

## EDITORIAL COMMENT

# Stenosis Is in the Eye of the Observer: Impact of Pressure Recovery on Assessing Aortic Valve Area\*

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**Severity of aortic stenosis.** The study by Garcia et al. (1) in this issue of the *Journal* has bearing on a long-standing clinical quandary: Doppler continuity aortic valve areas (2,3), taken in the context of typical catheterization valve area cutoff values (4,5), often appear to produce misclassification toward higher degrees of severity, particularly in patients with relatively mild obstruction (6–8). Several technical factors may produce apparent area overestimation, such as the assumption of a uniform subvalvular velocity profile in the Doppler calculation. This study, however, shows that an independent cause of Doppler–catheter discrepancy is pressure recovery, a real physical phenomenon.

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**Pressure recovery.** There has been a growing awareness that the simplified Bernoulli equation applied to Doppler velocities does not completely describe the pressure loss across native or prosthetic valves. The kinetic energy of blood accelerated through the orifice is partially recovered as pressure downstream in the aorta. This diminishes the net loss of pressure across the valve, compared with the predicted  $4v^2$  that would result if no pressure were recovered (9–23). Pressure recovery therefore reduces the work load on the left ventricle, which is proportional to the net pressure head loss  $\times$  flow rate (19,20).

**Effect on area: principles.** To date, studies have focused mainly on the recovery of pressure and the discrepancies between catheter and Doppler pressure gradients. These same discrepancies, however, should translate into corresponding differences between Doppler- and catheter-derived effective orifice areas (EOAs), as well as variation in catheter-derived areas depending on the measurement site. As flow re-expands beyond the valve, energy is conserved, and kinetic energy is converted back to pressure to the

extent permitted by minor frictional losses and turbulence. The pressure gradient measured by a catheter withdrawn through the orifice will therefore progressively decrease over several centimeters beyond the valve, as compared with that measured at the narrowest flow stream or vena contracta. The aortic valve area calculated from these pressure gradients by the Gorlin equation will thus increase to a downstream plateau. The Doppler EOA, on the other hand, is linked to the maximal velocity at the vena contracta and will agree with the catheter-derived area only when pressure is measured directly at the vena contracta. The Gorlin area, typically derived from downstream pressure measurements, will therefore tend to exceed the Doppler value (6).

**Effect on area: catheter data.** This translation of pressure recovery into what might be termed “area recovery” was first described by Schöbel et al. (17) in 37 patients with aortic stenosis, in whom pressure recovery was  $14 \pm 7\%$  (up to 29%) of the maximal pressure drop at the vena contracta, and the corresponding aortic valve areas derived from recovered pressures were  $15 \pm 8\%$  (up to 44%) higher than the vena contracta values, both measured invasively. These authors validated an equation that corrects the vena contracta area for pressure recovery, based on angiographic data.

**Noninvasive area correction.** In the current study, Garcia et al. (1), from the group of Dumesnil and Pibarot, confirm the effect of pressure recovery on area and use a relatively simple equation to correct the Doppler continuity valve area for this effect, documenting its validity comprehensively in vitro, in vivo (24,25), and in the patients studied by Schöbel and Karsch. They propose an “energy loss coefficient” that essentially modulates the Doppler EOA by factors that determine the net loss of pressure head, or energy, across the stenosis. Doppler EOA is multiplied by  $A_A/(A_A - EOA) = 1 + (EOA/[A_A - EOA])$ , where  $A_A$  is the area of the ascending aorta at the sinotubular junction. The form of this equation reflects the basic mechanisms causing and limiting pressure recovery: as post-stenotic high-velocity flow expands toward the ascending aortic walls, it encounters relatively stagnant fluid within the aortic sinuses. It is turbulence at this boundary that dissipates energy and limits pressure recovery. The smaller the aorta relative to the orifice, the less opportunity for such turbulence and the greater the pressure recovery, the magnitude of which directly relates to the ratio of EOA to the ascending aortic area (15–17,26).

**Effect on classification.** By looking at the preceding equation of Garcia, therefore, we see that in tight aortic stenosis, with EOA much smaller than the ascending aortic area, the “energy loss coefficient” is essentially equal to the Doppler EOA, as little pressure recovery can occur. However, a patient with an ascending aortic diameter of 2.6 cm and Doppler EOA of  $0.9 \text{ cm}^2$  would have a catheter-derived area (incorporating pressure recovery) of  $1.1 \text{ cm}^2$ , shifting the classification around a cutoff of  $1.0 \text{ cm}^2$ ; this occurred in

\*Editorials published in the *Journal of the American College of Cardiology* reflect the views of the authors and do not necessarily represent the views of JACC or the American College of Cardiology.

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3 (8%) of 37 patients in Garcia's study (36/37 with catheter areas  $<1.1 \text{ cm}^2$ ). This effect would be greater for a patient with the same aortic diameter, a Doppler EOA of  $1.2 \text{ cm}^2$ , and a recovered catheter area of  $1.6 \text{ cm}^2$ , shifting the classification around a cutoff of  $1.5 \text{ cm}^2$ . The need for correction occurs therefore primarily in patients with mild to moderate stenosis and smaller aortas (Doppler EOA  $>0.8 \text{ cm}^2$  with ascending aortic diameter  $<3.0 \text{ cm}$ ). In routine practice, Doppler EOAs  $>1.0$  to  $1.1 \text{ cm}^2$  have not, in general, been associated with important pressure gradients, in contrast to a recently proposed classification (5) in which catheter areas between  $1.0$  and  $1.5 \text{ cm}^2$  are considered moderately stenotic. Of note, in the study of Oh et al. (7), more than half of patients with catheter areas between  $1.0$  and  $1.5 \text{ cm}^2$  had Doppler areas between  $0.5$  and  $1.0 \text{ cm}^2$ .

**Caveats.** Variable aortic stenosis classifications have been proposed (5,27), with cutoff points at  $0.75 \text{ cm}^2$ , based on an inability to increase flow substantially despite large pressure gradients, and at  $1.0 \text{ cm}^2$ , based on a steep rise in the pressure gradient at typical outputs (4). The work load depends on both the area and output, and therefore on body size, with an energy loss index (based on the corrected area as described)  $\leq 0.55$  to  $0.60 \text{ cm}^2/\text{m}^2$  correlating best with adverse outcomes (21). In any classification, we must bear in mind the standard deviation of  $0.2 \text{ cm}^2$  in the Doppler-catheter area correlation (7), potentially reflecting variability in both measures based on the location of velocity and pressure measurement, variable aortic size and pressure recovery, and aortic insufficiency affecting the Gorlin equation.

**Other clinical implications.** The severity of aortic stenosis, therefore, depends not only on the anatomic orifice area and the inlet geometry (which determines flow contraction and thus vena contracta area [28]) but also on the outlet geometry—that is, the size of the ascending aorta. Progression of hemodynamic stenosis severity therefore might not depend on progressive valvular disease alone, but on progressive aortic dilation as well. As a result, a pathophysiologic cycle is generated similar to that in aortic regurgitation: progressive aortic dilation caused by aortic stenosis may, in itself, aggravate the hemodynamic severity of the lesion by increasing kinetic energy dissipation, thus minimizing pressure and effective orifice area recovery. It would be worthwhile to investigate to what extent the natural history of aortic stenosis and rate of effective orifice area decline are influenced by the rate of aortic dilation. Processes causing aortic dilation—for example, in patients with congenitally bicuspid valves—may independently influence the progression of effective stenosis severity. Therapeutically, it may be as important to prevent progressive aortic dilation in aortic stenosis as it is in aortic regurgitation, particularly in patients with hypertension and calcific stenosis.

The resolution of the ongoing controversy about concomitant aortic valve replacement in patients with mild to moderate aortic stenosis scheduled for coronary artery by-

pass graft surgery requires clarity about the true lesion severity in the mild-to-moderate range (29,30). This is exactly the range in which correction for ascending aortic size may make all the difference, because underestimation of the valve area by the continuity equation occurs predominantly in that range. Valve area correction may result in avoiding unnecessary valve replacement, balancing concerns regarding the risk of repeat operation.

**Conclusions.** A more complete understanding of the impact of stenosis, based on the resulting energy loss, can improve concordance between Doppler and catheter assessments and their use in clinical decision-making.

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