10

Calculation of Stenotic Valve Orifice Area

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The normal cardiac valve offers little resistance to blood flow, even when flow velocity across it is high. As valvular stenosis develops, the valve orifice produces progressively greater resistance to flow, resulting in a pressure drop (pressure gradient) across the valve. At any stenotic orifice size, greater flow across the orifice yields a greater pressure gradient. Using this principle together with two fundamental hydraulic formulas, Dr. Richard Gorlin and his father developed a formula for the calculation of cardiac valvular orifices from flow and pressure-gradient data (1).

GORLIN FORMULA

The first hydraulic formula that the Gorlins used was Torricelli's law, which describes flow across a round orifice:

\[ F = A V C_C \]  

where \( F \) = flow rate, \( A \) = orifice area, \( V \) = velocity of flow, and \( C_C \) = coefficient of orifice contraction. The constant \( C_C \) compensates for the physical phenomenon that, except for a perfect orifice, the area of a stream flowing through an orifice will be less than the true area of the orifice.

Rearranging the terms,

\[ A = \frac{F}{V C_C} \]  

The second hydraulic principle used in the derivation of Gorlin's formula relates pressure gradient and velocity of flow:

\[ V^2 = (C_v)^2 \cdot 2gh \quad \text{or} \quad V = (C_v) \sqrt{2gh} \]  

where \( V \) = velocity of flow; \( C_v \) = coefficient of velocity, correcting for energy loss as pressure energy is converted to kinetic or velocity energy; \( g \) = acceleration due to gravity (980 cm/sec/sec), and \( h \) = pressure gradient in cm H\textsubscript{2}O.
Combining the two equations,

\[
A = \frac{F}{C_v \sqrt{2ghC_C}} = \frac{F}{C_vC_c \sqrt{2.980h}} = \frac{F}{(C)(44.3) \sqrt{h}} \quad (10.4)
\]

where \(C\) is an empirical constant accounting for \(C_v\) and \(C_c\), the expression of \(h\) in mm Hg, and correcting calculated valve area to actual valve area measured at surgery or autopsy.

It is obvious that antegrade flow across the mitral and tricuspid valves occur only in diastole, whereas that across the aortic and pulmonic valves occur only in systole. Accordingly, the flow \(F\) for Eq. (10.4) is the total cardiac output expressed in terms of the seconds per minute during which there is actually forward flow across the valve. For the mitral and tricuspid valves, this is calculated by multiplying the diastolic filling period (seconds per beat) times the heart rate (beats per minute ‘bpm’), yielding the number of seconds per minute during which there is diastolic flow. The cardiac output in milliliters per minute (or cm\(^3\)/min) is then divided by the seconds per minute during which there is flow, yielding diastolic flow in cubic centimeters per second. For the aortic and pulmonic valves, the systolic ejection period is substituted for the diastolic filling period. The manner in which the diastolic filling period and systolic ejection period are measured is shown in Fig. 10.1. The diastolic filling period begins at mitral valve opening and continues until end-diastole. The systolic ejection period begins with aortic valve opening and proceeds to the dicrotic notch or other evidence of aortic valve closure.

**FIG. 10.1.**

Left ventricular (LV), aortic (Ao), and pulmonary capillary wedge (PCW) pressure tracings from a patient without valvular heart disease, illustrating the definition and measurement of diastolic filling period (DFP) and systolic ejection period (SEP). See text for discussion.

Thus the final equation for the calculation of valve orifice area \(A\) (in cm\(^2\)) is

\[
A = \frac{CO/(DFP \text{ or } SEP)(HR)}{44.3C \sqrt{\Delta P}} \quad (10.5)
\]

where \(CO\) = cardiac output (cm\(^3\)/min), \(DFP\) = diastolic filling period (sec/beat), \(SEP\) = systolic ejection period (sec/beat), \(HR\) = heart rate (beats/min), \(C\) = empirical constant, and \(\Delta P\) = pressure gradient. The DFP is measured directly from left ventricular versus pulmonary capillary wedge or left atrial pressure tracings as shown in Fig. 10.1.

An empirical constant of 0.7 (later adjusted to 0.85) was derived by comparing calculation and actual mitral valve areas \((1,2)\). Using this constant, the maximum deviation of calculated valve area from measured valve area was 0.2 cm\(^2\). The empirical constant for the aortic tricuspid and pulmonic valve was never derived. The constant for these valves has been assumed to be 1.0 (i.e., 1.0 \(\times\) 44.3 = 44.3) for lack of data comparing actual with calculated valve areas for those valves. Nonetheless, the Gorlin formula remains the “gold standard” for assessing the severity of stenotic cardiac valves.

**MITRAL VALVE AREA**
By rearranging the terms of Eq. (10.5), one sees that for the mitral valve,

\[
\Delta P = \left[ \frac{CO/(HR)(DFP)}{(MVA)(44.3)(0.85)} \right]^2 \quad (10.6)
\]

where \( P \) = mean transmitral pressure gradient, and \( MVA \) = mitral valve area. Thus by doubling cardiac output, one will quadruple the gradient across the valve, if heart rate and diastolic filling period remain constant. The normal mitral orifice in an adult has a cross-sectional area of 4.0 to 5.0 cm\(^2\) when the mitral valve is completely open in diastole. Considerable reduction in this orifice area can occur without symptomatic limitation, but when the area is 1.0 cm\(^2\) or less, a substantial resting gradient will be present across the mitral valve, and any demand for increased cardiac output will be met by increases in left atrial and pulmonary capillary pressure that lead to pulmonary congestion and edema.

Figure 10.2 demonstrates that a cardiac output of 5 L/min can be maintained with only a minimal mitral diastolic gradient as the mitral orifice area contracts from its normal 4.0 to 5.0 cm\(^2\) to a moderately stenotic area of 2.0 cm\(^2\).

**FIG. 10.2.**

After that, the gradient rises so that at an orifice area of 1.0 cm\(^2\) a resting gradient of 8 to 10 mm Hg is required to maintain cardiac output at 5 L/min, with a normal resting heart rate of 72 bpm (Fig. 10.2A). Note that even at this level of cardiac output, substantial increases in gradient may occur in response to tachycardia (Fig. 10.2B,C), which reduces the total time per minute available for diastolic filling. Thus 1.0 cm\(^2\) is generally viewed as the “critical” mitral valve area because only small increases in cardiac output lead to pulmonary congestion and severe dyspnea. Some allowance, however, needs to be made for the patient’s size in assessing critical valve area. Larger patients need greater flows to maintain tissue perfusion than smaller patients and have higher gradients because of higher cardiac output for any given valve orifice area. Thus 1.2 cm\(^2\) could be a critical mitral valve area for a larger patient.

Currently, no uniform agreement exists on indexing critical valve area to body size.

**Example of Valve Area Calculation in Mitral Stenosis**

Figure 10.3 shows pulmonary capillary wedge (PCW) and left ventricular (LV) pressure tracings in a 40-year-old woman with rheumatic heart disease and severe mitral stenosis. This woman also had hypertension and significant elevation of her LV diastolic pressure. The valve orifice area is calculated with the aid of a form reproduced as Table 10.1. In this patient, five beats were chosen from the recordings taken closest in time to the Fick cardiac output determination. Planimetry of the area between PCW and LV pressure tracings (Fig. 10.3) was done for these five beats, and these areas were divided by the length of the diastolic filling periods for each beat, giving an average gradient deflection in millimeters. The mean gradient in millimeters of mercury (Table 10.1, part B) was calculated as the average gradient deflection in millimeters multiplied by the scale factor (mm Hg/mm deflection). In this case, the mean gradient was 30 mm Hg. Next, the average diastolic filling period is calculated (Table 10.1, part C) using the average measured length between initial PCW–LV crossover in early diastole and end-diastole (peak of the R wave by ECG). This average length in millimeters is divided by the paper speed (mm/sec) to give the average diastolic filling period, which in this case was 0.40 sec. Heart rate and cardiac output (Table 10.1, parts D and E) are recorded, ideally from data collected simultaneously with the recording of the PCW–LV pressure gradient. Heart rate was 80 bpm and cardiac output was 4,680 cm\(^3\)/min in the case illustrated in Fig. 10.3. Note that cardiac output must be expressed in cubic centimeters per minute if valve area is expressed in square centimeters of cross-sectional area.
Pulmonary capillary wedge (PCW) and left ventricular (LV) pressure tracings in a 40-year-old woman with severe mitral stenosis. This woman also had systemic arterial hypertension and significant elevation of her LV diastolic pressure. See text for discussion.

Entering these values in the formula given in Table 10.1, part F and using a constant of 0.85(44.3) = 37.7 for the mitral valve, we get

\[
\text{Mitral orifice area} = \frac{(4,680 \text{ cm}^3/\text{min})/(80 \text{ beats/min})(0.40 \text{ sec/beat})}{\sqrt[3]{30 \text{ mm Hg}}} = 0.71 \text{ cm}^2
\]

Because the accuracy of the method to hundredths of a square centimeter has not been demonstrated, the resulting valve area is rounded off and expressed as 0.7 cm$^2$.

**Pitfalls**

**Pulmonary Capillary Wedge Tracing**

In most cases, PCW pressure is substituted for left atrial pressure under the assumption that a properly confirmed wedge pressure accurately reflects left atrial pressure. Nishimura et al. (3) found that transmitral gradient was overestimated by 3.3 ± 3.5 mm Hg when a Swan-Ganz catheter was used to measure wedge pressure compared with actual left atrial pressure. However, these “wedge” pressures were not confirmed as true wedge pressures, using the techniques described in Chapter 5. Conversely, Lange et al. (4) measured left atrial pressure directly (transseptal) and compared it with oximetrically confirmed wedge pressure obtained using a stiff woven Dacron catheter. In this study, overestimation of true left atrial pressure was only 1.7 ± 0.6 mm Hg. Thus we and others believe that the weight of evidence (5) and our own experience support the use of the PCW pressure as a satisfactory substitution for left atrial pressure, except in some patients with pulmonary venoocclusive disease or cor triatrum. Failure to wedge the catheter properly may, however, cause one to compare a damped pulmonary artery pressure with the LV pressure, yielding a falsely high gradient. To ensure that the right heart catheter is properly wedged, one should verify that

1. The mean wedge pressure is lower than the mean pulmonary artery pressure.
2. Blood withdrawn from the wedged catheter is 95% or more saturated with oxygen, or at least equal in saturation to arterial blood.

**Alignment Mismatch**

Alignment of the PCW and LV pressure tracings does not match alignment of simultaneous left atrial and LV tracings because there is a time delay in the transmission of the left atrial pressure signal back through the pulmonary venous and capillary beds. The resulting pressure mismatch is small when PCW pressure is measured in the distal pulmonary arteries using a 7F or 8F Courand or Gooodle-Lubin catheter, but may be larger when wedge pressure is measured more proximally in the pulmonary arterial tree using a balloon-tipped flow-directed catheter. As illustrated in Fig. 5.8, the A and V waves in an optimally damped PCW tracing are delayed typically by 50 to 70 msec compared with a simultaneous left atrial pressure tracing. Thus, ideally, the wedge pressure should be realigned with the LV pressure (using tracing paper) by shifting it leftward by 50 to 70 msec.

The V wave, which is normally present in the left atrium (where it represents pulmonary venous return), peaks
Relationships between cardiac output and mean aortic systolic pressure gradient in patients with aortic stenosis, calculated using Eq. (10.7), derived from the Gorlin equation. Individual curves represent orifice areas of 4.0, 2.0, 1.0, 0.7, 0.5, and 0.3 cm², and represent flow-gradient relations at differing heart rates and systolic ejection periods. (Courtesy of Dr. James J. Ferguson III.)

Calibration Errors

Failure to calibrate pressure transducers properly and adjust them to the same zero reference point may yield an erroneous gradient. A quick way to check the validity of an unsuspected transmitral pressure gradient is to switch left and right heart catheters to opposite transducers, which if calibrated equally yields the same gradient.

Cardiac Output Determination

Cardiac output must be determined accurately using the techniques described in Chapter 8. The cardiac output used in valve area calculation should be the value measured simultaneously with the gradient determination. The measurement used in the valve area formula is usually the forward cardiac output determined by the Fick method or the thermodilution method. If mitral valvular regurgitation exists, the gradient across the valve will reflect not only net forward flow but forward plus regurgitant or total transmitral diastolic flow. Thus using only net forward flow to calculate the valve orifice area will underestimate the actual anatomic valve area in cases where regurgitation coexists with stenosis. It is worth noting that many patients with mitral stenosis have coexistent tricuspid regurgitation. As indicated in Chapter 8, tricuspid regurgitation may cause the thermodilution technique of measuring cardiac output to be inaccurate.

Early Diastasis

Even when left atrial and LV pressures equalize (diastasis) before the end of diastole, there will generally still be flow through the mitral valve after the point of diastasis. The diastolic filling period to be used in valve area calculation should include all of nonisovolumic diastole, not just the period during which a gradient is present.

AORTIC VALVE AREA

An aortic valve area of 0.7 cm² or less is generally considered severe enough to account for the symptoms of angina, syncope, or heart failure in a patient with aortic stenosis. Since the development of symptoms in patients with aortic stenosis portends an abrupt worsening of prognosis, this valve area is termed “critical.” However, it must be pointed out that no unique “critical” valve area has been established and that an aortic valve area as large as 1.0 cm² can cause symptoms and thus be “critical,” especially in a large individual. Conversely, smaller calculated valve orifice areas in a totally asymptomatic patient may not be “critical.” Figure 10.4 illustrates the relationship between cardiac output and aortic pressure gradient over a range of values for aortic valve area at three values for heart rate and systolic ejection period. For the aortic valve, Eq. (10.4) can be rearranged as

FIG. 10.4.

Relationships between cardiac output and mean aortic systolic pressure gradient in patients with aortic stenosis, calculated using Eq. (10.7), derived from the Gorlin equation. Individual curves represent orifice areas of 4.0, 2.0, 1.0, 0.7, 0.5, and 0.3 cm². (A), (B), and (C) represent flow-gradient relations at differing heart rates and systolic ejection periods. (Courtesy of Dr. James J. Ferguson III.)

\[ \Delta P = \left[ \frac{CO/(HR)(SEP)}{44.3AVA} \right]^2 \]  

(10.8)
As can be seen in Fig. 10.4A, at a normal resting cardiac output of 5.0 L/min, an aortic orifice area of 0.7 cm² will result in a gradient of approximately 33 mm Hg across the aortic valve. Doubling of the cardiac output, as might occur with exercise, would increase the gradient by a factor of 4 to 132 mm Hg if the systolic time per minute did not change. This increase in gradient would require a peak LV pressure in excess of 250 mm Hg to maintain a central aortic pressure of 120 mm Hg. Such a major increase in LV pressure obviously increases myocardial oxygen demand and limits ejection performance. These factors contribute to the symptoms of angina and congestive heart failure, respectively (6),(7). The limitations in cardiac output imposed by high afterload may contribute to hypotension when peripheral vasodilation occurs during muscular exercise. Actually, the systolic time per minute does not remain constant during the increase in cardiac output associated with exercise. As heart rate increases during exercise, the systolic ejection period tends to become shorter, but the tendency is counteracted by both increased venous return and systemic arteriolar vasodilation, factors that normally help to maintain LV stroke volume constant (or even allow it to increase) during exercise. Thus, heart rate is increasing but systolic ejection period is diminishing only slightly so that their product (systolic ejection time per minute) increases. This is the counterpart of the decreased diastolic filling time per minute during exercise discussed earlier. Examining Eq. 10.8, it can be seen that the increase in cardiac output will be partially offset by the increase in (HR)(SEP) so that the gradient will not quadruple with a doubling of cardiac output during exercise.

**FIG. 10.5.**

Left ventricular (LV) and right femoral artery (RFA) pressure tracings in a patient who presented with exertional syncope due to aortic stenosis. **A:** The tracings actually recorded, demonstrating the significant time delay for the pressure waveform to reach the RFA. **B:** Realignment using tracing paper. (See text for discussion.)

Figure 10.4B and C show that with decreasing heart rate, the gradient increases in aortic stenosis for any value of cardiac output. This is opposite to the effect of heart rate in mitral stenosis and reflects the opposite effects of heart rate on systolic and diastolic time per minute. Viewed another way, as the heart rate slows in aortic stenosis, the stroke volume increases if cardiac output remains constant. Thus the flow per beat across the aortic valve increases and so does the pressure gradient.

As with mitral stenosis, some allowance must be made for body size in deciding what is a critical valve area in patients with aortic stenosis; larger patients who require higher output may become symptomatic at somewhat larger valve areas. Thus, a very large man with a body surface area of 2.4 m² and a cardiac index of 3.0 L/min/m² would have a cardiac output of 7,200 mL/min. At a heart rate of 68 bpm (Fig. 10.4C), this man might have a 50-mm Hg aortic valve gradient with an orifice area of 0.9 to 1.0 cm². Thus, for him, this might be a critical valve area.

**Example**

Figure 10.5. demonstrates simultaneous pressure tracings from the left ventricle (LV) and right femoral artery (RFA) in a patient with exertional syncope. Since the pulse wave takes a finite period of time to travel from the left ventricle to the femoral artery, the femoral artery tracing is somewhat delayed (Fig. 10.5A). Figure 10.5B shows the LV and RFA tracings realigned to correct for the delay in transmission time. This is accomplished by using tracing paper and aligning the arterial upstroke to coincide with the LV upstroke. After such alignment, the mean pressure gradient can now be obtained by planimetry, and the orifice area can be calculated using the form given in Table 10.1. For this example, the average aortic pressure gradient was 40 mm Hg, the systolic ejection period is 0.33 sec, the heart rate is 74 bpm, and the cardiac output is 5,000 mL/min. Using these values together with an aortic valve constant of (1)(44.3) = 44.3 in the equation in Table 10.1 gives
As discussed in Chapter 7, peripheral arterial pressure waveforms are distorted in ways other than time delay. These distortions include systolic amplification and spreading out (widening) of the pressure waveform. To assess possible errors introduced by the use of peripheral arterial pressure as a substitute for ascending aortic pressure, Folland et al. (8) compared the LV-ascending aortic (LV-Ao) mean gradient in 26 patients with aortic stenosis with the LV-femoral artery (LV-FA) systolic gradient, with and without realignment (Fig. 10.6). Without realignment, the LV-FA gradient overestimated the LV-Ao gradient by about 9 mm Hg. In contrast, aligned LV-FA gradients underestimated the LV-Ao gradient by about 10 mm Hg, possibly representing the fact that peak systolic arterial pressure is higher in peripheral arterial pressure tracings than in central aortic tracings so that the planimetered gradient will be smaller when using LV-FA. Without realignment, this effect is offset by the fact that much of the arterial systolic waveform is outside and to the right of the LV pressure tracing (Fig. 10.6). A second error in gradient measurement can occur if the LV catheter is placed in the LV outflow tract (9). As shown in Fig. 10.7, a gradient usually exists between the body of the left ventricle and outflow tract, produced as blood accelerates when it enters this relatively narrow portion of the left ventricle. A catheter tip placed in the LV outflow tract will measure a typical LV pressure tracing but can underestimate the true LV-aorta gradient by 30 mm Hg. Assey et al. (10) measured the transaortic valve gradients in 15 patients from eight different combinations of catheter locations using the schema shown in Fig. 10.8. The average mean gradient recorded between positions 1 and 3 was the greatest, while the gradient between positions 1 and 5 using the alignment technique produced the smallest value. In some patients, the differences in gradient among the different measurement sites were as much as 45 mm Hg. In calculating aortic valve area, the gradient between sites 1 and 3, which records gradient before pressure recovery, is probably the most accurate reflection of the pressure drop across the valve. When the aortic catheter is placed at a more distal site, it records the effect of pressure recovery, which reduces gradient as blood flow again becomes laminar. The more proximal aortic position is probably the ideal location for measuring the gradient for the valve area calculation; the more distal positions may better reflect the actual overload on the myocardium. When the transvalvular gradient recorded from positions 1 and 5a in Fig. 10.8 is larger than 60 mm Hg, these differences are of little clinical importance. When a small transvalvular gradient is present in conjunction with a low cardiac output, however, the differences between aligned and unaligned tracings and between gradients recorded at different catheter locations may affect the decision about whether to replace the valve. In such instances, we recommend that the problem be obviated by placing a second catheter in the proximal ascending aorta without need for alignment (8) (Fig. 10.6A). As an alternative, the difference between peak central aortic and peripheral arterial pressure is added to the planimetered gradient measured during the Fick output determination. This compensates for the fact that the planimetered gradient with realignment (Fig. 10.6C) underestimates the true gradient (Fig. 10.6A). The most accurate approach, however, involves the use of a second catheter positioned in the ascending aorta, as discussed earlier.

FIG. 10.6.


FIG. 10.7.

A: Pressure tracings recorded from two catheters placed within the body of the left ventricular chamber. Both are
nearly identical. **B:** Pressure recorded by one catheter placed in the body of the left ventricular chamber and by a second catheter placed in the left ventricular outflow tract, proximal to the aortic valve. Both catheters record characteristic left ventricular pressure tracings; however, there is a substantial pressure gradient between the body of the left ventricle and the outflow tract. This is not due to anatomic subvalvular stenosis but rather to acceleration of blood as it enters the relatively narrow outflow tract. **C:** Pressures recorded from the catheter in the body of the left ventricle and from a second catheter in the proximal aorta. These tracings demonstrate the gradient across the aortic valve and outflow tract. (Reproduced from Pasipoularides A. Clinical assessment of ventricular ejection dynamics with and without outflow obstruction. *J Am Coll Cardiol* 1990;15:859, with permission.)

FIG. 10.8.

Two sites for recording left ventricular pressure (1 and 2) and three sites for recording distal pressure (3, 4, and 5) are shown. 5u represents the actual femoral artery pressure tracing which is unaligned with the left ventricular pressure tracing. 5a represents the recording obtained from the femoral artery, which is then manually aligned to match the left ventricular pressure tracing in time. The following are the potential recording sites for obtaining the transaortic valve pressure gradient in aortic stenosis: 1–3, 1–4, 1–5a, 1–5u, 2–3, 2–4, 2–5a, and 2–5u. Gradients recorded at these different sites may vary widely in any given patient. (Reproduced with permission from Assey ME, Zile MR, Usher BW, Karavan MP, Carabello BA. Effect of catheter positioning on the variability of measured gradient in aortic stenosis. *Cathet Cardiovasc Diagn* 1993; 30:287.)

Another approach to increasing the accuracy of transaortic valve gradient measurement using simultaneous LV and femoral artery pressures has been introduced by Krueger et al. (11) at the University of Utah. As seen in Fig. 10.9, the mean LV systolic pressure during interval A and the mean femoral artery systolic pressure during interval B are determined by planimetry. Their difference was nearly identical to the gradient measured by planimetry of simultaneous LV and central aortic pressures and was more accurate than other techniques commonly used (11).

FIG. 10.9.

Simultaneous recordings of left ventricular (LV) and femoral artery (FA) pressures in a patient with aortic stenosis. The mean LV systolic pressure during interval A and the mean FA systolic pressure during interval B are determined by planimetry, and the systolic LV-aortic gradient is estimated as the difference between these mean pressures. (Reproduced with permission from Krueger SK, Orme EC, King CS, Barry WH. Accurate determination of the transaortic valve gradient using simultaneous left ventricular and femoral artery pressures. *Cathet Cardiovasc Diagn* 1989;16: 202.)

If a second catheter is not used to obtain simultaneous LV and peripheral pressures, the gradient may be obtained by recording LV pressure and superimposing it on the aortic pressure obtained immediately after the LV catheter is pulled back into the aorta.

**Pitfalls**

**Transducer Calibration**

As with calculation of mitral valve area, attention to cardiac output determination and transducer calibration is critical. Assurance that proper transducer calibration has been accomplished can be obtained by comparing the left heart catheter pressure with the peripheral arterial catheter pressure before insertion of the left heart catheter into the left ventricle. Since in the absence of peripheral stenosis mean arterial pressure will be the same throughout the arterial tree, the mean pressure recorded by both catheters should be identical, confirming identical transducer calibration. Further gradient verification is made by comparing the LV pressure with aortic pressure obtained by the left heart catheter during catheter pullback. In this case, both LV and aortic pressures are recorded by the same catheter and transducer, eliminating the second transducer as a source of error.

**Pullback Hemodynamics**
When the aortic valve area is diminished to 0.6 cm\(^2\) or less, a 7F or 8F catheter placed retrograde across the valve takes up a significant amount of the residual orifice area, and the catheter may actually increase the severity of stenosis. Conversely, removal of the catheter reduces the severity of stenosis. We have observed that a peripheral pressure rise occurs in severe aortic stenosis when the LV catheter is removed from the aortic valve orifice (12). In our experience, an augmentation in peripheral systolic pressure of more than 5 mm Hg at the time of LV catheter pullback indicates that significant aortic stenosis is present. This sign is present in more than 80% of patients with an aortic valve area of 0.5 cm\(^2\) or less, a point that is discussed further in Chapter 29.

**AREA OF TRICUSPID AND PULMONIC VALVES**

Because of the rarity of tricuspid and pulmonic stenosis in adults, no general agreement exists as to what constitutes a critical orifice area for these valves. In general, a mean gradient of 5 mm Hg across the tricuspid valve is sufficient to cause symptoms of systemic venous hypertension. Gradients across the pulmonic valve of less than 50 mm Hg are usually well tolerated, but gradients of more than 100 mm Hg indicate a need for surgical correction. Between 50 and 100 mm Hg, decisions regarding surgical correction depend on the clinical features in each case.

**ALTERNATIVES TO THE GORLIN FORMULA**

A simplified valve formula for the calculation of stenotic cardiac valve areas was proposed by Hakki et al. (13) and tested in 100 consecutive patients with either aortic or mitral stenosis. The simplified formula is

\[
\text{Valve area} = \frac{\text{cardiac output (liters/min)}}{\sqrt{\text{pressure gradient}}} \quad (10.10)
\]

and is based on their observation that the product of heart rate, SEP or DFP, and the Gorlin equation constant was nearly the same for all patients whose hemodynamics were measured in the resting state, and the value of this product was close to 1.0. For the examples given earlier in this chapter, the simplified formula works reasonably well. Thus for the patient with mitral stenosis (Fig. 10.3) with a cardiac output of 4,680 mL/min and a mitral diastolic gradient of 30 mm Hg, mitral valve area = 4.68 ÷ (30 = 0.85 cm\(^2\) using the simplified formula as opposed to the value of 0.71 cm\(^2\) calculated using the Gorlin formula. For the patient with aortic stenosis whose tracings are shown in Fig. 10.5 (cardiac output 5 L/min, aortic gradient 40 mm Hg), the aortic valve area by the simplified formula is 5 ÷ (40 = 0.79 cm\(^2\) as opposed to 0.73 cm\(^2\) by the Gorlin formula. Because the percentage of time per minute spent in diastole or systole changes substantially at higher heart rates, the simplified formula may be less useful in the presence of substantial tachycardia. This point, however, has not been tested adequately.

**ASSESSMENT OF AORTIC STENOSIS IN PATIENTS WITH LOW CARDIAC OUTPUT**

In the patient with a forward cardiac output of 3 L/min, a mean transvalvular gradient of 20 mm Hg will yield a calculated valve area of 0.7 cm\(^2\), indicating critical aortic stenosis. However, not all such patients actually have severe aortic stenosis. Valve calculations made using the Gorlin formula are flow dependent. That is, as cardiac output increases, calculated area increases, and as cardiac output decreases, calculated area decreases (14),(15). Two potential mechanisms exist by which calculated valve orifice area increases with cardiac output: (a) Increased flow through the stenotic aortic valve in conjunction with increased LV pressure physically opens the valve to a greater orifice area, and thus the valve orifice really is wider during increased flow, and (b) inaccuracies in the Gorlin formula cause the calculated area (but not necessarily the actual orifice area) to be flow dependent. The Gorlins themselves noted that they had no data from which to calculate an empirical constant for the aortic valve (1). Indeed, such a constant has never been calculated but has been assumed to be 1.0 by the cardiologic community. The issue remains in doubt, but in all probability both explanations are correct in part. On one hand, Tardif and coworkers have shown that two-dimensional transesophageal echocardiographic imaging of the stenotic aortic valve has failed to
demonstrate true change in valve orifice areas when increased flow caused calculated area to increase (16). These data suggest calculated area-flow dependence resides within the calculation rather than in representing a true change in area. However, it remains unclear whether the echocardiographic method used is sensitive enough to detect tiny (0.2 to 0.4 cm²) changes in actual valve area. On the other hand, Voelker and colleagues working in vitro concluded that changes in calculated orifice area with changes in flow were probably due to actual changes in valve area (17). Flow dependence of calculated valve orifice area appears less in bicuspid than in tricuspid valves (18) but is greater at lower than at higher flows (19). These problems in assessing stenosis severity have substantial clinical importance. Consider a patient with reduced cardiac output and low LV ejection fraction who has both cardiomyopathy and mild aortic stenosis. Despite a calculated valve area of 0.7 cm², such a patient will probably not benefit from aortic valve replacement because aortic stenosis was not the cause of the LV dysfunction. On the other hand, although patients with low aortic valve gradients are generally at higher risk for perioperative death associated with aortic valve replacement (7), (20), Brogan et al. (21) have demonstrated that some patients with low gradients may improve substantially following surgery. It is likely that such patients have truly severe aortic stenosis, which is the cause of their hemodynamic decompensation; in these patients, correcting the aortic stenosis is beneficial. Preliminary data from three studies suggest that cautious hemodynamic manipulation in the catheterization laboratory can distinguish between these two different clinical entities (22–25). In patients with mild aortic stenosis, an infusion of nitroprusside or dobutamine increases forward output substantially but may actually decrease the transvalvular gradient. In such cases, the calculated aortic valve area increases dramatically and is no longer within the “critical” range. On the other hand, in patients with truly severe aortic stenosis, infusion of nitroprusside widens the transvalvular gradient and increases the calculated aortic valve area only slightly, if at all. The results of nitroprusside infusion in a patient with mild aortic stenosis are demonstrated in Table 10.2 (24). The patient's initial calculated valve orifice area was 0.6 cm², which would indicate a need for surgery. However, following nitroprusside infusion, the gradient actually fell and calculated valve area increased. The patient improved on chronic vasodilator therapy, usually contraindicated in aortic stenosis unless the disease is mild. It must be emphasized that infusion of nitroprusside in patients with aortic stenosis must be performed with great caution, since if true aortic stenosis is present, hypotension may result. If it is known that the patient has normal coronary arteries, dobutamine, which produces similar changes in cardiac output, can be infused instead of nitroprusside. However, dobutamine infusion may be dangerous in patients who also have coronary disease, in whom it may precipitate ischemia.

**VALVE RESISTANCE**

Valve resistance is simply the mean aortic valve gradient divided by the cardiac output per second of systolic flow. It has the advantage of being calculated from two directly obtained pieces of data (output and gradient) and requires no discharge coefficient (26). A simplified formula for calculating aortic valve resistance is

\[
\text{Valve resistance} = \frac{\text{Mean gradient}}{\left(\text{Systolic ejection period} \times \text{Heart rate}\right) \times 1.33}
\]

\[
\text{Cardiac output (liters/min)}
\]

\[\text{(10.11)}\]

Valve resistance has been shown by Cannon et al. (22) to help separate patients with severe aortic stenosis from those patients who had similarly small calculated aortic valve areas, but who were subsequently demonstrated to have mild disease. Resistance appears less flow dependent than valve area (22), (26). Resistance is unlikely to supplant the Gorlin formula in assessing stenosis severity but may be an important adjunct to it in patients with low cardiac output.

Currently, we recommend cautious hemodynamic manipulation with dobutamine or nitroprusside for patients with a cardiac output of less than 4.5 L/min who have a transvalvular gradient of less than 40 mm Hg and a valve resistance of less than 275 dyn-sec-cm⁻². If patients respond by substantially increasing the measured gradient, they probably have truly severe aortic stenosis and may benefit from aortic valve replacement. However, if cardiac output increases substantially but gradient increases only slightly, or actually declines, the aortic stenosis is mild and the patient is unlikely to benefit from aortic valve replacement.
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