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Planimetry and Transthoracic Two-Dimensional Echocardiography in Noninvasive Assessment of Aortic Valve Area in Patients With Valvular Aortic Stenosis

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Objectives. **The aim of this study was to evaluate the reliability of transthoracic two-dimensional echocardiography in measuring aortic valve area (AVA) by planimetry.**

Background. **Planimetry of AVA using two-dimensional transesophageal echocardiographic images has been reported to be a reliable method for measuring AVA in patients with aortic stenosis. Recent advances in resolution of two-dimensional echocardiography permit direct visualization of an aortic valve orifice from the transthoracic approach more easily than before.**

Methods. **Forty-two adult patients with valvular aortic stenosis were examined. A parasternal short-axis view of the aortic valve was obtained with transthoracic two-dimensional echocardiography. AVA was measured directly by planimetry of the inner leaflet edges at the time of maximal opening in early systole. AVA was also measured by planimetry using transesophageal echocardiography, by the continuity equation and by cardiac catheterization (Gorlin formula).**

Results. **In 32 (76%) of the 42 study patients, AVA could be detected by using the transthoracic planimetry method. There were good correlations between results of transthoracic two-dimensional echocardiographic planimetry and the continuity equation ($y = 0.90x + 0.09$, $r = 0.90$, $p < 0.001$, SEE = 0.09 cm^2), transesophageal echocardiographic planimetry ($y = 1.05x - 0.02$, $r = 0.98$, $p < 0.001$, SEE = 0.04 cm^2) and the Gorlin formula ($y = 1.02x + 0.05$, $r = 0.89$, $p < 0.001$, SEE = 0.10 cm^2).**

Conclusions. **Transthoracic two-dimensional echocardiography provides a feasible and reliable method in measuring AVA in patients with aortic stenosis.**

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Aortic valve area (AVA) calculated by the Gorlin formula (1) or continuity equation (2–8) has been utilized as a reliable index of the severity of valvular aortic stenosis. However, previous reports indicated that valve area derived from the Gorlin formula (9–14) and continuity equation (14,15) vary with changes in transvalvular volume flow rate. Recently, direct planimetry of AVA using monoplane or multiplane (omniplane) transesophageal echocardiography has been reported to provide reliable measurements of anatomic AVA in patients with aortic stenosis (16–20). In contrast, early studies (21–24) suggested that, because of limited resolution, the transthoracic echocardiographic approach was inadequate for quantitating the severity of aortic stenosis. However, recent advances in resolution of two-dimensional echocardiography permit direct visualization of an aortic valve orifice from the transthoracic approach more clearly than before (25,26). We therefore undertook this study to determine the feasibility and reliability of using planimetry with transthoracic two-dimensional echocardiography in evaluating AVA. Our transthoracic planimetry measurements were then compared with previously validated values derived from the continuity equation, transesophageal echocardiographic planimetry and the Gorlin formulas.

Methods

Study patients. We studied 42 consecutive adult patients (20 men and 22 women, mean age 63 ± 12 years [range 36 to 85]) with valvular aortic stenosis as assessed by physical and echocardiographic examination. Thirty-seven patients had sinus rhythm, and five had atrial fibrillation. All gave informed consent according to a protocol approved by the Human Study Committee of the Kobe General Hospital. Transthoracic and transesophageal echocardiography were performed in all patients. The patients underwent cardiac catheterization within 1 week after echocardiographic examination.

Cardiac catheterization. Retrograde left and right heart catheterization was performed by femoral approach in all cases. The mean pressure gradient across the aortic valve was recorded with use of a fluid-filled catheter during pullback maneuver of the catheter across the aortic valve into the ascending aorta. Three pressure measurements were averaged in patients with normal sinus rhythm and five in those with

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Abbreviations and Acronyms

AVA	= aortic valve area
CSALVOT	= cross-sectional area of the left ventricular outflow tract
DLVOT	= diameter of the left ventricular outflow tract
VLVOT	= flow velocity in the left ventricular outflow tract
Vmax	= maximal flow velocity across the aortic valve

atrial fibrillation. Cardiac output was determined by thermodilution technique, averaging three measurements. AVA was calculated by the Gorlin formula. The severity of aortic stenosis was defined by the Gorlin formula–derived AVA as follows: severe $\leqq 0.75\ cm^2$, moderate >0.75 to $1.0\ cm^2$ and mild $>1.0\ cm^2$.

Transthoracic two-dimensional echocardiography. Transthoracic two-dimensional echocardiography was performed by using a commercially available echocardiographic machine (Sonos 1500 and 2500. Hewlett-Packard, Medical Products Group) with a 64-element, 3.5-MHz phased array transducer. All echocardiographic images were recorded on 0.5-in. (1.27-cm) SVHS videotape for subsequent analysis. The parasternal short-axis view was obtained at aortic valve level to visualize the leaflet edges completely during peak systole. Gain was adjusted at the lowest setting possible without losing definition of the commissural edges. AVA was defined as the smallest orifice area between the aortic cusps during peak systole obtained by shifting and tilting the transducer. AVA was measured by planimetry of the inner leaflet edges at the time of maximal opening in a zoom mode (Fig. 1). All measurements were performed by an observer who had no knowledge of the transesophageal echocardiographic and cardiac catheterization findings. Three orifice area measurements were averaged in each patient with normal sinus rhythm; five measurements were averaged in patients with atrial fibrillation.

To determine interobserver variability for AVA, 20 studies were randomly selected and analyzed by two independent observers. To determine intraobserver variability, 20 studies were also repeated by the same observer. Interobserver and intraobserver variabilities for AVA measurements were 9.7% and 7.1%, respectively.

Doppler echocardiography (continuity equation). Doppler echocardiography was also performed by using a Hewlett-Packard Sonos 1500 and 2500 echocardiographic machine with a 2.5-MHz phased array duplex transducer, and recorded on 0.5 in. SVHS videotape for later analysis. Parasternal long-axis views of the left ventricular outflow tract were obtained in the left lateral decubitus position, and the diameter of the left ventricular outflow tract (DLVOT) was measured immediately below the aortic valve. Three diameter measurements were averaged. Cross-sectional area of the left ventricular outflow tract (CSALVOT) was calculated from DLVOT by assuming it to be a circular as follows: $CSA_{LVOT} = \pi \times (D_{LVOT}/2)^2$. Apical long-axis views were obtained, and flow velocity in the left ventricular outflow tract flow (VLVOT) was detected by the pulsed Doppler method. The maximal flow velocity across the aortic valve (Vmax) was recorded by the continuous wave Doppler method. AVA was calculated by using the continuity equation as follows: $AVA = CSA_{LVOT} \times (V_{LVOT}/Vmax)$.

Transesophageal echocardiography. Transesophageal echocardiography was performed with a 64-element 5-MHz phased array probe (Omniplane, Hewlett-Packard) capable of mechanically changing plane orientation from 0° to 180°. The echocardiographic images were recorded on SVHS video tape for later analysis. After application of a topical pharyngeal anesthetic agent (lidocaine), the probe was inserted with the transducer at 0° in a left lateral decubitus position and placed at the aortic valve level. Short-axis viewing of the aortic valve was manipulated to obtain a cross-sectional view of the aorta with complete visualization of leaflet edges during peak systole. Gain was adjusted at the lowest setting possible without losing definition of the commissural edges. AVA was measured by planimetry of the inner leaflet edges at the time of maximal opening. An average of three valve area measurements during normal sinus rhythm and five measurements during atrial fibrillation was obtained.

Statistical analysis. Data are presented as mean value ± SD. The correlation between two variables was tested by linear regression analysis. To examine further the comparison of two methods of clinical measurements, the method of Bland and Altman (27) was used. A p value < 0.05 was considered statistically significant.

Results

Hemodynamic data. Clinical findings and echocardiographic and catheter-derived data in 42 patients are shown in Table 1. Cardiac index ranged from 1.58 to 3.71 liters/min per m^2 (mean 2.46 ± 0.70). Mean transvalvular pressure gradients ranged from 8 to 94 mm Hg (mean 48 ± 23). Aortic valve area

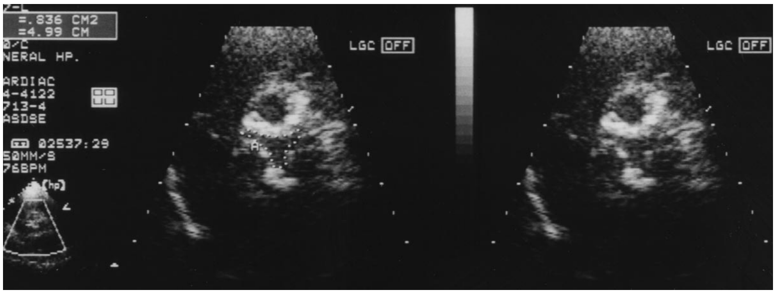

Figure 1. Measurement of AVA by transthoracic echocardiography in a patient with moderate aortic stenosis. The parasternal short-axis image of the aortic valve orifice was visualized during peak systole (**right**). Orifice area determined by planimetry was 0.84 cm^2 (**left**).

Table 1. Aortic Valve Area Determined by Transthoracic Planimetry, Continuity Equation, Transesophageal Planimetry and Gorlin Formula in 42 Patients

Pt No.	Age (yr)/ Gender	Rhythm	AR	CI (liters/min per m^2)	mPG (mm Hg)	Aortic Valve Area (cm^2)			
						TTE	Cont	TEE	Gorlin
1	75/F	SR	None	2.71	55	0.76	0.44	0.70	0.77
2	67/M	SR	Mild	1.76	77	0.67	0.50	0.56	0.36
3	57/M	SR	Mild	1.79	19	1.92	2.20	1.95	1.92
4	62/M	SR	Mild	1.82	64	0.63	0.55	0.62	0.55
5	53/F	Af	Mild	2.02	54	*	1.20	0.77	0.41
6	50/M	SR	Mild	2.38	44	0.52	0.59	0.57	0.38
7	63/M	SR	None	2.91	69	0.55	0.90	0.59	0.56
8	57/M	Af	None	1.97	17	1.13	0.87	1.06	1.10
9	57/M	Af	None	2.29	25	1.35	1.10	1.17	0.85
10	37/F	SR	Mild	3.34	55	0.93	1.03	0.92	0.72
11	58/F	SR	None	3.27	94	0.48	0.34	0.54	0.41
12	70/M	SR	None	3.04	83	0.50	0.52	0.46	0.48
13	69/M	SR	None	3.04	66	0.48	0.25	0.55	0.51
14	60/F	SR	None	2.02	18	*	1.50	0.90	1.13
15	66/F	SR	None	1.89	36	0.77	0.75	0.60	0.40
16	76/F	SR	None	1.93	97	*	0.45	*	0.27
17	63/F	SR	None	2.45	52	0.66	0.64	0.65	0.73
18	70/M	SR	None	2.45	63	0.60	*	0.45	0.58
19	72/F	SR	Mild	2.86	14	1.20	*	1.10	1.17
20	49/F	SR	None	2.50	20	1.40	1.43	1.30	0.79
21	70/F	SR	Mild	2.50	40	*	0.36	0.70	0.89
22	74/F	SR	None	3.51	66	*	0.45	0.50	0.58
23	71/M	SR	Mild	2.25	47	*	0.39	0.55	0.48
24	53/F	SR	None	2.88	64	0.48	0.42	0.50	0.57
25	74/F	SR	Mild	1.94	78	0.71	0.55	0.80	0.80
26	65/M	SR	None	2.79	8	2.00	1.60	2.00	2.10
27	40/M	SR	None	2.65	50	0.72	0.50	0.71	0.74
28	79/F	SR	None	2.35	28	0.84	0.90	0.78	0.88
29	54/M	SR	Mild	2.13	15	1.25	1.33	1.30	0.92
30	77/M	SR	Mild	2.28	16	0.41	0.65	0.41	0.41
31	71/M	SR	Mild	2.15	58	0.36	0.52	0.41	0.39
32	36/F	SR	Mild	3.34	55	0.66	1.03	0.79	0.72
33	70/M	SR	None	2.82	42	0.54	0.50	0.56	0.80
34	55/M	SR	Mild	2.49	57	*	*	0.48	0.50
35	78/F	Af	None	1.58	54	*	0.66	0.68	0.50
36	62/F	SR	Mild	2.09	47	0.69	0.56	0.59	0.61
37	60/F	SR	Mild	3.71	31	0.85	*	0.89	0.80
38	75/F	SR	None	2.53	67	0.24	0.50	0.26	0.50
39	45/F	Af	Mild	3.30	17	1.96	*	1.70	1.35
40	71/M	SR	Mild	2.30	36	*	0.44	0.77	0.80
41	85/M	SR	None	2.79	66	0.28	0.33	0.32	0.40
42	55/F	SR	Mild	3.08	54	*	0.80	0.60	0.52
Mean	63			2.46	48	0.83	0.75	0.78	0.72
SD	12			0.70	23	0.47	0.43	0.40	0.38

*Orifice area could not be determined. Af = atrial fibrillation; AR = aortic regurgitation; CI = cardiac index; Cont = continuity equation; F = female; Gorlin = Gorlin formula; M = male; mPG = mean pressure gradient measured by cardiac catheterization; Pt = patient; SR = sinus rhythm; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography.

calculated by the Gorlin formula ranged from 0.27 to 2.10 cm^2 (mean 0.72 ± 0.38).

Doppler echocardiography (continuity equation). In 37 (88%) of the 42 patients, AVA could be determined by the continuity equation. In the remaining five patients, adequate continuous wave or pulsed wave Doppler signals could not be obtained because of severe aortic valve calcification or pulmonary emphysema. AVA detected by the continuity equation ranged from 0.25 to 2.20 cm^2 (mean 0.75 ± 0.43).

Transesophageal echocardiography. In 41 (98%) of the 42 patients, the aortic valve orifice could be delineated and thus was suitable for planimetry of AVA. In one patient with a severely calcified aortic valve, AVA could not be determined by transesophageal echocardiography. AVA detected by transesophageal echocardiography ranged from 0.26 to 2.00 cm^2 (mean 0.78 ± 0.40).

Transthoracic two-dimensional echocardiography. In 32 (76%) of the 42 study patients, transthoracic two-dimensional echocardiographic image quality was adequate for planimetry of AVA (Fig. 1). In 6 of the other 10 patients, there was inadequate visualization of the valve orifice because of acoustic shadowing of a severely calcified aortic valve leaflet and annulus. In the remaining 4 patients, the aortic valve itself could not be visualized from the parasternal approach because of thorax deformity, adiposity or lung emphysema. AVA determined by transthoracic two-dimensional echocardiography ranged from 0.24 to 2.00 cm^2 (mean 0.83 ± 0.47).

Comparison between transthoracic two-dimensional echocardiography and the continuity equation and transesophageal echocardiography. AVA was determined by both transthoracic two-dimensional echocardiography and the continuity equation in 28 (67%) of 42 patients. Transthoracic planimetry–derived AVA correlated well with the continuity equation–derived AVA ($y = 0.90x + 0.09$, $r = 0.90$, $p < 0.001$, SEE = 0.09 cm^2) (Fig. 2) and with transesophageal echocardiographic planimetry–derived AVA ($y = 1.05x - 0.02$, $r = 0.98$, $p < 0.001$, SEE = 0.04 cm^2) (Fig. 3). The mean differences between aortic valve area determined by transthoracic two-dimensional echocardiography and the continuity equation and between that determined by transthoracic two-dimensional echocardiography and transesophageal echocardiography were 0.02 ± 0.20 cm^2 and 0.002 ± 0.01 cm^2, respectively.

Comparison between transthoracic two-dimensional echocardiography and cardiac catheterization (Gorlin formula). AVA was determined by both transthoracic two-dimensional echocardiography and cardiac catheterization (Gorlin formula) in 32 patients. The correlation between transthoracic two-dimensional echocardiography and cardiac catheterization–derived AVA was good ($y = 1.02x + 0.05$, $r = 0.89$, $p < 0.001$, SEE = 0.10 cm^2) (Fig. 4). The mean difference between AVA determined by transthoracic two-dimensional echocardiography and the Gorlin formula was 0.07 ± 0.22 cm^2. In 28 (88%) of 32 patients, the results of both methods were in agreement with the diagnosis of either severe or not severe aortic stenosis. The sensitivity and specificity of transthoracic two-dimensional echocardiography for detecting severe aortic stenosis were 89% and 85%, respectively.

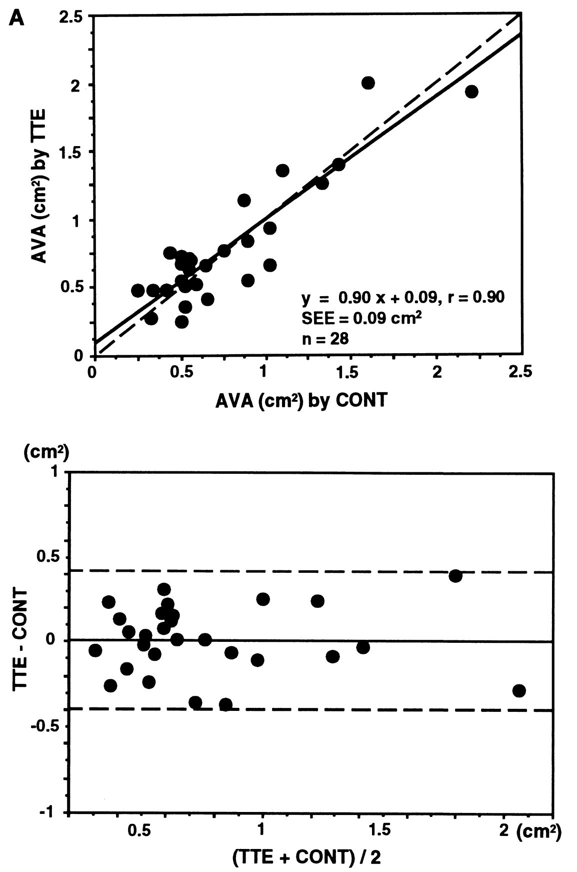

Figure 2. **A,** Scattergram of the correlation between AVA determined by two-dimensional transthoracic echocardiographic (TTE) planimetry and AVA determined by the continuity equation (CONT) in 28 patients. **B,** Plots of the average mean versus the differences between TTE planimetry–derived AVA and CONT-derived AVA. In **A,** the **solid line** is the regression line; the **dashed line** is the line of identity; in **B,** the **solid line** is the mean difference; the **dashed lines** are 2 SD of the mean difference.

Discussion

This study demonstrates that the two-dimensional echocardiographic planimetry method using a transthoracic approach provides a feasible and accurate measurement of AVA in adult patients with valvular aortic stenosis. Transthoracic two-dimensional echocardiography has been widely used in the clinical evaluation of aortic valve stenosis because it allows comprehensive evaluation of valvular anatomy and ventricular function in a short period of time, is less invasive than the transesophageal echocardiographic approach and does not require the multiple step measurements needed in the continuity equation method. However, previous reports (16,23) showed that the transthoracic echocardiographic approach using a 2.25-MHz transducer with 32 elements visualizes a complete aortic valve orifice in only a small number of patients with aortic stenosis. However, recent improvements in echocardiography have increased the clarity of the images we have obtained with transthoracic two-dimensional echocardiography images. Therefore, as Weyman et al. (22) predicted in 1975, these developments have revived interest in use of this approach for determination of AVA (25,26).

Previous methods. The Gorlin formula has been utilized as the standard for assessment of the severity of aortic stenosis (1). However, this method needs invasive procedures to assess AVA. Previous studies (9–14) have demonstrated that AVA derived from the Gorlin formula is flow dependent. That the empiric constant in this formula has different values at different flow rates may be the reason for the flow dependency

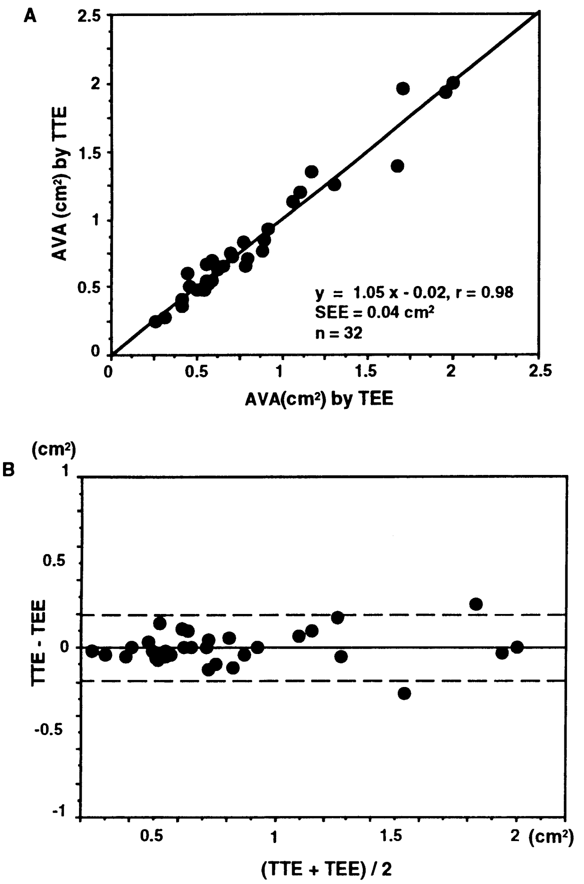

Figure 3. **A,** Scattergram of the correlation between AVA determined by transthoracic echocardiographic (TTE) planimetry versus AVA by transesophageal echocardiographic (TEE) planimetry. **B,** Plots of the average mean versus the differences between TTE planimetry–derived AVA and TEE planimetry–derived AVA. **Solid** and **dashed** lines as in panels **A** and **B** of Figure 2.

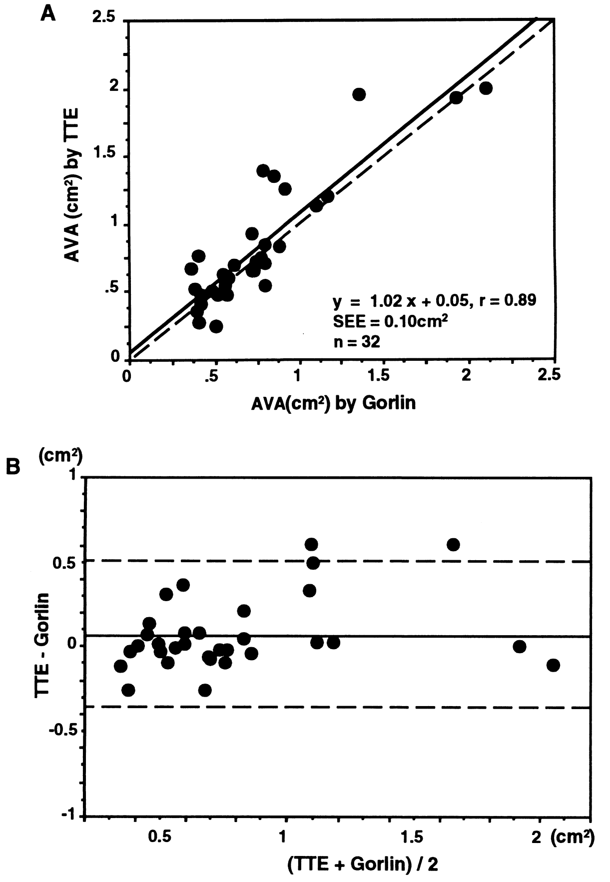

Figure 4. **A,** Scattergram of the correlation between AVA determined by transthoracic echocardiographic planimetry (TTE) versus AVA by the Gorlin formula (Gorlin). **B,** Plot of the average mean versus the differences between TTE planimetry–derived AVA and Gorlin-derived AVA. **Solid** and **dashed** lines as in panels **A** and **B** of Figure 2.

(11,12). Noninvasive attempts to assess the severity of aortic stenosis using two-dimensional and Doppler echocardiography have been reported (2–8,16–20).

Maximal and mean pressure gradients across the aortic valve measured by continuous wave Doppler echocardiography have correlated well with catheter-determined pressure gradients (28–36). However, transvalvular pressure gradients are flow dependent and can be underestimated in patients with decreased cardiac function and overestimated in patients with high output status or aortic regurgitation (37). AVA measured by Doppler technique has correlated well with AVA measured at catheterization (2–8). Previous studies (2–4) showed that determination of AVA by the continuity equation is possible in 85% to 95% of patients. However, the Doppler measurement of AVA depends on several assumptions. The left ventricular outflow tract is assumed to be circular and its cross-sectional area to be constant during systole. The velocity profile in the left ventricular outflow tract is assumed to be laminar and flat. AVA calculated by the continuity equation is not an anatomic valve area but an area of the vena contracta, which is always smaller (4) than the anatomic one. Furthermore, it has been reported (9,15) that AVA determined by the continuity equation also varies with changes in transvalvular volume flow rate.

Recently, transesophageal echocardiography was found (16–21) to provide accurate and reliable measures for detecting AVA. Hofmann et al. (16) and Stoddard et al. (17) demonstrated that AVA can be accurately measured by using monoplane transesophageal echocardiography. Hoffmann et al. (18) used multiplane transesophageal echocardiography to obtain more accurate planimetry of aortic valve orifice with a high correlation of r = 0.95. Tribouilloy et al. (19) reported that the correlation between transesophageal echocardiographic and catheter-determined AVAs was significantly greater with multiplane transesophageal echocardiography than with a monoplane transducer. Tardif et al. (38) showed that the planimetric AVA using transesophageal echocardiography does not change significantly with variations in cardiac output and stroke volume in patients with aortic stenosis.

Transthoracic planimetry method. In our present study, direct visualization of the aortic valve orifice was possible in 32 (76%) of 42 patients from the transthoracic approach. Development of the resolution and high frequency (short wavelength) transducer of echocardiography is thought to be the most important factor in the difference between the detection rate in the current study and previous studies. Clinical differences between subjects in the previous studies and the current study may in part account for this difference. Our study patients had relatively smaller body size (mean body surface area 1.55 cm^2). Furthermore, the valve area was >0.75 cm^2 in $\sim 40\%$ of these patients. In these patients with noncritical aortic stenosis, planimetry of the valve area may be easier.

Limitations of the study. Our present study has some limitations. 1) We could determine the aortic valve orifice from the transthoracic planimetry approach in only 76% of our patients with aortic valve stenosis, which is a lower detection rate than that obtained with the transesophageal approach. Thorax deformity, adiposity, pulmonary emphysema and severely calcified valve were thought to be the reasons for this high failure rate. However, we could assess AVA in 98% of patients from the transthoracic approach by using either planimetry or the Doppler (continuity equation) method. 2) Echocardiographic and cardiac catheterization studies were not performed simultaneously. Temporal differences in hemodynamic variables, such as cardiac output, mean pressure gradient, heart rate and blood pressure, might result in differences in value for the two areas. 3) Although AVA derived from the Gorlin formula was taken as the reference standard, there were several potential sources of error in the calculation of AVA with the Gorlin formula. The limitations of the Gorlin formula might affect the correlation between the two methods. In this study, although we excluded patients with significant aortic regurgitation, some study patients had low cardiac output.

Clinical implications. The transthoracic two-dimensional echocardiographic planimetry method, which is completely noninvasive, provides feasible and reliable measurements for quantitating AVA in adult patients with valvular aortic stenosis. Therefore, this method should be utilized repeatedly in combination with the Doppler method in daily clinical settings.

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