Pressure Recovery in Aortic Valve Stenosis

Warren K. Laskey, MD; William G. Kussmaul, MD

Background Pressure recovery is the variable increase in lateral pressure downstream from a stenotic orifice. The magnitude and clinical significance of pressure recovery in aortic valve stenosis are poorly defined.

Methods and Results We obtained high-fidelity pressure and velocity recordings in 11 patients with isolated significant aortic valve stenosis at the time of diagnostic cardiac catheterization. Systematic catheter pullback from the left ventricular cavity revealed a consistent although variable subvalvular gradient. Further pullback across and distal to the region of the stenosed aortic valve revealed a consistent and progressive increase in the ascending aortic pressure. This increase in lateral pressure occurred pari passu with a diminution in amplitude of the velocity pulse. The extent of pressure recovery was directly related to systemic blood flow and transvalvular flow but inversely related to the Gorlin-derived aortic valve area.

Conclusions These findings have potentially important implications for the hemodynamic evaluation of mild to moderately severe aortic valve stenosis. The extent of pressure recovery may be of additional utility in the assessment of aortic valve stenosis under varying physiological states.

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Key Words • aorta • valves • stenosis

Transvalvular pressure differences are the hemodynamic hallmark of valvular obstruction. The severity of obstruction in the clinical context is represented by a derived orifice area, with the latter a function of the transvalvular pressure difference and transvalvular flow.1 Hydrodynamic theory of flow through stenotic orifices indicates convergence of the flow field proximal to and within the stenosis, with subsequent flow acceleration.2-5 Considerable effort has been expended in the quantitation and evaluation of transvalvular pressure gradients in aortic valve stenosis in humans.6 More recently, theoretical predictions and empirical studies have indicated that a significant minor pressure loss occurs proximal to the valvular stenosis.7,8 These same model-based predictions indicate variable degrees of expansion of the flow field distal to the stenosis with subsequent flow deceleration and turbulence.4,9 Although these theoretical approaches and experimental observations9-11 have cogently suggested the existence of pressure recovery, no direct confirmation of pressure recovery has been reported in humans.

We analyzed systematic high-fidelity catheter pullbacks during diagnostic cardiac catheterization in patients with clinically significant aortic valve stenosis in order to (1) ascertain the presence of pressure recovery, (2) quantify the extent of pressure recovery, and (3) relate the extent of pressure recovery to traditional hemodynamic indices of stenosis severity.

Methods

Patient Population

Eleven patients (nine men, two women) with clinically significant aortic valve stenosis were referred for diagnostic cardiac catheterization and coronary angiography before planned aortic valve surgery. All patients were in sinus rhythm and had normal left ventricular systolic function assessed by contrast ventriculography and/or two-dimensional echocardiography. No subject had more than trace aortic regurgitation, again assessed by contrast aortography and/or Doppler echocardiography. Finally, no subject had significant obstructive coronary artery disease on angiography.

Study Protocol

All patients underwent cardiac catheterization via the brachial approach. A custom-designed multisensor catheter (Millar Instruments, Inc, Houston, Tex) consisting of a distal (tip) pressure sensor, a more proximal pressure sensor (5 cm from the tip), and an electromagnetic velocity probe located at the site of the proximal pressure sensor was advanced from the right brachial artery to the left ventricle under fluoroscopic control. The outputs from the pressure transducers were amplified and displayed in real time and recorded on FM magnetic tape. The electromagnetic velocity probe was energized by a 500-Hz square wave electromagnetic flowmeter (Carolina Medical Electronics, King, NC). The output of the velocity probe was amplified, filtered at the 100-Hz setting, and displayed in real time and recorded on tape.

The study protocol, performed before administration of radiocontrast, consisted of the following. The catheter was advanced to the deep left ventricular body, where both pressure sensors recorded left ventricular pressure. Both tracings were adjusted so that the left ventricular late diastolic contour was superimposed on a simultaneous recording of the pulmonary capillary wedge pressure. The proper gain setting for recording the velocity waveform was obtained and not adjusted thereafter. At this point, the catheter was slowly withdrawn, with strict attention paid to the following landmarks: (1) a zone in which a subvalvular gradient was noted, (2) a zone identified by the traversal of the aortic valve by the proximal sensor, (3) a zone in which continued withdrawal resulted in maintaining the distal sensor within the left ventricle and the proximal sensor within the ascending aorta, and (4) withdrawal of the distal (ventricular) sensor into the proximal ascending aorta. This sequence was performed twice in each patient, and on each occasion strict attention was paid to the acquisition of steady-state data, ie, absent ventricular ectopy.

After completion of the protocol the remainder of the diagnostic procedure was performed. No complications were
Table 1. Hemodynamic Profile of Patient Population

<table>
<thead>
<tr>
<th>Patient</th>
<th>LVSP (mm Hg)</th>
<th>Grad</th>
<th>AVA (cm²)</th>
<th>CO (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>47</td>
<td>0.35</td>
<td>2.3</td>
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<td>2</td>
<td>206</td>
<td>40</td>
<td>0.80</td>
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<td>4</td>
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<td>0.97</td>
<td>6.6</td>
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<tr>
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<td>97</td>
<td>0.43</td>
<td>5.3</td>
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<td>196</td>
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<td>5.3</td>
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<tr>
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<td>56</td>
<td>0.71</td>
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<td>5.8</td>
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<tr>
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<td>6.4</td>
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<tr>
<td>SD</td>
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<td>26</td>
<td>0.22</td>
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</tbody>
</table>

LVSP indicates peak left ventricular systolic pressure (mm Hg); Grad, mean transvalvular gradient using minimum recorded aortic pressure (mm Hg); AVA, Gorlin-derived valve area (cm²); and CO, thermodilution cardiac output (L/min).

encountered as a result of the protocol. All subjects gave written consent in accordance with guidelines established by the University of Pennsylvania Committee on Studies Involving Human Beings.

Data Analysis

Results are expressed as mean±SD. Least squares and multiple linear regression were used to analyze the relation between selected variables.

Results

Table 1 describes the hemodynamic profile of the study population. The Gorlin-derived aortic valve area was obtained from recordings of the maximum transvalvular gradient, ie, the location of the proximal (aortic) pressure sensor yielding the lowest aortic systolic pressure during catheter withdrawal (compare with below). Fig 1 demonstrates the presence of pressure recovery during catheter pullback in a patient with significant aortic valve stenosis. In Fig 1A, both pressure sensors are located in the deep left ventricular cavity and the velocity waveform is of small amplitude. Withdrawal of the catheter into zone 1 (compare with “Methods”) depicts the development of a subvalvular pressure gradient coincident with an increase in the amplitude of the velocity waveform. In Fig 1B, further pullback results in the passage of the proximal sensor across the aortic valve into the proximal ascending aorta (zone 2). Note the further increase in amplitude of the velocity waveform at this location. In Fig 1C, further catheter withdrawal with the distal sensor remaining in the left ventricular cavity reveals a progressive increase in the ascending aortic systolic pressure with destabilization and diminution of the (now aortic) velocity waveform (zone 3). The difference in peak systolic pressure between zones 2 and 3 is the extent of pressure recovery. Finally, withdrawal of the distal pressure sensor into the ascending aorta reveals matched aortic pressure waveforms and a chaotic, destabilized aortic velocity trace (Fig 1D).

Table 2 summarizes the extent of pressure recovery and the effect of pressure recovery on the transvalvular gradient. The results represent the average of the two catheter pullback sequences. The correlation between the two pullback sequences was excellent (r= .99, SEE=1.3 mm Hg). It should be noted that in each patient, the zone in which the gradient was highest (zone 2) was characterized by a high-amplitude, organized velocity waveform and that further catheter withdrawal consistently resulted in the recording of a higher aortic systolic pressure and a lower-amplitude, disorganized waveform (zone 3). Fig 2 illustrates the effect of pressure recovery on the assessment of the mean systolic transvalvular gradient. It can be seen that the mean gradient in zone 3 is consistently and significantly lower than the mean gradient in zone 2. The implications of this disparate assessment of the transvalvular gradient are demonstrated in Fig 3. The Gorlin-derived valve area is consistently and significantly higher if the recovered aortic pressure is used in the transvalvular gradient. Furthermore, this disparity appears greater when the aortic valve area exceeds 0.7 cm².

There was no significant relation between pressure recovery and the maximum transvalvular gradient (r²=.125, P=NS). Fig 4 illustrates the dependence of pressure recovery on mean flow (cardiac output) and stroke (transvalvular) flow. Although statistically significant, these relations are not precise and therefore indicate the importance of additional unquantified variables. One such variable is the aortic valve area itself. Fig 5 indicates that pressure recovery is inversely related to the severity of the valvular stenosis, although the latter can only explain 70% of the variation in the pressure recovery. Multiple linear regression indicated that the combination of stroke flow, aortic valve area, and aortic valve gradient accounted for 80% of the variability in pressure recovery.

Discussion

In this report, we provide direct evidence for the presence of pressure recovery in humans with aortic valve stenosis. The observations are in accord with theoretical predictions and validate models of flow through stenotic orifices under physiological conditions. The extent of pressure recovery, in absolute terms, varies directly with the transvalvular flow and indirectly with the severity of the stenosis.

Hydrodynamic theory and experimental observations indicate that the flow field tapers proximal to a stenotic orifice, further converges within the stenosis itself, and reaches its smallest dimension just beyond the physical obstruction (vena contracta). We reproducibly demonstrated in each subject the presence of a subvalvular gradient upon catheter withdrawal from the deep left ventricular cavity. Such observations have been previously reported⁷,⁸ and verify the theoretical predictions of an intracavitary tapering flow field in subjects with aortic valve stenosis.¹² Simultaneous recordings of lateral pressure and fluid velocity immediately upon entry into the proximal ascending aorta reveal a zone of minimum aortic pressure beyond which the pressure increases, or recovers, along with a region of maximum velocity. That this latter region corresponds to the vena contracta is supported by the demonstration of the simultaneous occurrence of minimum aortic pressure...
and maximum velocity in each subject studied. It should be emphasized that the vena contracta was consistently demonstrated distal to the aortic valve, as it was only with further catheter withdrawal, after the immediate transition from left ventricular outflow tract to proximal ascending aorta, that the vena contracta was identified. Distal to the vena contracta, the zone of pressure recovery was consistently demonstrated by the simultaneous increase in (lateral) aortic pressure and decrease in amplitude of the velocity pulse.

The true extent of pressure recovery may have been underestimated by the noncalibrated withdrawal sequence. We think this unlikely because (1) pressure recovery reached its maximum value whenever the velocity waveform was no longer recognizable (indicating either transducer location within turbulent eddies or in immediate proximity to the inner curve of the aortic wall), and (2) withdrawal of both sensors into the proximal ascending aorta and beyond failed to reveal any further recovery of pressure. We could not, however, be certain of the linear extent of the zone of pressure recovery because the withdrawal sequence was not calibrated. An upper limit of 5 cm is reasonable given the distance between distal and proximal sensors and the failure to record further increments in proximal transducer pressure when both sensors were located within the aorta.

The clinical utility of the quantitation of the extent of pressure recovery in aortic valve stenosis is as yet untested. A number of investigators have invoked pressure recovery as an explanation for the discrepancy between Doppler-derived transvalvular gradients and

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**Fig 1.** Tracings. A shows the two laterally mounted pressure sensors located within the deep left ventricular cavity, revealing superimposed intracavitary pressures. Middle tracing is the intracavitary velocity signal. Note the development of a small intracavitary pressure gradient along with an increase in the amplitude of the velocity signal as the catheter is withdrawn. B shows continued slow catheter withdrawal, which results in the passage of the proximal transducer across the left ventricular outflow tract (left side) and into the region of the stenotic aortic valve. Note the progressive increase in amplitude of the velocity signal along with the decrease in peak pressure recorded from the proximal transducer. C shows further catheter withdrawal, which results in the recording of a distinctive ascending aortic pressure pulse. Continued slow withdrawal reveals a progressive decrease in amplitude of the velocity signal along with an increase in the systolic aortic pressure. D shows withdrawal of both pressure sensors into the ascending aorta; this reveals superimposed aortic pressures and a destabilized velocity waveform.
Table 2. Extent of Pressure Recovery and Effect on Measured Gradient

<table>
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<tr>
<th>Patient</th>
<th>AoP_{min}</th>
<th>AoP_{max}</th>
<th>P_{rec}</th>
<th>Grad_{max}</th>
<th>Grad_{min}</th>
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<td>4</td>
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</table>

AoP_{min} indicates smallest valve of ascending aortic systolic pressure (mm Hg); AoP_{max}, highest value of ascending aortic systolic pressure (mm Hg); P_{rec}, extent of pressure recovery (mm Hg); Grad_{max}, difference between peak left ventricular systolic pressure and AoP_{min} (mm Hg); and Grad_{min}, difference between peak left ventricular systolic pressure and AoP_{max} (mm Hg).

invasively determined gradients at catheterization.\textsuperscript{9,10,13} It must be recalled, however, that these techniques, while assessing the severity of the valvular stenosis, do so in different ways. Doppler-derived measures rely on the identification of the maximum velocity in the region of the vena contracta and thus reflect the physiological importance of the stenosis, whereas the Gorlin-derived aortic valve area yields an estimate of the anatomic severity of the stenosis. Furthermore, the implication of pressure recovery for accurate gradient and, therefore, valve area assessment, is potentially important. Although the magnitude of pressure recovery is a small fraction of the peak (maximal) gradient, Gorlin-derived valve areas are consistently underestimated in the presence of pressure recovery. These considerations are particularly important in the setting of moderate severity, eg, >0.7 cm\(^2\), when clinical decision making often rests on these calculations.

We extend the clinical implications of pressure recovery with the empiric correlations between the extent of pressure recovery and transvalvular flow and the Gorlin-derived aortic valve area. Unfortunately, the small study population precludes analysis of the low-flow,
small-valve-area subgroup of patients. However, the potential for an additional means of assessment of valve area in this difficult clinical situation is clear. Under physiological conditions at the other end of the hemodynamic spectrum—high flow—the importance and relevance of pressure recovery is also demonstrated. High flows across a relatively unchanged stenotic orifice, such as might occur with exercise, result in higher peak transvalvular gradients but also a greater extent of pressure recovery (Fig 6). The maintenance of an effective input pressure to the systemic arterial circulation is clear. Finally, pharmacologically mediated alterations in transvalvular flow would be expected from these data to influence the extent of pressure recovery. Although none of our patients were receiving inotropic medications or afterload-reducing agents, the expected effect of these medications can be surmised from Figs 4 and 5. The extent of pressure recovery for any given valve area is greater at higher transvalvular flows. Similarly, at equivalent levels of transvalvular flow, pressure recovery is less when the stenosis is more severe.

**Study Limitations**

Several limitations in this study merit discussion. We did not routinely perform aortic root angiography, which would have allowed for quantitative assessment of aortic dimension. Theoretical predictions hold that not only the dimension of the stenotic orifice (or more properly, the vena contracta) but also the dimension of the aorta in the zone of pressure recovery contribute importantly to the extent of pressure recovery. The absence of these data may explain the variance in the correlations between pressure recovery and transvalvular flow and valve area. In this regard, ascending aortic distensibility as well as geometry may impact importantly on the extent of pressure recovery. Age-related changes in aortic compliance may very likely contribute to this process. Although there was no significant relation between age and the extent of pressure recovery in our small series, the narrow range of patient age (48 to 69 years) precludes drawing any conclusions. Thus, age-related influences on pressure recovery remain a distinct possibility. The lack of quantitative velocity data precluded us from validating a model of pressure recovery in aortic valve stenosis. At the outset, we were unsure of the quality and stability of the velocity signal with the velocity transducer within and distal to the vena contracta. These concerns were formidable, given the variable degree of destabilization and diminution of the velocity signal in this region. Thus, the qualitative observations are emphasized. Last, it must be acknowledged that these considerations are overlooked in routine clinical practice wherein transvalvular gradients are assessed with fluid-filled catheter systems and multiple side-hole or end-hole catheters. It is unlikely that pressure recovery would be detected under such circumstances. Nevertheless, clinicians must be aware of the potential for gradient underestimation using these latter techniques. Given the importance of accurate assessment of the gradient in the setting of moderately severe aortic valvular stenosis, the operator should
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strive for placement of the catheter measuring ascending aortic pressure at the most proximal location.

Summary

In this article, we report the presence and extent of pressure recovery in aortic valve stenosis in humans. Although quantitatively small in comparison to the transvalvular gradient, the recovery of pressure is a function of stenosis severity and transvalvular flow. These observations have important clinical implications for the assessment of the severity of valvular stenoses under varying physiological conditions encountered during clinical evaluation.

Acknowledgments

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References

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