Limitations of Doppler Echocardiography in the Evaluation of Serial Stenoses

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A 71-year-old man with a history of coronary artery disease with multiple previous interventions, atrial fibrillation, and moderate aortic stenosis presented with progressive angina. Physical examination revealed decreased peripheral pulses and a 2 to 3/6 mid- to late peaking systolic ejection murmur at the upper right sternal border, unchanged with squat-to-stand maneuver. Coronary angiography ruled out significant obstructive coronary disease. Echocardiography revealed a septal thickness of 19 mm (Figure 1A). Doppler interrogation of the left ventricular (LV) outflow tract revealed a late peaking systolic velocity of 4.2 m/s (Figure 1B) and an early peaking systolic velocity of 3.8 m/s with a mean gradient of 40 mmHg (Figure 1C), with 2 discrete levels of color aliasing noted on 2-dimensional (2D) Doppler color imaging (Movies I and II in the online-only Data Supplement). Because of patient reluctance to undergo surgery based on noninvasive evaluation, hemodynamic catheterization was performed to characterize the degree of obstruction accurately at each level.

A fluid-filled catheter was positioned in the LV apex via a transseptal approach and a high-fidelity micromanometer-tipped catheter was introduced into the LV outflow tract through a retroaortic multipurpose guide, which was subsequently withdrawn into the aorta (Figure 1D). At baseline, there was a 48 mmHg mean gradient between the LV apex and aorta because of 2 separate levels of obstruction: a 25 mmHg mean gradient fixed subvalvular obstruction between the LV apex and outflow tract and a 25 mmHg mean gradient between the LV outflow tract and aorta.

This case demonstrates a significant discrepancy between the measured gradients at echocardiography and cardiac catheterization in the presence of tandem stenoses. Application of the modified Bernoulli equation in ultrasonography revolutionized the management of valvular heart disease,1 resulting in a significant decline in the use of cardiac catheterization for evaluation of aortic stenosis.2 However, limitations of applying this mathematical construct to obstructions in series become apparent by reviewing the derivation of the simplified equation.

The Bernoulli principle is based on the principle of conservation of energy and posits that the total energy (the sum of potential energy, manifest as pressure and kinetic energy, manifest as velocity of flow) before a stenosis is the same as that after, given that energy contributed by gravitational acceleration is constant and the system is frictionless and noncompressible (ie, the total energy in the system should be the same at any point along the streamline; Figure 2A). When used in the evaluation of stenosis, the full equation is modified to

\[ \Delta P = \frac{1}{2} \rho (V_x^2 - V_y^2) = 4 \left(V_x^2 - V_y^2\right) \]

and then further simplified to \( \Delta P \approx 4V_x^2 \) given modeling assumptions.

However, in situations where the proximal velocity is >1 m/s, its exclusion is no longer appropriate.3 Tandem stenoses, as in our patient, are one such example. As depicted in Figure 2B, the proximal stenosis (x) results in a significant velocity \( V_x \) approaching the distal stenosis (y). The pressure gradient across y would, therefore, be calculated by \( \Delta P = 4(V_x^2 - V_y^2) \), and not as \( \Delta P = 4(V_x^2 - V_y^2) \), as would be measured with typical application of Doppler echocardiography. Doppler also produces a systematic overestimation of the pressure gradient because of the phenomenon of pressure recovery. As deceleration occurs, kinetic energy is transformed to potential energy (pressure). Therefore, the actual pressure distal to a stenosis is higher than that at the stenosis and the true pressure gradient is lower than what is estimated by the modified Bernoulli. This effect is emphasized in the setting of long stenoses (or tandem stenoses as in our case) and narrower distal diameters. Viscosity and friction may also become more relevant in longer stenoses and the modified Bernoulli does not account for these.

The clinically relevant hemodynamic finding is the severity of valvular aortic stenosis. This was overestimated by applying the simplified Bernoulli equation to the aortic valve velocity. Figure 3 illustrates the incorporation of a higher proximal velocity into this equation, demonstrating that the exact gradient cannot be estimated noninvasively. Thus, if 2D or 3D echocardiographic evaluation is unable to distinguish the degree of severity of tandem stenoses and clinical decision making will be affected by differentiating the magnitude of hemodynamic contributions, then invasive hemodynamic evaluation is recommended.
Disclosures
None.

References

Key Words: aortic stenosis ▪ cardiac catheterization ▪ Doppler echocardiography ▪ hypertrophic cardiomyopathy ▪ serial stenoses

Figure 1. Hemodynamic assessment of tandem stenoses. **A**, Transthoracic echocardiography via a parasternal window revealed thickening of the intraventricular septum, measuring 19 mm. **B**, Doppler interrogation of the left ventricular outflow tract revealed a late peaking systolic velocity of 4.2 m/s. **C**, In addition, Doppler interrogation of the left ventricular outflow tract demonstrated a distinct early peaking systolic velocity of 3.8 m/s with a mean gradient of 40 mmHg. **D**, High-fidelity left heart catheterization revealed mean gradient of 48 mmHg between the LV apex and aorta (highlighted in grey) because of 2 separate levels of obstruction: a fixed subvalvular obstruction with a 25 mmHg mean gradient between the LV apex and the LV outflow tract (highlighted in blue) and a 25 mmHg mean gradient between the LV outflow tract and the aorta (highlighted in green).

Figure 2. Derivation and application of the simplified Bernoulli equation. **A**, The full Bernoulli equation:

$$\Delta P = P_1 - P_2 = \frac{1}{2} \rho \left( V_2^2 - V_1^2 \right) + \rho \int_{1}^{2} \frac{dV}{dt} ds + R(V)$$

where $\rho$ is the mass density of blood, $V_1$ is the velocity in the outflow tract obtained by pulse wave Doppler. The equation is further simplified to

$$\Delta P = \frac{1}{2} \rho \left( V_2^2 - V_1^2 \right) = 4V_2^2$$

as the latter 2 terms, the inertial and viscous friction terms, are negligible and $\frac{1}{2} \rho$, where $\rho$ is the mass density of blood, equates to a constant of 4. $V_1$=maximum velocity across the system obtained by continuous wave Doppler; $V_2$=velocity in the outflow tract obtained by pulse wave Doppler. The equation is further simplified to

$$\Delta P = 4V_2^2$$

as $V_2$ is assumed to be <1 m/s and, therefore, negligible, as it contributes minimally to the overall equation. **B**, In the case of tandem stenoses, the proximal stenosis (x) results in a significant velocity $V_2$ approaching the distal stenosis (y), negating modeling assumptions made in derivation of the simplified Bernoulli equation.
Figure 3. Invasively and noninvasively derived aortic pressure gradients. A, Aortic valve pressure gradient at catheterization (left ventricular outflow tract [LVOT] pressure minus aortic [AO] pressure; green) and echocardiographically derived pressure via the modified Bernoulli equation (blue) are shown for a single beat. For noninvasive pressure calculation, the distal velocity (denoted $V_3$ in Figure 2B) is determined from the AV continuous wave Doppler (B). The proximal velocity (denoted $V_2$ in Figure 2B) is determined from the LVOT continuous wave Doppler (C), differentiated from the AV trace by the late peaking signal. The difference in echocardiographically derived pressures (blue) does not approximate the true valvular stenosis (green).
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doi: 10.1161/CIRCIMAGING.113.000575
Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-9651. Online ISSN: 1942-0080

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