Aortic Stenosis
Correlations between Pressure Gradient and Left Ventricular Angiocardiography

By VIKING OLOV BJÖRK, M.D., INGEMAR CULLHED, M.D., and HERMAN LODIN, M.D.

In the study of hemodynamics in aortic stenosis until the last decade more or less indirect methods were used, such as electrocardiography and phonocardiography, and direct and indirect pulse tracings. At best the results would confirm a clinical diagnosis but often were of little help in the surgical evaluation of the valvular defect.

Only after the introduction of left-sided heart catheterization with pressure measurements in the left ventricle, by the trans-thoracic or transbronchial left atrial routes or percutaneous left ventricular puncture, can the aortic stenosis be evaluated with respect to the systolic pressure gradient.

Thoracic aortography can give valuable information in aortic stenosis concerning the mobility of the aortic cusps and the degree of post-stenotic aortic dilatation but does not reveal subaortic stenosis. Thoracic aortography is the routine method in assessing aortic regurgitation.

Aortic catheterization may be extended to the left ventricle, where contrast injection may be done. This method carries the danger of damaging an aortic cusp or obstructing a coronary ostium. Further, the roentgenologic study of the mobility of the aortic leaflets is disturbed by the presence of the catheter, and in tight stenosis the catheter adds to the obstruction and artificially exaggerates the systolic pressure in the ventricle.

We have in the last years performed percutaneous puncture of the left ventricle with contrast injection in more than 120 cases. The value of left ventricular angiocardiography in the diagnosis of valvular heart disease was shown early. It is the aim of this paper to discuss the correlations between the pressure measurements and the findings at the left ventricular angiocardiography in aortic stenosis.

Material and Methods

The material consists of 36 patients with aortic stenosis of whom 21 had a pressure gradient over the aortic orifice. The patient lies supine, and with the caudal part of the thorax at some distance from the lateral film (see below). A thin polythene catheter, 10 to 15 cm. long, was introduced percutaneously into a peripheral artery, usually the left femoral artery. The left ventricle was punctured under local anesthesia as suggested by Brock. The catheter and the puncture needle were connected each to a strain-gage electrometer, with the anterior axillary line as reference level. The pressure curves were recorded on a direct-writing four-channel oscillograph, together with a standard limb-lead electrocardiogram and the x-ray exposures. On a single-beam cathode-ray oscilloscope, the electrocardiogram or a pressure curve was continually observed.

On undamped curves the systolic pressures were determined in the ventricle and the artery. From these values the gradient between the peak systolic pressures was calculated. After the position of the needle in the ventricle was controlled by observation of the ventricular pressure curve when the direction of the needle was altered, 1 ml. of 76 per cent "Urografin" per Kg. of body weight was injected, an electrocardiogram being run at the same time. Six frames per second (maximum exposure time 0.03 second) were exposed in two planes during inspiration. During and immediately after the injection the carotid arteries were compressed in order to reduce the flow of contrast material to the brain.

After the withdrawal of the needle an electrocardiogram and a chest x-ray were taken to look for signs of cardiac tamponade or pneumothorax.

We have encountered two fatal complications among our first 120 left ventricular punctures. In a 55-year-old man cardiac tamponade occurred and in a 9-month-old boy the contrast was injected into the myocardium. A detailed report of our minor and major complications is under preparation.

Roentgenologic Aspects

In the ideal roentgen visualization of the aortic orifice the beams should be parallel with the valve plane. In true frontal and lateral projections, however, the incident beam generally forms an angle with that plane (fig. 1). This disadvantage can, however, to some extent be reduced by adjusting the position of the patient or the tubes to bring the beams more parallel to the valve plane.

The position of the lateral tube or the patient should be arranged so that the beam falls in a somewhat caudo-cranial direction; the more so, the more transverse is the position of the heart. Adjustment of the frontal tube, however, has little effect, since the angle between the valve plane and the vertical beam (fig. 1) generally is small and since the valve plane may be inclined either cranially or caudally. The orientation of the valve plane thus cannot be exactly predicted.

In assessing the thickness and mobility of the individual cusps, it is essential that the beam be directed along the cusp and its base "tangentially" (fig. 2). It is impossible to get more than one of the three cusps in an ideal position with use of two planes at right angles to each other. In a true lateral projection a "tangential" picture is obtained of the right coronary cusp, whereas in pure frontal and lateral projections a more oblique view is obtained of the other two cusps.

Accordingly we have been using true frontal and lateral projections, the inferior part of the thorax being at a slight distance from the vertical film plane.

Owing to this arrangement it may be necessary to tolerate projections that are at times highly unsatisfactory, rendering difficult the assessment of the morphology of the aortic orifice and the mobility of the cusps. Thus two or even all of the cusps may be shown in some degree of "oblique" projection, and it may be incorrectly assumed that the mobility of the cusps and caliber of the aortic orifice are reduced. Further, the valve plane may be completely masked in the lateral projection by an enlarged left ventricle when there is transverse position of the heart. With use of two planes at right angles to each other, however, some of the difficulties are eliminated, the assessment of the orifice being based on a comparison between the two planes.

The normal orifice is characterized by thin scarcely visible cusps, which open fully in systole and close tightly in diastole.

The diagnosis of aortic stenosis is made on the grounds of the thickness and mobility of the cusps, the caliber of the orifice, the rate of contrast flow through the orifice, the appearance of the ascending aorta, and the
emptying capacity of the left ventricle and the thickness of its wall.

In estimating the thickness of the cusps, care must be taken to measure only cusps that are seen in "tangential" view, or misleading figures will be obtained.

The mobility of the cusps is estimated by observing their position during different phases of the cardiac cycle, regard being paid to any disturbances of rhythm and the rate of flow. The rate of flow may be so slow, for example in marked mitral stenosis and mitral incompetence, that it is insufficient to open the valves fully, especially if the aortic cusps are thickened. If the rate of flow is neglected, therefore, a "morphologic" stenosis may be diagnosed when in fact the cusps are normal or thickened but not fused.

In many cases of stenosis a typical dome is seen. The caliber of the orifice is assessed with greatest accuracy if there is a jet. In many cases, however, the caliber may be measurable in only one plane, or not at all, because the orifice is eccentric on the dome and the incident beam therefore does not meet it tangentially.

The degree of stenosis when the orifice cannot be measured exactly may to a certain extent be assessed by estimating the rate of the flow and the degree of post-stenotic dilatation of the ascending aorta. If other causes of delayed flow can be excluded, incomplete mixture of contrast medium and blood in the ascending aorta indicates grave stenosis. In cases of pure stenosis conclusions can even be drawn from the thickness of the ventricular wall which is measured in diastole above the apex. The emptying time of the ventricle after the completion of the injection, in relation to the number of ventricular contrac-

---

**Table 1**

<table>
<thead>
<tr>
<th>Pressure gradient in mm. Hg</th>
<th>S-T depression and negative T waves</th>
<th>Diagnosis's</th>
<th>Femoral artery pressure</th>
<th>Systolic and end-diastolic left ventricular pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>128</td>
<td>-</td>
<td>AS</td>
<td>120/75</td>
<td>248/15</td>
</tr>
<tr>
<td>105</td>
<td>+</td>
<td>AS MI</td>
<td>100/65</td>
<td>205/10</td>
</tr>
<tr>
<td>102</td>
<td>+</td>
<td>AS MI</td>
<td>140/60</td>
<td>242/20</td>
</tr>
<tr>
<td>100</td>
<td>+</td>
<td>AS MS TS</td>
<td>105/80</td>
<td>200/0</td>
</tr>
<tr>
<td>89</td>
<td>+</td>
<td>AS MS MI AI</td>
<td>188/84*</td>
<td>277/20</td>
</tr>
<tr>
<td>80</td>
<td>+</td>
<td>AI AS</td>
<td>150/73</td>
<td>230/13</td>
</tr>
<tr>
<td>80†</td>
<td>+</td>
<td>AS</td>
<td>115/49</td>
<td>195/6</td>
</tr>
<tr>
<td>75</td>
<td>-</td>
<td>AS MS</td>
<td>110/62</td>
<td>185/15</td>
</tr>
<tr>
<td>52</td>
<td>+</td>
<td>AS AI MI</td>
<td>142/64</td>
<td>194/32</td>
</tr>
<tr>
<td>50</td>
<td>-</td>
<td>AS MI MS AI</td>
<td>190/72</td>
<td>240/20</td>
</tr>
<tr>
<td>40</td>
<td>-</td>
<td>MS AS MI AI</td>
<td>118/70</td>
<td>158/0</td>
</tr>
<tr>
<td>40</td>
<td>-</td>
<td>MS AS AI</td>
<td>125/95</td>
<td>165/0</td>
</tr>
<tr>
<td>32</td>
<td>-</td>
<td>AS</td>
<td>125/72</td>
<td>157/15</td>
</tr>
<tr>
<td>30</td>
<td>-</td>
<td>AS</td>
<td>140/84</td>
<td>170/14</td>
</tr>
<tr>
<td>27</td>
<td>-</td>
<td>AS</td>
<td>137/59</td>
<td>164/0</td>
</tr>
<tr>
<td>25</td>
<td>-</td>
<td>AS MS</td>
<td>185/93</td>
<td>210/15</td>
</tr>
<tr>
<td>20</td>
<td>-</td>
<td>AS</td>
<td>135/64</td>
<td>156/4</td>
</tr>
<tr>
<td>20</td>
<td>-</td>
<td>AI AS</td>
<td>210/84</td>
<td>230/7</td>
</tr>
<tr>
<td>15</td>
<td>1</td>
<td>AI AS</td>
<td>185/80</td>
<td>200/5</td>
</tr>
<tr>
<td>12</td>
<td>-</td>
<td>MS AS</td>
<td>130/55</td>
<td>142/0</td>
</tr>
<tr>
<td>8</td>
<td>-</td>
<td>MS AS AI</td>
<td>159/86</td>
<td>167/8</td>
</tr>
</tbody>
</table>

*Brachial artery pressure.
†In this case no angiocardiogram was obtained. The patient died in connection with the puncture and was reported recently.12

(AS = aortic stenosis, AI = aortic insufficiency, etc.)
In RCC, between and mentions, probably maintained. The femoral pulses were found again, the difference being in Hg. In R, right coronary cusp; R, right; L, left. The direction of the beam (→).

**Figure 2**

*Schematic drawing illustrating the relations between the cusps and the incidence of the beam. In the lateral projection the right coronary cusp meets the beam ideally. An oblique view is obtained of the two other cusps in both projections. RCC, right coronary cusp; R, right; L, left. The direction of the beam (→).*

**Results**

Undamped pressure curves were obtained in all cases but two. These patients (cases no. 6 and 31) suffered from obliterating, probably thromboembolic aortic disease. The femoral pulses were absent in one and weak in the other patient. This source or error in the determination of the pressure gradient should be considered in the elder patients.

The pressure gradient in four cases was again measured during operation, prior to valvulotomy. A reasonable correlation was found with the preoperative values, the difference being -10, +13, +11, and -58 mm Hg. The significant lower value in the last case (no. 100) may be due to altered hemodynamics, as the operation was performed in hypothermia and extracorporeal circulation. The remaining gradient, 70 mm., was reduced to 0 mm. through the operation. Other authors, too, have found a good correlation between the pressure gradients before and during surgery.3, 13, 14

In normal cases as well as in aortic stenosis there is usually a higher systolic pressure in the femoral arteries than in the central aorta.15-17 Even a small positive systolic pres-

---

**Table 2**

*Correlations between the Gradient and the Incidence of Calcifications*

<table>
<thead>
<tr>
<th>Pressure gradient in mm. Hg</th>
<th>Number of cases</th>
<th>Number with calcifications</th>
<th>Mean age</th>
</tr>
</thead>
<tbody>
<tr>
<td>128-52</td>
<td>8</td>
<td>7</td>
<td>29</td>
</tr>
<tr>
<td>50-25</td>
<td>7</td>
<td>4</td>
<td>32</td>
</tr>
<tr>
<td>20-8</td>
<td>4</td>
<td>1</td>
<td>42</td>
</tr>
<tr>
<td>0</td>
<td>10</td>
<td>1</td>
<td>41</td>
</tr>
</tbody>
</table>

**Table 3**

*The Pressure Gradient Correlated with the Left Ventricular Wall Thickness*

<table>
<thead>
<tr>
<th>Pressure gradient in mm. Hg</th>
<th>Number of cases</th>
<th>Thickness of ventricle in mm. mean</th>
<th>range</th>
</tr>
</thead>
<tbody>
<tr>
<td>128-52</td>
<td>8</td>
<td>16</td>
<td>12-20</td>
</tr>
<tr>
<td>50-25</td>
<td>7</td>
<td>14</td>
<td>9-25</td>
</tr>
<tr>
<td>20-8</td>
<td>5</td>
<td>11</td>
<td>8-15</td>
</tr>
<tr>
<td>0</td>
<td>15</td>
<td>12</td>
<td>6-25</td>
</tr>
</tbody>
</table>

---

The pressure gradient between the left ventricle and the femoral artery will thus probably be due to some degree of stenosis. Because of the jet effect pressure measurements just distal to the aortic valve will differ considerably according to the situation of the tip of the catheter.18-20 It should thus be safer to measure the pressure in the femoral artery.

In the following our search for different correlations with the pressure gradient is related. We wish to emphasize once more that "pressure gradient" means the gradient between the left ventricular and the peripheral artery pressures.

**Correlations between the Gradient and the Electrocardiographic Changes**

The material was grouped according to the pressure gradient, (table 1). All cases but one, with a gradient of at least 80 mm. Hg show S-T depression and T-wave negativity in at least two of leads V4, V5, and V6. This is in general agreement with Fleming et al.,21 although the exceptional case with a gradient of 128 mm. Hg (the diagnosis was verified at open-heart surgery) shows the possibility of severe aortic stenosis with an essentially normal electrocardiogram. This point was also made by Matthews et al.22 The same electro-
AORTIC STENOSIS

Table 4

The Pressure Gradient Correlated with the Size of the Aortic Orifice

<table>
<thead>
<tr>
<th>Pressure gradient in mm. Hg</th>
<th>Number of cases</th>
<th>Diameter in mm. mean</th>
<th>range</th>
</tr>
</thead>
<tbody>
<tr>
<td>105-52</td>
<td>7</td>
<td>8</td>
<td>3-15</td>
</tr>
<tr>
<td>50-8</td>
<td>8</td>
<td>10</td>
<td>5-15</td>
</tr>
<tr>
<td>0</td>
<td>14</td>
<td>11</td>
<td>8-15</td>
</tr>
</tbody>
</table>

cardiographic findings were found in a case with a gradient of only 52 mm., with severe combined aortic valvular disease and left heart failure. Of the 15 cases with aortic stenosis without a pressure gradient only one had the same electrocardiographic picture, a 40-year-old man with aortic stenosis and insufficiency and mitral insufficiency.

Correlation between the Gradient and the Incidence of Aortic Valvular Calcification

The presence of calcifications in the material was investigated by means of tomography, which was performed in 29 of the 36 cases. When the incidence of calcifications was correlated to the pressure gradients (table 2), a higher incidence was found in those with a larger gradient. In the different groups the cases with calcification were found in the older patients.

Correlation between the Pressure Gradient and the Degree of Left Ventricular Hypertrophy

On the angiocardiograms the thickness of the left ventricle in diastole was measured as an index of the degree of hypertrophy (table 3).

Since the groups are small, very limited conclusions can be drawn. There seems to be a positive correlation, however, between the gradient and the degree of left ventricular hypertrophy, as would be anticipated.

Correlation between the Pressure Gradient and the Left Ventricular End-Systolic Volume

The volume of the contrast-filled ventricular cavity at the end of systole was assessed on the angiocardiograms. No correlation was found between the gradient and this volume. Nor was any correlation found between this volume and the total heart volume, expressed in milliliters per square meter of body surface area. The degree of arrhythmia induced by the contrast injection could not be shown to influence the magnitude of the residual volume.

Correlation between the Gradient and the Degree of Stenosis

The normal aortic orifice has a diameter of about 2.6 cm. The diameter in vivo can be estimated indirectly in pure aortic stenosis according to the formulas introduced by Gorlin. More directly the diameter can be determined during open-heart surgery, though palpation may overestimate the width of the ostium. With due regard to the limitations discussed earlier in this paper we have tried to measure the diameter of the aortic orifice on the angiocardiograms. This was possible in 29 cases, and these were grouped according to the pressure gradient (table 4). The orifice is smaller in the cases with the largest gra-

Circulation, Volume XXIII, April 1961
dieters: in this group only one case had a diameter of over 10 mm. That case had a gradient of 102 mm. with a ventricular pressure of 240/20 mm. Hg in the absence of any clinically significant aortic incompetence.

There was no correlation between the gradient and the thickness of the aortic cusps, which in all cases measured 2 to 4 mm. The mobility of the cusps was good in only four of 20 cases with a pressure gradient, contrasted with the good mobility in eight of 15 cases without gradient.

No correlations were found between the gradient and the heart volume, the left ventricular emptying time or the physical working capacity, as measured on an ergometer cycle in kilograms per meter per minute.

The findings in four cases of aortic stenosis are presented as representative examples.

**Case Reports**

**Case 1**

G. K., a 39-year-old man (201024/59). No rheumatic fever. For 2 or 3 years dyspnea and dizziness on work. Clinically he was classified as isolated aortic stenosis. Right heart catheterization: normal pressure values in rest and at work. Electrocardiogram: no definite signs of hypertrophy. Chest radiogram revealed a normal-sized heart (340 ml./M.2 BSA), on tomography rather extensive intracardiac calcifications were seen in the aortic valvular region. On left ventricular puncture a pressure gradient over the aortic orifice of 128 mm. Hg was detected. Left ventricular angiogram: see figure 3. The patient was operated upon with the aid of deep hypothermia (24 C.) and extracorporeal circulation. The noneorony and the fused right and left coronary cusps were found heavily calcified, leaving a long but narrow passage for the blood stream. By curettage the noneorony cusp could be decalcified and mobilized, resulting in elimination of the pressure gradient, as measured during surgery. No attempt at valvulotomy was regarded as possible because the mobility would not be influenced. When the patient left the hospital 5 weeks after the operation he was subjectively better and had less calcifications on tomography. The patient died 2 months later of a brain embolism from an incisional aortic aneurysm.

**Case 2**

M. J., a 45-year-old woman (140921/58). Rheumatic fever when 19 years old. For the last 5 years decompensated—grade III (New York Heart Association). She was admitted for the first time in 1957. A tricuspid and mitral stenosis was diagnosed and later was operated upon.25 For the first half year she felt better but then her symptoms came back and she was again admitted in 1958. The clinical diagnosis now became aortic, mitral, and tricuspid stenosis, possibly mitral regurgitation. Right heart catheterization revealed moderate pulmonary hypertension.

Figure 4

(M. J.) Aortic valvular stenosis + mitral stenosis. Angiocardiograms after ventricular puncture. Ventricular systole, (left, center) lateral plane, ventricular diastole, (right) lateral plane. Typical aortic stenosis with a 3-mm. broad jet through the orifice. The jet is eccentric, the contrast stream directed against the anterior aortic wall (left). The cusps are very thick (center) and practically without any mobility (right). Slight dilatation of the middle part of the ascending aorta. Typical mitral dome26 (right): mitral stenosis.
and a diastolic gradient between the right atrium and ventricle. Electrocardiogram: right axis deviation, no ventricular hypertrophy. Chest radiogram: heart size 1080 mL/M.2 BSA. Tomography: mitral and aortic calcifications. On left ventricular puncture a systolic pressure gradient of 100 mm Hg was found. The angiocardiogram (fig. 4) visualized a valvular aortic stenosis. On reoperation a transventricular dilatation of the aortic and mitral valves was done. However, a rupture of the aortic valve occurred, resulting in severe aortic regurgitation, which overburdened the heart. The autopsy confirmed the preoperative diagnosis.

Case 3

R. E., a 20-year-old man (390321/59). No rheumatic fever. A murmur heard since the early school years. Slight symptoms on exercise (grade I). Clinical diagnosis: isolated aortic stenosis. Electrocardiogram: normal. Chest radiogram: heart size 400 mL/M.2 BSA. Tomography: no calcifications. Left ventricular puncture: valvular aortic stenosis (fig. 5) with a pressure gradient of 32 mm. Hg. So far operation has not been advised.

Case 4

R. J., a 40-year-old woman (180613/58). Rheumatic fever when 34 years old, since that increasingly decompensated, functionally grade III. The clinical diagnosis was aortic and mitral stenosis. Right heart catheterization revealed definite pulmonary hypertension, Electrocardiogram: left atrial enlargement, but no signs of ventricular hypertrophy. Chest radiogram: heart size 495 mL/M.2 BSA. Tomography: no calcifications. Left ventricular puncture was performed. There was no pressure gradient but the angiocardiograms (fig. 6) showed a valvular aortic stenosis with fused but mobile cusps. At operation the mitral stenosis was very tight, open only for the tip of the finger. A transventricular dilatation was performed, first of the mitral valve to about 1½ fingerbreadths, and then of the aortic valve, which offered a hard resistance to the instrument. With some force the valve could be dilated to about 2 fingerbreadths. After the first postoperative week in a respirator the patient made a good recovery and left the hospital after 7 weeks.

Discussion

Leonardo da Vinci, as quoted by McMillan,24 has shown the triangular form of the normal aortic orifice. The internal diameter of this orifice averages 2.6 cm., which makes an area of about 3 cm.2 No hemodynamic changes occur until the orifice is reduced to at least one quarter,18 corresponding to a diameter of about 10 mm. In isolated aortic stenosis the valve area may be estimated when the rate and magnitude of flow across the valve are known. This is possible if the pressures on both sides of the valve are determined at the same time as the cardiac output. In coexisting aortic regurgitation the total left ventricular stroke volume cannot be determined and thus the valve area during systole cannot be assessed either. In spite of insignificant stenosis a large systolic pressure gradient will occur in aortic regurgitation,27 due to the large systolic flow. However, in acute dog experiments Moscovitz et al.28 obtained no systolic pressure gradient in aortic regurgitation.

On the other hand, in mitral stenosis and insufficiency the aortic flow will be diminished, as in left ventricular failure. When
significant aortic stenosis exists, the diminished flow will give only a small gradient.

From a practical point of view Brock and Wood stated that a pressure gradient over the aortic orifice of at least 50 mm. points to a significant aortic stenosis. In co-existing mitral stenosis or aortic regurgitation, the gradient should exceed 25 or 100 mm. respectively.

The combination in left ventricular puncture of pressure measurement and left ventricular angiocardiography has not been reported earlier in the study of aortic stenosis. Cregg et al. mentioned "the excellent visualization of the aortic mitral valves," but reported no case of aortic stenosis. Recently Connolly performed pressure measurements in aortic stenosis and mentioned contrast injection. No results were given, however.

Our material of 36 cases is rather heterogeneous, with six cases of isolated aortic stenosis and 30 cases of different combinations of valvular diseases. In this report we have chosen to present the whole material together, grouped according to the pressure gradient, since the different diagnostic subgroups would otherwise be very small.

With one exception the gradient was found to correlate well with the T-wave negativity over the left ventricle. The same applies to the degree of aortic valvular calcifications and, though less marked, to the size of the orifice and to the ventricular wall thickness. In estimations of the size of the orifice due attention must be paid to the risk of over-diagnosis by underestimating the width, if due to low stroke volume (e.g., in mitral insufficiency or stenosis) or to unsatisfactory, oblique projections.

Probably hemodynamic changes may occur even before the aortic ostium is reduced to one fourth of the normal area, i.e., to a diameter of about 10 mm. Our two cases with isolated aortic stenosis and gradients, respectively 20 and 27 mm. Hg, had ostium widths of 15 and 12 to 13 mm., respectively, and these are minimum measurements. However, these cases were young men with only slight symptoms and have so far not been operated upon.

On the other hand a tight aortic stenosis may exist without any pressure gradient, if combined with mitral valvular disease or in left ventricular failure. In this material two

Figure 6

(R. J.) Aortic valvular stenosis + mitral stenosis. Angiocardiograms after ventricular puncture. Ventricular systole: (left) lateral plane, (center) frontal plane. Ventricular diastole: (right) frontal plane. Typical aortic stenosis with thick cusps and dome formation in both planes. The orifice is about 1.5 cm. in diameter. The aortic cusps have a good mobility. Slight dilatation of the middle part of the ascending aorta. The free borders of the mitral leaflets are thickened (left) and in ventricular diastole a typical mitral dome is seen (right), causing a defect in the ventricular contrast : mitral stenosis.
cases with mitral and aortic stenosis without gradient had aortic ostium widths of 8 and 10 mm. Both cases were operated upon with closed transventricular dilatation of both valves. The aortic valves made a tough resistance to the instrument in both cases, but a successful dilatation was performed. So far no case without a gradient has been operated upon with open technic.

It is thus our impression that left ventricular angiocardiography is a valuable addition to the technic of left ventricular puncture, in the study of aortic stenosis. Especially in combined valvular disease we think it may sometimes be of vital importance in order to detect preoperatively and to grade an aortic stenosis. The absence of a gradient at rest in these cases is probably due to lessened diastolic filling. Perhaps more information could be gained by measuring the ventricular pressure at rest and graded exercise.

The value of left ventricular angiocardiography in the diagnosis of subvalvular aortic stenosis was recently reported.22 The same should apply to the diagnosis of supravalvular stenosis and a coexistent aortic coarctation.

Post-stenotic aortic dilatation is usual in valvular aortic stenosis and can be detected in chest radiograms. A more exact outline may be obtained by contrast injection, as is shown in our case 3, where the considerable aortic dilatation awoke suspicions of medionecrosis cystica.23

Summary

In 36 cases of valvular heart disease with a clinical diagnosis of isolated or significant aortic stenosis we have performed percutaneous intercostal puncture of the left ventricle for pressure measurements and, in all but one case, left ventricular angiocardiography. The possible correlations between the pressure gradient and the angiocardiographic findings are discussed.

With the exception of the unsatisfactory angiocardiograms due to oblique projection of the valvular planes, valuable information was obtained regarding the degree of aortic stenosis. This was especially the case if the aortic stenosis was combined with mitral valve disease, resulting in a small or no systolic pressure gradient.

References


To the physician particularly a scientific discipline is an incalculable gift, which leavens his whole life, giving exactness to habits of thought and tempering the mind with that judicious faculty of distrust, which can alone, amid the uncertainty of practice, make him wise.—SIR WILLIAM OSLER. Aphorisms from His Bedside Teachings and Writings. Edited by William Bennett Bean, M.D. New York, Henry Schuman, Inc., 1950, p. 114.
Aortic Stenosis: Correlations between Pressure Gradient and Left Ventricular Angiocardiography

VIKING OLOV BJÖRK, INGEMAR CULLHED and HERMAN LODIN

_Circulation._ 1961;23:509-518
doi: 10.1161/01.CIR.23.4.509

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1961 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/23/4/509

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/