters measured echocardiographically. The subvalvular integral was multiplied with the cross-sectional area of the LVOT and the heart rate to give an estimate of total cardiac output (effective cardiac output + regurgitant volume).

In eight patients the velocities in the LVOT were recorded at multiple levels, and the differences in integral size and peak velocity from one level to the next were calculated.

**Catheterization.** The patients received a mild sedative 1 hr before catheterization; otherwise there was no change in medication between the noninvasive and invasive measurements.

The pressures were recorded with a fluid-filled No. 7F pigtail catheter connected to an Elema 746 pressure transducer. The pressure difference was calculated by superimposing pressure recordings obtained by pullback of the catheter from the left ventricle to the ascending aorta. Beats with comparable RR

**FIGURE 3.** Velocities recorded with pulsed Doppler in the distal part of the LVOT. The aortic valve is located at about 10.7 cm from the transducer (the level showing maximal aliasing of the velocities). In this patient, steeply increasing velocities were recorded from 9.5 cm. The velocities recorded at 9.0 cm were therefore chosen as representing the spatial mean.

**FIGURE 4.** Representative subvalvular (A) and valvular (B) velocity recordings. The paper was run at a speed of 100 mm/sec to increase the accuracy when the curves were integrated.
DIAGNOSTIC METHODS—DOPPLER ECHOCARDIOGRAPHY

TABLE 1
Catheterization data*

<table>
<thead>
<tr>
<th>Patients</th>
<th>Pressure drop (mm Hg)</th>
<th>Cardiac output (l/min)</th>
<th>Aortic valve area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Peak</td>
<td>Mean</td>
<td>F</td>
</tr>
<tr>
<td>All (n = 30)</td>
<td>89 ± 31</td>
<td>62 ± 22</td>
<td>5.5 ± 1.9</td>
</tr>
<tr>
<td>With no or mild AI (n = 16)</td>
<td>90 ± 35</td>
<td>64 ± 24</td>
<td>5.4 ± 1.6</td>
</tr>
<tr>
<td>With &gt; mild AI (n = 14)</td>
<td>88 ± 29</td>
<td>60 ± 20</td>
<td>5.6 ± 2.3</td>
</tr>
<tr>
<td>Doppler &lt; 4 days from cath. (n = 24)</td>
<td>91 ± 32</td>
<td>64 ± 23</td>
<td>5.7 ± 2.0</td>
</tr>
<tr>
<td>Doppler &gt; 4 days from cath. (n = 6)</td>
<td>84 ± 32</td>
<td>54 ± 16</td>
<td>4.8 ± 1.4</td>
</tr>
</tbody>
</table>

F = Fick method; A = single-plane angiography; G = Gorlin’s formula; AI = aortic regurgitation.

*In groups including patients with more than mild aortic regurgitation, only the valve areas for which the calculations are based on single-plane angiographic estimates of flow are shown. Values presented as mean ± 1 SD.

Results

There were no statistically significant differences in heart rate (p > .05) and systolic and diastolic blood pressure (p > .1 and p > .5) between the noninvasive and invasive examinations.

The invasive results are presented in table 1 and the noninvasive results in table 2. The “large” diameter was 2.2 ± 0.3 cm and the “inner” diameter was 2.0 ± 0.3 cm (mean ± SD). In patients with no or mild aortic regurgitation two invasive estimates of the valve area were calculated, one with cardiac output measured by single-plane angiographic images and one with measurements obtained by the Fick method, assuming that the regurgitant volume in patients with mild aortic regurgitation is within the error of the Fick method. This is supported by the findings in figure 5, where cardiac output measurements by the two methods are compared. There is no tendency for the Fick method to underestimate flow across the valve in patients with mild aortic regurgitation when compared with single-plane angiographic measurements. The same assumption was made about the regurgitant volume in patients with mild mitral regurgitation when total cardiac output was quantitated from single-plane angiographic images.

Table 3 shows the correlations obtained when measurements of pressure drops, cardiac output, and valve areas were compared. The rather poor correlation between noninvasive and invasive measurements of flow might be explained by the time interval of 1 to 3 months between the two measurements in some patients. Therefore, noninvasive measurements of cardiac output were also compared with invasive measurements (Fick) in a subgroup of patients (patients with no or mild aortic regurgitation, examined with echocardiography/Doppler within 4 days of catheterization).

TABLE 2
Noninvasive results*

<table>
<thead>
<tr>
<th>Patients</th>
<th>Pressure drop (mm Hg)</th>
<th>Cardiac output (l/min)</th>
<th>Aortic valve area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Peak</td>
<td>Mean</td>
<td>D/Ei</td>
</tr>
<tr>
<td>All</td>
<td>92 ± 32</td>
<td>61 ± 23</td>
<td>5.9 ± 1.8</td>
</tr>
<tr>
<td>With no or mild AI</td>
<td>92 ± 35</td>
<td>64 ± 25</td>
<td>4.9 ± 1.3</td>
</tr>
<tr>
<td>With &gt; mild AI</td>
<td>92 ± 30</td>
<td>58 ± 20</td>
<td>7.0 ± 1.7</td>
</tr>
<tr>
<td>Doppler &lt; 4 days from cath.</td>
<td>93 ± 33</td>
<td>62 ± 24</td>
<td>5.9 ± 1.9</td>
</tr>
<tr>
<td>Doppler &gt; 4 days from cath.</td>
<td>88 ± 30</td>
<td>58 ± 21</td>
<td>5.7 ± 1.5</td>
</tr>
</tbody>
</table>

D/Ei = Doppler/echo measurements, “large” diameter used; D/Ei = Doppler/echo, “inner” diameter used; A’ = valve area calculated by equation 5, “large” (El) and “inner” (Ei) diameter; A’ = valve area calculated by equation 2, same diameters.

*Values presented as mean ± 1 SD.
FIGURE 5. Effective cardiac output measurements obtained by the Fick method compared with total cardiac output measurements from single-plane angiographic images. Symbols represent patients with the following degrees of aortic regurgitation: • = no aortic regurgitation; ▲ = mild; ○ = moderate; △ = moderate to severe; □ = severe. Data points marked with an asterisk represent patients who also had mild mitral regurgitation.

Figures 6 to 9 graphically present comparisons between noninvasive and invasive estimates of valve areas. The noninvasive areas were calculated with the "large" diameter. There is a tendency for the larger valve areas to be underestimated, especially when calculated by equation 5. When the "inner" diameter was used, this tendency was more pronounced, affecting also smaller valve areas (table 3).

The valve areas calculated from equations 2 and 5 are compared in figure 10. The largest difference between the estimates in one patient was 37% (0.41 cm²).

In five patients a second diameter measurement was made on another occasion. In two of the diameter was identical to the first measurement, in two there was a difference of 1 mm, and in one there was a difference of 2 mm.

In the eight patients in whom the velocities were recorded at several levels in the LVOT, it was decided which was the last level to show only a gradual increase in velocities and which was the first level to definitely indicate the steeper rise. Peak velocities and velocity integrals were measured from recordings made at these two levels. Differences in peak velocity and integral size in paired recordings ranged from 5% to 14% (mean 9%).

TABLE 3
Comparison of pressure drops, cardiac output, and valve areas

<table>
<thead>
<tr>
<th>Comparisons</th>
<th>r</th>
<th>p</th>
<th>Regression equation</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure drops</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak</td>
<td>.93</td>
<td>&lt;.001</td>
<td>y = 0.94x + 7.60</td>
<td>11.75 30</td>
</tr>
<tr>
<td>Mean</td>
<td>.92</td>
<td>&lt;.001</td>
<td>y = 0.95x + 2.48</td>
<td>9.23 30</td>
</tr>
<tr>
<td>Cardiac output</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D/Ei-A</td>
<td>.70</td>
<td>&lt;.001</td>
<td>y = 0.52x + 2.53</td>
<td>1.29 30</td>
</tr>
<tr>
<td>D/Ei-A</td>
<td>.71</td>
<td>&lt;.001</td>
<td>y = 0.46x + 1.99</td>
<td>1.13 30</td>
</tr>
<tr>
<td>D/Ei-F</td>
<td>.46</td>
<td>&lt;.1</td>
<td>y = 0.37x + 2.93</td>
<td>1.12 16</td>
</tr>
<tr>
<td>D/Ei-F</td>
<td>.45</td>
<td>&lt;.1</td>
<td>y = 0.32x + 2.52</td>
<td>0.99 16</td>
</tr>
<tr>
<td>A-F</td>
<td>.49</td>
<td>&lt;.1</td>
<td>y = 0.34x + 3.89</td>
<td>1.39 16</td>
</tr>
<tr>
<td>D/Ei-F^3</td>
<td>.71</td>
<td>&lt;.01</td>
<td>y = 0.54x + 1.76</td>
<td>0.88 12</td>
</tr>
<tr>
<td>D/Ei-F^3</td>
<td>.69</td>
<td>&lt;.02</td>
<td>y = 0.47x + 1.44</td>
<td>0.76 12</td>
</tr>
<tr>
<td>Valve area</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A''/EI-G/A</td>
<td>.80</td>
<td>&lt;.001</td>
<td>y = 0.59x + 0.27</td>
<td>0.20 30</td>
</tr>
<tr>
<td>A''/EI-G/A</td>
<td>.82</td>
<td>&lt;.001</td>
<td>y = 0.51x + 0.23</td>
<td>0.17 30</td>
</tr>
<tr>
<td>A''/EI-G/A</td>
<td>.87</td>
<td>&lt;.001</td>
<td>y = 0.79x + 0.20</td>
<td>0.21 30</td>
</tr>
<tr>
<td>A''/EI-G/A</td>
<td>.87</td>
<td>&lt;.001</td>
<td>y = 0.68x + 0.16</td>
<td>0.17 30</td>
</tr>
<tr>
<td>A''/EI-G/F</td>
<td>.87</td>
<td>&lt;.001</td>
<td>y = 0.70x + 0.18</td>
<td>0.13 16</td>
</tr>
<tr>
<td>A''/EI-G/F</td>
<td>.86</td>
<td>&lt;.001</td>
<td>y = 0.59x + 0.17</td>
<td>0.11 16</td>
</tr>
<tr>
<td>A''/EI-G/F</td>
<td>.89</td>
<td>&lt;.001</td>
<td>y = 0.74x + 0.21</td>
<td>0.12 16</td>
</tr>
<tr>
<td>A''/EI-G/F</td>
<td>.89</td>
<td>&lt;.001</td>
<td>y = 0.62x + 0.20</td>
<td>0.10 16</td>
</tr>
<tr>
<td>G/A-G/F</td>
<td>.73</td>
<td>&lt;.01</td>
<td>y = 0.49x + 0.37</td>
<td>0.15 16</td>
</tr>
</tbody>
</table>

Abbreviations as in tables 1 and 2.

^Subgroup: patients with no or mild aortic regurgitation examined with echo/Doppler within 4 days of catheterization.

Discussion

The obvious difficulty in evaluating a new method for estimating valve areas is the problem of what to compare it with. Even in the absence of aortic regurgitation, Gorlin’s formula may produce errors of up to 20% to 40% by including an empirical constant, the discharge coefficient. Additional errors may be introduced by incorrect measurements of cardiac output and pressure drop. Besides, in the presence of aortic regurgitation, the valve area will be underestimated. It is possible to substitute effective cardiac output in the formula with total cardiac output calculated from single-plane angiographic images, except when there is significant mitral regurgitation. However, cardiac output measurements with this method may be less accurate in hypertrophic than in nonhypertrophic ventricles because of the influence of the larger papillary muscles. This may explain the poor correlation between invasive measurements of cardiac output and of valve area in these patients (table 3) and also the larger standard error of the estimate in figures 8 and 9. Considering these limitations, the correlations obtained in figures 6 to 9 indicate that the present method is...
reasonably accurate for estimating the aortic valve area.

Figure 10 shows that valve areas calculated by equation 2 are equal to or larger than those calculated from equation 5. The assumption that equation 2 calculates a maximal area and equation 5 an effective area is thus supported. This difference seems to be less marked when the valve area is small, possibly indicating that those valves are more calcified and less elastic. The difference cannot be explained by the peak velocity in the stenotic jet being less well recorded than the rest of the velocity curve, since both the peak and mean pressure drops calculated from the velocity recordings correlated well with the corresponding invasive measurements (table 3).

Valve areas calculated from equation 2 correlated better with invasive results (figures 7 and 9) than did the valve areas calculated from equation 5 (figures 6 and 8). Especially the larger areas seemed to be underestimated by equation 5. The reason for this is difficult
to ascertain because there were few patients with large valve areas in this series and because the measurements were not done simultaneously. However, according to Rodrigo, the discharge coefficient in Gorlin’s formula can only be assumed to be constant when the valve area is small compared with the area proximal to the valve. Larger valve areas will be overestimated. One therefore cannot exclude that the larger valve areas in figures 6 and 8 are overestimated by Gorlin’s formula rather than underestimated by equation 5. Thus in figures 7 and 9 the overestimation of effective valve area made by equation 2 may seem to be balanced by an overestimation of similar magnitude by assuming the discharge coefficient in Gorlin’s formula to be constant, accidentally causing the two methods to correlate well. The tendency for Gorlin’s formula to overestimate large valve areas was also shown by Warth et al.

Reliability of velocity and diameter measurements. The reliability of the estimates of the right sides of equations 2 and 5 is best established for \( V' \) and \( \text{int} V'(t) \). In accordance with the results of Hegrenaes and Hatle, the peak and mean pressure drops calculated from the velocity recordings correlated well with the invasive results (table 3), indicating that the velocity curve of the stenotic jet was adequately recorded in all patients. The accuracy of valve area estimations will therefore largely depend on the reliability of subvalvular velocity and diameter recordings.

Ihlen et al. and Gussenhoven et al. found the reproducibility of subvalvular diameter measurements to be good in patients with noncalcified aortic valves. In the case of calcific aortic stenosis, the accuracy is probably less good. Cohen et al. measured the subvalvular diameter to predict the prosthetic valve size in adult patients with aortic stenosis. Errors up to 2 mm were made. This is similar to the difference in diameters on repeated measurements that we found in five patients and indicates that an error of about 10% to 20% in the assessment of subvalvular area has to be expected in some patients.

In some patients the diameter was measured in late diastole because the upper LVOT was poorly visualized in systole. In the case of different diameters in systole and diastole, the systolic value should probably be used. However, there is reason to believe that this diameter does not change much during the cardiac cycle.

Assessment of the reliability of the subvalvular velocity recordings is difficult. In some patients stable velocities were recorded over a distance of 1 to 2 cm in the upper LVOT, making the decision on which velocities to use easy. In general, however, the velocities increased gradually until the more rapid increase 0.5 to 1.5 cm proximal to the valve. Even if this rapid increase could occur quite suddenly, there was necessarily a range of velocities from which the correct one was difficult to choose. In figure 3 this range is represented by the velocities recorded at 9.0 and 9.5 cm. These recordings are from a patient in whom the change from gradually to steeply increasing velocities was less marked than usual. The resulting differences in maximal velocity and integral size, about 14%, therefore probably represent the upper limit of the error in calculated valve area, introduced by subvalvular velocity recordings. In seven other patients this range gave differences of 12% or less.

It may be argued that diameter and velocity measurements should be made at the same level in the LVOT (compare figures 2 and 3). Because the LVOT may show considerable axial movement during systole (we have noticed up to 1 cm), this is not possible. Pasipoularides et al. found that the distance between the level just below the valve and the level of the stenosis ranged from 0.26 to 0.95 cm (mean 0.52). When this is added to the distance of the systolic movement of the LVOT, our procedure for diameter and velocity measurements appears to be acceptable.

The combined error produced by subvalvular velocity and diameter recordings can be evaluated by calculating cardiac output and comparing the results with invasive measurements (table 3). The poor correlation obtained is probably partly explained by the fact that
the measurements were done simultaneously, partly by the inaccuracy of subvalvular velocity and diameter measurements as discussed above, but also by the inaccuracies of the invasive methods (table 3). Nevertheless, the standard error of the estimate as obtained in the subgroup of patients is not very different from that reported in other studies in which the noninvasive and invasive measurements of cardiac output were made simultaneously. In the same group of patients the largest underestimation of cardiac output occurring when the “inner” diameter was used was 49%, compared with 38% when the “large” diameter was used. This may indicate that the “large” diameter is to be preferred for calculations of volume flow and valve area, but again, due to the fact that measurements were not made simultaneously, it is difficult to make any final evaluation of the problem.

The reasons why noninvasive measurements of pressure drops correlated better with the invasive results than did the measurements of cardiac output are probably that both the invasive and noninvasive methods are more accurate in assessing pressure drops than volume flow and that variations in flow will produce comparatively smaller variations in pressure drop because the ejection time also varies. Variations in heart rate will also influence cardiac output and pressure drop differently.

If the errors in diameter and velocity measurements are added, they may be of the same magnitude as the difference between the valve areas estimated from equation 2 and 5. The simplicity of equation 2 probably justifies its use in the clinical routine. If the estimated area is borderline regarding the decision of how to treat the patient, it might be helpful to calculate the area from equation 5 as well.

Our results show that this method can also be used when there is significant aortic regurgitation, which was present in 14 patients. Even though it was not present in this series, significant mitral regurgitation should not invalidate the method. Thus the limitations of other methods are eliminated. No empirical constant is used, reducing errors caused by individual variations. The approximation made by Kosturakis et al. by assuming the temporal mean velocity in the stenotic jet to be 0.88 max velocity is also avoided. According to our observations, the quotient Vmean/Vpeak can range from 0.69 to 0.87. By assuming a fixed quotient, the method will tend to average the results. On the other hand, the error made may not be important compared with other errors inherent in any method used to estimate valve areas.

The left and right sides of equation 4 describe the stroke volume at two levels that are in series and closely located. Hemodynamic changes should therefore affect both sides of the equation similarly, making the method independent of left ventricular performance.

Fujii et al. have simultaneously and independently of our study used equation 2 to calculate the valve area in aortic stenosis and have derived similar results.

There may be some practical problems in using equations 2 and 5. Especially in patients with emphysema, in whom the subvalvular diameter can be very difficult to measure, the results should be accepted with some reservation. Caution should also be exercised when calcifications are seen to protrude into the LVOT. This was seen in two patients in the present series. We attempted to solve the problem by placing the sample volume just proximal to the calcification. Thereby the increase in velocity, secondary to the decrease in cross-sectional area caused by the calcifications, was avoided. The calculation of the subvalvular area was made as described earlier, disregarding the calcifications. This apparently was successful, since the noninvasive estimates of valve area were quite close to the invasive results (invasive results, based on Fick method, 0.59 and 0.86 cm²; noninvasive results with the “large” diameter, 0.50 and 0.66 cm² [equation 5], 0.56 and 0.79 cm² [equation 2]).

If subvalvular obstruction caused by septal hypertrophy is present, the subvalvular velocity profile may be unpredictable and calculated valve area should be looked upon only as a rough estimate. Significant subvalvular obstruction was not seen in any patient in this study. In fact, problems were more frequently encountered when collecting the invasive than the noninvasive data, emphasizing the usefulness of the noninvasive method.

Appendix

If the stenotic area (A') of a flow channel varies because of the changing pressure drop across the obstruction during pulsatile flow, the equation of continuity can be developed to calculate the temporal mean of A'.

The equation of continuity is valid at any moment during pulsatile flow:

\[ A \times k(prof) \times V = A' \times k(prof') \times V' \]

A and V are the area and maximal velocity proximal to the obstruction, and V' is the maximal velocity at the obstruction; k(prof) is a constant describing the velocity profile [k(prof) = 1 if the profile is flat, k(prof) = \(\frac{1}{2}\) if the profile is parabolic]. If k(prof) = k(prof'), the equation can be reduced and rearranged to:

\[ A'/A = V/V' \]

Both sides are then integrated over one flow period:

\[ \int A'/A \] = \[ \int V/V' \]
If A is constant:

$$\int(A') = A \times \int(V/V')$$

Temporal mean of $$A' = \int(A')/t$$, where t is the duration of one flow period:

$$\text{mean } A' = A \times \int(V/V')/t$$

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