

# Estimation of Aortic Valve Effective Orifice Area by Doppler Echocardiography: Effects of Valve Inflow Shape and Flow Rate

Damien Garcia, Eng, PhD, Philippe Pibarot, DVM, PhD, Champlain Landry, Eng, Amélie Allard, Eng, Boris Chayer, Eng, Jean G. Dumesnil, MD, and Louis-Gilles Durand, Eng, PhD, Sainte-Foy and Montreal, Quebec, Canada

**Background:** The effective orifice area (EOA) is the standard parameter for the clinical assessment of aortic stenosis severity. It has been reported that EOA measured by Doppler echocardiography does not necessarily provide an accurate estimate of the cross-sectional area of the flow jet at the vena contracta, especially at low flow rates. The objective of this study was to test the validity of the Doppler-derived EOA.

**Methods:** Triangular and circular orifice plates, funnels, and bioprosthetic valves were inserted into an in vitro aortic flow model and were studied under different physiologic flow rates corresponding to cardiac outputs varying from 1.5 to 7 L/min. For each experiment, the EOA was measured by Doppler and compared with the catheter-derived EOA and with the EOA derived from a theoretic formula. In bioprostheses, the geometric orifice area (GOA) was estimated from images acquired by high-speed video recording.

**Results:** There was no significant difference between the EOA derived from the 3 methods with the rigid orifices (Doppler vs catheter:  $y = 0.97x + 0.18$

$\text{mm}^2$ ,  $r^2 = 0.98$ ; Doppler vs theory:  $y = 1.00x - 3.60 \text{ mm}^2$ ,  $r^2 = 0.99$ ). Doppler EOA was not significantly influenced by the flow rate in rigid orifices. As predicted by theory, the average contraction coefficient (EOA/GOA) was around 0.6 in the orifice plates and around 1.0 in the funnels. In the bioprosthetic valves, both EOA and GOA increased with increasing flow rate whereas contraction coefficient was almost constant with an average value of 0.99. There was also a very good concordance between EOA and GOA ( $y = 0.94x + 0.05 \text{ mm}^2$ ,  $r^2 = 0.88$ ).

**Conclusions:** In rigid aortic stenosis, the Doppler EOA is much less flow dependent than generally assumed. Indeed, it depends mainly on the GOA and the inflow shape (flat vs funnel-shaped) of the stenosis. The flow dependence of Doppler EOA observed in clinical studies is likely a result of a variation of the valve GOA or of the valve inflow shape and not an inherent flow dependence of the EOA derived by the continuity equation. (J Am Soc Echocardiogr 2004;17:756-65.)

The aortic valve effective orifice area (EOA) is the minimal cross-sectional area of the flow jet, ie, the cross-sectional area of the vena contracta, downstream of a native or bioprosthetic aortic heart valve. The EOA is the standard parameter used for the clinical assessment of aortic valve stenosis severity. It is determined either from Doppler echocardiography by using the continuity equation or from cath-

eterization by applying the Gorlin formula. Many clinical studies have reported that EOA determined by Doppler or by catheter may vary with increasing flow rate.<sup>1-13</sup> Moreover, some studies have suggested that the EOA estimated by Doppler with the use of the continuity equation<sup>14</sup> may underestimate the actual EOA and that a change in the velocity profile within the vena contracta (ie, flat profile at normal flow rates becoming parabolic or semiparabolic at low flow rates) may be the cause for this underestimation.<sup>14</sup> It is also well known that the geometric orifice area (GOA) and, thus, necessarily the EOA of the aortic valve may also increase with flow.<sup>7,15-18</sup> It is, therefore, not clear whether the flow dependence of the Doppler-derived EOA that is often reported in patients with native or prosthetic heart valves undergoing stress echocardiography is due to an actual change in the GOA and/or to an error related to the limitations of the continuity equation in the context of low flow rates. Our in vitro study was, therefore, designed to: (1) determine whether Doppler echocardiography measures

From the Laboratoire de génie biomédical, Institut de Recherches Cliniques de Montréal (D.G., P.P., C.L., A.A., B.C., L-G.D.); and Research Center of Laval Hospital/Quebec Heart Institute, Laval University, Ste-Foy (P.P., J.G.D.).

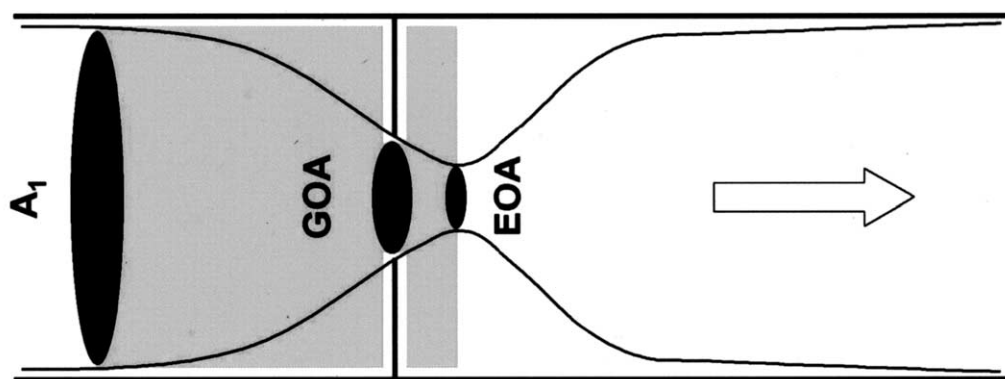
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Reprint requests: Philippe Pibarot, DVM, PhD, Laval Hospital, 2725 Chemin Sainte-Foy, Sainte-Foy, Quebec, Canada, G1V 4G5 (E-mail: [philippe.pibarot@med.ulaval.ca](mailto:philippe.pibarot@med.ulaval.ca)).

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**Figure 1** Schematic representation of flow through rigid orifice plate.  $A_1$ , Inlet cross-sectional area;  $EOA$ , effective orifice area;  $GOA$ , geometric orifice area.

the actual  $EOA$ ; (2) determine whether  $EOA$  is flow dependent under physiologic conditions; and (3) analyze the relationship between  $GOA$  and  $EOA$ . For this purpose, we tested several flat sharp-edged orifice plates, funnel-shaped orifices, and bioprosthetic valves under physiologic flow rates corresponding to cardiac outputs varying between 1.5 and 7 L/min.  $EOA$  was measured using 3 independent methods: Doppler; catheter; and theory of jets.  $GOA$  was measured by high-speed videocamera.

## METHODS

### Theoretic Background

As the flow passes through an orifice plate (area =  $GOA$ ), a jet is formed (Figure 1). The location where the cross-sectional area of the jet is minimal is known as the vena contracta (area =  $EOA$ ). Downstream of the vena contracta a turbulent mixing region occurs, which induces an energy loss ( $E_L$ ).<sup>19,20</sup>

#### Relationship between $EOA$ and $GOA$ for a rigid stenosis.

We have previously shown that the  $E_L$  caused by a stenosis can be expressed as a function of the  $EOA$ , the flow rate  $Q$ , and the outlet cross-sectional area  $A_2$ . For an aortic stenosis,  $A_2$  corresponds to the aortic cross-sectional area measured at the sinotubular junction,<sup>19,21</sup> and under steady conditions:

$$E_L = \frac{1}{2} \rho Q^2 \left( \frac{1}{EOA} - \frac{1}{A_2} \right)^2 \quad (1)$$

where  $\rho$  is the density of the fluid. In 1953, Idelchik<sup>22,23</sup> showed that the pressure loss associated with a flat sharp-edged (Figures 1 and 2) orifice plate located in a straight tube can be written as a function of the  $GOA$  as follows<sup>23</sup>:

$$E_L = \frac{1}{2} \rho Q^2 \frac{1}{GOA^2} \left( 1 + \frac{\sqrt{2}}{2} \sqrt{1 - \frac{GOA}{A_1}} - \frac{GOA}{A_2} \right)^2 \quad (2)$$

where  $A_1$  and  $A_2$  are the inlet and outlet cross-sectional areas, respectively. By combining equations 1 and 2, one can deduce the relation between  $EOA$  and  $GOA$  for a flat, sharp-edged, rigid stenosis:

$$EOA = GOA \left( 1 + \frac{\sqrt{2}}{2} \sqrt{1 - \frac{GOA}{A_1}} \right)^{-1} \quad (3)$$

According to this equation, the  $EOA$  depends both on the inlet cross-sectional area and the  $GOA$ . Interestingly, 30 years later, Grose<sup>24</sup> developed a similar equation from a semi-empirical formulation. Although this equation has been derived under the assumption of a high Reynolds steady flow, we assume it is still valid with a transvalvular flow because the latter is highly transient. According to equation 3, the contraction coefficient ( $C_c$ ) defined as  $EOA/GOA$  is not dependent on the outlet geometry. One can also note that when  $GOA/A_1$  tends toward 0,  $C_c$  converges toward the limit value  $2/(2 + \sqrt{2}) \approx 0.59$ . For a funnel-shaped orifice (Figure 2) the theoretic relation between  $EOA$  and  $GOA$  is straightforward<sup>23</sup>:

$$EOA = GOA \quad (4)$$

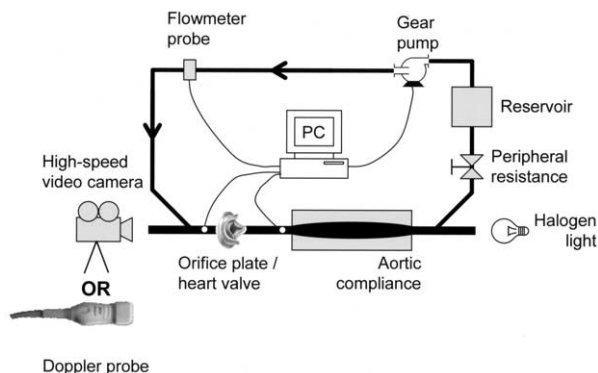
It is hypothesized that this equation is also applicable to noncalcified native or bioprosthetic aortic valves. Hence, for a funnel-shaped orifice,  $C_c$  is close to 1.0.

### In Vitro Study

**Mock flow model.** The mock flow circulation model used in this study was made up of a reservoir, a stenotic orifice or bioprosthetic valve, a compliant aortic chamber, and valve resistance (Figure 3). The pulsatile flow was provided by a computer-controlled direct-current motor coupled to a gear pump (Vi-Corr, Viking Pump, Cedar Falls, Ia). The left ventricular (LV) outflow tract and the ascending aorta were both circular and rigid. The aortic compliance consisted of a rubber tube enclosed within a hermetic plexiglas box, filled with water and air, and connected to an air vacuum/compressor. To avoid any



**Figure 2** Schematic representation of flat sharp-edged and funnel-shaped orifices used for this in vitro study.



**Figure 3** Schema of the aortic mock flow model. *PC*, Personal computer; *two white dots*, locations where ventricular and aortic pressures were measured.

regurgitation with the fixed orifices, a large mitral bioprosthesis (33 mm) (Medtronic Intact, Minneapolis, Minn) was inserted in the rigid aortic section just before the compliant one at 25 cm downstream of the stenotic orifice. Its size and position were chosen so that the bioprosthesis did not disturb the flow through the stenosis or the measurements. The fluid was composed of two-thirds water and one-third glycerol so that its density (1.08<sup>o</sup>) and viscosity (3.5 cP) were similar to that of blood under high shear rate. The scattering of the Doppler ultrasound waves was obtained by the addition of microbubbles in the liquid. The flow control and data acquisition were performed with a custom software (LabView, National Instruments, Austin, Tex). The flow rate was measured by an electromagnetic flowmeter (Cliniflow II, Carolina Medical Electronics, King, NC), and the LV and aortic pressures with Millar catheters under a sampling frequency of 300 Hz. The LV pressure was measured 2 cm upstream from the stenotic (or valve) orifice. The aortic pressure was measured in the rigid aortic section 15 cm downstream of the stenotic (or valve) orifice where the pressure is totally recovered. The velocity at the venia contracta was measured by continuous wave Doppler using an Ultramark 9 HDI or a HP Sonos 5500 (Philips Medical Systems, Boston, Mass). Ten cycles were recorded for each experiment, and the average values were used to calculate the hemodynamic parameters.

**Video recordings of bioprostheses.** To quantify their GOA, bioprosthetic valves were back-illuminated and filmed with a high-speed video camera (Dalstar CA-D6, Dalsa, Waterloo, Ontario, Canada) at 860 frames/s with a

256 × 256 pixel resolution. The camera was coupled with a board and driver (Viper-Digital, Coreco, Montreal, Quebec, Canada). The video images were converted to binary by thresholding. For every movie, the threshold was computed from the most luminous frame (corresponding to the largest GOA) using the method of Otsu.<sup>25</sup> The values of instantaneous GOA were determined after having calibrated the system with fixed orifices having precise known values of GOA. The mean GOA was calculated from the curve of instantaneous GOA.

**Protocol.** A total of 7 circular (60, 80, 120, 190, 250, 320, and 500 mm<sup>2</sup>) and 3 triangular (80, 110, and 155 mm<sup>2</sup>) rigid flat sharp-edged orifice plates (Figure 2) were tested under 7 pulsatile flow rates (cardiac outputs varying from 3-7 L/min). Because, according to equation 3, the size of the inflow section may have an impact on the EOA, 3 inflow sections with different cross-sectional areas (300, 500, and 800 mm<sup>2</sup>; diameters of 19, 25, and 32 mm, respectively) were tested. The cross-sectional area of the rigid aortic section was 800 mm<sup>2</sup> (diameter: 32 mm). The duration of the cardiac cycle and of the systole was maintained at 857 ± 0 milliseconds and 341 ± 27 milliseconds, respectively (70 bpm with a 40% systolic period). To further study the effect of very low flow rates on Doppler and catheter EOAs, the 60 mm<sup>2</sup> circular, 80 mm<sup>2</sup> circular, and 80 mm<sup>2</sup> triangular orifices were also tested under the additional following cardiac outputs: 1.5; 2.2; and 3.0 L/min. Three rigid funnels (65, 100, and 120 mm<sup>2</sup>) were evaluated under the same conditions. Four bioprostheses (Mosaic, Medtronic) (19, 21, 23, and 25 mm) were also tested under 6 pulsatile flow rates (cardiac outputs varying from 1.3-7.5 L/min).

**Calculation of Doppler and catheter EOAs.** Doppler EOA was estimated using the standard continuity equation by dividing the stroke volume measured by the electromagnetic flowmeter by the Doppler velocity-time integral. It should be noted that we calculated the stroke volume during the ejection period, ie, when the flow rate *Q* was greater than 0. Catheter EOA was calculated from the mean systolic net pressure gradient (ie, mean gradient after pressure recovery [*TPG<sub>net</sub>*]), the instantaneous flow *Q*, and the cross-sectional areas of the inlet (*A<sub>1</sub>*) and outlet (*A<sub>2</sub>*) rigid sections, by using the following formula (appendix):

$$EOA = \left[ \frac{1}{A_2} + \sqrt{\left( \frac{1}{A_1^2} - \frac{1}{A_2^2} \right) + \frac{2 \overline{TPG_{net}}}{\rho Q^2}} \right]^{-1} \quad (5)$$

where  $\rho$  is the fluid density (1080 g/L). The overline denotes the systolic mean. This formula, rather than the Gorlin formula, has been chosen because we have previously shown that the Gorlin formula may underestimate the EOA, especially in the presence of a small aorta.<sup>21</sup> Because the measurement errors of the catheters are of the order of 1 to 2 mm Hg, the catheter EOA was not calculated when the mean *TPG<sub>net</sub>* was less than 5 mm Hg to avoid large calculation errors.

It is important to emphasize that the equation of the catheter-derived EOA used in the current study (equation

5) is quite different from the Gorlin formula. Because this equation takes into account the pressure recovery and the dynamic pressure gradient, it reflects the actual cross-sectional area of the vena contracta. As opposed to equation 5, we have previously shown that the Gorlin formula overestimates the actual EOA as a result of the pressure recovery phenomenon.<sup>21</sup> In this previous study, the observed overestimation was, on average, 24% and it varied markedly depending on the size of the aorta.

### Data and Statistical Analyses

The Doppler EOAs were compared with the EOAs obtained from theoretic equations 3 and 4 for the flat sharp-edged and the funnel-shaped orifices, respectively. The EOA measurements obtained by Doppler were also compared with those obtained by catheter (equation 5). All comparisons were performed using a linear regression and a Bland-Altman plot.<sup>26</sup> The Doppler EOAs of the bioprostheses were compared with their mean GOAs determined by the video recordings.

## RESULTS

### EOA: Doppler Versus Catheter

As shown in Figure 4, there was a very good agreement between Doppler and catheter EOAs ( $y = 0.97x + 0.18$ ,  $r^2 = 0.98$ , standard error of the estimate =  $5.4 \text{ mm}^2$ ). The mean relative difference between the two methods was  $-3.5\% \pm 5.2\%$ . This result confirms that the Doppler EOA derived from the standard continuity equation and the catheter EOA derived from the mean  $TPG_{\text{net}}$  using equation 5 are equivalent parameters that reflect the cross-sectional area of the vena contracta.

### EOA: Doppler Versus Theory

As shown in Figure 5, there was also a very good agreement ( $y = 1.0x - 3.6$ ,  $r^2 = 0.99$ , standard error of the estimate =  $4.6 \text{ mm}^2$ ) between Doppler EOA and theoretic EOA derived from the GOA and the inlet cross-sectional area (equations 3 and 4). The mean relative error was  $-3.4\% \pm 4.8\%$ . This result further confirms that Doppler EOA reflects the cross-sectional area of the vena contracta.

### EOA Versus GOA in Bioprostheses

As shown in Figure 6, a very good concordance between Doppler EOA and GOA was observed ( $y = 0.94x + 5.3$ ,  $r^2 = 0.88$ , standard error of the estimate =  $8.4 \text{ mm}^2$ ). The mean relative error was  $-0.9\% \pm 8.2\%$ . This result suggests that the EOA tends to be equal to the GOA in normal bioprosthetic heart valves.

### Effect of Flow Rate on GOA and EOA

The mean values  $\pm$  SD of the Doppler EOAs calculated with the  $60 \text{ mm}^2$  circular,  $80 \text{ mm}^2$  circular, and

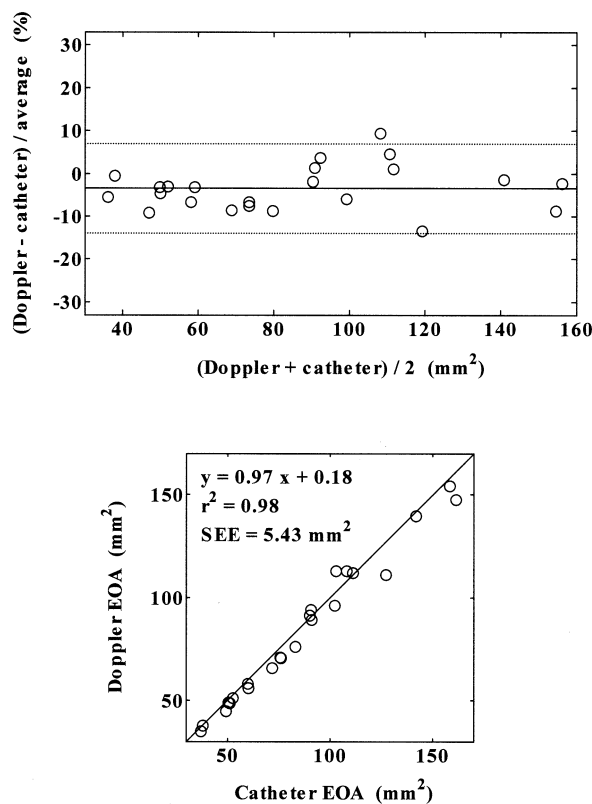
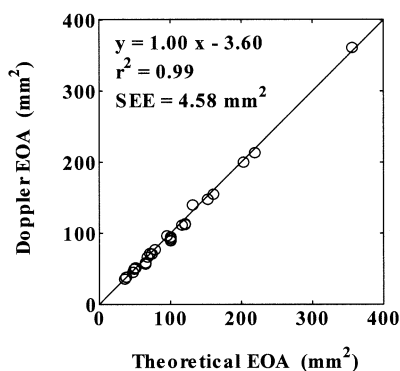
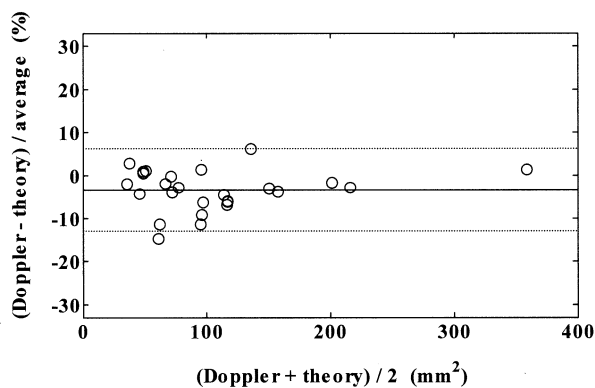


Figure 4 Comparison between Doppler effective orifice area (EOA) and catheter EOA as calculated by equation 5. Differences in Bland-Altman plot are expressed as percentage of averages (*top*). SEE, Standard error of the estimate; solid line in bottom panel, identity line.

$80 \text{ mm}^2$  triangular orifices at cardiac outputs varying from 1.5 to 6.5 L/min were  $37.5 \pm 1.63 \text{ mm}^2$ ,  $47.5 \pm 1.63 \text{ mm}^2$ , and  $47.5 \pm 0.82 \text{ mm}^2$ , respectively, by Doppler and  $38.8 \pm 0.78 \text{ mm}^2$ ,  $49.9 \pm 1.11 \text{ mm}^2$ , and  $49.3 \pm 1.10 \text{ mm}^2$ , respectively, by catheter. Overall, the relative deviation from the mean value remained less than 6%. No correlation was observed between mean transvalvular flow rate and EOA. Figure 7 illustrates the effect of flow rate on GOA and Doppler EOA. These results show that, when GOA is constant as in rigid orifice plates, EOA does not significantly depend on flow under normal and low cardiac output conditions. Conversely, Figure 7 shows that the GOA and the Doppler EOA increased with increasing cardiac output for the bioprostheses. For a cardiac output of  $1.37 \pm 0.04 \text{ L/min}$ , the bioprostheses opened at about 70% and 80% of their maximal EOA and GOA capacities, respectively. These results are concordant with the changes in EOA observed for patients undergoing stress echocardiography.<sup>3,13,27</sup>

Figure 8 shows the effect of flow rate on the  $C_C$ . There was no significant change in the  $C_C$  with decreasing flow rate in rigid orifices and its value

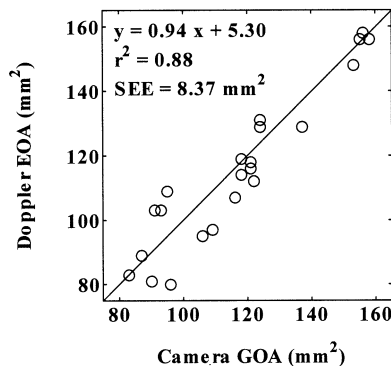
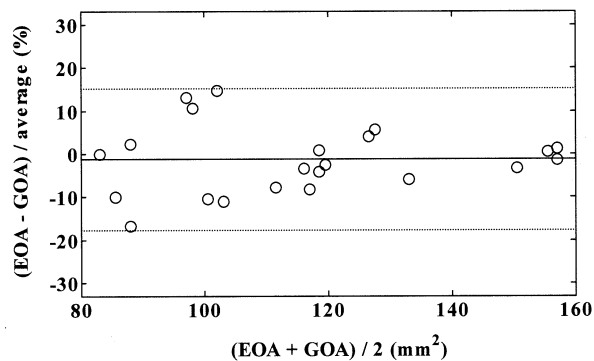


**Figure 5** Comparison between Doppler effective orifice area (EOA) and theoretic EOA as calculated by equations 3 (flat sharp-edged orifice) and 4 (funnel-shaped orifice). Differences in Bland-Altman plot are expressed as percentage of averages (*top*). SEE, Standard error of the estimate; *solid line in bottom panel*, identity line.

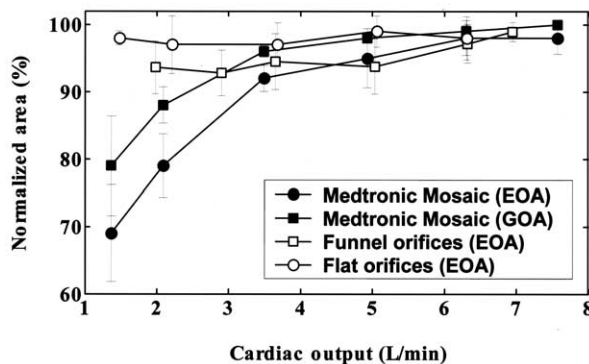
was around 0.6 ( $0.63 \pm 0.05$ ,  $n = 126$ ). In bioprosthetic valves,  $C_c$  was around 1.0 ( $0.99 \pm 0.28$ ,  $n = 14$ ) and tended to decrease at low flow rates. In the rigid funnel-shaped orifices  $C_c$  was around 0.9 ( $0.92 \pm 0.03$ ,  $n = 56$ ) and did not change significantly with flow.

**DISCUSSION**

Historically,<sup>28</sup> Gorlin and Gorlin<sup>29</sup> first proposed to calculate the aortic valve area by catheter using pressure and flow measurements. In their article of 1951,<sup>29</sup> the Gorlin formula, written as  $EOA = Q / (C \cdot 44.3 \sqrt{TPG})$ , contains an empirical constant C that was not tested with aortic valve stenoses and was assumed to be 1 in further studies. Thus, the classic Gorlin formula is computed from the mean systolic flow rate Q and the mean transvalvular pressure gradient (TPG) as follows:  $EOA = Q / (44.3 \sqrt{TPG})$ . Because the discrepancy between the catheter and the Doppler estimates of valve EOA has been shown

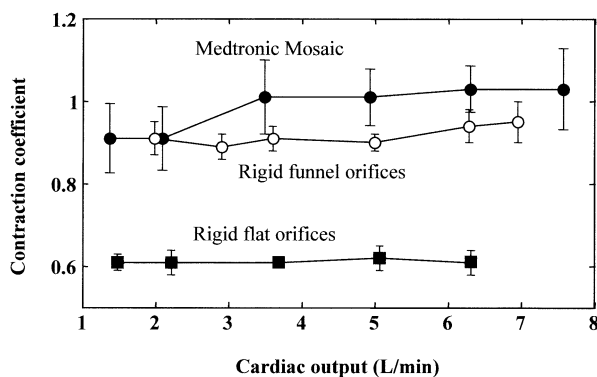


**Figure 6** Comparison between Doppler effective orifice area (EOA) and geometric orifice area (GOA) for bioprosthetic valves. Differences in Bland-Altman plot are expressed as percentage of averages (*top*). SEE, Standard error of the estimate; *solid line in bottom panel*, identity line.



**Figure 7** Doppler effective orifice area (EOA) and geometric orifice area (GOA) both normalized to maximal values. *Closed circles*, EOA of bioprosthetic valves; *closed squares*, GOA of bioprosthetic valves; *open circles*, EOA of sharp-edged orifice plates studied from low to high flow rates (see protocol description); *open squares*, funnel orifices.

to increase with decreasing flow rate,<sup>6,9,12,30</sup> the error has been attributed either to the flow dependence of the empiric constant in the Gorlin formu-



**Figure 8** Contraction coefficient as function of cardiac output. *Closed circles*, bioprosthesis valves; *open circles*, funnel orifices; *closed squares*, flat orifices.

la<sup>6,12</sup> or to the overestimation of the mean velocity by continuous wave Doppler in the continuity equation at low flow rates.<sup>14</sup>

#### Effect of Flow Rate on Doppler EOA and Validity of the Continuity Equation

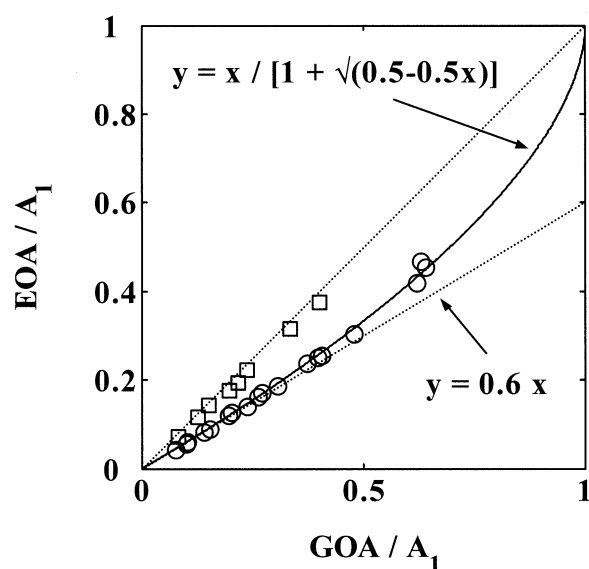
In a numerical study using finite element method, DeGroff et al<sup>14</sup> concluded that the velocity profile at the vena contracta becomes parabolic at low flow rates, thus, causing an underestimation of the EOA by Doppler echocardiography. However, this study was performed using flow conditions that are not representative of the flow conditions observed for patients. First, they obtained these results in conditions of extremely low flow rates (cardiac output as low as 0.4 L/min) that are not compatible with the human circulatory physiology. Indeed, even for patients with severe LV dysfunction, the cardiac output is rarely below 1.5 L/min. Second, they only considered steady-flow conditions, which predispose to the establishment of parabolic flow pattern. In pulsatile flow conditions, the effects of transient flow contribute to flatten the velocity profiles. Finally, the results of this numerical study have not been validated by experimental in vitro or in vivo data. It is, therefore, difficult to ascertain that these theoretic results are directly applicable to the clinical situation. Conversely in the current study, we have performed pulsatile flow experiments using a range of cardiac outputs that is more representative of the normal and abnormal conditions that may occur in adult patients. In addition, the results of this study suggest that, in severely stenotic and rigid orifices, the Doppler EOA is not significantly influenced by flow rate. Moreover, the strong agreement between EOA measurements obtained from 3 different methods suggests that the Doppler estimation of EOA using the continuity equation remains valid at low flow rates in moderate/severe aortic stenosis. Given that the  $C_C$  of bioprosthesis valves tended to

decrease at low flow rates (Figure 8), one can, however, not exclude that the Doppler-derived EOA may slightly underestimate the actual EOA of mildly stenotic or normal valves at low flow rates. This is not a major limitation because this situation is not necessarily of clinical relevance. Indeed, dobutamine stress echocardiography is generally performed to discriminate between moderate and severe aortic stenosis.

The results of this study, therefore, suggest that the flow dependence of Doppler EOA reported in patients with aortic stenosis in the literature may not be a result of an inherent limitation of the continuity equation but rather a variation of GOA with flow. Indeed, as observed in our in vitro study and as mentioned in the literature, the GOA of native and bioprosthesis aortic valves is not necessarily constant. At low flow rates, the force applied against the valve leaflets may not be high enough to completely open the valve. However, when transvalvular flow is increased by exercise or pharmacologic stimulation, the valve leaflets open more widely because of an increase inflow force and the valvular orifice, thus, increases. The resulting increase in GOA is necessarily associated with an increase in EOA. As opposed to previous beliefs, it should be emphasized that more recent studies have shown that the vast majority of patients with aortic stenosis have some valve opening reserve, ie, the stenotic aortic valve is not always completely rigid and has the capacity to enlarge its orifice when flow and, thus, pressure gradient are increased during stress echocardiography.<sup>3,13,27,31</sup>

#### Effect of Valve Inflow Shape on EOA

Figure 9 shows the relationship between EOA and GOA both normalized to the inlet area. As predicted by the theory, sharp-edged orifice EOA was related to GOA and inlet area according to equation 3, and EOA was almost equal to GOA with the 3 funnel-shaped orifices according to equation 4. Because an aortic stenosis becomes significant when EOA/A is less than one-fourth,<sup>32</sup> one can assume, according to Figure 9, that the contribution of the LV outflow tract area to the variation of EOA is minimal in the case of patients with aortic stenosis. Hence, for a significant aortic stenosis, EOA depends principally on the valve orifice inflow shape and the GOA. Table illustrates how the inflow shape may influence the value of the  $C_C$ . With a rigid sharp-edged orifice, the  $C_C$  depends on the angle formed by the aperture. For such an orifice, the  $C_C$  can be estimated from the theory of jets in ideal fluids.<sup>33</sup> As illustrated in Table, the  $C_C$  decreases from 0.8 to 0.6 when the angle increases from 30 to 90 degrees in sharp-edged orifices. In the presence of a rounded edge, assuming a significant curvature radius, the  $C_C$  tends to converge to 1.0.<sup>23</sup> From this latter observation, and



**Figure 9** Relationship between effective orifice areas (EOA) and geometric orifice area (GOA) both normalized to inlet area.  $A_1$ , Inlet cross-sectional area; circles, sharped orifices; squares, funnel-shaped orifices.

as confirmed by our results obtained with the bioprostheses, one may hypothesize that the  $C_C$  is close to 1.0 for a normal native or bioprosthetic valve. The average  $C_C$  calculated from our overall measurements with the bioprostheses was  $0.99 \pm 0.08$  ( $n = 23$ ). Consistently, Chambers et al<sup>7</sup> observed a very good concordance between Doppler EOA and GOA measured in vitro at the peak systole in native and bioprosthetic aortic valves.

Gilon et al<sup>34</sup> have recently studied the effect of valve inflow shape on the  $C_C$  for patients with aortic stenosis. They concluded that the  $C_C$  varied prominently with valve funnel shape, and was largest for long, tapered domes. In their 35 patients,  $C_C$  was significantly lower for flat compared with doming bicuspid valves ( $0.73 \pm 0.14$  vs  $0.94 \pm 0.14$  cm<sup>2</sup>). On the basis of our results and those of Gilon et al,<sup>34</sup> the planimetry method that measures the GOA is likely inadequate to estimate EOA in calcified stenotic valves. Consistently, de la Fuente et al<sup>35</sup> showed that transesophageal planimetry and Doppler provide similar valvular areas in mildly or moderately calcified stenoses. However, Doppler EOA was systematically lower than planimetry GOA for patients with severely calcified valves. Accordingly, the  $C_C$  was  $1.11 \pm 0.28$  in patients with mild/moderate valvular calcification and  $0.79 \pm 0.28$  for patients with severely calcified valves.

Gilon et al<sup>34</sup> also suggested that the increase in pressure gradient occurring during dobutamine or exercise stress echocardiography may change the valve inflow shape. Indeed, the inflow shape that would be relatively flat at low flow rate may become

doming at higher flow rates. This would result in an increase in the  $C_C$  and, thus, of the EOA, and this could occur even in the absence of any increase in GOA. These findings suggest that, besides the measurement errors, the two main mechanisms that may be responsible for the flow-related increase in Doppler EOA are: (1) an increase in GOA as a result of more complete opening of valve leaflets; and/or (2) a change in the valve inflow shape because of the accentuation of the forces applied against the valve leaflets.

### Limitations of the Study






An evident limitation of our study is the lack of a gold standard for determining EOA. Particle image velocimetry could have been used as a gold standard method; however, such a procedure would have limited the number of experiments. Instead, we chose 3 independent methods. The strong agreement obtained between these 3 methods confirms the coherence of the results and suggests that Doppler echocardiography accurately measures the actual EOA.

### Clinical Implications

**Planimetry versus Doppler.** Planimetry of valve orifice area using transesophageal echocardiography<sup>15,36,37</sup> or magnetic resonance imaging<sup>38,39</sup> has been proposed as an alternative to measure the aortic valve area in some patients with aortic stenosis. These methods provide the GOA with a satisfactory accuracy. However, because it is the EOA and the aortic cross-sectional area that determine the LV  $E_L$  induced by the stenosis,<sup>19</sup> GOA is, therefore, not appropriate for the assessment of the hemodynamic burden caused by the stenosis. Indeed, the ratio between EOA and GOA can theoretically be as low as 0.6 with severely calcified stenoses. Moreover, this ratio may vary markedly depending on valve inflow shape (Table). For example, in the patients with a relatively flat stenosis studied by Gilon et al,<sup>34</sup> the  $C_C$  was, on average, 0.73. In these patients, the planimetry method would have greatly underestimated the severity of the stenosis. These findings, therefore, suggest that planimetry is not reliable to assess the hemodynamic severity of aortic stenosis.

**Reliability of Doppler EOA.** Potential sources of error in the determination of the EOA from the continuity equation have been described. First of all, the velocity profile in the outflow tract is not flat but skewed, although the skew is less pronounced for patients with aortic stenosis.<sup>40-43</sup> Nonetheless, it has been shown that the central velocity accurately reflects the average velocity<sup>40,44</sup> and more importantly, Doppler echocardiography has been shown to adequately measure the stroke volume in noncalcified stenotic valves.<sup>45</sup> Other potential sources of error include the measurement of the LV outflow

**Table 1** Contraction coefficient calculated in this study and reported in the literature for different orifice inflow shapes

Valve inflow shape						Mildly/ noncalcified valve	Severely calcified valve
Theoretic $C_c$	0.8 <sup>(33)</sup>	0.7 <sup>(33)</sup>	0.6 <sup>(33)</sup>	1 <sup>(23)</sup>	1 <sup>(23)</sup>	1	$0.6 \leq C_c < 1$
In vitro $C_c$ (this study)			$0.63 \pm 0.05$ n = 126	$0.92 \pm 0.03$ n = 56		$0.99 \pm 0.08$ n = 23	
In vivo $C_c$ (patients)						$1.11 \pm 0.28$ n = 10 <sup>(35)</sup>	$0.79 \pm 0.28$ n = 14 <sup>(35)</sup>
						$0.94 \pm 0.14$ n = 14 <sup>(34)</sup>	$0.73 \pm 0.14$ n = 21 <sup>(34)</sup>

$C_c$ , Contraction coefficient.

Parenthetical numbers are the reference numbers of the corresponding studies.

tract area,<sup>46</sup> the jet eccentricity,<sup>47</sup> and the quality of the Doppler image.<sup>48</sup> In this in vitro study, we also noted that the signal-to-noise ratio of the continuous wave Doppler signal decreases substantially with decreasing velocity, which could contribute to errors in EOA measurements at low flow rates, especially in mild or moderate stenoses. However, this phenomenon appeared less important for severe stenoses in which velocities were relatively high irrespective of flow rate.

As discussed above, there are potential sources of error in the determination of EOA by Doppler echocardiography. However, Doppler echocardiography remains the most reliable method for calculating the actual area of the vena contracta in clinical practice. Indeed, we have previously demonstrated<sup>21</sup> that catheter EOA estimated from the Gorlin formula does not predict EOA, but rather the  $E_L$  coefficient, which also depends on the aortic cross-sectional area. Also the planimetry method provides the GOA and not the EOA. The results of this study suggest that, in severe rigid aortic stenosis, Doppler-derived EOA is flow independent under physiologic conditions when GOA and valve inflow shape are invariant. Thus, changes in Doppler EOA observed during stress echocardiography are likely a result of a concomitant change in the GOA, valve inflow shape, or both.

### Conclusion

In rigid aortic stenosis, the Doppler EOA is much less flow dependent than generally assumed. Indeed, it depends mainly on the inflow area, the GOA, and the inflow shape of the stenosis. The flow dependence of Doppler EOA observed in clinical studies is likely a result of a variation of the valve GOA or of the valve inflow shape and not an inherent flow dependence of the EOA derived by the continuity equation.

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### APPENDIX

We describe hereafter the derivation of the EOA from the mean systolic net transvalvular pressure gradient  $TPG_{net}$ .

The mean energy loss caused by an aortic stenosis can be written as follows<sup>19</sup>:

$$\overline{E_L} = \frac{1}{2} \rho \overline{Q^2} \left( \frac{1}{EOA} - \frac{1}{A_2} \right)^2$$

where  $A_2$  is the outlet cross-sectional area, ie, the aortic cross-sectional area,  $Q$  is the flow rate and  $\rho$  is the fluid density. The overline denotes the systolic mean. The mean energy loss is the sum of the mean  $TPG_{net}$  and the mean dynamic pressure gradient so that:

$$\overline{TPG_{net}} = \overline{E_L} - \frac{1}{2} \rho (\overline{V_1^2} - \overline{V_2^2})$$

where  $V_1$  and  $V_2$  are the inlet and outlet velocities, respectively. If we assume flat velocity profiles, the mass conservation gives  $Q = A_1 V_1 = A_2 V_2$ . Therefore:

$$\overline{TPG_{net}} = \overline{E_L} - \frac{1}{2} \rho \overline{Q^2} \left( \frac{1}{A_1^2} - \frac{1}{A_2^2} \right)$$

Using the expression of the energy loss, one can extract the EOA:

$$EOA = \left[ \frac{1}{A_2} + \sqrt{\left( \frac{1}{A_1^2} - \frac{1}{A_2^2} \right) + \frac{2 \overline{TPG_{net}}}{\rho \overline{Q^2}}} \right]^{-1}$$



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