Mechanism of Elevated Innominate Artery
Pressures in Supravalvular Aortic Stenosis

By ROBERT E. GOLDSTEIN, M.D., AND STEPHEN E. EPSTEIN, M.D.

SUMMARY
Patients with supravalvular aortic stenosis frequently have higher phasic and mean blood pressures in the innominate artery than in the aorta distal to the stenosis, a finding never fully explained. Theoretically, this phenomenon could result from the impact of a jet upon blood within the innominate artery. Slowing of the jet would convert kinetic into potential energy, thus increasing pressure within the innominate artery. The aorta distal to the innominate artery would receive blood whose kinetic energy had been dissipated and would consequently remain at the same pressure as the aorta proximal to the origin of the innominate artery. This hypothesis was tested in a model simulating supravalvular aortic stenosis. Cinematography showed a jet directed into the innominate artery. Only after this jet was dispersed, did fluid turn backward to flow into the distal portion of the aorta. Observed pressure rises in the innominate artery corresponded closely with values predicted from the estimated kinetic energy of the jet. Dispersion of the jet by replacement of the stenotic orifice with an equally obstructive screen or proximal displacement of the stenosis (simulating valvular aortic stenosis) abolished the pressure difference. The results obtained were thus entirely consistent with the mechanism proposed to explain the elevated innominate artery pressures seen in supravalvular aortic stenosis.

Additional Indexing Words:
Kinetic energy    Jet    Potential energy    Blood pressure

PATIENTS with supravalvular aortic stenosis frequently have higher systolic and mean blood pressures in the innominate artery and its radicals than in the aorta beyond the stenosis.1-5 Similarly, supravalvular aortic stenosis produced acutely in dogs resulted in elevations in brachiocephalic artery pressure above pressures recorded immediately distal to the stenosis.6 Despite its familiarity, the phenomenon of a higher pressure in the innominate artery than in the aorta (and the seemingly paradoxical implication that blood flows into the innominate artery against a pressure gradient) has never been fully explained.

The proximity of the supravalvular stenosis to the origin of the innominate artery suggested that this phenomenon may be the result of the impact of a jet issuing from the stenosis directly upon the column of blood within the innominate artery. The rapidly moving jet would thereby be slowed and its kinetic energy converted into potential (pressure) energy, a possibility noted previously.2,3 Consequently, blood pressure in the innominate artery would be increased above aortic blood pressure. The aorta distal to the innominate origin, however, only receives blood whose kinetic energy has been dissipated in whorls and eddies. Thus, pressure in the aorta distal to the innominate origin would not differ from aortic pressures proximal to the innominate origin.

To test these hypotheses experimentally, we constructed a model of the aorta and innominate artery based on the angiographically demonstrated anatomy found in supravalvular aortic stenosis. Measurements made with this
Diagrammatic representation of the model used to simulate anatomic relationships in supravalvular aortic stenosis. Arrows indicate direction of fluid flow.

Figure 1

The model consistently supported the hypothesized explanation for the elevated innominate artery pressures.

Methods

A glass simulation of the aortic arch and innominate artery was blown to conform exactly to the roentgenographic appearance of these structures in a patient with documented supravalvular aortic stenosis. No attempt was made to compensate for the minor amount of magnification associated with cineangiography (fig. 1). A neoprene stopper with an 11-mm hole provided a critical orifice. Fluid was pumped from a reservoir via Tygon tubing, through the glass simulation and past resistances (provided by clamps), then returned to the reservoir. The ratio of aortic arch to innominate flow was maintained between 3.4 and 3.7, a value approximating physiologic flow distribution.6 Lateral pressures were measured by a Statham 23-Db gauge connected to needles in the aorta proximal and distal to the stenosis and in the innominate artery. Rotation of the needles or displacement of the needle tips across the vessel diameters during steady perfusion produced no measurable alteration in pressure. In addition to the fixed sites of pressure measurement, shown in figure 1, pressures were also measured through the side hole of an 8.5 F double-lumen catheter during withdrawal from aortic arch or innominate artery, across the stenosis, and into proximal aorta. Flows were measured directly by time collections. In addition, high speed cinematography of the model (at 1,000 frames per second) was performed after injection of dye to visualize the pattern of fluid flow.

Results

When water was pumped through the model at a steady (nonpulsatile) rate, pressure was consistently higher in the innominate artery than in the aorta distal to the stenosis (fig. 2). This pressure difference increased markedly as total flow through the stenosis was progressively increased. Rates of flow were chosen to simulate hemodynamic conditions obtaining during systole. Under these conditions, the innominate artery pressure increment, as well as the gradient across the

Figure 2

Plot of pressures within various parts of the model as a function of total flow. Progressive increases in flow (necessitating higher pressures proximal to the stenosis) lead to corresponding increases in innominate artery pressures, but no alteration in aortic pressure distal to the stenosis.
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Figure 3

Record of pressures within the model during pulsatile perfusion with blood. Higher "systolic" pressures are evident in the innominate artery than in the distal aorta.

stenosis, corresponded closely to measurements observed during systole in patients with supravalvular aortic stenosis. A similar pressure increment was observed in the innominate artery when heparinized blood was pumped through the model by a pulsatile pump (fig. 3). Catheterization of the model during steady perfusion with water (fig. 4) demonstrated that lateral pressure was essentially the same throughout the entire aorta distal to the stenosis. But when a catheter was moved distally from a position in the aorta just beyond the stenosis into the innominate artery, pressure began to rise at the innominate origin and continued to rise progressively within the first 5 cm of this vessel. Pressure changes distal to the stenosis were thus confined entirely to the innominate artery, where a pressure rise was seen to develop within a short segment.

To evaluate the possible influence of a jet issuing from the site of stenosis, the stenotic orifice was replaced by a fine mesh screen designed to furnish a similar degree of obstruction, but without creating a jet. This substitution virtually eliminated the increase in innominate artery pressure despite a pressure gradient of 102 mm Hg across the screen (fig. 5 middle). Obstruction per se was thus insufficient to cause an increase in innominate pressures even when the obstruction was situated at the same site as the supravalvular stenosis. Progressive proximal displacement of the stenotic orifice, so that the geometric relationships eventually approximated those present in valvular aortic stenosis (fig. 5 bottom), resulted first in a diminution and then elimination of the increase in innominate pressure.

High-speed cinematography and bolus injections of dye permitted a direct evaluation of fluid motion (fig. 6). After crossing the stenosis, a jet of dye was visualized which flowed rapidly and directly into the most proximal portions of the innominate artery; fluid reached the aorta distal to the innominate origin only after dispersion of the jet within the proximal innominate artery.

Discussion

Using a mechanical model perfused with water or blood under nonpulsatile or pulsatile conditions, we were able to reproduce the increase in innominate artery pressure above aortic pressure seen frequently in patients with supravalvular aortic stenosis. The ability to reproduce this phenomenon under these
conditions suggests that the increase results from anatomic relationships rather than from the pulsatile nature of blood flow or specific properties of blood or vessel walls. High-speed cinematography demonstrated that the proximity and orientation of the stenotic orifice relative to the innominate origin caused a jet to be directed straight into the innominate artery. The fact that dispersion or elimination of the jet abolished the pressure increase in the innominate artery indicates that the phenomenon is a result of the jet.

When the stenosis was displaced proximally and thus simulated valvular aortic stenosis, the pressure increment in the innominate artery disappeared, presumably due to a dispersion of the jet proximal to the innominate origin. Dispersion of the jet probably explains why elevated pressures in the innominate system are only rarely observed in patients with valvular aortic stenosis.

An analysis of the energetics of fluid flow in this system provides a satisfactory explanation for the observed results. The rapidly moving

Figure 4

Pressure record during withdrawal of a catheter from innominate artery (upper panel) and from aortic arch (lower panel) across the stenosis into proximal aorta. Pressures simultaneously obtained from stationary needles are shown at the right of each panel. Catheter was positioned via an opening in the proximal aorta. Small vertical artifacts occurred when catheter was withdrawn about 1 cm. As the catheter was withdrawn from the innominate artery, lateral pressure began to fall at a point approximately 4 cm from the innominate origin and continued to fall progressively up to the innominate origin. At this point pressure was virtually the same as that within the distal aorta. No further changes in pressure were encountered within the aorta until the catheter crossed the stenosis and entered the proximal aorta, at which point pressure abruptly rose. No pressure changes were observed in the aorta distal to the stenosis.
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**Figure 5**
Comparison of pressures in simulated supravalvular aortic stenosis produced by a single discrete stenotic orifice (top) with those obtained when a screen was substituted for the stenotic orifice (middle) or when the orifice was displaced proximally (bottom). Numbers in figures refer to pressures (mm Hg) in each locus. Increment of innominate pressure above aortic (Innom-Ao) is given below each figure. See text for discussion.

Fluid in the jet has a high kinetic energy. Within the proximal portion of the innominate artery fluid flow divides into that portion which continues smoothly down the innominate to its distal radicals and that portion turning backward to flow into the distal aorta. That fluid remaining within the innominate artery is slowed by distal resistance and its kinetic energy is converted into potential energy (pressure). Hence, innominate pressure rises as the fluid is slowed. This process represents a conservation of mechanical energy. Fluid turning backward toward the aorta, however, loses its kinetic energy in whorls and eddies. This is a dissipative process in which kinetic energy is converted into heat, rather than pressure. There is thus no pressure rise within the aorta comparable to that in the innominate artery. In summary, the increase in innominate artery pressure relative to the pressure in the aorta in supravalvular aortic stenosis appears to result from a difference in the energy content of the blood entering the two vessels, the innominate receiving blood in which kinetic energy has been conserved as pressure and the aorta distal to the innominate origin receiving blood in which kinetic energy has been lost.

**Figure 6**
High-speed cinematography after dye injection revealed a jet issuing from the stenotic orifice directed exclusively into the innominate artery. Only after the jet was dispersed within the most proximal portions of the innominate artery did the fluid turn backward to flow into the distal aorta.
A quantitative evaluation of this explanation can be made by comparison of measured gains in innominate artery pressure (or potential) energy with estimates of the kinetic energy lost by the jet. According to Bernoulli's theorem, the sum of kinetic and potential energy per unit volume along a streamline is constant:

\[ \frac{1}{2} \rho V_1^2 + P_1 = \frac{1}{2} \rho V_2^2 + P_2 = \text{constant} \]

where \( V \) = velocity, \( P \) = pressure, \( \rho \) = density, and subscripts 1 and 2 designate different points along the streamline. Rearranging the above equation,

\[ \frac{1}{2} \rho (V_1^2 - V_2^2) = P_2 - P_1 = \Delta P \]

Thus if mechanical energy is completely conserved, the rise in pressure, \( \Delta P \), should equal the fall in kinetic energy, \( \frac{1}{2} \rho (V_1^2 - V_2^2) \). Mean velocities of the jet and of flow in the innominate artery were estimated by dividing measured flows by the cross-sectional areas of the stenotic orifice and innominate artery, respectively. The implied assumption of a uniform velocity profile in both instances is consistent with turbulent flow, which is probably present at both loci. Figure 7 is a plot of observed \( \Delta P \) or potential energy rise against kinetic energy loss, \( \frac{1}{2} \rho (V_1^2 - V_2^2) \), calculated in this manner. In each instance the observed \( \Delta P \) values were the same as, or somewhat less than, the corresponding estimated kinetic energy losses. Thus the kinetic energy lost in the innominate artery as a result of slowing of the jet was sufficient in each instance to account for the potential energy gain associated with the observed pressure rise. On the average, the pressure rise represented a 78% conversion of the estimated available kinetic energy. The remaining kinetic energy may have been lost in dissipative processes. In these calculations a minimum figure was used for the estimate of kinetic energy. A non-uniform velocity profile or a "vena contracta" at the stenotic orifice could only serve to increase kinetic energy loss above the values estimated. Thus, the efficiency of energy conversion may be as low as 50%. Nevertheless, these calculations clearly indicate that enough energy is available as a result of slowing of the jet to account fully for the observed pressure increases; no other source of energy is required.

Consideration of the energetics of fluid flow can also explain the seemingly paradoxical observation, noted previously, that flow from the aorta into the innominate artery occurs against a pressure gradient. Impelled by its momentum, blood does indeed flow up a pressure gradient. But in doing so, it experiences a corresponding loss in velocity and kinetic energy. The total energy content of blood, that is, the sum of kinetic and potential energies, is actually decreased after entering the innominate artery because the efficiency of energy conversion is generally less than 100%.

It should be emphasized that this model reproduces and explains the differences in mean pressures between innominate artery and aorta seen in patients. Particular attention was paid to the mean pressure difference...
ELEVATED INNOMINATE ARTERY PRESSURES because it necessarily implies a difference in the energy content of the blood in the two vessels. When mean pressures and flows are studied, elastic properties of vessel walls and transient phenomena, such as reflected waves, exert relatively little influence on the results. Thus, with respect to mean values our model closely resembles the in vivo situation. The difference in phasic pressures observed in patients, of course, will be influenced by the elasticity of the vessels, reflection phenomena, diastolic runoff, and other factors as well as the effects of the kinetic energy of the jet.

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References

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