Fluid Dynamics of Aortic Stenosis: Subvalvular Gradients Without Subvalvular Obstruction

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SUMMARY Analysis of a tapering, pulsatile flow field predicts that substantial subvalvular pressure gradients exist in patients with valvular aortic stenosis (AS) without invoking a second anatomic site of obstruction. Using a catheter with two laterally mounted micromanometers, we examined the left ventricle in 11 patients with AS, mean age 64 ± 11 years (± sd); the mean valve area was 1.0 ± 0.3 cm². Simultaneous measurements were made in (1) the left ventricular (LV) chamber and the LV outflow tract (LVOT) and (2) the LVOT and ascending aorta (AO). No patient had anatomic evidence of a subvalvular obstruction, but large subvalvular gradients were present in all. The average peak LV-LVOT and LV-AO gradients were 41 ± 17 mm Hg and 58 ± 23 mm Hg, respectively. Flow velocity was electromagnetically derived in two patients. The LV-LVOT gradient was associated with an increased flow velocity in the LVOT. This study suggests that large subvalvular gradients are present in AS and help overcome blood's inertia to convective and local accelerations in the tapering subvalvular flow field.

SUBSTANTIAL intraventricular pressure differences and gradients* are traditionally considered to reflect an organic "outflow obstruction" in the region across which they are measured.1-9 It is commonly postulated that organic obstructions can be fixed or dynamic, and range from the collar type to the hypertrophic cardiomyopathy variants of subvalvular stenosis.10-13 Aortic valvular stenosis can coexist with various types of subvalvular obstruction.2-7, 14-19 These associated lesions cannot always be demonstrated angiographically, even by left ventriculography in several views. In such cases, obstructive lesions are best diagnosed from pressure gradients detected by catheter pullback from the ventricle to the proximal aorta.2, 3, 10, 12, 19 Persistence or recurrence of intraventricular gradients shown by repeated or serial postoperative catheterization signals inadequate resection or regrowth of subvalvular obstructive lesions and possibly the need for further surgical resection.6, 20-25

Clinical investigation of normal ejection dynamics has shown that dynamic factors associated with flow contribute to physiologic transvalvular pressure gradients.26 Studies of flow dynamics in hypertrophic cardiomyopathy have cast strong doubt on the premise that large intraventricular pressure gradients are always a consequence of an anatomic obstruction of the outflow tract region.27 Improved instrumentation for measuring pressure28-33 has led to the frequent observation of subvalvular gradients in patients with valvular aortic stenosis without subvalvular pathology in our institution. Nonetheless, such gradients are traditionally taken as evidence of subvalvular obstruction and are relied upon for the identification, the operative management and the follow-up of isolated or multiple obstructive lesions.6, 20-25, 34 Therefore, we studied intraventricular hydrodynamic pressure gradients and their relationship to transvalvular decreases in pressure in patients with isolated aortic valvular stenosis.

Methods

Patients

Eleven adult patients, 10 males and one female, mean age 64 years (range 23-77 years), constituted our study group. All had moderate-to-severe aortic valvular stenosis, minimal or mild aortic regurgitation and varying degrees of coronary artery disease. Before catheterization, each patient underwent an evaluation that included a complete history and physical examination, chest x-rays, ECGs, and M-mode and two-dimensional echocardiography. Cardiac catheterization and angiography were performed on all patients as described below. The study protocol was approved by the Clinical Investigation and Human Use Committees at Brooke Army Medical Center. All patients gave informed consent.

The diagnosis of valvular aortic stenosis without coexistent subvalvular obstruction was based on the clinical and laboratory evaluation. No patient had echocardiographic or angiographic evidence of asymmetric septal hypertrophy, systolic anterior motion of the mitral valve or fixed subvalvular obstruction. A summary of patient characteristics and basic hemodynamic and angiographic data is presented in table 1.

Catheterization

All patients underwent cardiac catheterization in a fasting, postabsorptive state and were unsedated or lightly premedicated with oral diazepam, 10 mg, administered 30 minutes before the procedure. All catheterizations were performed through a right brachial arteriotomy; a left-heart catheter with two laterally mounted, solid-state micromanometers (Mikro-Tip, Millar Instruments), one at the catheter tip and the

*The term gradients in this paper is used in the conventional hemodynamic sense, but in fluid dynamic terms represents a driving pressure difference evaluated across a finite distance in the direction of flow.
second 5 cm from the first, was used. In two patients, the catheter also contained an electromagnetic flow-velocity probe (Millar Instruments) at the level of the proximal micromanometer. The solid-state pressure sensors and the flow-velocity probe have been described in detail elsewhere.26-32 Simultaneous right-heart catheterization was performed on all patients through a median basilic or brachial comitans venotomy with a #7F balloon-tipped thermal dilution catheter (Swan-Ganz, Edwards Laboratories) or a Courand Positrol catheter (USCI), which was manipulated through the right-heart chambers into the pulmonary artery and the pulmonary capillary wedge positions.

After hemodynamic data were obtained, the left-heart catheter was withdrawn and replaced with an NIH (USCI) or a Lehman (USCI) angiography catheter. Biplane left ventriculography was then performed at 60 frames/sec while 30-50 ml of USP diatrizoate meglumine and diatrizoate sodium (Renografin-76) were injected with the patient in the 30° right anterior oblique (RAO) and 60° left anterior oblique (LAO) projections. Next, biplane aortography was performed by injecting 50 ml of Renografin-76 and filming at 60 frames/sec. All patients also underwent coronary arteriography by the Sones technique.

### Hemodynamic Protocol

All patients were studied at rest in the supine position. Steady-state conditions were inferred from a stable heart rate and stable sequential pulmonary artery hemoglobin oxygen saturations. Cardiac outputs were measured by the Fick method (duplicate determinations) or by thermodilution measurements. All right- and left-heart pressures were measured during the same steady-state conditions. The left-heart multisensor catheter was manipulated through the aortic valve and advanced retrogradely into the left ventricular cavity so that both micromanometers were located within the chamber. The catheter was then withdrawn slowly:

The proximal sensor traversed the left ventricular outflow tract, the aortic valve orifice and the root of the ascending aorta, but the distal sensor stayed in the main chamber throughout the pullback. Simultaneous pressure recordings were obtained (fig. 1) with both the tip sensor and the proximal sensor in the main left ventricular chamber; the tip sensor in the main chamber and the proximal sensor in the outflow tract just below the aortic valve; and the tip sensor in the main chamber and the proximal sensor in the ascending aorta.

### Data Processing and Analysis

Left ventricular and aortic pressures, an electrocardiographic signal and, from two patients, the flow-velocity signal were recorded on a Honeywell 5600 analog tape recorder. The data were later replayed from the FM tape and displayed on an 1858 fiberoptic strip-chart recorder at paper speeds of 100 and 50 mm/sec. Peak-to-peak systolic pressure gradients in the left ventricle and across the aortic valve were measured during stable rhythm over at least 10 cardiac cycles during normal respiration. Aortic valve areas were measured as described by Gorlin and Gorlin.35

### Results

#### Hemodynamic Measurements

A typical uninterrupted withdrawal sequence and the simultaneous ECG recording are displayed in figure 2. In panel A, the first three cardiac cycles illustrate simultaneous pressure recordings with both pressure sensors in the left ventricular chamber. As the catheter is slowly pulled back, a prominent subvalvular gradient becomes manifest. Slight differences in contour among these three pulses reflect, in part, minor positional changes of the downstream sensor. In panel B, a continuation of the withdrawal maneuver, the proximal sensor is in the outflow tract during the first three cycles. The subvalvular pressure wave form

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### Table 1. Patient Characteristics and Basic Hemodynamic and Angiographic Data

<table>
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<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>BSA (m²)</th>
<th>HR (beats/min)</th>
<th>CO (l/min)</th>
<th>LVSP (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>AoSP (mm Hg)</th>
<th>AoDP (mm Hg)</th>
<th>AVA (cm²)</th>
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<td>± 9</td>
<td>± 1.6</td>
<td>± 20</td>
<td>± 20</td>
<td>± 21</td>
<td>± 12</td>
<td>± 0.3</td>
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</table>

Abbreviations: BSA = body surface area; HR = heart rate; CO = cardiac output; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; AVA = aortic valve area; EDV = end-diastolic volume; ESV = end-systolic volume; EF = ejection fraction; AI = aortic insufficiency; AoSP = aortic systolic pressure; AoDP = aortic diastolic pressure; CAD = coronary artery disease.
is rounded, and pressure is maximal near mid-systole. The downstream sensor traverses the aortic orifice during diastole between the third and fourth beats, and a clear transition from left ventricular to aortic diastolic pressure patterns is shown. At this site, the downstream sensor records a systolic wave form with a delayed upstroke and a late peak pressure, in contrast to the pressure contour in the subvalvular region. A further decrease in the peak pressure amplitude is also present. The upstream sensor still records deep left ventricular pressures, as shown by the absence of significant amplitude or contour changes between the first and the second trio of cycles. In every patient, the sequential pullback patterns were qualitatively similar.

Figure 3 shows the pressure contours from the three regions of interest. In panel A, the left-heart catheter was placed so that both micromanometers were in the main left ventricular chamber. A small, flow-associated gradient is evident between the upstream and downstream sensor sites. Panel B shows the pressures recorded when the downstream micromanometer was in the immediate subvalvular area and the upstream sensor remained in the main chamber; a large subvalvular ejection gradient is demonstrated. The matched diastolic pressures attest to the intraventricular placement of both pressure sensors. The contour of the subvalvular pressure gradient is rounded and symmetric. The peak-to-peak subvalvular gradient in this example is 50 mm Hg. In panel C, the withdrawal sequence is complete; the downstream sensor has traversed the aortic valve orifice and is in the ascending aorta, while the tip sensor remains in the main chamber. A slow-rising, highly asymmetric aortic stenosis pressure wave form is recorded in systole by the downstream sensor. The peak-to-peak transvalvular gradient was 65 mm Hg, 30% greater than the corresponding subvalvular gradient.

The peak-to-peak transvalvular and subvalvular gradients for each patient are shown in figure 4, which graphically displays the high correlation between the magnitudes of these gradients in any given patient. All patients had large subvalvular pressure gradients (average 41 ± 17 mm Hg); the transvalvular gradients averaged 58 ± 23 mm Hg.
In two patients, the left-heart catheter included an electromagnetic flow velocity probe at the level of the downstream micromanometer that allowed simultaneous flow-velocity recordings. A withdrawal sequence with this additional signal is shown in figure 5, with ECG, flow velocity and pressure tracings displayed from top to bottom at a slow strip-chart recorder speed. As the flow probe enters the tapering subvalvular region, velocity increases and a prominent subvalvular gradient appears. Once the aortic orifice is crossed, the velocity sensor no longer demonstrates the familiar ejection velocity wave form. The velocity patterns in the other subject were similar.

**Angiographic Findings and Measurements**

Proximal aortography revealed absent, trivial or minimal aortic regurgitation (0 to 2+) (table 1). Conventional criteria were used to assess mitral regurgitation. No patient in this study had greater than trivial mitral regurgitation. There was no evidence of fixed or dynamic subvalvular pathology by biplane angiography or M-mode and two-dimensional echocardiography. Echocardiography showed that all patients had concentric left ventricular hypertrophy. A wide range of coronary artery disease was present.

**Discussion**

We examined ejection fluid dynamics in the human left ventricle to characterize intraventricular pressure gradients in the presence of isolated aortic valvular stenosis. Large subvalvular gradients were present in all patients in this study.

Analysis of the fluid dynamics of a pulsatile, tapering flow field is necessary to explain these observations. A theoretical analysis has been presented elsewhere. However, our theoretical results show that in the ventricular ejection field, inertial forces predominate substantially over viscous forces. Blood's inertia is associated with its mass per unit volume, or mass density, and is totally independent of its viscous behavior. According to Newton's Second Law of Motion, the total acceleration of a unit volume of blood in the tapering subvalvular flow field is equal to the total force exerted on this volume divided by its mass. It follows that instantaneous values of the total measured subvalvular pressure gradient (that is, force per unit volume of moving blood) help overcome blood's inertia to both local and convective acceleration components:

\[
\frac{\partial v}{\partial t} + v \frac{\partial v}{\partial s} = -\frac{1}{\rho} \frac{\partial p}{\partial s}.
\]

Here, \(v\), \(p\) and \(t\) denote velocity, pressure and time, respectively; \(s\) denotes distance along a streamline, and \(\rho\) the mass density of blood. The right-hand side of the equation is the applied force per unit mass in the streamwise direction; the first term on the left-hand side is the resultant local acceleration and the second the convective acceleration component.

The terms will be clarified by considering a unit volume of blood located at the upstream site of a taper-
FIGURE 6. In a tapering flow field, streamwise velocity ($v$) is a function of position. $s =$ streamwise distance.

ing flow field (fig. 6). We must also take into account that this field changes with time. The first (local) acceleration component is due to the local rate of change of the velocity with respect to time. The second (convective) component is due to the streamwise motion (i.e., motion along the $s$ direction) of the blood volume, whose position is a function of time irrespective of whether the flow is steady or pulsatile. Because the flow field tapers, the velocity is a function of position, in accordance with conservation of mass requirements. Considering that velocity in the tapering field is a function of position and the position of the moving blood is a function of time, the latter must undergo an acceleration as it moves in the streamwise direction. This is the convective acceleration, which gives rise to the Venturi effect. The two acceleration components, when combined, give the total acceleration in the pulsatile, tapering velocity field proximal to the stenosed orifice. Our hemodynamic measurements demonstrate that the driving intraventricular, particularly subvalvular, pressure gradients are very pronounced in ventricles with stenotic outflow valves.

The undisturbed velocity wave forms we recorded in the subvalvular region as the large intraventricular pressure gradients became manifest clearly show that turbulence is not necessary to cause major pressure drops and contour changes in systolic pressure wave forms, despite the suggestions of other investigators. In the rapidly tapering flow field upstream of a stenosed orifice, the nonturbulent acceleration mechanisms underlie the striking pressure gradients in the streamwise direction. Turbulent mechanisms are set in motion downstream from the stenotic orifice and preclude complete recovery of static pressure.

The observation of residual intraventricular and transvalvular gradients after aortic valve replacement can also be explained by applying the same fluid dynamic considerations. It is unlikely that aortic valve replacement could establish normal outflow orifice areas or subvalvular flow field tapering. It would certainly not remove poststenotic aortic root dilatation, which is conduclive to flow disturbance and turbulent pressure losses. Thus, residual subvalvular and transvalvular gradients may be present without indicating a need for further surgical intervention.

Since ejection velocity patterns are heavily influenced by loading conditions and wall stresses, they will be changed after operation. In view of the inverse relationship between muscle shortening velocity and afterload, wall collapse during ejection will be faster. Faster patterns of wall collapse against the reduced afterload should strongly enhance ejection velocity and its time rate of change after valve replacement. These augmented velocities and accelerations in turn enhance measured pressure gradients from the postoperative levels expected if preoperative contraction velocities still prevailed. Accordingly, "residual" subvalvular and transvalvular gradients may be found in postoperative studies without necessarily implying the existence of a subvalvular obstruction.

In conclusion, this study has demonstrated that prominent subvalvular gradients exist in the tapering subvalvular flow field of left ventricles with aortic stenosis. These gradients are a result of blood’s inertia to convective and local accelerations. Viscous losses become important beyond a stenotic valve, but make only a nominal contribution to large gradients recorded within the ejection chamber. Only careful consideration of fluid dynamics including flow field geometry and ejection velocity patterns, will permit correct delineation, surgical management and follow-up of obstructive lesions.

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References

5. Harrison EE, Sbar SS, Martin H, Pupello DF: Coexisting right and left hypertrophic subaortic stenosis and fixed left ventricular outflow obstruction due to aortic valve stenosis. Am J Cardiol 40: 133, 1977
10. Maron BJ, Redwood DR, Roberts WC, Henry WL, Morrow AG, Epstein SE: Tunnel subaortic stenosis: left ventricular outflow tract obstruction produced by fibromuscular tubular narrowing. Circula-
tion 54: 404, 1976
18. Morton BR, Castellanos JF,出发的段落

42. Sabbah GN, Blick EF, Anbe DT, Stein PD: Effect of turbulent blood flow on systolic pressure contour in the ventricles and great vessels: significance related to anatomic and bipherferous pulses. Am J Cardiol 45: 1139, 1980
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