Aortic Stenosis: Physics and Physiology—What Do the Numbers Really Mean?

Arthur E. Weyman, MD, Marielle Scherrer-Crosbie, MD, PhD
Cardiology Division, Massachusetts General Hospital, Harvard Medical School, Boston, MA

Cardiac catheterization and Doppler echocardiography are two methods used to measure transvalvular gradients and valve area in the assessment of aortic stenosis severity. Although both approaches are based on the same hemodynamic concepts and report data using the same units of measure, each method measures pressure drop or gradient at a different place; hence they produce fundamentally different quantities. Likewise, cardiac catheterization formulas for valve area attempt to obtain the anatomic area whereas the Doppler continuity equation reports the area to which flow is constricted. To use these two methods appropriately, it is necessary to understand the underlying hemodynamic principles and the effects of the methods of measurement on the values obtained. This article examines these variables and shows how they affect the reported gradients and valve areas and how differences can affect clinical application.

[Rev Cardiovasc Med. 2005;6(1)23-32]

© 2005 MedReviews, LLC

Key words: Aortic stenosis • Catheterization • Doppler echocardiography • Pressure gradients • Valve area

Assessment of the severity of aortic stenosis depends on measurement of the transvalvular gradients and valve area. Historically these parameters were determined by cardiac catheterization based on direct measurement of pressures in the left ventricle and aorta and estimation of transvalvular flow using the Fick, dye dilution, or thermodilution method. At present, however, gradients and valve areas are generally determined using noninvasive Doppler echocardiographic methods. Although both of these approaches derive from
similar hemodynamic concepts and report their results in the same units of measure, the catheter and Doppler measurements of pressure drop or gradient are not made at the same place and therefore are fundamentally different quantities. Likewise, catheterization formulas for valve area attempt to derive the anatomic area whereas the Doppler continuity equation reports the area to which flow is constricted or the effective valve area. The effect of these differences is exaggerated because important changes in the classification of severity are based on differences in valve areas of only tenths of a square centimeter. This is further complicated by the fact that although clinical decisions are now commonly made based on Doppler echocardiographic data, criteria for severity are based on more traditional catheterization data in which outcomes have been more extensively studied. The appropriate use of these frequently differing data therefore depends on an understanding of the underlying hemodynamic principles and effects of the methods of measurement on the resulting values. The purpose of this review is to examine these variables and to show how they affect the reported gradients and valve areas and how differences can affect clinical application.

Although differences between Doppler echocardiographic and catheterization data are often a result of technical errors in data acquisition, inherent errors in the component methods, or simply differences in the parameter measured (eg, the difference between peak instantaneous and peak-to-peak gradients), for the purposes of this review, we assume that all the data are appropriately recorded and instantaneously compared.

**Characteristics of Flow Through Stenotic Valves**

To understand the differences in catheter and Doppler measurements of gradients and valve areas, it is first necessary to appreciate the hydrodynamic pattern of flow through a stenosis. In any closed system, the law of conservation of mass requires the volume flow to be constant at all points. Because flow equals the product of mean velocity and vessel area, as the area of the flow stream decreases as blood approaches a stenosis, the velocity must increase proportionately in order to maintain constant flow. As flow exits the stenotic orifice, the streamlines continue to converge for a short distance (the vena contracta effect) because inertia prevents the peripheral streamlines entering the stenotic orifice from the side from changing directions instantly. By the time the flow lines have again become parallel, the cross-sectional area of the jet will be smaller than the orifice area and the velocity of the jet will be proportionately higher. The ratio of the jet area at the vena contracta (the area to which flow is constricted or effective orifice area \( A_{\text{vcon}} \)) to the anatomic area \( A_{\text{anat}} \) is called the coefficient of contraction (Figure 1). This ratio is affected by the size and shape of the orifice and by the inlet geometry. To determine the anatomic orifice size, some correction must be introduced to account for this coefficient of contraction, which is unknown in most cases. Such calculations are facilitated by a term used in engineering, the coefficient of discharge, which compares the actual flow through the valve with the predicted flow if no contraction or viscous losses occur. The coefficient of discharge is specific for an individual valve shape and size and converts the effective area to the anatomic area.

Distal to the stenosis, there is an abrupt separation of the jet from the vessel wall, resulting in the jet shearing against the stagnant blood in the sinuses of Valsalva, causing vortex formation and a zone of recirculation (Figure 2). The abrupt change in vessel diameter (orifice diameter to aortic diameter) suddenly increases the ratio of kinetic to viscous forces, represented by the Reynolds number, up to...
to fivefold. The Reynolds number (Re = blood density velocity x vessel diameter/blood viscosity) is a measure of the tendency of the blood to become turbulent, and this abrupt increase is associated with flow instability and turbulence. In this turbulent zone, kinetic energy is lost as heat that is nonrecoverable.

Pressure Gradients

Doppler measurement of the transvalvular pressure drop or gradient is based on the law of conservation of energy, which states that for flow in a closed system, the total energy at all points must remain constant. Because the linear velocity in centimeters per second through a stenotic valve must increase as the valve area decreases (law of conservation of mass), its kinetic energy, which is proportional to the square of the linear velocity, increases. As a result, its potential energy (lateral pressure) must decrease to maintain total energy constant. In pulsatile systems, additional energy may be required to overcome inertia and accelerate blood to its peak velocity. Energy may also be lost as heat as a result of viscous friction. These relationships can be expressed mathematically using the Bernoulli equation,

$$\Delta P = \frac{1}{2} \rho (v_2^2 - v_1^2) + \rho \frac{dv}{dt} \int ds + R(\rho, v)$$

Eq.1

where $\Delta P$ is the difference in the pressures proximal and distal to the stenosis, $v_1$ and $v_2$ are the velocities proximal to the stenosis and at the vena contracta, $s$ is the distance over which flow accelerates, $R$ is viscous resistance, $\rho$ is the mass density of blood, and $\mu$ is the viscosity. The first term in the equation accounts for convective acceleration of flow through the stenosis, the second term for flow acceleration, and the third for viscous friction. In the clinical situation, viscous friction has been shown to be negligible for discrete orifices $\geq 0.25$ cm$^2$, because blood velocity is approximately constant across the orifice, and as a result, there is no frictional loss between adjacent fluid layers. In addition, although the need for flow to accelerate from zero delays the velocity waveform slightly relative to the pressure waveform, it does not significantly alter the calculation of the peak gradient (because at peak velocity, $dv/dt$ [acceleration] = 0). The mean gradient is also unaffected because the lag in velocity is roughly symmetric during acceleration and deceleration. At other points in the cardiac cycle, the acceleration term produces small discrepancies between pressure gradient and velocity, but these are not clinically important. Because the viscous friction and flow acceleration terms are negligible, they can be ignored.

Figure 2. Flow visualization study illustrating the pattern of flow distal to a stenosis. Immediately beyond the orifice, the jet shears against the stagnant blood in the parajet region, causing turbulent eddies that erode the laminar core, resulting in the loss of energy as heat. Reproduced with permission from Levine et al.7
Aortic Stenosis continued

and equation 1 simplifies to
\[ \Delta P = \frac{1}{2} \rho (\nu_2^2 - \nu_1^2) \]

Eq. 2

In addition, in most stenotic lesions, \( \nu_2^2 \gg \nu_1^2 \), so that \( \nu_1 \) can be ignored, and the pressure gradient after correction for different units \( \rho = 1.06/981 \text{ g s}^2/\text{mL} \times 1/1.36 \) (to convert dyne centimeters to millimeters of mercury [mm Hg]) \( x \frac{1}{2} \), which after appropriate conversion of measurement units \( (x 10^4) = 3.972 \), or roughly 4, so that
\[ \Delta P = 4\nu^2 \]

Eq. 3

The simplified Bernoulli equation permits calculation of the transvalvular gradient at each instant in systole; however, two measures of transvalvular pressure gradient are usually reported from Doppler aortic velocity profiles: the peak gradient and the mean gradient. The peak gradient is determined from the peak velocity, whereas the mean gradient is the mean of the squared instantaneous velocities recorded during the systolic ejection period.

It is important to remember that continuous-wave Doppler measures the change in velocity from a point proximal to the onset of convective acceleration toward the stenosis to the peak velocity at the vena contracta. A critical but unstated assumption in using this increase in velocity as a measure of the pressure gradient across the valve is that all of the pressure that is converted to kinetic energy (velocity) is then lost as heat in the turbulent eddies downstream from the stenosis and that none of the kinetic energy is reconverted to pressure. It is this downstream loss of energy that is the important effect of the stenosis, because if velocity were simply reconverted to pressure (pressure recovery), there would be no energy loss to the system and the stenosis would have no significant hemodynamic effect. This lack of effect would occur despite the fact that Doppler would record an increase in velocity at the stenosis and pressure taps proximal to the stenosis and at the vena contracta would register a corresponding fall in pressure. In practice, the conversion of kinetic energy to heat is not always complete and some pressure can be recovered (see below).

Catheter Measures of Transvalvular Gradients

Experimental studies comparing Doppler gradients measured using the peak velocity at the vena contracta with catheter values obtained by estimating those reported at catheterization. Some of this overestimation has been attributed to failure to account for the proximal velocity in the Bernoulli equation in high-flow states. However, a number of experimental studies have demonstrated that as the streamlines of flow reattach to a vessel wall downstream from a stenosis, some of the momentum in the jet is reconverted to lateral or pressure energy, a phenomenon known as pressure recovery.\(^5,6,7\) Because the energy loss in aortic stenosis is the result of flow separation and vortex formation (turbulence), the extent of this phenomenon depends on the size relationship between the orifice and aorta.\(^5,6,8\) The smaller the valve orifice relative to the size of the aorta, the more turbulence will occur, and because turbulence results in the conversion of kinetic energy to heat that is nonrecoverable, less energy will be available to be recovered as pressure. Conversely, the larger the valve orifice or the smaller the aorta, the less turbulence will occur and the greater the
pressure recovered. Although the distance required for maximal pressure recovery varies with orifice size and aortic diameter, the majority of the pressure is usually recovered within a few centimeters (~5 cm) beyond the vena contracta.\textsuperscript{5,6}

When pressure recovery occurs, the actual pressure loss to the system, termed the head loss, will be less than that reflected by the increase in kinetic energy at the vena contracta, and the pressure gradient between the left ventricle and aorta (the net pressure gradient) will be less than the gradient between the left ventricle and the vena contracta.\textsuperscript{5,6} Figure 3 illustrates the effect of vena contracta area pressure recovery on the actual pressure loss to the system, termed the head loss, and the pressure gradient between the left ventricle and aorta. As illustrated, the sizes of the smallest aortic diameters were associated with the greatest discrepant pressures between the Doppler and catheter gradients. Figure 4 illustrates the effect of valve area on pressure recovery for a fixed aortic size. As predicted, the larger the orifice, the greater the amount of pressure recovery. In patients with aortic sizes larger than 3 cm, only small differences in catheter and Doppler gradients were observed (peak, 7.3 ± 8.7 mm Hg; mean, 2.6 ± 6.1 mm Hg), whereas in patients with aortas ≥ 3 cm, greater degrees of pressure recovery were observed (peak, 24.8 ± 19.7 mm Hg; mean, 16.2 ± 13.2 mm Hg).\textsuperscript{9}

On the basis of fluid mechanics theory, the Doppler-predicted pressure drop can be corrected for the size of the aorta in order to derive the actual pressure drop (head loss) or net pressure gradient after pressure recovery. This area-based correction factor is given as

$$\Delta P = 4V^2 (1 - C)$$

with \(C = 2[(A_{\text{eff}}/A_\Lambda) - A_{\text{eff}}^2/A_\Lambda^2]\),

\textbf{Eq. 4}

where \(A_{\text{eff}}\) is the vena contracta area and \(A_\Lambda\) is the area of the aorta. The calculated \(A_{\text{eff}}\) assumes a circular orifice, which is appropriate given that pressure recovery is not affected by the shape of the orifice.\textsuperscript{10} Figure 5 compares peak and mean Doppler gradients with the Doppler gradients predicted using equation 4 with the peak and mean catheter gradients in a group of patients with valvular aortic stenosis. Using this correction decreased the slopes of the peak and mean Doppler gradients from 1.36 and 1.25, respectively, for the peak and mean uncorrected values to 1.03 and 0.96 for the corrected Doppler values when compared with catheter-derived gradients.

For any given valve area and aortic diameter, the orifice velocity and therefore the gradient will depend on the flow rate. The absolute amount of pressure recovery increases...
with flow rate (orifice velocity). The percent overestimation, however, remains approximately the same and therefore is independent of flow.

Jet eccentricity also affects pressure recovery, with more eccentric jets showing decreasing Doppler-catheter differences (less pressure recovery because momentum is lost when the jet strikes the vessel wall). In this case, the correction factor described above is no longer applicable.

Clinically, pressure recovery is most relevant in patients with moderate aortic stenosis, small aortas, and high flow rates. In these cases, the maximal pressure drop at the vena contracta (Doppler) and net pressure gradient (catheter after pressure recovery) may differ significantly and therefore affect management, particularly when the gradients are used to calculate valve area.

In addition to the ratio of the stenotic orifice to the aorta, both the inlet and outlet geometry of the stenosis importantly affect the amount of pressure recovery and thus the relationship between the catheter- and Doppler-measured gradients. Valvular stenoses are usually discrete, with abrupt narrowing and expansion, and although pressure recovery is present, it is usually not great. For more tapering stenoses—such as those that often characterize subvalvular and supravalvular obstruction and coarctation—the shape of the outlet becomes more important than the simple orifice diameter to downstream vessel diameter ratio, and pressure recovery may cause a greater disparity between Doppler- and catheter-measured gradients than would be predicted simply for the orifice aortic ratio. Figure 6 illustrates this effect for a Venturi tube with a gradually tapering outlet, which permits almost immediate reattachment of the flow streamlines to the vessel wall. In this example, although there is a significant pressure loss at the valve orifice, the streamlines almost immediately reattach to the vessel wall and the head loss is only 15% (pressure recovery 85%).

Importantly, when pressure recovery occurs, the head loss (which is the loss of energy to the system and is measured at catheterization where the gradient after pressure recovery is recorded) is the appropriate measure of energy loss to the system and determines the left ventricular pressure required to maintain a given aortic pressure. However, the Doppler gradient, which measures the conversion of pressure to kinetic energy induced by the stenosis, is the appropriate gradient to use to calculate the effective orifice area.

Valve Area Determination

Because the pressure drop or gradient across a stenotic valve varies with flow, it has become common practice to calculate the valve area, which is a flow-independent measure of severity.

Doppler Echocardiographic Estimation of Valve Area: the Effective Valve Area

The calculation of the aortic valve area from Doppler recordings is based on the law of conservation of mass, which states that for an incompressible fluid in a closed system, flow (Q) at all points must remain constant. As illustrated in

Figure 7. Calculation of aortic valve area using the continuity principle. Aortic outflow is calculated as the product of the subvalvular area $A_2$ and velocity $V_1$. The aortic valve area is then equal to $A_2 \times V_1$ divided by the peak transvalvular velocity $V_2$. LA, left atrium; Ao, aorta; LV, left ventricle.
Figure 7, the flow through the outflow tract must be the same as the flow through the valve ($Q_1 = Q_2$) at any point in time. In addition, because flow equals mean velocity times area at any point, 

$$Q_1 = Q_2 = A_1 \cdot V_1 = A_2 \cdot V_2 \quad \text{Eq. 5}$$

If flow through the valve is known or can be determined from the product of area and velocity at a reference level, and the velocity at the stenosis can be recorded, then the area at the point of stenosis can be calculated as follows:

$$A_2 = \frac{A_1 \cdot V_1}{V_2} \quad \text{Eq. 6}$$

where $V_2$ is the velocity and $A_2$ the area at the vena contracta and $V_1$ and $V_2$ are used, it can be assumed that the maximal $A_2$ is calculated because the gradient is greatest at this point, forcing the valve to open maximally. Conversely, using the mean velocity will give the average area occurring throughout systole. One may also use the instantaneous velocities throughout systole to calculate the instantaneous valve areas and thereby detect any flow-related changes.

Because the peak Doppler velocity is the velocity at the vena contracta, the calculated valve area will be the smallest area to which the flow stream is reduced, which will be equal to the anatomic area reduced by the coefficient of discharge. The effective area is the appropriate hydrodynamic area, but should be smaller than the area calculated at catheterization using the Gorlin formula, which includes a constant to account for the coefficient of discharge and thus attempts to convert hydrodynamic area to anatomic area.

**Calculation of Valve Area at Catheterization**

**Theoretical Background**

Valve area can also be calculated using the pressure drop or gradient across the valve. To do this requires use of both the continuity and Bernoulli equations, where

$$Q = A_{ef} \cdot V$$

and

$$\Delta P = \frac{1}{2} \rho V^2.$$  

Solving for $V$ gives

$$V = \frac{Q}{A_{ef}} \text{ and } V = (2\Delta P/\rho)^{1/2}.$$ 

In this equation, $\Delta P$ is in metric units (dyne/cm$^2$), whereas clinically it is usually expressed in mm Hg.

Thus, substituting ($1 \text{ mm Hg} = 1333 \text{ dyne/cm}^2$) and $\rho = 1.05 \text{ g/mL}$ gives

$$V = (2 \times 1.333 \Delta P/1.05)^{1/2} = (2.539 \Delta P)^{1/2} = 50.4 \Delta P^{1/2} \quad \text{Eq. 7}$$

and

$$A_{ef} = \frac{Q}{50.4 \Delta P^{1/2}} \quad \text{Eq. 8}$$

This equation calculates the effective orifice area and yields the same result as the Doppler continuity equation.

Because the effective orifice area, or the area to which flow is constricted, is related to the anatomic area of the valve by the coefficient of discharge:

$$A_{anat} = A_{eff} \cdot CD$$

or

$$A_{anat} = \frac{A_{eff}}{CD} \quad \text{Eq. 9}$$

The Gorlin Equation

The first clinical application of these concepts was by Gorlin and Gorlin in 1951.11 In their original formula, they included a constant to account for the coefficient of contraction and thereby attempted to correct the flow area to the anatomic area, given that their standard of reference was excised valves. Thus,

$$A_{anat} = \frac{Q}{C \cdot 44.3 \Delta P^{1/2}} \quad \text{Eq. 10}$$

where $44.3 = \sqrt{2 \cdot 981}$. The empiric constant $C$ in the original formulation included the coefficients of contraction $CC$ and viscosity $CV$ as well as correction for the conversion of centimeters of water (cm H$_2$O) to mm Hg. Recognizing that blood viscosity, turbulence, pulsatile flow, and the inconstant shape of deformed valves made it almost impossible to predict the discharge coefficient analytically, they determined an empiric coefficient from direct measurement of mitral valves at surgery or autopsy. For the mitral
Aortic Stenosis continued

...valve, they concluded that the anatomic valve area equals

\[ A_{\text{anat}} = \frac{Q}{31\sqrt{P_1 - P_2}} \]

Eq. 11

which corresponds to a discharge coefficient of roughly 0.62.

For the aortic valve, they used the constant \( C = 1 \), which results in

\[ A_{\text{anat}} = \frac{Q}{44.3\sqrt{\Delta P}} \]

Eq. 12

Use of the constant 44.3 ignores the conversion of cm H₂O to mm Hg and the mass density of blood but differs from equation 6 only in the constant (50.4 vs 44.3). Because anatomic area can in theory be calculated as

\[ A_{\text{anat}} = \frac{Q}{(CD50.4\sqrt{\Delta P})} \]

Eq. 13

the Gorlin constant corresponds to \( CD \times 50.4 \), which implies a discharge coefficient of 0.879.

This correction is partially offset by the use of the square root of the mean pressure gradient in the Gorlin formula instead of the mathematically correct mean of the square roots of the instantaneous gradients, which systematically underestimates the valve area and results in a value closer to the physiologic area.12,13

Thus the Gorlin formula actually calculates the flow area (\( A_{\text{eff}} \)) offset by a small constant and will correspond to the anatomic area only by chance (ie, when the empiric coefficient of discharge happens to be appropriate for the valve in question).

Does the Gorlin constant bear a consistent relationship with the anatomic area? Recent studies have shown that the true coefficient of discharge is not constant but rather varies with valve orifice size and shape, but, importantly, is independent of flow, except at very low flow rates.13 The discharge coefficient increases with increasing valve area and decreases with increasing eccentricity. Figure 8 illustrates the combined effects of orifice area and eccentricity on the discharge coefficient. Because flow contraction by itself should not cause these changes, they have been attributed to the viscosity,13 because for an eccentric jet, the jet perimeter—where viscous losses occur—is larger relative to the cross-sectional area than for a circular jet. Likewise, for a small jet, the perimeter-to-area ratio is greater than for a large jet.

The shape of the valve inlet also affects the coefficient of discharge.13 Figure 9 illustrates the relationship of valve shape to coefficient of contraction as a function of valve area derived from three-dimensional models of clinical echocardiographically imaged aortic valves. As illustrated, the impact of a stenosis on pressure and flow depends not only on the cross-sectional area of the orifice but also on the three-dimensional geometry of the leaflets proximal to the orifice. The geometry determines the pattern of flow convergence and thus the relationship of \( A_{\text{eff}} \) to \( A_{\text{anat}} \). Patients with flat valves and steeper flow convergence have smaller \( A_{\text{eff}} \) than those with more gradually tapered domed valves for the same anatomic area and flow rate. Corresponding pressure losses may be increased by up to 40%, increasing the ventricular workload proportionately. Theoretically, hemodynamic severity may increase because of changes in valve shape without change in orifice area, as with the progressive calcification of a bicuspid valve.

**Effects of Pressure Recovery on Valve Area Measurements**

Pressure recovery also affects the measurement of valve area at catheterization because the net pressure gradient (measured between the left ventricle and aorta after pressure recovery) is smaller than the gradient at the vena contracta, therefore resulting in a larger calculated valve...
Aortic Stenosis

The valve area calculated using the net gradient does not correspond to either the effective or anatomic valve area but rather represents the area that would have occurred had there been no pressure recovery: a theoretic value, but one that reflects the hydrodynamic impact of the stenosis. Use of the net gradient further increases the difference between Doppler measures of the effective valve area and the area calculated at catheterization.

Because the amount of pressure recovery is determined by the ratio of the orifice area to the aortic area, a correction factor has been proposed to correct the Doppler continuity valve area for the effect of pressure recovery.\textsuperscript{14} Using this correction, Doppler $A_{\text{eff}}$ is multiplied by

$$A_a/A_s - A_{\text{eff}} = 1 + A_{\text{eff}}/A_s - A_{\text{eff}}$$

where $A_a$ is the area of the ascending aorta. As can be appreciated from this equation, in patients with tight aortic stenosis in which the $A_{\text{eff}}$ is much smaller than the ascending aortic area, there will be little difference between the actual and corrected $A_{\text{eff}}$. Table 1 illustrates the relationship between the Gorlin-derived effective areas and the Doppler areas for a series of aortic diameters.\textsuperscript{14} Relating these changes to current criteria for severity, it can be shown that a patient with an ascending aortic diameter of 2.6 cm and a Doppler $A_{\text{eff}}$ of 0.9 cm$^2$ would have a valve area of 1.1 cm$^2$ after correcting for pressure recovery.\textsuperscript{1,15} This effect would be greater for a patient with the same aortic diameter and a Doppler $A_{\text{eff}}$ of 1.2 cm$^2$, in which the corrected area would correspond to a recovered catheter-derived area of 1.6 cm$^2$.\textsuperscript{13} In both of these cases, the change would shift the patient to a lower grade of severity based on current American Heart Association/American College of Cardiology guidelines.\textsuperscript{1} Further, although $A_{\text{eff}}$ and $C_D \cdot A_{\text{anat}}$ are measures of the severity of stenosis, $A_{\text{anat}}$ is not because the hemodynamic severity of stenosis may increase with no change in the anatomic area, as a result of progressive dilation of the aorta with a corresponding decrease in pressure recovery.

**Summary**

The area of a stenotic aortic valve calculated using standard hemodynamic methods will be consistently larger than that measured using Doppler echocardiography when these data are accurately recorded and simultaneously compared, even in the absence of pressure recovery. When pressure recovery is present, this difference will increase and may result in significant misclassification of severity. Understanding these differences allows appropriate corrections to be introduced and the total effect of the stenosis on the system to be appreciated. Alternatively, more conservative standards may be more

<table>
<thead>
<tr>
<th>Table 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Doppler-Derived Effective Orifice Area (EOA)</strong></td>
</tr>
<tr>
<td><strong>Catheter-Derived EOA (cm$^2$) (Constant 50)</strong></td>
</tr>
<tr>
<td>1.50</td>
</tr>
<tr>
<td>1.00</td>
</tr>
<tr>
<td>0.75</td>
</tr>
<tr>
<td>0.50</td>
</tr>
</tbody>
</table>

Adapted from Garcia et al.\textsuperscript{14}
appropriate for estimating severity using Doppler echocardiographic data than those currently recommended for catheterization values.

References

Main Points
- To understand the differences in catheter and Doppler measurements of gradients and valve areas, it is first necessary to appreciate the hydrodynamic pattern of flow through a stenosis.
- Doppler measurement of the transvalvular pressure drop or gradient is based on the law of conservation of energy, which states that for flow in a closed system, the total energy at all points must remain constant. Catheter pressures are usually recorded in the ascending aorta after pressure recovery has occurred. Because clinical Doppler and catheter measurements of pressure occur at different places, they measure fundamentally different quantities and may report significantly different results when pressure recovery is present.
- The calculation of the aortic valve area from Doppler recordings is based on the law of conservation of mass, which states that for an incompressible fluid in a closed system, flow at all points must remain constant. Catheterization calculates valve area by measuring the pressure drop or gradient across the valve using continuity and Bernoulli equations.
- The area of a stenotic aortic valve calculated using standard hemodynamic methods will be consistently larger than that measured using Doppler echocardiography when these data are accurately recorded and simultaneously compared, even in the absence of pressure recovery. When pressure recovery is present, this difference will increase and may result in significant misclassification of severity.