Variation of Anatomic Valve Area During Ejection in Patients With Valvular Aortic Stenosis Evaluated by Two-dimensional Echocardiographic Planimetry: Comparison With Traditional Doppler Data

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Objectives. Flow variations can affect valve-area calculation in aortic stenosis and lead to inaccuracies in the evaluation of the stenosis. Knowing that transvalvular flow varies normally within one beat, we designed this study to assess the response of the valve to intrabeat variation of flow during systole. Results were compared with flow-derived measurements.

Background. Technological improvements now allow us to evaluate aortic valve area directly by short axis planimetry. This offers the possibility to perform serial planimetrised during one ejection phase and analyze the intrabeat dynamic behavior of the stenotic-aortic valve and compare these measurements with flow-derived measurements.

Methods. Forty echocardiograms displaying different degrees of aortic stenosis were analyzed by frame-by-frame planimetry of the valve area from onset of opening to complete closure. Maximal-mean area, opening and closing rates and ejection times were obtained and compared with Doppler-derived data.

Results. Valve area varied during ejection. Stenotic valves opened and closed more slowly than normals and remained maximally open for a shorter period. Mean area by Doppler data corresponded more closely to maximal than to mean-planimetered area. Duration of flow was shorter than valve opening in severely stenotic valves. Discrepancies between Doppler-derived and two-dimensional (2D) measurements decreased in less stenotic valves.

Conclusions. Our observations reveal striking differences between normal and stenotic valves. Surprisingly, Doppler-derived mean-valve area correlated better with maximal-anatomic area than with mean-anatomic area in patients with aortic stenosis. Discrepancies between duration of flow and valve opening could explain this phenomenon.

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Echocardiography has been used for almost two decades in the evaluation of valvular aortic stenosis and is accepted as an accurate noninvasive method of quantification of aortic stenosis severity using Doppler flow measurements (1–7). Recently, transthoracic and transesophageal two-dimensional echocardiographic planimetry of the valve in short axis views have also been validated as accurate and reliable methods to measure aortic valve area (8–15).

Although maximal anatomic aortic valve area is measured by planimetry at one time point of the ejection, Doppler-derived continuity equation, if flow-velocity integrals are used for its calculation, is thought to yield mean area throughout ejection. The good correlation found so far between these two methods (8–15) implies little variation of valve area during ejection because mean (Doppler-derived) and maximal (planimetry) areas seem to be equal. However, others have suggested that normal “intrabeat” variations in flow affect valve area and that valve area is not constant during ejection (16–17). If valve area varies significantly during the ejection phase in patients with aortic stenosis, then a single two-dimensional measurement of the maximal orifice during ejection could potentially yield inaccurate data regarding the true severity of the stenosis. Moreover, if there is significant variation of valve area during ejection, mean and maximal measurements of valve area would not be expected to be equal. To address those issues, we designed this study to investigate the dynamic behavior of the aortic valve with different degrees of stenosis during the ejection phase of the cardiac cycle and see how the mean and maximal areas obtained by planimetry relate to flow-derived Doppler measurement of valve area.

Methods

Forty patients with different degrees of calcific degenerative aortic stenosis underwent a prospective complete two-dimensional and Doppler echocardiographic examination us-
ing a state-of-the-art ultrasound system (HP Sonus 2500, Hewlett-Packard). The echo studies of those patients (25 women, 15 men; mean age of 69) were recorded on standard VHS videotapes at a standard video frame rate of 30 per s and analyzed off-line using a digital analysis system. If the quality of the two-dimensional imaging was adequate, the transthoracic (TTE) echo study was used for valve planimetry; if not, then a transesophageal study (TEE) was used if available (overall: TTE = 32 patients, TEE = 8 patients).

For all these patients, a frame-by-frame planimetry of the aortic valve area from the onset of opening of the valve to its complete closure was performed on the zoomed short axis view of the valve after careful optimization of the gray-scale and gain settings (Fig. 1). Because systolic movement of the aortic root can affect cutting plane position, care was taken to position the cutting plane at the exact tips of the leaflets by tilting and shifting the transducer. Excessive movement of the cutting plane was suspected if the image of the valve was lost at some point during ejection and the patient was excluded from the study. Other criteria for rejecting images on a suspicion of excessive movement were the detection of any changes in leaflet body or edge morphology during ejection and changes in other landmarks’ locations near the aortic valve on the same view. Depending on the heart rate, this method yielded for each patient 6 to 16 sequential planimetries each being 1/30 of a s apart (or 33 msec). Two beats were analyzed and averaged. All were in normal sinus rhythm. Extrasystolic and postextrasystolic beats were rejected because of altered hemodynamics. Study protocol was approved by the Institutional Review Board, and patients gave informed consent.

**Planimetry data.** Maximal valve area was traced at the widest opening during ejection. Mean valve area was calculated by dividing the sum of all the planimetered areas by the number of tracings for that beat. Total ejection time was calculated by multiplying the total number of frames where the valve was seen open by 33 ms (1 frame). Time to maximal opening was determined by multiplying the number of frames from onset of opening until maximal opening was reached by 33 ms and is expressed as percentage of total ejection time. Opening rate was obtained by dividing the maximal valve area by the time needed to reach it (number of frames × 33 ms) and is expressed in cm²/s. Similarly, closing rate was calculated by dividing the maximal area by the time needed from maximal area to complete closure of the valve (number of frames × 33 ms) and is expressed in cm²/s.

**Doppler data.** Mean valve area was calculated by continuity equation using flow-velocity integrals (FVI) from the available pulsed and continuous wave Doppler data and the left ventricular outflow tract (LVOT) diameter (d) using the following widely accepted equation: Aortic valve area (AVA) = LVOT area × FVI LVOT/FVI Aortic. Ejection time was obtained by measuring the duration of flow through the valve using continuous wave Doppler tracings through the valve.

**Statistical analysis.** Simple linear regression analysis was used to evaluate the relations between the different valve measurements and degree of stenosis. Unpaired t tests were performed to compare groups with different degrees of aortic stenosis. Statistical significance was assessed at p < 0.05. Intra- and inter-observer variability were calculated on half the

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<th>Abbreviations and Acronyms</th>
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<tr>
<td>AVA = aortic valve area</td>
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<td>FVI = flow-velocity integrals</td>
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<td>LVOT = left ventricular outflow tract</td>
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<td>TEE = transesophageal echocardiogram</td>
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<td>TTE = transthoracic echocardiogram</td>
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**Figure 1.** Examples of two-dimensional planimetry. A, Top row: transthoracic echocardiography (TTE). Bottom row: transesophageal echocardiography (TEE) (NB: two different patients). The valve is seen closed, then maximally open and finally with superimposed planimetry of the valve area. B, Frame by frame planimetry (dotted lines) for one ejection period in moderate aortic stenosis (different patient from top panel). Left, the valve is closed. Progressive opening and closing can be seen in the following frames, returning to complete closure on the right.
patients selected randomly and are expressed as the mean difference between the measurements of the same and of two different blinded observers respectively.

Results

Patient data. Screening of 53 patients was needed in order to obtain 40 adequate quality studies on which a complete Doppler evaluation and frame-by-frame planimetry could be performed. Using the continuity equation on these 40 patients, 17 had mean aortic valve areas under 1 cm², 11 had valve areas of more than 1 but less than 2 cm² and 12 had areas over 2 cm² (normal or only sclerotic valves). Mean left ventricular ejection fraction was 53% ± 2.2% (mean ± SEM).

Valve dynamics. Aortic valve area as measured by sequential planimetry was not constant throughout the ejection phase in our patients. Figure 2 shows typical opening and closing patterns for the aortic valve of 3 patients with valve areas under 1 cm² (A), with valve areas between 1 and 2 cm² (B) and 3 with valve areas over 2 cm² (C). The variation of valve area can easily be appreciated in those examples and seems more pronounced in patients with more severe aortic stenosis (A) compared to the other two groups. The valves of patients with valve areas under 1 cm² (A) opened and closed slowly and stayed maximally open for a very short period. The valves of patients with valve areas between 1 and 2 cm² (B) showed an intermediate variability pattern compared to the other two groups. In patients without any significant stenosis (C), the valve opened and closed very quickly with a “stable” plateau in the middle thereby resulting in very little variation of valve area through ejection in this group.

Figure 3 demonstrates the opening (3A) and closing rate (3B) of the aortic valve related to valve area obtained from Doppler data (CE) in square centimeters. Opening and closing rates are calculated by dividing the maximal valve area obtained by planimetry by the time needed to reach that maximal opening or by the time needed to go back to complete closure from that maximal opening respectively and are expressed in square centimeters per s.
Comparison of maximal and mean valve areas by planimetry. The degree of variation of valve area during one ejection period can be appreciated by comparing the mean and maximal areas obtained from the planimetry. Identical mean and maximal valve areas imply a stable valve area through ejection; on the other hand, if the valve area varies a lot then the difference between mean and maximal areas increases proportionally.

Results of mean and maximal valve areas for the different groups are shown in Figure 4. A significant difference was noted between the two measurements, maximal area being consistently larger than mean valve area ($y = 0.9x - 0.2$, $r = 0.99$, $p < 0.001$) where $y = mean area by planimetry$ and $x = maximal area by planimetry; mean diff. of 0.38 cm$^2$, $p < 0.001$).

The relative difference between mean and maximal areas depending on the degree of stenosis became smaller as valve area increased ($y = 42.8 - 8.3x$, $r = 0.7$, $p < 0.01$) where $y = relative difference between maximal and mean area by planimetry$ and $x = valve area by Doppler in cm$^2$).

Comparison with doppler data

Mean versus maximal areas. Mean effective valve area by continuity equation derived from Doppler measurements was compared to the mean and maximal anatomic valve areas obtained by planimetry (Fig. 5). We found an excellent correlation and little differences between mean effective area by continuity equation and maximal anatomic area as reported by others (Fig. 5A). An equally good correlation was found between mean effective and mean anatomic areas (Fig. 5B), but their values differed on average by 0.25 cm$^2$ ($p < 0.01$). The relative difference between these two methods of measuring mean valve area increased as the valve became more stenotic ($y = 0.5x - 0.15x$, $r = 0.7$, $p < 0.001$) where $y = relative difference between mean valve area by planimetry and mean area by continuity equation and $x = valve area by Doppler in cm$^2$).

Ejection time by Doppler and planimetry. Doppler ejection time (duration of flow on continuous wave Doppler) was compared to ejection time by planimetry (time from onset of opening to complete closure of the valve as seen on the 2D short axis view of the valve) according to severity of the stenosis. No significant difference was detected between the two measurements in the group of patients without significant stenosis (valve area over 2 cm$^2$) (mean diff. of 25 ms, $p = 0.37$). In patients with valve areas between 1 and 2 cm$^2$, a larger difference is noted, but did not reach statistical significance (mean diff. of 41 ms, $p = 0.36$). However, in patients with valve areas under 1 cm$^2$, ejection time as evaluated by Doppler was significantly shorter than the ejection time calculated from the planimetries (mean diff. of 78 ms, $p < 0.05$).

Observer variability. Intraobserver variability for maximal valve area measurement by planimetry was 0.01 cm$^2$ ± 0.06 and for mean area by planimetry was 0.04 cm$^2$ ± 0.03 (mean ± SE). Interobserver variability was 0.05 cm$^2$ ± 0.04 for maximal area and 0.03 cm$^2$ ± 0.06 (mean ± SE) for mean area by planimetry. Those results reflect the differences between the
two sets of measurements by the same and two different blinded observers respectively and none of these differences reached statistical significance when compared by a paired t test to the initial set of measurements.

**Discussion**

Improvements in ultrasound technology have allowed us to look directly at the aortic valve in its short axis view and reliably measure its opening at different time points in the cardiac cycle. Using this technology in patients with different degrees of aortic stenosis, we observed that the aortic leaflets behave a lot differently depending on the degree of the stenosis and that this altered behavior affects the relationship between planimetric (anatomic valve area) and flow-derived measurements (effective valve area).

**Valve dynamics.** Our observations indicate that valve area does not remain constant during the ejection phase of the cardiac cycle. This variability of valve area increases with increasing severity of the stenosis. The results of our study are in agreement with a few previous observations: in two studies that examined this issue in normal canine aortic valves, definite ejectional variation of valve area was detected (18–20). In another study on patients with congenital aortic stenosis of mild severity, similar variation was noted (21).

Our data shows a slower opening and closing rate of stenotic valves. The mechanism of opening and closing of the valve might be disturbed by different factors. One reason might be that in aortic stenosis, the valve leaflets are thickened, calcified and lose mobility. This increases their inertia and the force needed to make them open and close.

The mechanism of opening and closing of the normal aortic valve have been previously studied in instrumented dogs: the authors noted that early systolic expansion of the aortic root is possibly the first mechanism inducing leaflet separation before onset of transvalvular flow (18,20). Calcification of the aortic annulus and root can sometimes occur in calcific aortic stenosis and limit its pulsatility and systolic expansion. This would affect one of the early opening mechanism of the aortic valve. With worsening of the stenosis, increased leaflet inertia and loss of normal aortic root expansion combined together would thus result in slower opening and closing as we found in our patients.

The time needed to reach peak valve area increases with the severity of the stenosis, leaving less time for the valve to stay maximally open. Compared to a normal valve, a stenotic valve not only has a smaller anatomic area, but it also remains maximally open for a relatively shorter period of time. This implies that two valves with the same calculated areas, either by continuity equation or by short axis planimetry area, may need different times to reach this peak opening and stay maximally open also for a different period of time.

**Comparison with doppler data.** Our findings have confirmed that mean valve area calculated from Doppler data (continuity equation) can be used interchangeably with maximal area by planimetry as shown in previous studies (8–15). However, it cannot be used as an estimation of mean anatomic valve area.

The good correlation between maximal valve on planimetry and continuity equation raises some questions. Since the continuity equation, using flow-velocity integrals in its calculation, yields a mean effective and not an anatomic valve area (22), we expected that mean anatomic valve area as calculated by planimetry would be different from the effective mean valve area by continuity equation. We would nevertheless expect a closer correlation between both methods that calculate mean areas, namely continuity equation and mean area by planimetry, than between two methods where one calculates the mean area (continuity equation) and the other maximal area (maximal planimetry). Our findings suggest the opposite.

One possible explanation for this phenomenon could be that no significant flow occurred at very small valve openings in aortic stenosis, namely at the early opening and late closing phases. This would limit significant flow through the valve at openings closer to maximal opening which occur in the middle of the ejection phase. Continuity equation, which uses Doppler-flow measurements, would therefore include in its calculation only flow that occurs at valve areas closer to maximal valve area. This would explain why maximal area by planimetry correlates more closely than mean area by planimetry to mean valve area by continuity equation. In an attempt to confirm the hypothesis that valve opening does not correspond to significant onset of flow in significant aortic stenosis, we compared ejection time by Doppler and ejection time by planimetry, expecting to find a larger difference between the two (shorter ejection time by Doppler) as the degree of stenosis increased. As expected, the ejection time by Doppler was significantly shorter in the groups with valve areas under 1 cm$^2$ compared to the time when the valve was seen open on the echo and there was no significant difference in patients with valve area over 1 cm$^2$. Previous studies have reported the possible discrepancy between onset of flow and valve opening (18–20): the normal aortic valve is already open before any forward flow is detected, maximal valve area is reached before peak flow, the valve has already started closing while flow continues and final closure of the valve occurs after all forward flow has ceased.

**Potential clinical implications.** The presence or absence of symptoms in patients with aortic stenosis is a key factor that greatly influences the prognosis (23). However, why a patient with moderate or severe aortic stenosis is symptomatic and another with a similar valve area is not cannot always be explained. Many factors other than reaching a critical valve area or transvalvular gradient have been incriminated to explain the development of symptoms in those patients (24–27). From our findings, it seems possible that two valves with identical maximal areas could have different mean areas throughout systole. This phenomenon would imply different total afterload burdens for two valves with identical maximal areas, which could potentially influence the occurrence of symptoms. To our knowledge, only one study has suggested that not only the increase of the anatomic valve area but also...
improvement of orifice variability due to opening of fused commissures and decreased leaflet inertia could play a role in the symptomatic improvement and response to flow variation in patients with aortic stenosis after valvuloplasty (28).

Valve area measurement by the flow-derived continuity equation complemented by short axis planimetry when necessary in difficult cases provide sufficient information in the vast majority of clinical cases. However, it could be interesting to try to obtain maximal and mean areas by planimetry in some clinical circumstances where discrepancies between clinical findings and calculated valve area, either by continuity equation or short-axis planimetry of maximal area, are found.

**Limitations.** The results of our study must be viewed under the light of certain limitations. First, ejection time by Doppler and by planimetry were not simultaneously recorded. We cannot be sure that the hemodynamic status was exactly similar at the time of both recordings; although it is most likely the case since no procedures or medications were given during the examination. We also tried to match the R-R intervals of the cycles we have used for the calculation of both ejection times in order to minimize the potential effects of beat-to-beat variations. The noncompressible time resolution of 33 ms that results from the video frame rate of 30 images per s also limits the accuracy of our measurements. This could be an important factor, especially in very fast heart rates where fewer frames were available for analysis. However, few patients in this study had fast heart rates and no significant difference in heart rates was found according to the degree of stenosis. Thus, if the measurements were indeed affected by this factor, all patients were affected to the same extent and this could not explain the results we obtained.

Second, our data could be affected by flow conditions (29–33). To minimize this factor, only patients in regular normal sinus rhythm were selected. Ejection fraction was also close to the normal range for most of our population, thereby limiting low flow conditions which may have produced incomplete opening of the valve. We found no relation between ejection fraction and our measurements. Maximal area by planimetry should not have been affected by flow conditions (17).

Although great care was taken to avoid analyzing images with excessive movement of the cutting plane during ejection, some movement may have still been present. However, it must be remembered that the plane of the aortic valve is very narrow and cannot be compared, for example, to that of the mitral valve which is much more dome-like. Excessive movement resulted in most cases in complete loss of the image of the valve. The reproducibility of our results argues against the potential random effects of such movement.

Finally, our results can only be applied to a selected subgroup of patients with calcific degenerative aortic stenosis with high quality echocardiograms and cannot be generalized to all patients with aortic stenosis of other etiology. A learning curve is certainly necessary to feel comfortable with this technique and great technical care is required in image acquisition which should be done by experienced sonographers.

**Conclusions.** While our findings raise some questions, we do not imply that planimetry of the maximal valve area and the use of the continuity equation are inadequate for the clinical evaluation of the majority of patients with aortic stenosis. Current literature confirms that continuity equation is reliable and that transthoracic and transesophageal planimetry can be used to complement continuity equation in patients with adequate images when performed adequately and with great technical care in the hands of experienced personnel. However, our study has revealed some striking differences in the dynamics of the stenotic aortic valve compared to normal valves and also outlined some major differences between measurements of Doppler-derived valve areas and values obtained from planimetry. When comparing different methods to measure valve area by echocardiography, we need to know that, although maximal planimetry and continuity equation-derived valve area can be used interchangeably, those two methods do not give us any information about the mean anatomic valve area for that specific patient.

Valve dynamics and the differences between mean and maximal valve areas could potentially play a role together with other pathophysiologic factors in explaining the occurrence of symptoms in patients with nonsevere aortic stenosis. It would also be of interest to assess the effects of systematic flow variation on these parameters. These issues will be addressed in future studies.

**References**


