Simultaneous Determination of Aortic Valve Area by the Gorlin Formula and by Transesophageal Echocardiography Under Different Transvalvular Flow Conditions

Evidence That Anatomic Aortic Valve Area Does Not Change With Variations in Flow in Aortic Stenosis

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Objectives. The purpose of this study was to determine the impact of changes in flow on aortic valve area (AVA) as measured by the Gorlin formula and transesophageal echocardiographic (TEE) planimetry.

Background. The meaning of flow-related changes in AVA calculations using the Gorlin formula in patients with aortic stenosis remains controversial. It has been suggested that flow dependence of the calculated area could be due to a true widening of the orifice as flow increases or to a disproportionate flow dependence of the formula itself. Alternatively, anatomic AVA can be measured by direct planimetry of the valve orifice with TEE.

Methods. Simultaneous measurement of the planimetered and Gorlin valve area was performed intraoperatively under different hemodynamic conditions in 11 patients. Left ventricular and ascending aortic pressures were measured simultaneously after transventricular and aortic punctures. Changes in flow were induced by dobutamine infusion. Using multiplane TEE, AVA was planimetered at the level of the leaflet tips in the short-axis view.

Results. Overall, cardiac output, stroke volume and transvalvular volume flow rate ranged from 2.5 to 7.3 liters/min, from 43 to 86 ml and from 102 to 306 ml/min, respectively. During dobutamine infusion, cardiac output increased by 42% and mean aortic valve gradient by 54%. When minimal flow was compared with maximal flow, the Gorlin area varied from (mean ± SD) 0.44 ± 0.12 to 0.60 ± 0.14 cm² (p < 0.005). The mean change in Gorlin area under different flow rates was 36 ± 32%. Despite these changes, there was no significant change in the planimetered area when minimal flow was compared with maximal flow. The mean difference in planimetered area under different flow rates was 0.002 ± 0.01 cm² (p = 0.86).

Conclusions. By simultaneous determination of Gorlin formula and TEE planimetry valve areas, we showed that acute changes in transvalvular volume flow substantially altered valve area calculated by the Gorlin formula but did not result in significant alterations of the anatomic valve area in aortic stenosis. These results suggest that the flow-related variation in the Gorlin AVA is due to a disproportionate flow dependence of the formula itself and not a true change in valve area.

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Aortic valve area (AVA) has proved to be a valuable index for the clinical management of patients with valvular aortic stenosis. Historically, this value has been derived from the Gorlin formula utilizing catheterization data and, more recently, from the continuity equation using both two-dimensional and Doppler data. Hemodynamic studies have revealed that the AVA calculated from the Gorlin equation is not constant but varies with flow (1–7). The meaning of these flow-related changes in AVA remains controversial. Flow dependence could be due to a true widening of the orifice as flow increases or to a disproportionate flow dependence of the formula. Thus, the data presently available do not delineate clearly whether the degree of stenosis really does vary with changes in the valve-opening force. The determinants of this force are the transvalvular flow and velocity and systolic left ventricular pressure. Therefore, hemodynamic and Doppler assessment of the severity of aortic stenosis can present problems in low output states (7).

Alternatively, the anatomic AVA can be measured reliably by planimetry from short-axis images obtained with transesophag-
ageal echocardiography (TEE) (8–13). The planimetered AVA correlated well with both the Gorlin and continuity equations in three studies (8,10,13). Good correlation was also obtained when the planimetered valve area was compared with direct intraoperative measurement of the anatomic area by the surgeon in another study (9). Recently, it was demonstrated (13) that acute changes in stroke volume do not result in significant alterations in the AVA measured with multiplane TEE. However, no study exists in which simultaneous catheterization and TEE were performed to evaluate fluctuations in the Gorlin valve area and to compare them with anatomic planimetered AVA. Such data are essential to understand the significance of flow-related changes in Gorlin valve area and, in particular, to test the concept of overdependence of flow of this calculated area. The purpose of the present study was to determine the impact of changes in flow on the AVA by simultaneous determination of Gorlin formula and TEE planimetry valve areas in patients with aortic stenosis.

Methods

Study patients. The study protocol was approved by the institutional research and ethics committees of the Montreal Heart Institute. Written informed consent was obtained from each patient. The study included 11 patients (3 men, 8 women; 38 to 82 years old, mean age 63) who were undergoing aortic valve replacement. AVA measured with transthoracic echocardiography ranged from 0.24 to 0.70 cm² (mean ± SD) 0.58 ± 0.15). Mean pressure gradient measured with transthoracic echocardiography ranged from 38 to 95 mm Hg (mean 61 ± 15). Aortic regurgitation was mild or absent in all patients, according to standard angiographic and transthoracic echocardiographic criteria. Because of the need for dobutamine infusion during the study, patients with a coronary artery narrowing >50% in diameter at angiography were excluded. Simultaneous determination of Gorlin formula and TEE planimetry AVAs were performed in the operating room under different transvalvular flow conditions.

Hemodynamic monitoring. A pulmonary artery catheter was inserted before intubation of the patient. After sternotomy and before going on cardiopulmonary bypass, left ventricular and ascending aortic punctures were performed using fine needles (21 gauge). The fluid-filled catheters were manipulated to avoid left ventricular entrapment and to obtain artifact-free ascending aortic pressure tracings. Left ventricular and ascending aortic pressures were measured simultaneously. Monitoring of heart rate and left ventricular, ascending aortic, right atrial and pulmonary artery pressures was continuously done throughout the protocol. Cardiac output was determined by thermodilution with averaging of three measurements.

Multiplane TEE. We used a multiplane TEE instrument (Omniplane, Hewlett-Packard) composed of a 64-element phased-array transducer that has a dual-frequency feature allowing two-dimensional imaging at 5 and 3.7 MHz (14). The transducer array can be rotated through a 180° arc. The multiplane probe is interfaced to the ultrasound imaging console (HP Sonos 1500, Hewlett-Packard) and the operation of the console is similar to that of conventional echocardiography. The entire examination was recorded on 0.5-in. (1.27 cm) VHS videotape. The procedure was performed as previously described (14). The probe was introduced after the induction of general anesthesia and tracheal intubation. The first part of the examination consisted of image acquisition at different levels and in various planes to perform a systematic evaluation of cardiovascular structures. Examination of the aortic valve was then performed. To define the optimal level of the transducer location for subsequent planimetry of the aortic valve in the short-axis view, the leaflets tips were initially positioned in the center of the two-dimensional sector in the long-axis view of the aortic valve and ascending aorta (110° to 160°). With the transducer position held stable, the ultrasound array was steered to obtain a short-axis view of the aortic valve. This was usually possible between 30° and 70°. Minimal probe manipulation was performed to ensure that the smallest orifice of the aortic valve (at its tips) was identified. The view was considered adequate for planimetry if the aorta had a circular shape and all aortic cusps were visualized simultaneously. Special care was taken to optimize gain settings and grayscale. The short-axis view of the aortic valve was then held stable before and during changes in cardiac output.

Analysis of hemodynamic data. Mean transvalvular aortic pressure gradient was derived from planimetry of simultaneous high speed left ventricular and ascending aortic pressure recordings. AVA was calculated using the Gorlin formula: Cardiac output/(Heart rate × Systolic ejection period × 44.3 × √ΔP). Mean aortic pressure gradient and Gorlin valve area were calculated off-line separately at baseline and at steady state dobutamine infusion, with the observer blinded to results of transthoracic and TEEs, and to results obtained with different hemodynamic conditions. Transvalvular flow rate was obtained by dividing stroke volume (Cardiac output/Heart rate) by the systolic ejection period.

Planimetry of aortic valve by multiplane TEE. AVA was determined off-line at baseline and at steady state dobutamine
infusion. All measurements were done by planimetry of the aortic valve in the short-axis view on the same imaging console using the multiplane images considered most appropriate by an experienced observer. The observer had no knowledge of the results of cardiac catheterization and TEE, the changes in intraoperative hemodynamic findings and the results of planimetry obtained with different hemodynamic profiles. At least three consecutive measurements of AVA were averaged for a given hemodynamic profile in patients in sinus rhythm; no patient had atrial fibrillation in this study. Intraobserver variability was determined by reanalysis by a single observer at an interval of at least 2 weeks. Interobserver variability was determined by reanalysis of all the measurements by a single observer at an interval of at least 2 weeks. Interobserver variability was determined by independent analysis by two blinded experienced observers. Intraobserver and interobserver variability for TEE measurements of aortic valve area is 7.9 ± 6.2% and 11.6 ± 8.8%, respectively.

**Ability of multiplane TEE to detect changes in AVA.** The true orifice area of aortic bioprostheses is known to decrease at very low flow (1). To demonstrate the ability of multiplane transesophageal imaging to detect actual changes in valve area, we studied four other patients with aortic bioprostheses under different flow conditions. After aortic valve replacement, multiplane TEE was performed as patients were very slowly weaned from cardiopulmonary bypass (from total extracorporeal circulation), in 0.25-liter/min increments for 1 liter and in 0.5-liter/min increments thereafter.

**Statistical analysis.** Results are presented as mean value ± SD. Because we were studying the effect of flow on valve area, and not the effect of dobutamine itself, data were compared at minimal (lower flow rate) and maximal (higher flow rate) flow conditions for each patient. For patients in whom there was a decrease in flow rate despite an increase in cardiac output during dobutamine infusion, results obtained during dobutamine infusion represented minimal flow rate, and baseline values had to be taken as maximal flow rate data. A similar analysis was performed comparing minimal with maximal stroke volume. Measurements of Gorlin formula or TEE planimetry AVAs were compared at minimal and maximal transvalvular flow conditions (both flow rate and stroke volume) by using a paired t test. Differences were considered significant at p < 0.05.

**Results**

**Description of valve morphology and mobility.** In all patients, we were able to obtain short-axis images of the aortic valve adequate for measurement (Fig. 1 and 2). The valve orifice at its tips, and its shape, were well displayed in the images at all hemodynamic settings. The leaflets were thickened in all patients and mildly or moderately calcified in seven patients; four aortic valves were severely calcified. Differentiation of bicuspid from tricuspid valve morphology was occasionally difficult in these patients with aortic valve calcification. However, five patients appeared to have a bicuspid aortic valve. After quantitative measurements were done in blinded and random manner, the recordings of each patient at baseline and during infusion of dobutamine were reviewed together. There was no significant difference in the systolic mobility of the aortic valve leaflets from minimal to maximal transvalvular flow rate, in the long-axis view.

**Effects of changes of transvalvular flow on Gorlin formula AVA.** Overall, cardiac output and mean transvalvular aortic pressure gradient ranged from 2.5 to 7.3 liter/min and from 34 to 111 mm Hg, whereas stroke volume and transvalvular volume flow rate ranged from 43 to 86 ml and from 102 to 306 ml/s, respectively (Table 1). Cardiac output increased from 3.9 ± 1.1 liters/min at baseline to 5.6 ± 1.1 liters/min during dobutamine infusion, representing a 42 ± 27% increase from baseline (p < 0.0001). Mean aortic pressure gradient varied from 51 ± 12 mm Hg at baseline to 78 ± 19 mm Hg during dobutamine infusion, a 54 ± 24% increase (p < 0.0001) (Fig. 1 and 2). Transvalvular volume flow rate increased from 145 ± 33 to 217 ± 41 ml/s and stroke volume varied from 50 ± 12 to 66 ± 10 ml from minimal to maximal flow conditions (p ≤
The percent increases in transvalvular flow rate and stroke volume were 53% and 38%, respectively.

When minimal and maximal transvalvular volume flow rates were compared, the Gorlin formula AVA varied from $0.45 \pm 0.12 \text{ cm}^2$ to $0.60 \pm 0.14 \text{ cm}^2$ ($p < 0.01$). Because there was a minor divergent response of flow rate and stroke volume during dobutamine infusion in one patient, we also compared valve area from minimal to maximal stroke volume. In this analysis, the Gorlin valve area increased from $0.44 \pm 0.12 \text{ cm}^2$ to $0.60 \pm 0.14 \text{ cm}^2$ ($p < 0.005$). The mean difference in Gorlin valve area under different stroke volumes ranged from 0.02 to 0.48 cm$^2$ (Fig. 3).

Effects of changes of transvalvular flow on TEE planimetry AVA. Despite these changes in transvalvular flow and Gorlin valve area, there was no significant change in AVA measured with TEE planimetry when minimal and maximal transvalvular volume flow rates were compared (from $0.51 \pm 0.15 \text{ cm}^2$ to $0.51 \pm 0.15 \text{ cm}^2$). The mean difference in planimetered aortic valve area under different flow rates was $0.002 \pm 0.01 \text{ cm}^2$ ($p = 0.86$). The absolute change ranged from 0.02 to 0.02 cm$^2$; the largest increase in planimetered valve area from minimal to maximal transvalvular flow rate was 0.02 cm$^2$ in one patient (Fig. 3).

Ability of multiplane TEE to detect changes in AVA. The bioprosthetic orifice area (measured with multiplane TEE) gradually increased from 0.87 to 1.30 cm$^2$ for 21-mm valves and from 1.46 to 1.75 cm$^2$ for 23-mm valves when normal anterograde flow was increased from 0.25 to 2.0 liters/min (Fig. 4) as patients were weaned from cardiopulmonary bypass after aortic valve replacement. The orifice area remained stable thereafter. The smallest change in valve area detected in these patients was 0.05 cm$^2$.

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*Data are presented in the text at minimal and maximal flow rates for each patient. For patients in whom there was a decrease in flow rate (despite an increase in cardiac output) during dobutamine infusion (Patients 1 and 11), results obtained during dobutamine infusion represent minimal flow rate data. AVA = Gorlin aortic valve area (cm$^2$); AVR = aortic valve resistance (dynes s x cm$^{-5}$); CO = cardiac output (liters/min); FR = flow rate (ml/s); HR = heart rate (beats/min); ΔP = mean pressure gradient using catheterization data (mm Hg); SEP = systolic ejection period (s); SV = stroke volume (ml); TEE = transesophageal echocardiographic planimetry of aortic valve area (cm$^2$).
In our study, simultaneous determination of Gorlin formula and TEE planimetry AVAs shows that changes in transvalvular flow and in the Gorlin valve area were not associated with alterations of the directly planimetered valve area.

Development of Gorlin formula. Peak to peak and mean pressure gradients measured invasively are useful indexes of the severity of aortic stenosis. However, these values are known to vary with flow, which can lead to underestimation of the severity of the lesion in patients with low cardiac output and low pressure gradient. By taking into account both cardiac output and pressure gradient, Gorlin and Gorlin (17) in their pioneer work of 1951 expected to eliminate dependence on flow. They developed their formula based on the Torricelli model of nonturbulent flow through an orifice where flow was proportional to the square root of the pressure gradient. A constant, accounting for orifice contraction and energy loss as pressure energy is converted into kinetic energy, had to be introduced. For calculation of mitral valve area, a constant (0.83) was determined by correlating the calculated mitral valve areas of 11 patients with measurements obtained at autopsy or by intraoperative digital palpation (17). When calculating the AVA, Gorlin and Gorlin empirically decided to use a constant of 1.

Limitations of Gorlin formula in aortic stenosis. Although the formula has been used clinically for many years, confirmation of its accuracy as applied to AVA is still uncertain (18). Indeed changes have frequently been observed in the calculated AVA with modifications in cardiac output (1–7). This may be explained by two factors. A true widening of the orifice area could occur with an increase in the valve-opening force (19), which is related to left ventricular pressure, stroke volume and transvalvular flow rate. Alternatively, the increased area with increased flow could be due to a disproportionate flow dependence of the formula itself. Efforts have been made to try to solve the controversy. In one study (2), the AVA calculated with the Gorlin formula increased in all the patients who received dobutamine, whereas the AVA measured with the continuity equation did not change. This finding suggested that changes in the calculated valve area reflected flow dependence of the Gorlin formula. Additionally, it has been demonstrated (1) that the constant in the Gorlin formula is not constant but varies directly with the square root of the pressure gradient. A new equation was therefore proposed by Cannon et al. (1) who used the first power, rather than the square root of the gradient, to increase the importance of the gradient and make the formula less flow dependent. However, this equation has not yet been validated with native valvular
aortic stenosis and is not in routine clinical use. Using the first power of the pressure gradient, aortic valve resistance has also been shown (20) to be less dependent on flow than the area calculated with the Gorlin equation. Importantly, resistance has also been shown (21) to help distinguish truly severe from milder aortic obstruction with similar calculated valve areas.

Our data also point to another potential limitation of area calculation with the Gorlin formula. Although the formula is based on measurements of mean flow and mean pressure gradient, Gorlin and Gorlin (17) added correction factors to allow for correlation with the area of maximally opened valves measured at autopsy or by intraoperative digital palpation. Thus, the Gorlin valve area was corrected to correlate with the instantaneous maximal anatomic area, similar to that obtained with direct echocardiographic planimetry. In our two patients (Patients 1 and 11) who had a decrease in transvalvular flow rate and stroke volume and a higher pressure gradient during dobutamine infusion, there was a resultant decrease in the valve area calculated with the Gorlin formula. However, the planimetric valve area remained stable in these two patients. Because of the predetermined relation between flow rate, stroke volume and pressure gradient in the Gorlin equation, it is impossible to have a higher pressure gradient with a smaller stroke volume and flow rate without a simultaneous decrease in the calculated valve area. However, determinants of fluid dynamics other than changes in kinetic effects are not taken into consideration in the Gorlin formula. It is possible to explain why the anatomic (planimetered) orifice area did not change in Patients 1 and 11 by considering the complexities of pulsatile flow across a stenotic aortic valve and, in particular, the effects of viscous forces and local acceleration on instantaneous pressure drop. An increase in pressure drop due to viscous forces may have occurred during dobutamine infusion in these patients because of the significant decrease in flow rate. Indeed it has been demonstrated (22) that the hydraulic discharge coefficient (Cd) in the Gorlin formula is not constant but can decrease under low flow conditions, causing an underestimation of valve area. The pressure drop caused by flow acceleration, or the force necessary to overcome inertia, may also have increased significantly in Patients 1 and 11, partly due to the large increases in heart rate (from 75 to 115 beats/min and from 48 to 129 beats/min) during dobutamine infusion (23). Indeed these two patients were the only ones in our study with heart rates >100 beats/min and an increase in heart rate >50% with dobutamine infusion. The mean change in heart rate was 111% in these patients but only 16% in the other patients. Thus, although the Gorlin formula was originally intended to correlate with the maximal anatomic valve area, it is occasionally impossible to predict changes in this instantaneous maximal value from the relatively simple Gorlin relation, which uses mean data and only assesses changes in kinetic effects.

Use of continuity equation for calculation of AVA. A noninvasive alternative for the evaluation of aortic stenosis has been made available with the advent of Doppler echocardiography. Using experimental models, Segal et al. (22) have suggested that the continuity equation may be more accurate than the Gorlin formula because a constant does not need to be assumed for energy loss (discharge coefficient) in the former. In a clinical study (2), AVA measured with the continuity equation and valve resistance did not change with increases in cardiac output, despite fluctuations in the values derived with the Gorlin equation. In a more recent animal study with experimentally created aortic stenosis (7), valve area measured with the continuity equation also varied with changes in flow, leading to the hypothesis that the anatomic valve area may change with flow. However, no significant change in the planimetric area of a stenotic valve was observed in vitro over a 250% change in flow rate in another study (1). Potential pitfalls associated with use of the continuity equation include the difficulty of accurately measuring the left ventricular outflow tract diameter and carefully defining the maximal aortic flow velocity and left ventricular outflow tract obesity before flow acceleration. All these observations suggested the absence of an ideal reference standard for assessment of AVA.

TEE planimetry in aortic stenosis. The value of TEE in the assessment of the severity of aortic stenosis has previously been demonstrated (8–13). The anatomic valve area measured with biplane and multiplane TEE has been shown to correlate well with calculation of AVA utilizing the Gorlin formula at catheterization, with the continuity equation during trans-thoracic echocardiography and with direct intraoperative measurement (8–10,13). Nevertheless, potential problems exist for planimetry of the aortic valve with TEE. To measure the AVA accurately, the transducer must be located at the tips of the valve leaflets. The longitudinal motion of the aortic root during the cardiac cycle needs to be taken into account. Confirmation of the appropriate imaging level can be obtained by carefully positioning the valve tips in the center of the sector in the long-axis orientation, and subsequently rotating the ultrasound array from that location to a short-axis view. Furthermore, simultaneous visualization of all the cusps in an adequate short-axis orientation is important before considering an image plane adequate for assessment. In our study, planimetry was possible in all patients, even those with severe calcification or very small valve areas. However, planimetry may occasionally be more difficult to perform in the presence of heavy calcifications. Intraobserver and interobserver variability was low and similar to that reported in previous studies. Importantly, we demonstrated the ability of multplane TEE to detect changes in valve area by studying aortic bioprostheses under different flow conditions. Changes in orifice area had previously been demonstrated (1) in aortic bioprostheses at very low flow, with direct videotape of valve operation in vitro. The smallest change in planimetered area that we detected in our study in aortic bioprostheses was smaller than the flow-related variations in native AVA calculated with the Gorlin formula. Thus, the possibility that multplane TEE was insensitive to true flow-related changes in Gorlin valve area was excluded.

Sprigings et al. (24) observed in three excised stenotic aortic valves that the anatomic valve area (measured from direct
videotaping) increased by an average of 38% when stroke volume was augmented in vitro from 40 to 100 ml. Although their results vary from ours, several aspects of this in vitro model may differ from the clinical scenario (25). In particular, expansion of the aortic root may be an important mechanism contributing to valve opening in vivo, whereas the expansibility of the in vitro model was limited.

**Limitations of the study.** The changes in flow obtained in our patients with aortic stenosis were significant but limited. Previous studies (2–6) that have demonstrated variation in the Gorlin formula valve area had changes in flow that were similar to those obtained in our study. The relative change in Gorlin AVA that we observed with alterations in flow was also similar to the variations reported in those studies (2–7). The data presented here were acquired in patients with severe aortic stenosis. However, similar results were recently obtained with moderate aortic stenosis (13). Although similar data are not available for milder obstruction, possible flow-related variations in patients with greater native valve areas would nevertheless be less significant clinically. Because the 32 patients studied in the present and previous studies (13) were >35 years old, our results may not apply to younger patients with congenital, noncalcific aortic stenosis. Finally, the relatively small size of our patient cohort was another study limitation.

**Clinical implications.** Simultaneous catheterization and multiplane TEE data demonstrating flow-related variation in the Gorlin valve area with a stable anatomic planimetered valve area provides strong evidence for an overdependence of flow in the Gorlin formula in the evaluation of AVA. We also demonstrated in this study the ability of multiplane TEE to detect changes in valve area in other circumstances (aortic bioprostheses). In view of these considerations, planimetry of the AVA with multiplane TEE could offer a more accurate calculation of the orifice area than that obtained with the use of the Gorlin formula in patients with a moderate to severe limitation.

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**References**


