An Explanation of Asymmetric Upper Extremity Blood Pressures in Supravalvular Aortic Stenosis

The Coanda Effect

By James W. French, M.D., and Warren G. Guntheroth, M.D.

SUMMARY

The Coanda effect, the tendency of a jet stream to adhere to a wall, was investigated as an explanation of the unequal pressures in the upper extremities in patients with supravalvular aortic stenosis (SVAS). Of 56 patients with SVAS reviewed, 48 had unequal blood pressures in the upper extremities. The average difference was 18 mm Hg systolic. Although 11 of the 20 patients in the control group (valvular aortic stenosis) had some blood pressure asymmetry, the average difference was 3.5 mm Hg systolic. In valvular aortic stenosis, the velocity of the jet is quickly dissipated beyond the stenotic orifice, preventing any sustained high-velocity stream. However, the smooth, annular narrowing of SVAS creates a "step" between the orifice and the ascending aortic wall which enhances the natural affinity of a jet for a boundary wall and conserves the kinetic energy of the jet stream. In most patients with SVAS, the high-velocity stream is along the right aortic wall, causing disproportionately high pressure in the right arm.

Additional Indexing Words: Annular stenosis Jet stream Kinetic energy

Supravalvular aortic stenosis (SVAS) is one of several lesions which are included in the congenital aortic stenosis group. Unequal systolic blood pressure in the upper extremities, which has been frequently reported with supravalvular stenosis,2-10 is an important diagnostic sign because it has only rarely been reported in other congenital cardiovascular lesions, except coarctation or interruption of the transverse aortic arch.11

We believe that aspects of fluid control theory, specifically the Coanda effect, offer a logical explanation of this phenomenon.

The Coanda effect,12-14 the tendency of a jet stream to adhere to a boundary wall, was first noted in 1910 by Henri Coanda15 and probably represents a special case of the more familiar Bernoulli principle. As a jet exits from a nozzle, it progressively broadens by entraining the surrounding fluid, the peak velocity is proportionately diminished, and the kinetic energy of the jet is dissipated downstream. An area of low pressure is produced at the margins of the jet, and a counterflow is created to equalize the pressures (fig. 1A). If a random disturbance causes the jet to deviate, inequalities will develop in the counterflow, and the stream may "attach" to one of the boundary walls. The course of the stream is then maintained by a combination of

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a low pressure area adjacent to the boundary wall and the countercurrent flow from the opposite side of the jet. A similar deviation is produced if the space or chamber around the nozzle is not symmetrical. The simplest example of this is shown in figure 1B. One wall of the chamber has been moved closer to the nozzle exit. It can be shown that the jet will consistently attach to this near wall,\textsuperscript{12} even following interruptions in the jet such as diastole. This tendency of the jet stream to reestablish a specific pattern is termed "reset" or "memory."\textsuperscript{4} Other variations in the dimensions and the configuration of the chamber will produce consistent changes in the course of the jet stream. One important modification is the introduction of a "shoulder" or "step" in the wall close to the nozzle. Experimental work has shown that this particular configuration enhances the Coanda effect if the step is not too abrupt.\textsuperscript{13} If the step is too abrupt, the jet will not follow the boundary wall, and the peak energy of the jet will be dissipated in an expanding volume downstream. The reason for the separation is the large angle of reflection necessary to reach the boundary wall.\textsuperscript{12, 14} This is termed an "anti-Coanda configuration."

By introducing a splitter or bifurcation downstream (figs. 1A and B), it is possible to demonstrate the kinetic energy of the jet stream as differential pressures or flows.\textsuperscript{12, 14} The bifurcation must be at least two nozzle widths distant from the nozzle (2:1 ratio) to establish the Coanda effect.

To investigate the frequency and significance of the unequal pressures, we have reviewed the data in six of our cases of SVAS, pressure and dimension data from 50 cases presented in the literature, and compared the results with the data from 20 of our patients with valvular aortic stenosis (VAS). The VAS group was chosen as a control because of the similarities between the two lesions; thus, if there is a significantly greater pressure differential in the supravalvular group, it should be

\begin{figure}
\centering
\includegraphics[width=\linewidth]{figure1.png}
\caption{A jet stream flowing into a bounded chamber causes entrainment of fluid by the jet, generation of low pressures at the jet margins, and a counterflow (represented by arrows) of chamber fluid. (A) The jet kinetic energy is dissipated downstream, as the high-velocity stream broadens. \(B\) Unequal counterflow as a result of asymmetry of the chamber causes deviation and "attachment" of the jet stream, with conservation of its kinetic energy and direction of the stream into the right branch.}
\end{figure}
EFFECT COANDA

20– R > L
left arm

Differences in systolic pressures in the upper extremities. Each dot represents one patient (total number of cases, 76). SVAS = supravalvular aortic stenosis group; VAS = control group with valvular aortic stenosis; R > L = systolic pressure in right arm exceeds that in left arm. L > R = systolic pressure in left arm exceeds that in right arm.

due to the specific configuration of the supravalvular lesion.

Methods

The blood pressures in patients with supravalvular or valvular stenosis seen in the University of Washington Hospital were obtained with a standard sphygmomanometer cuff by auscultation. The proper-sized cuff was judged to be 20% wider than the diameter of the extremity where the cuff was applied. All patients were examined either in the Pediatric Cardiology Clinic or as inpatients on the Pediatric Cardiology Service at University Hospital. Cases were accepted from literature if blood pressures in both upper extremities were recorded in the report. Angiocardiographic films were reviewed for step configuration (gradual transition between the stenotic annulus and the poststenotic wall) and to determine the ratio of the chamber length (from the level of stenosis to the orifice of the innominate artery) to the stenotic orifice.

Results (Table 1; Fig. 2)

Of the 56 patients with SVAS under review, 48 or 86% had unequal systolic blood pressures in the upper extremities. The average difference in all cases of SVAS was 18 mm Hg (± 3.1). The range was 0 to 60 mm Hg. Higher pressures were obtained in the right arm in 40 or 83% of the patients with a difference in pressures. In the control group of 20 patients with valvular aortic stenosis, 11 or 55% had a systolic pressure differential in the upper extremities, but the difference for all valvar cases was only 3.5 mm Hg (± 0.2). The range was 0 to 10 mm Hg. Higher pressures were obtained on the right in five or 45% of patients with differential pressures. The average differences in systolic pressure between the two arms in SVAS, 18 mm Hg, is significantly greater (P < 0.01) than the average difference in systolic pressures in VAS, 3.5 mm Hg. In all cases reviewed, the ratio of chamber length to stenotic diameter exceeded the minimum 2:1 ratio necessary for the Coanda effect to be established.

### Table 1

**Data on Upper Extremity Systolic Blood Pressures in SVAS and VAS**

<table>
<thead>
<tr>
<th></th>
<th>SVAS</th>
<th>VAS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of cases</td>
<td>56</td>
<td>20</td>
</tr>
<tr>
<td>No. with unequal pressures</td>
<td>48 (86%)</td>
<td>11 (55%)</td>
</tr>
<tr>
<td>Right greater than left</td>
<td>40</td>
<td>5</td>
</tr>
<tr>
<td>Left greater than right</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Average difference (all cases) in blood pressures (mm Hg)</td>
<td>18 (±3.1)*</td>
<td>3.5 (±0.2)*</td>
</tr>
<tr>
<td>Range of difference (mm Hg)</td>
<td>0-60</td>
<td>0-10</td>
</tr>
</tbody>
</table>

*Standard error of mean.

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Discussion

Two principal explanations have been proposed for the differential pressures in the upper extremities. A selective stenosis at the origin of arch vessels has been suggested as a cause of the unilaterally diminished pressure.\(^4,\,8,\,20,\,26,\,27\) However, recent reviews of pathologic specimens by Peterson and associates\(^28\) and radiologic material by Kupic and Abrams\(^22\) have suggested that the incidence of significant selective aortic arch vessel stenosis is in the range of only 15 to 20%. Based on the radiologic report, selective stenoses of either the left common carotid or left subclavian vessels are more common, although only 22 of the 121 cases reviewed had any significant stenosis of arch vessels. While it is probable that selective arch vessel stenosis is significant in some cases, the frequency is too low to explain the rather high incidence of differential pressures in patients with SVAS.

We support the alternate theory that the cause of the differential pressures is a high velocity jet streaming into the origin of a specific arch vessel. In 1963, Lurie and Mandelbaum,\(^29\) working with a canine model, and more recently Goldstein and Epstein,\(^30\) with an aortic arch model, have confirmed that the kinetic energy developed in a jet stream under simulated pathologic conditions (that is, with SVAS) is sufficient to cause the clinically observed difference in pressures. It has been suggested that the differential was the result of the alignment of the ascending aorta and the orifice of the right innominate artery. However, this would not explain patients with higher pressures on the left side. In addition, patients with VAS without SVAS have high velocity jets, but rarely unequal pressures. The high incidence of differential pressures in SVAS logically suggests a unique mechanism controlling the course of the stream between the stenotic area and the arch vessel bifurcation.

The major difference between the valvular and supravalvular lesions occurs in the immediate poststenotic area. In valvular aortic stenosis, the initial step between the valve orifice and the ascending aortic wall is large and abrupt (fig. 3A). This is made even more extreme by the commonly associated poststenotic dilatation. Consequently, the jet stream does not attach to the aortic wall. By contrast, the annular hourglass narrowing of the supravalvular lesion creates an optimal step or shoulder transition between the stenotic area and the wall of the ascending aorta (figs. 3B and 4). The limiting effect of the boundary wall on one surface and the confining effect of the counterflow on the opposite surface tend to stabilize the jet and retard its natural tendency to broaden and disperse. With the aortic wall as a boundary, a positively directed, sustained jet stream is produced. Although the higher pressures are usually recorded in the right arm, the Coanda effect would apply equally well to patients who have higher pressures in the left arm. This is logical if the aorta is considered in its three dimensions (fig. 4). The jet stream could easily flow up the posterior wall of the aorta, bypassing the right innominate artery and entering the left common carotid or the left

Figure 3

Diagrammatic representations. (A) Valvular aortic stenosis with mild poststenotic dilatation. Note abrupt transition between stenotic valve and ascending aortic wall. (B) Supravalvular aortic stenosis. Note gradual transition (step) between annular stenosis and ascending aortic wall.
subclavian artery without separating from the aortic wall.

In summary, we have proposed an explanation for the difference in systolic blood pressures in the upper extremities seen in SVAS. We have emphasized that although both valvular and supravalvular aortic stenosis produce jet streams, the unique step configuration of the supravalvular aortic stenosis and the Coanda principle provide an explanation for the unequal carotid and brachial artery systolic pressures commonly seen in the patients with supravalvular lesions.

References


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