Hemodynamic Assessment of Aortic Stenosis
Are There Still Lessons to Learn?*
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Doppler echocardiography with estimation of pressure gradients and valve area has become the mainstay for assessing the hemodynamic severity of aortic stenosis (AS). The method has been validated extensively, showing its strength. Nevertheless, it has also been recognized that a number of sources of error must be considered to provide data solid enough for guiding appropriate patient management. Although well known, in particular an inappropriate recording angle and less frequently recording of the wrong velocity (i.e., proximal velocity or mitral regurgitation velocity), failure to account for an increased subvalvular velocity and selection of nonrepresentative velocity data in the presence of arrhythmias remain major reasons for miscalculations of Doppler-derived gradients in daily practice when high technical expertise is lacking (1). Over the years we have also learned that more complex fluid dynamic phenomena such as pressure recovery need to be considered in certain subsets of patients. Although aortic diameters <30 mm—the cut-off point at which pressure recovery may become clinically relevant—are present in a minority of patients, Doppler-derived gradients may be largely misleading in this subset of AS patients (2). Doppler gradients measure the conversion of pressure to kinetic energy induced by the stenosis. As soon as significant pressure recovery occurs, the head loss of energy (i.e., net pressure decrease measured after pressure recovery), which determines the left ventricular pressure required to maintain a given systemic pressure and therefore the actual hemodynamic burden, is significantly overestimated by the Doppler measurement.

Because pressure gradients are highly flow dependent, it has generally been accepted that reliable assessment of AS requires estimates of the aortic valve area (AVA), particularly in the presence of low flow rates. Again, catheterization is no longer considered the gold standard. Although the hemodynamic principle may be valid, difficulties in accurately estimating transvalvular flow rates and the question-
is obvious that an increase in GOA with flow is possible, the observation of flow-dependent variation in Doppler-derived EOA for rigid orifices (10,14) supports the idea that additional phenomena must exist. DeGroff et al. (12) suggested that viscous effects may cause a more parabolic flow profile in the vena contracta at low flow rates, resulting in underestimation of the actual EOA by the Doppler method. However, such phenomena can only occur at Reynolds numbers much lower than those present in the clinical setting, even at low cardiac output.

The study by Kadem et al. (15) in this issue of the Journal adds important new information to our understanding of this matter. In an elegant in-vitro study using particle image velocimetry, the investigators were able to show that there is good agreement between Doppler-derived EOA and the measurements obtained by this technique even at low flow rates. More importantly, they were able to show that changes in EOA observed with increasing flow are real and not caused by artifact. Furthermore, they were able to provide an explanation of why EOA may increase with flow even in a rigid orifice: they hypothesized that these flow-related changes in EOA are caused by the predominance of unsteady effects at low flow rates. Using an equation that takes this phenomenon into account, they were indeed able to predict changes in EOA observed in their study as well as in a study previously published by Voelker et al. (10). The results suggest that the predominance of the unsteady effects at low flow rates has significant impact on the flow configuration downstream from a stenotic lesion. At normal flow rates, the kinetic energy of the fluid crossing the obstruction is sufficient to break down the vortex structures generated downstream from the stenosis and thus enables the formation of a large and well-established flow jet. However, at low flow rates, the reduction in kinetic energy may predispose to the formation of vortices, which tend then to squeeze the flow jet and thus the vena contracta, resulting in a smaller EOA. The phenomenon is apparently less important in the presence of very small orifices, but may become clinically relevant in moderately severe AS. The fact that flow-dependent changes in EOA may occur in the absence of changes of the geometric orifice would have important clinical consequences. It can no longer be considered an ideal measure of AS severity to visualize the valve orifice and calculate the GOA even if this were possible with high precision, not only because such measurements concentrate on the peak valve area rather than the mean orifice area (the relation between these two may obviously vary depending on valve extensibility). If it is true that the EOA for a given GOA can vary more than 50% depending on the flow rate, then estimation of the GOA would indeed be of limited value for characterizing the hemodynamic burden imposed on the ventricle by a stenotic valve. Again, it must be taken into account that such changes may not reach clinical relevance in patients with definitely small or large orifices. However, in those with moderate disease and particularly at low flow rates, consideration of such phenomena may result in different judgment of stenosis severity and therefore imply changes in patient management.

In conclusion, Doppler echocardiography has become the gold standard for the assessment of AS. Despite a number of simplifying assumptions in the generally used way of generating pressure gradients and valve areas, the method can provide reliable measurements in the majority of patients as long as the echocardiographer is well trained and the study is carefully performed to avoid any technical reasons for measurement errors. However, in certain subsets of patients, more sophisticated approaches considering increased LVOT velocity, pressure recovery, or flow-dependent variation of EOA due to either valve extensibility, the predominance of unsteady effects at low flow rates, or both may be necessary to provide measurements precise enough to guide appropriate clinical management. This seems to be of particular importance for EOA in the range between 0.8 and 1.0 cm² and in patients with reduced cardiac output.

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