Discrepancies Between Catheter and Doppler Estimates of Valve Effective Orifice Area Can Be Predicted From the Pressure Recovery Phenomenon

Practical Implications With Regard to Quantification of Aortic Stenosis Severity

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OBJECTIVES
We sought to obtain more coherent evaluations of aortic stenosis severity.

BACKGROUND
The valve effective orifice area (EOA) is routinely used to assess aortic stenosis severity. However, there are often discrepancies between measurements of EOA by Doppler echocardiography (EOA Dop) and those by a catheter (EOA cath). We hypothesized that these discrepancies might be due to the influence of pressure recovery.

METHODS
The relationship between EOA cath and EOA Dop was studied as follows: 1) in an in vitro model measuring the effects of different flow rates and aortic diameters on two fixed stenoses and seven bioprostheses; 2) in an animal model of supravalvular aortic stenosis (14 pigs); and 3) based on catheterization data from 37 patients studied by Schöbel et al.

RESULTS
Pooling of in vitro, animal, and patient data showed a good correlation (r = 0.97) between EOA cath (range 0.3 to 2.3 cm²) and EOA Dop (range 0.2 to 1.7 cm²), but EOA cath systematically overestimated EOA Dop (24 ± 17% [mean ± SD]). However, when the energy loss coefficient (ELCo) was calculated from EOA Dop and aortic cross-sectional area (A A) to account for pressure recovery, a similar correlation (r = 0.97) with EOA cath was observed, but the previously noted overestimation was no longer present.

CONCLUSIONS
Discrepancies between EOA cath and EOA Dop are largely due to the pressure recovery phenomenon and can be reconciled by calculating ELCo from the echocardiogram. Thus, ELCo and EOA cath are equivalent indexes representing the net energy loss due to stenosis and probably are the most appropriate for quantifying aortic stenosis severity.

According to the American College of Cardiology/American Heart Association (ACC/AHA) recommendations (1), the aortic valve effective orifice area (EOA) can be used to grade aortic stenosis severity as follows: mild at >1.5 cm²; moderate at 1.0 to ≤1.5 cm²; and severe at ≤1.0 cm². In the clinical situation, the valve EOA is routinely determined by using either the Gorlin formula during cardiac catheterization or the continuity equation during Doppler echocardiography (2–6). However, there are often discrepancies between catheter- and Doppler-derived valve EOAs, resulting in potentially divergent estimations of aortic stenosis severity. Because catheter pressure measurements are generally performed a few centimeters downstream from the aortic valve, we hypothesized that these discrepancies might be due to the pressure recovery phenomenon, which mainly depends on the size of the ascending aorta (7–16). Thus, the objective of this study was to more closely examine the relationship between catheter (EOA cath) and Doppler (EOA Dop) measurements of EOA to reconcile such measurements and present more coherent evaluations of aortic stenosis severity.

Theoretical background. The transvalvular pressure gradient through a stenotic valve is maximal (TPG max) at the level of the vena contracta. However, it is generally difficult to obtain an adequate measurement of TPG max by a catheter because of the difficulty in adjusting and maintaining the position of the pressure sensor or pressure lumen orifice at the level of the vena contracta, as well as the position instability caused by flow-jet turbulences. Nonetheless, when TPG max (mm Hg) is successfully measured by a catheter, the EOA at the vena contracta (cm²; EOA cath/max) can be calculated as follows using the Gorlin formula:

$$EOA_{cath/max} = \frac{Q}{50 \cdot TPG_{max}}$$

[1]
by this equation:

$$\text{energy loss (EL)}$$

due to aortic stenosis (16), as demonstrated

pressure recovery phenomenon. Hence, the energy loss

measurements of $$\text{EOADop}$$ and $$\text{AA}$$. Because the transvalvu-

that $$\text{ELCo}$$ can be calculated from the echocardiogram using

the traditional Gorlin equation. However, instead of the

aortic stenosis.

where $$\text{AA}$$ is in cm² and $$\text{EL}$$ is in mm Hg. It should be noted

account the cardiac output requirements of the patient. In a

previous study (16), we found that the indexed $$\text{ELCo}$$ (i.e.,

stream from the aortic valve annulus (19).

Abbreviations and Acronyms

$$\text{AA}$$ = cross-sectional area of the aorta
$$\text{EL}$$ = energy loss
$$\text{ELCo}$$ = energy loss coefficient
$$\text{EOA}$$ = effective orifice area
$$\text{EOA}_{\text{carh}}$$ = effective orifice area measured by catheter
$$\text{EOA}_{\text{carh/max}}$$ = effective orifice area measured by catheter

with use of maximal transvalvular pressure gradient

$$\text{EOADop}$$ = effective orifice area measured by Doppler

echocardiography
$$\text{TPG}_{\text{net}}$$ = net transvalvular pressure gradient
$$\text{TPG}_{\text{max}}$$ = maximal transvalvular pressure gradient

where $$\text{Q}$$ is the flow rate in ml/s. Previous studies have
demonstrated that the original Gorlin formula contains
several errors that can be corrected by using a constant of 50
instead of 44.3 (17). To ensure the coherence of the results
from both a theoretical and physiologic standpoint, we thus
elected to use a constant of 50 in Equation 1. Because the
EOA measured by Doppler using the continuity equation
($$\text{EOA}_{\text{Dop}}$$) is also meant to represent the EOA at the vena
contracta, there should theoretically be a close agreement
between $$\text{EOA}_{\text{carh/max}}$$ and $$\text{EOA}_{\text{Dop}}$$.

After the vena contracta, part of the jet kinetic energy
is recovered in pressure, resulting in a net pressure grad-

ient ($$\text{TPG}_{\text{net}}$$) lower than $$\text{TPG}_{\text{max}}$$ and the magnitude of

$$\text{TPG}_{\text{max}} - \text{TPG}_{\text{net}}$$ (i.e., pressure recovery) is dependent on

the valve EOA and the size of the ascending aorta (7–16).

It should be noted that the measurement generally
recorded during cardiac catheterization is $$\text{TPG}_{\text{net}}$$, and

consequently, the EOA reported corresponds to:

$$\text{EOA}_{\text{carh}} = \frac{\text{Q}}{50 \sqrt{\text{TPG}_{\text{net}}}}$$  \[2\]

Recently, we proposed a new index based on $$\text{EOA}_{\text{Dop}}$$ and
aortic cross-sectional area ($$\text{AA}$$) that takes into account the
pressure recovery phenomenon. Hence, the energy loss
coefficient ($$\text{ELCo}$$) provides an accurate estimation of the
energy loss (EL) due to aortic stenosis (16), as demonstrated
by this equation:

$$\text{ELCo} = \frac{\text{EOA}_{\text{Dop}} \times \text{AA}}{\text{AA} - \text{EOA}_{\text{Dop}}} = \frac{\text{Q}}{50 \sqrt{\text{EL}}}$$  \[3\]

where $$\text{AA}$$ is in cm² and EL is in mm Hg. It should be noted that
$$\text{ELCo}$$ can be calculated from the echocardiogram using
measurements of $$\text{EOA}_{\text{Dop}}$$ and $$\text{AA}$$. Because the transvalvu-
lar flow rate at rest is mainly dependent on body size, $$\text{ELCo}$$
can also be indexed for body surface area to take into
account the cardiac output requirements of the patient. In a
previous study (16), we found that the indexed $$\text{ELCo}$$ (i.e.,
EL index) was superior to either $$\text{EOA}_{\text{Dop}}$$ or indexed
$$\text{EOA}_{\text{Dop}}$$ in predicting adverse outcomes in patients with
aortic stenosis.

It is interesting to note that Equation 3 is very similar to
the traditional Gorlin equation. However, instead of the
valve EOA, the left-hand side of the equation represents
$$\text{ELCo}$$, and the right-hand side represents EL in terms of
pressure instead of $$\text{TPG}_{\text{net}}$$.

The EL is the sum of $$\text{TPG}_{\text{net}}$$ and the dynamic pressure
gradient:

$$\text{EL} = \text{TPG}_{\text{net}} + 4(V_V^2 - V_A^2)$$ \[4\]

where $$V_V$$ and $$V_A$$ are the blood velocities (expressed in m/s)
in the left ventricular outflow tract and ascending aorta,
respectively. In patients with aortic stenosis, the dynamic
pressure gradient $$4(V_V^2 - V_A^2)$$ is negligible compared with
$$\text{TPG}_{\text{net}}$$, so that $$\text{EL} \sim \text{TPG}_{\text{net}}$$ and thus $$\text{ELCo} \sim \text{EOA}_{\text{carh}}$$,
according to Equations 2 and 3. Hence, it should theore-
ically be possible to estimate $$\text{EOA}_{\text{carh}}$$ from Doppler echo-

cardiographic data by calculating $$\text{ELCo}$$ using the left-hand
side of Equation 3.

METHODS

In vitro study. The pulse duplicator used for the in vitro
study has been previously described in detail (16,18). Two
fixed stenoses (2 plates with circular orifices of 1.0 and 1.5
cm²) and seven aortic bioprosthetic heart valves (Medtronic
Intact 19, 21, 23, and 25 mm, and Medtronic Mosaic 21,
23, and 25 mm) were tested in this model under 10 levels of
flow rate ranging from 90 to 430 ml/s and using two aortic
sizes: 2.54 cm (cross-sectional area: 5.07 cm²) and 3.8 cm
(11.34 cm²).

Flow rate was measured with an electromagnetic flow-

meter, and pressure measurements were performed using
fluid-filled, side-hole catheters. Ventricular pressure was
measured 20 mm upstream from the valve, and aortic
 pressures at 5 and 100 mm downstream of the valve to
calculate $$\text{TPG}_{\text{max}}$$ and $$\text{TPG}_{\text{net}}$$, respectively. $$\text{EOA}_{\text{carh}}$$
calculated from $$\text{TPG}_{\text{net}}$$ and mean flow rate using Equation
2, and $$\text{EOA}_{\text{carh/max}}$$ was calculated from $$\text{TPG}_{\text{max}}$$ and mean
flow rate using Equation 1.

An Ultramark 9 HDI (Philips Medical Systems/ATL,
Bothell, Washington) was used for Doppler velocity mea-
surements. $$\text{EOA}_{\text{Dop}}$$ was determined by the standard con-
tinuity equation using stroke volume measured by the
electromagnetic flowmeter and the velocity–time integral of
the continuous-wave Doppler aortic jet signal. The $$\text{ELCo}$$
was calculated using the left-hand term of Equation 3.

Animal study. Animal care and experiments were con-
ducted in accordance with the Guidelines of the Canadian
Council for Animal Care. The protocol was approved by the
institutional Animal Care Committee of Laval University,
Sainte-Foy, Quebec, Canada. Fourteen pigs weighing be-
tween 27 and 35 kg were anesthetized, and a lateral
thoracotomy was performed in the fourth left intercostal
space. A supravalvular aortic stenosis was created using
umbilical tape tightened around the aorta ~2 cm down-
stream from the aortic valve annulus (19).

The pressure measurements were performed using a
Millar catheter (customized model, Millar Instruments,
Houston, Texas) with a distal (P1), intermediary (P2), and
proximal (P3) sensor. The P2 was positioned at the level of the vena contracta (minimal pressure downstream from the stenosis). The P1, which is at 1.5 cm of the intermediary sensor, was therefore located ~1 cm upstream from the stenosis. The P3, located at 4 cm of the intermediary sensor, was used to measure the aortic pressure after recovery. Cardiac output was measured using an ultrasonic flowmeter (T206, Transonic Systems, Ithaca, New York), with the probe positioned around the main pulmonary artery. The electrocardiogram, the three pressure signals, and the flow signal were simultaneously recorded and digitized (Digidata 1322, Axon Instruments, Foster City, California). The systolic trans-stenotic pressure gradients were calculated as follows: TPGmax = P1 – P2; and TPGnet = P1 – P3. EOAcath and EOAcath/max were calculated as described in the in vitro study.

The Doppler echocardiographic measurements were performed with a Sonos 5500 (Phillips Medical Systems/Agilent Technologies, Andover, Massachusetts). An upper laparotomy was performed, and the ultrasound probe was introduced in the abdominal cavity and positioned on the diaphragm at the level of the cardiac apex. This window allowed the visualization of high-quality apical five-chamber images and optimal recording of the left ventricular outflow tract pulsed-wave velocity and aortic jet continuous-wave velocity. EOADop was calculated using the standard continuity equation. The diameter of the ascending aorta was measured at 2 to 3 cm downstream of the stenosis by epicardial bi-dimensional echocardiography, using a 12-MHz probe. The AA was calculated assuming a circular shape. The ELCo was calculated using Equation 3.

These measurements were obtained under the following experimental conditions: 1) moderate stenosis; 2) severe stenosis; 3) severe stenosis plus a mild increase in systemic resistance; 4) severe stenosis plus a moderate increase in systemic resistance; and 5) severe stenosis plus a marked increase in systemic resistance. The increase in resistance was obtained by constriction of the descending thoracic aorta. The objective of this intervention was to increase the aortic pressure downstream of the stenosis to produce dilatation of the ascending aorta and thus an increase in AA.

**Patient data.** To further validate the results obtained in the pulsed duplicator and in the animals, we used the raw data published by Schöbel et al. (14). Their study was performed in 37 patients with aortic stenosis and no significant regurgitation. They simultaneously recorded the pressures within the left ventricle, at the vena contracta, and in the aorta at the site after pressure recovery. Cardiac output was determined by thermodilution, and the mean transvalvular flow rate was calculated. EOAcath (noted as A-V Δ in their report) and EOAcath/max (noted as A-V X) were determined from the Gorlin formula using TPGnet and TPGmax, respectively. However, Schöbel and colleagues used the original Gorlin formula with a constant of 44.3. Schöbel’s raw EOA data were therefore corrected by multiplying by 0.89 (44.3/50). In their study, the AA was derived from angiographic images in the middle part of the ascending aorta.

**Data analysis.** Data are expressed as the mean value ± SD. The EOA values obtained from different methods (EOADop, EOAcath/max and EOAcath) were compared within each data subset (in vitro, animals, and patients) using one-way analysis of variance for repeated measures. Statistical analysis of the association between variables was performed with the Pearson correlation coefficient, and graphs were constructed with the corresponding regression equation. Values of p < 0.05 were considered significant.

**RESULTS**

Table 1 presents the Doppler echocardiographic and catheter data obtained in vitro and in the animals, as well as the catheter data from the patients studied by Schöbel et al. (14). It should be noted that EOADop was not available in the latter study. In the in vitro model and the animals, EOADop was, on average, 24 ± 17% lower than the EOAcath values (p < 0.001), whereas EOADop and EOAcath/max were in close agreement and correlated strongly (in vitro data: y = 1.12x – 0.09, r = 0.97; animal data: y = 1.06x + 0.03, r = 0.92; all data: y = 1.02x + 0.03, r = 0.98) (Fig. 1). These experimental results confirm that EOAcath/max (EOA determined by a catheter using TPGmax) and EOADop are equivalent parameters reflecting EOA at the vena contracta.

Figure 2 shows the relationship (r = 0.97) found between EOAcath and EOADop. For the patient data, EOAcath/max was substituted for EOADop as the latter was not available in the study of Schöbel (14). This substitution is nonetheless valid because, as shown in Figure 1, there is a strong agreement between EOAcath/max and EOADop. Figure 2 confirms that EOADop is systematically lower than EOAcath (in vitro data: y = 1.36x – 0.16, r = 0.96; animal data: y = 1.47x – 0.04, r = 0.81; patient data: y = 1.38x – 0.13, r = 0.95; all data: y = 1.26x – 0.02, r = 0.97) (Fig. 2). The discrepancy between EOADop and EOAcath tended to be more important in animals than in patients; this may be due to the fact that pigs have relatively smaller aortas compared with humans.

Finally, Figure 3 shows that there is an excellent corre-
lution and concordance (in vitro data: $y = 0.93 + 0.10, r = 0.94$; animal data: $y = 0.87x + 0.12, r = 0.81$; patient data: $y = 1.15 - 0.04, r = 0.94$; all data: $y = 0.93x + 0.09, r = 0.97$) between EOAcath and ELCo and that, in contrast to EOADop, ELCo does not systematically underestimate EOAcath. This result thus confirms that EOAcath can be accurately predicted from Doppler echocardiographic data by calculating ELCo using Equation 3.

**DISCUSSION**

Because it is less flow-dependent than pressure gradients, the valve EOA is one of the main parameters on which clinicians have relied to assess aortic stenosis severity (1). However, although some investigators have found a relatively good agreement between Doppler and catheter EOA measurements (4,5,20,21), others have reported important discrepancies, and in the latter studies, the catheter EOAs were usually higher than the Doppler EOAs (9,22–27). Consistent with the latter studies, the present study also found catheter EOAs to be consistently higher than Doppler EOAs in the same individuals.

Moreover, the theoretical background shows that the systematic underestimation of EOAcath by EOADop is largely justified by the important following concepts: 1) EOADop is derived from the maximal velocity of the jet and reflects the cross-sectional area of the vena contracta. As confirmed by the present study, EOA cath/max is a parameter equivalent to EOADop that can also be used to estimate the area of the vena contracta. 2) The calculation of EOAcath/max requires the measurement of TPGmax which is rarely performed during routine catheterization because of the difficulty in obtaining adequate pressure measurements within the vena contracta. The fact that EOADop underestimates EOAcath is therefore not surprising, because

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**Figure 1.** Relationship between valve effective orifice area (EOA) measured by a catheter using the maximal transvalvular pressure gradient (EOA<sub>cath/max</sub>) and EOA measured by Doppler echocardiography (EOA<sub>Dop</sub>). Open triangles and open squares represent in vitro ($n = 172$) and animal ($n = 65$) data, respectively. The solid and dotted lines represent the identity and regression lines, respectively. The regression line was constructed including the whole data set (in vitro and animal data). Several data points are superimposed.
EOADop reflects the area at the vena contracta, whereas EOAcath is derived from TPGnet recorded after pressure recovery and thus downstream of the vena contracta. 3) Given 1) and 2), EOAcath will thus necessarily be higher than EOADop, but in varying proportions depending on the size of the ascending aorta and the severity of the stenosis (12–16,28). 4) The discrepancies between EOADop and EOAcath can be reconciled by calculating ELCo from the echocardiogram. This parameter takes into account pressure recovery, and, as shown by Equations 2 and 3, its formulation is very close to that of EOAcath. 5) Neither EOAcath nor ELCo represents the true EOA, but are rather dimensionless and relatively flow-independent indexes representing the relative loss of energy due to stenosis.

Our experimental results largely confirm these theoretical considerations. As shown in Figure 1, there is indeed a very good correlation and concordance between EOADop and EOAcath/max, as both parameters are a reflection of the cross-sectional area of the vena contracta. In contrast, EOAcath overestimated both EOADop and EOAcath/max, but in varying proportions depending on the diameter of the aorta (Table 1). Finally, when ELCo was calculated from the echocardiogram to account for pressure recovery, there was an excellent agreement between EOAcath and ELCo, and the aforementioned discrepancy between echocardiographic and catheter measurements was no longer present (Fig. 3). It should be noted that according to fluid mechanics considerations and previous in vivo studies (13,16,29), the diameter of the aorta used to calculate ELCo should be measured at the sino-tubular junction (i.e., at the site where pressure recovery is ongoing). Schöbel et al. (14) also proposed an equation that incorporates EOAcath/max and AA to predict EOAcath. Nonetheless, this equation is not readily applicable to reconcile the discrepancies between EOAcath and EOADop, as the latter was not measured in their study.

The clinical implications of these findings are important
because they may have a direct implication with regard to the criteria used to quantify aortic stenosis severity. As mentioned, measurements of $TPG_{\text{max}}$ and $EOA_{\text{cath/\max}}$ are rarely performed in the clinical setting because of the difficulty in obtaining adequate pressure measurements in the vena contracta, and the parameters routinely reported from catheter measurements are $TPG_{\text{net}}$ and $EOA_{\text{cath}}$. In this context, it should be emphasized that the ACC/AHA guidelines for defining aortic stenosis severity were established mainly based on data obtained from catheter measurements, as well as clinical outcomes in relation to these measurements (1,30–32). The same values for aortic stenosis severity (e.g., $<1.0 \, \text{cm}^2$) were then extended to the echocardiographic data on the assumption that $EOA_{\text{Dop}}$ and $EOA_{\text{cath}}$ were equivalent parameters, and indeed, the aforementioned guidelines do not distinguish between catheter and Doppler measurements.

A most important finding of this study is that the pressure recovery phenomenon may cause important discrepancies between $EOA_{\text{cath}}$ and $EOA_{\text{Dop}}$, and that $EOA_{\text{Dop}}$ systematically tends to overestimate aortic stenosis severity, compared with $EOA_{\text{cath}}$. The practical implications of this finding are best evidenced by considering Table 2, where Equations 2 and 3 are used to calculate the theoretical values of $EOA_{\text{Dop}}$ for different values of $EOA_{\text{cath}}$ and aortic size. The range of aortic sizes used in this table is based on the study of Gjertsson et al. (29), performed in a large group of patients with aortic stenosis (range of aortic diameters at the sino-tubular junction 2.1 to 4.1 cm, mean 3.0 cm). As expected, the greatest discrepancies between $EOA_{\text{cath}}$ and $EOA_{\text{Dop}}$ are observed in patients with smaller aortas (diameter $\leq 3.0 \, \text{cm}$), and when comparing Doppler and catheter EOAAs in a given patient, it is therefore important to remember that these parameters are not equivalent and that discrepancies up to 50% may be observed depending on the size of the aorta and the severity of the stenosis. Overall, 10% (27/274) of the stenoses examined in the present study would have been classified as
severe on the basis of EOA\textsubscript{Dop} and moderate on the basis of EOA\textsubscript{cath}. Of the 37 patients included in this study, three (8%) would have been misclassified. Furthermore, these discrepancies become even more important if one uses the Gorlin formula with a constant of 44.3, as routinely done in catheterization laboratories (Table 2). The present guidelines, based mostly on EOAs measured during catheterization, may therefore not be directly applicable to measurements made from EOA\textsubscript{Dop} and may result in overestimations of severity, thus affecting clinical management.

From a practical standpoint, there would appear to be two options. One would be to use a different threshold of severity (e.g., EOA \(\leq 0.75 \text{ cm}^2\)) when EOA is measured by Doppler echocardiography. However, the underestimation of EOA\textsubscript{cath} by EOA\textsubscript{Dop} will vary depending on the size of the aorta, and the large standard deviation (\(\pm 17\%\)) observed in this study suggests that it would not be appropriate to apply a single correction factor. The second alternative would be to use ELCo as the Doppler echocardiographic measurement of aortic stenosis severity. As shown in Figure 3, this would have the advantage of consistency with catheter measurements, and the present guidelines for severe aortic stenosis (EOA \(\leq 1.0 \text{ cm}^2\)) could then be directly applicable to this parameter. From a conceptual standpoint, it should also be emphasized that ELCo and EOA\textsubscript{cath}, both reflect the net EL due to stenosis and, as such, are more representative of the increased burden imposed on the left ventricle, compared with EOA\textsubscript{Dop} and EOA\textsubscript{cath}/max. In contrast, uncorrected EOA\textsubscript{Dop} has major disadvantages for clinical use because it does not account for differences in actual left ventricular burden, as the latter varies markedly depending on the magnitude of pressure recovery (10–16).

For these reasons, it would appear logical to use ELCo rather than EOA\textsubscript{Dop} as the preferred echocardiographic parameter for quantifying aortic stenosis severity, in which case the severity criteria proposed in the ACC/AHA guidelines would become directly applicable (1). Also, as previously shown, ELCo can be indexed for the patient’s body surface area to better account for differences in cardiac output requirements due to differences in body size (16). Previous studies are consistent in suggesting that an indexed EOA\textsubscript{cath} or EL index \(\leq 0.55\) to 0.60 \text{ cm}^2/m^2 is indicative of severe aortic stenosis (16,32).

### Study limitations.

An obvious limitation of this study is the absence of a gold standard method for the direct measurement of EOA at the vena contracta. Nonetheless, the strong agreement between the experimental results and the theoretical equations derived from fluid dynamics confirms the conceptual validity of our results and conclusions.

Ideally, it would also have been interesting to obtain simultaneous measurements of EOA\textsubscript{Dop} and EOA\textsubscript{cath} not only in vitro and in animals, but also in patients. However, the measurement of EOA\textsubscript{cath} requires complete left- and right-heart catheterization, a procedure that is not without risk for the patient. Indeed, the most recent ACC/AHA guidelines recommend that this procedure should be performed only if there is a discrepancy between the clinical and echocardiographic evaluations of aortic stenosis severity (1). Hence, systematic performance of such a procedure in patients would have been difficult to justify from an ethical standpoint, and for this reason, we elected to use the data previously published by Schöbel et al. (14). The fact that these data were collected independently and agree well with our own results further validates the conclusions of the present study.

### Conclusions.

Discrepancies between catheter and Doppler measurements of EOA are largely due to the pressure recovery phenomenon and can be reconciled by calculating ELCo from the Doppler echocardiogram. Although EOA\textsubscript{Dop} better represents the actual cross-sectional area of the vena contracta, ELCo and EOA measured from the catheter net gradient are equivalent indexes that primarily reflect the net EL due to stenosis rather than the EOA, per se. As such, the latter indexes better reflect the increased burden imposed by the stenosis upon the left ventricle and are probably the most appropriate for quantifying aortic stenosis severity.

### Acknowledgment

We thank Guy Rossignol for his technical assistance in the realization of the animal study.

### Table 2. Theoretical Values of Doppler-Derived Effective Orifice Areas for Given Catheter-Derived Effective Orifice Areas and Aortic Diameters

<table>
<thead>
<tr>
<th>Catheter-Derived EOA (cm(^2))</th>
<th>Aortic Diameter = 2.0 cm</th>
<th>Aortic Diameter = 3.0 cm</th>
<th>Aortic Diameter = 4.0 cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>((A_L = 3.14 \text{ cm}^2))</td>
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</tr>
<tr>
<td>1.50 (1.69)</td>
<td>1.02</td>
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<td>0.43</td>
<td>0.47</td>
<td>0.48</td>
</tr>
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</table>

*These values were derived from Equations 2 and 3. †The EOA value in parentheses was calculated from the Gorlin equation with the use of a constant of 44.3.

EOA = effective orifice area; \(A_L\) = cross-sectional area of the aorta.
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REFERENCES


