Hydraulic estimation of stenotic orifice area: a correction of the Gorlin formula

SCOTT R. CANNON, PH.D., KENT L. RICHARDS, M.D., AND MICHAEL CRAWFORD, M.D.

ABSTRACT To determine the source of errors in the Gorlin formula for estimating stenotic valvular orifice area, we used a pulsatile flow model that emulated left ventricular and aortic pressures and flow and allowed control of ventricular outflow orifice area. After comparing orifice areas calculated by the Gorlin formula with actual orifice areas, the Gorlin formula constant (k) was found to be highly correlated with the square root of the mean transvalvular gradient (r = .95). A new formula was derived empirically and predicted areas more accurately and with smaller standard errors than the Gorlin formula in the model (r = .98, SEE = 0.11 and r = .87, SEE = 0.28, respectively) in a series of 19 patients with Hancock porcine xenograft valves (r = .89, SEE = 0.07 and r = .60, SEE = 0.12, respectively) and in the original series of patients reported by Gorlin and Gorlin in proposing the Gorlin formula (r = .93, SEE = 0.11 and r = .91, SEE = 0.12, respectively).


THE GORLIN FORMULA,1 introduced in 1951 and later modified in 1972,2 has become the standard method of estimating valvular orifice areas in the presence of stenosis. Unfortunately, significant questions exist concerning its accuracy. Several investigators3–9 have noted apparent errors in valve area calculations made with the Gorlin formula (involving both patient studies in vivo and models in vitro), particularly in the presence of extremes of pressure gradient or flow. After studying hemodynamic data from 175 patients with mitral xenografts who underwent catheterization shortly after surgery, Ubago et al.3 noted a wide disparity between valve areas calculated with the Gorlin formula and actual valve areas. This same disparity was reported by Bache et al.,4 who found questionable increases in valve areas calculated with the Gorlin formula during exercise in patients with significant native aortic stenosis. Richter5 suggested that errors may be as great as 100% in patients with low valvular flows and pressure gradients.

The Gorlin formula was derived from two simplified equations describing steady-state flow through an orifice:

\[ F = A \cdot V \]  
\[ V = (c\sqrt{2g}) \cdot \frac{V}{\text{MPG}} \]

where F represents valvular flow (ml/sec), A is valvular orifice area (cm²), V is flow velocity (cm/sec), c is a constant representing primarily the proportion of the pressure gradient causing flow (as opposed to friction, turbulence, etc.) and depending on valve operational characteristics, g is the gravitational constant (980 (cm/sec²)), and MPG represents the mean pressure gradient (mm Hg) across the orifice. Substituting equation 2 for V into equation 1 gives the Gorlin formula:

\[ A = \frac{F}{k\sqrt{\text{MPG}}} \]

where k is the combined proportionality constant representing c times the square root of twice the force of gravity. Gorlin and Gorlin empirically determined k for mitral stenosis from hemodynamic measurements (15 records) in 11 patients and compared them with valve areas determined during surgery (six patients) or at autopsy (five patients). Gradients ranged from 16 to 33 mm Hg and actual valve areas ranged from 0.5 to 1.4 cm². The published Gorlin constant of 31 (or 0.7\sqrt{2g}) represents an average of constants calculated from each patient record, which ranged from 23 to
40 (SD = 5.7). The constant k has never been adequately determined for aortic stenosis. Herman et al. suggested the use of a Gorlin constant of 44.3 (or 1.0V2 g), which implies that 100% of the pressure drop across the aortic valve results in flow.

Theoretic considerations from several authors suggest that pulsatile flow cannot be accurately modeled over a wide range of orifice areas, flows, and gradients with k as a constant. Gorlin and Gorlin predicted that k would be larger in aortic stenosis (where gradients and valvular flow rates are larger) than in mitral stenosis. Ubago et al. suggested that k is actually inversely proportional to flow in a pulsatile system.

It has been postulated that errors in the Gorlin formula may be due primarily to the fact that valvular orifices are not constant but rather are flow and pressure dependent. Static studies of porcine valves in vitro showed that they do in fact vary slightly in cross-sectional area with variations in gradient and flow at low pressures and flows. It has also been postulated that the Gorlin constant k may not be a constant at all in a pulsatile system but a function of flow or pressure.

Our purpose was threefold: (1) determine the source of error in the Gorlin orifice formula by means of a hemodynamically accurate pulsatile flow model and a wide range of orifice areas, valvular flows, and pressure gradients; (2) empirically derive an accurate orifice area formula based on the results in the pulsatile flow model; (3) assess this formula with a patient study in vivo.

Methods

Flow model. Because errors in the Gorlin formula would be expected to be more apparent in aortic stenosis, where gradients and flow inertia are large, we used a pulsatile flow model to simulate human left ventricular and aortic pressures, flow, and anatomy (figure 1). To simulate left ventricular hemodynamic conditions, a porcine valve holder was positioned over a 300 ml latex bag suspended in a water chamber. A 2.5 cm diameter plastic outlet tube, molded to simulate the anatomy of the ascending and transverse aorta, was attached to the valve holder. A 25 cm section of compliant latex tubing containing a variable restrictive orifice was added to allow control of systemic resistance and compliance. A constant pressure reservoir was positioned 20 cm above the ventricle and connected through a simple flap valve to provide diastolic filling. Pressures in the water chamber were controlled with an air bag connected to pressure and vacuum sources through digitally controlled valves. Flow was measured with an electromagnetic flowmeter (Zepeda Instruments SWF-4RD) calibrated for the model by timing the collection of pump output. Pump timing was set at 1 beat/sec with a 20% to 24% systolic pressure period.

To test the flow and pressure dependence of porcine valvular orifice areas, valve operation was first videotaped. A video camera was mounted above the outlet tube and directed down to the porcine valve. The system was filled with a clear mixture of glycerin and water with a viscosity matching that of whole human blood. A normal 25 mm Carpentier-Edwards porcine valve was mounted in the valve holder and valve operation was videotaped at pump outputs from 0.5 to 9.0 liters/min (valvular flow = 30 to 550 ml/sec). The normal porcine valve was then replaced with a stenotic 27 mm Hancock porcine valve (removed at operation) and valve operation was videotaped at pump outputs of 1.0 to 3.1 liters/min (valvular flow = 65 to 163 ml/sec, mean pressure gradient = 29 to 62 mm Hg). At 3.1 liters/min the stenotic valve required a ventricular pressure greater than 150 mm Hg (the limit of the flow model) and higher flows could not be achieved. Orifice areas from the maximally opened valve were measured by planimetry from the videotape with a MicroSonics video image analyzer and an IBM PC-XT computer.

For hemodynamic measurements, the plastic aorta was replaced with a compliant latex aorta and the system was filled with a mixture of glutaraldehyde-fixed canine red cells, glycerin, and water adjusted to produce a hematocrit of 40% and a viscosity matching that of whole human blood. A normal functioning 27 mm Carpentier-Edwards porcine valve was placed in the valve holder. To simulate stenosis, a flat supravalvular diaphragm snare (figure 1) was placed 0.5 cm downstream from the open porcine valve. The valve-snare combination was calibrated at multiple orifice sizes by timing the flow of a known volume of fluid from a constant pressure source through the orifice. The effective area results were then confirmed by replacing the valve-snare with a series of fixed diaphragms of known orifice size and repeating the calibration process. Systemic resistance was set to a normal human value of approximately 1300 dyne-sec-cm^{-5} with measured mean aortic pressure and flow. Compliance was adjusted to produce expected pressure curves (figure 2).

Pressures in the ventricle and aorta were measured simultaneously with micromanometer-tipped catheters and recorded on
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FIGURE 2. Flow model aortic and ventricular pressure tracings. Model output was 4.5 liters/min through an effective valve-snare orifice area of 1.0 cm². Vertical timing lines are at 1 sec intervals.

a strip chart recorder. Hemodynamic data were collected for seven valve-snare orifice areas (0.75, 1.0, 1.25, 1.5, 1.75, 2.0, and 2.25 cm²) at each of six flow rates (2.5, 3.0, 3.5, 4.0, 4.5, and 5.0 liters/min), which resulted in 42 hemodynamic measurements.

The mean valvular systolic pressure gradient was measured from each strip chart record by standard graphic techniques; the systolic pressure difference was integrated between the aortic and ventricular cross-over points with a planimeter, divided by the systolic ejection period measured between the aortic and ventricular cross-over points, and scaled. Valvular flow was calculated as pump output (ml/sec) divided by the systolic ejection period (the systolic time percentage of a beat).

Patient studies. Twenty-four patients who had received Hancock porcine xenografts (17 aortic, seven mitral) within 6 months underwent routine heart catheterization as part of a research protocol. None has symptoms or signs of valvular dysfunction and all gave informed consent on a form approved by our institutional review board. Pressures were measured by fluid-filled catheters connected to Gould-Statham transducers and recorded on an Electronics for Medicine VR-16 strip chart recorder. Left ventricular pressures were measured by means of transvalvular pressures and paired with pulmonary arterial wedge (mitral valve) or aortic (aortic valve) pressure tracings. Cardiac outputs were measured by Fick or green dye techniques when mild or no regurgitation was evident, or by single-plane left ventricular cineangiography when regurgitation greater than 30% was noted (four patients). Mean pressure gradient and valvular flow were again calculated by standard graphic techniques.

For a standard of reference, published orifice areas for 21 and 31 mm Hancock valves and for 33 mm Hancock valves determined photographically during operation were used to calculate areas for 23, 25, and 27 mm valves by linear interpolation techniques (table 1).

To ensure that accurate hemodynamic measurements were being made in nonstenotic valves, only patients meeting the following criteria were included in analysis: Aortic valve patients having gradients less than 20 mm Hg with outputs less than 5 liters/min or gradients less than 30 mm Hg with outputs over 5 liters/min were accepted. Mitral valve patients with gradients less than 10 mm Hg were accepted. In addition, patients with no measurable peak-to-peak gradients (a measurable transvalvular gradient during peak systolic or diastolic pressure) were excluded. Based on these criteria, one patient was rejected as having abnormally severe stenosis (aortic mean pressure gradient = 54 mm Hg) and four patients were rejected because of a lack of a peak-to-peak gradient (three aortic, one mitral). The remaining 19 patients had valve sizes from 21 to 33 mm, gradients from 3.5 to 27 mm Hg, and cardiac outputs from 3.2 to 9.9 liters/min.

Statistical analysis

Video data in vitro. Areas planimetered from video images were plotted against valvular flow. The percent change in orifice area for flow rates from 150 to 500 ml/sec was noted.

Hemodynamic data in vitro. Using the orifice area obtained with the calibrated snare as the standard of reference, we correlated calculated areas with actual areas. The Gorlin constant k was correlated with valvular flow and the square root of the pressure gradient by regression analysis techniques.

Patient data. With published orifice areas (table 1, Hancock porcine valves) used as a standard of reference, actual orifice areas were correlated with calculated areas, again by regression analysis techniques.

Results

Valve operation data in vitro. Valve orifice areas planimetered from the videotape of valve operation were plotted against valvular flow (figure 3) for both the normal and stenotic valves. The stenotic xenograft valve (removed at operation) varied by less than 0.05 cm² in orifice area over the range of pressures and flows tested. A definite decrease in orifice area was noted for the normal porcine valve at extremely low flow rates, yet less than a 0.1 cm² orifice area difference was noted in the normal valve through the valvular flow range of 150 to 500 ml/sec.

Hemodynamic data in vitro. For each model valve set-

TABLE 1

Hancock porcine valve areas

<table>
<thead>
<tr>
<th>Size (mm)</th>
<th>Area (cm²)</th>
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</thead>
<tbody>
<tr>
<td>21</td>
<td>1.29*</td>
</tr>
<tr>
<td>23</td>
<td>1.36</td>
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<tr>
<td>31</td>
<td>1.65*</td>
</tr>
<tr>
<td>33</td>
<td>1.79f</td>
</tr>
</tbody>
</table>

* Determined photographically during operation by *Wright16 and fThomson and Barratt-Boyes15 or estimated using linear interpolation techniques.

FIGURE 3. Planimetered maximum orifice area plotted against aortic valve flow (AVF) for a normal functioning porcine valve and for a stenotic porcine valve removed at operation.
(r > .87, actual valve area range 0.75 to 2.25 cm²). This relationship can be seen in a plot of the area predicted by the Gorlin formula vs valvular flow (figure 5) and the mean systolic pressure gradient (figure 6) for actual areas of 1.75 cm². When k was compared with valvular flow across all orifice areas, correlation decreased (r = .46). However, when k was compared with the mean gradient across all orifice areas, the strong relationship persisted; regression against the square root of the mean gradient provided the best fit (figure 7, r = .95, SEE = 1.32). The resulting regression line does not appear to pass through zero. These data imply, then, that the k factor in the Gorlin formula is not a constant at all but is proportional to the square root of the mean systolic gradient (k is equal to a constant times the square root of the mean gradient):

\[ k\sqrt{MPG} = (k'\sqrt{MPG})\sqrt{MPG} = k' MPG \]  

(5)

where k’ is a new proportionality constant that is reasonably independent of both valvular flow (r = .10) and mean pressure gradient (r = .40). With this rela-

FIGURE 4. Area predicted by the Gorlin formula plotted against the actual orifice area at six flow rates (5, 4.5, 4, 3.5, 3, and 2.5 liters/min) in the pulsatile flow model.

The average value of k (20.0, SD = 4.1, range 12.0 to 29.3) compared favorably with the average value of 21 seen in the nonpulsatile flow model experiments of Gorlin and Gorlin¹ with a native stenotic valve.¹ Orifice areas predicted by the Gorlin formula are plotted against actual orifice areas in figure 4. Fair correlation can be seen (r = .87, SEE = 0.28), again similar to the original results of Gorlin and Gorlin¹ (r = .90). Several interesting observations can be made from this plot. A small offset is noted in Gorlin area estimations. At each actual orifice area, formula-predicted areas increase as valvular flow and gradients increase. Predicted areas overestimated small orifice areas (1.5 cm² or less) by as much as 47% and underestimated large orifice areas (greater than 1.5 cm²) by as much as 40%.

When the Gorlin proportionality constant k was compared with valvular flow and mean pressure gradient for each orifice area, a strong correlation was noted

FIGURE 5. Area predicted by the Gorlin formula plotted against aortic valve flow (AVF) with the snare orifice area set at 1.75 cm² in the flow model.
The Gorlin formula were plotted against actual areas predicted by table 1 (figure 9). Only fair correlation was noted \((r = .60, \text{SEE} = 0.12)\). A Gorlin constant of 44.3 was used for aortic valves and a value of 38 was used for mitral valves.\(^1\) The new proportionality constant \(k'\) was calculated from each of the 19 patient records and averaged \((k' = 80.3)\). The \(h\) constant was calculated to be \(1.20\). When orifice areas predicted by the new formula (equation 6) were compared with actual areas (figure 10 and table 2), a much better correlation could be seen \((r = .89)\) with a smaller standard error \((\text{SEE} = 0.07)\). Areas predicted by equation 6 were closer to actual areas than those predicted by the Gorlin formula in 17 of the 19 patients. For actual areas less than 1.5 cm\(^2\), the largest error of the new formula was 7\% (compared with 59\% for the Gorlin formula). For actual areas greater than 1.5 cm\(^2\), the maximum error was 9\%.

**Patient studies.** Orifice areas calculated by the Gorlin

\[
A = F(k' \text{ MPG}) + h \quad (6)
\]

where \(h\) is a constant added to correct the small offset noted in Gorlin area predictions. The average value of \(k'\) from the model data was 1.92 (SD = 0.15, range 1.59 to 2.22), and the value of \(h\) was calculated to be \(-0.226\). When orifice area predicted by the new formula (equation 6) was plotted against actual orifice area (figure 8), a much better correlation could be seen \((r = .98, \text{SEE} = 0.11)\) than that previously noted for the Gorlin formula \((r = .87, \text{SEE} = 0.28)\). Accuracy was excellent in the range of moderate-to-severe stenosis. The largest estimate error for orifice areas 1.5 cm\(^2\) or less was only 14\% for the new formula (compared with 47\% for the Gorlin formula). For orifice areas greater than 1.5 cm\(^2\), the largest estimate error was only 12\% (compared with 40\% for the Gorlin formula).

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was less dependent on the square root of the mean gradient than the Gorlin constant k (r = .60 and .02, respectively).

One data point from the data of Gorlin and Gorlin was omitted from this analysis: The calculated orifice area for the patient labeled “MT” given in the Gorlin and Gorlin table comparing actual vs calculated areas does not correspond with the hemodynamic data given in a separate table for “MT,” leading one to suspect a possible error in data entry.

Discussion

The Gorlin formula is widely accepted today as the clinical standard in the hemodynamic assessment of valvular stenosis in native and porcine xenograft valves. It is also the “gold standard” of reference for the development of noninvasive techniques used in assessing valve area. Unfortunately, validation of the Gorlin equation over a wide range of pulsatile hemody-
by the mean gradient (equation 6) rather than valvular flow divided by the square root of the mean gradient (equation 3), as implied by the Gorlin formula. The new orifice formula provided better correlations with actual valve areas and lower standard errors than the Gorlin formula in the model, in the present human study, and with the patient data used to demonstrate the Gorlin formula originally.

Some of those who have noticed apparent pressure or flow dependencies in orifice areas predicted by the

<table>
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<tr>
<th>Valve position</th>
<th>Actual area (cm²)</th>
<th>Gorlin formula area (cm²)</th>
<th>New formula area (cm²)</th>
<th>Valve flow (ml/sec)</th>
<th>Mean gradient (mm Hg)</th>
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FIGURE 10. Area predicted by the new formula plotted against the actual orifice area for patients with normally functioning Hancock porcine valves. The scale is the same as in figure 9 comparing Gorlin formula areas with actual orifice areas. The solid and dashed lines represent the lines of identity (ident.) and regression (regr.), respectively.

namic conditions has never been adequately done. In addition, there is little reason to expect that the steady-state relationships used in the derivation of the Gorlin formula are sufficiently valid in a pulsatile flow model to provide accurate area estimates over a wide range of flows and gradients, particularly in aortic stenosis where gradients and valvular flow rates are large. Apparent errors in Gorlin formula estimates of both mitral and aortic valve area have been noted in patients and in models, involving both native and prosthetic valves where a broad range of hemodynamic conditions were present.3-9

Using a pulsatile flow model in which valve orifice area, pressures, and flow could be independently controlled, we were able to show that the Gorlin constant k is actually not a constant but is related to the square root of the mean pressure gradient. A new orifice formula was derived empirically; our data demonstrate that valve area is proportional to valvular flow divided

TABLE 3
Actual and predicted valve areas: original data of Gorlin and Gorlin

<table>
<thead>
<tr>
<th>Valve position</th>
<th>Actual area (cm²)</th>
<th>Gorlin formula area (cm²)</th>
<th>New formula area (cm²)</th>
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Gorlin formula have attributed the phenomenon to variability in actual orifice area.\(^3, 9, 12, 15, 16\) Unfortunately, studies in vitro to demonstrate orifice area changes in response to changes in flow or pressure gradient have been conducted in steady-state models\(^15\) or at low valvular flow rates.\(^9, 13\) Patient studies have used the Gorlin formula as the standard of reference.\(^3, 12, 16\) Our results indicate that there is significant anatomic orifice variability at low flows in porcine valves. However, in the range of valvular flows normally encountered in the evaluation of aortic valves (150 to 500 mm Hg), orifice area changes were small. Changes in orifice area in the stenotic porcine valve tested were insignificant. Further work needs to be done to confirm these results in various types of prosthetic valves over a wide range of valve sizes.

The most clinically significant differences between the Gorlin formula and the new orifice formula lies in the estimation of valve areas less than 1.5 cm\(^2\), a range at which surgical decisions are most likely to be influenced. Maximum errors with the Gorlin formula were 47% in the flow model, 59% in the patient study, and 26% in the original mitral valve data; this compared with 14%, 7%, and 21%, respectively, for the new orifice formula. Although the differences between the two formulas were not large in the original data of Gorlin and Gorlin,\(^1\) the range of valve areas was not large; 12 of the 14 areas in table 3 are between 0.5 and 0.9 cm\(^2\). The range of pressures and valvular flow was also small. One would expect that a wider range of hemodynamic conditions, significant differences between the two formulas would be even more apparent in native valves.

The constants k' and h in the new formula should be expected to be dependent on valve operation similar to the constant c in equation 2. Indeed, a different set of constants was calculated in each data set (the flow model, the present series of patients, and the original series of patients). However, the constant set (k' = 80.3, h = 1.20) calculated for the data from our patients was accurate in estimations of both aortic and mitral valve orifice area. The constant set (k' = 6.84, h = 0.08) calculated for the data of Gorlin and Gorlin\(^1\) was verified only in their mitral stenosis data. Although we would expect this same constant set to be applicable in native aortic stenosis, we have not verified this impression.

The new formula is, of course, still an approximation. The equations for valvular pulsatile flow (corresponding to equations 1 and 2) are quite complex. We have merely suggested a better orifice formula based on an empirical study of errors in the Gorlin formula.

Limitations of the study that should be considered are: (1) constant systemic resistance and capacitance and the symmetric snare orifice in the model are only approximations of the more complex situations found in human stenotic valves; (2) only two porcine valves were used to measure changes in actual orifice area with changes in flow and pressure gradient. Since results found in the flow model were confirmed in both the prosthetic valve data from our patients and the original native valve data of Gorlin and Gorlin,\(^1\) we believe these limitations were not significant.

In summary, we have determined in a pulsatile flow model of aortic stenosis in vitro that the “Gorlin constant” varies with transvalvular pressure gradient. Consequently, we derived a more correct relationship between valvular flow and mean pressure gradient and the actual valve orifice area. Confirmation of the results in vitro with patient studies and the original mitral stenosis data of Gorlin and Gorlin\(^1\) leads us to suggest that this new formula should provide a more accurate estimate of valve area than the Gorlin formula. Further work now needs to be done to accurately determine and verify the constants k' and h for stenotic native and prosthetic valves.

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