Two Types of Intraventricular Pressure Difference in the Same Patient

Left Ventricular Catheter Entrapment and Right Ventricular Outflow Tract Obstruction

By Allan G. Adelman, M.D., and E. Douglas Wigle, M.D.

SUMMARY

Two types of intraventricular pressure difference were encountered in a single patient who presented with clinical evidence of obstruction to right ventricular outflow and nonobstructive cardiomyopathy of the left ventricle. On the basis of measurements of inflow tract pressure, the pressure difference in the right ventricle was demonstrated to be due to muscular subvalvular obstruction to right ventricular outflow, whereas the pressure difference in the left ventricle resulted from catheter entrapment in the myocardium. This combination of intraventricular pressure differences must be differentiated from the situation in muscular subaortic stenosis, in which biventricular obstruction to outflow occurs.

Additional Indexing Words:
Initial inflow tract pressure  Cardiomyopathy  Muscular subaortic stenosis

Two types of intraventricular pressure difference may be encountered within the left ventricle of man.1-3 In muscular subaortic stenosis, all pressures proximal to the outflow tract obstruction, including that just inside the mitral valve (initial inflow tract pressure1,2), are elevated above the systolic pressure in the outflow tract distal to the stenosis. An intraventricular pressure difference may also result from catheter entrapment in the myocardium.4-7 In this instance, an elevated ventricular systolic pressure is recorded only by the entrapped catheter. All other truly intracavitary pressures are not elevated, including the initial inflow tract pressure.1-3,6,7

These same principles should apply to pressure differences within the right ventricle—that is, in the presence of infundibular obstruction to right ventricular outflow, all pressures proximal to the obstruction, including the pressure just inside the tricuspid valve (initial right ventricular inflow tract pressure), should be elevated above the systolic ventricular pressure distal to the obstruction.

Using measurements of initial ventricular inflow tract pressure as a means of differentiating between the two types of intraventricular pressure difference, this report describes a patient in whom the intraventricular pressure difference in the right ventricle resulted from outflow tract obstruction, whereas the intraventricular pressure difference in the left ventricle resulted from catheter entrapment in the myocardium.

Report of Case

Mrs. E. H., a 42-year-old married stenographer, was in good health until 1945, when, at age 20, she was hospitalized for acute glomerulonephritis. A heart murmur was discovered at that time. She had no cardiac symptoms and no family history of heart disease. Two years later she was admitted to the Toronto General Hospital in
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the sixth month of her first and only pregnancy with severe hypertension, edema, and proteinuria. An ejection systolic murmur was again noted and cardiac enlargement was evident in the chest x-rays. A diagnosis of toxemia of pregnancy superimposed on chronic glomerulonephritis was made. As the hypertension could not be controlled medically, an abdominal hysterotomy was performed. The blood pressure dropped from 240/140 to 160/120 mm Hg and remained at this level subsequently. From 1947 to 1963, mild intermittent ankle edema was noted. Dyspnea and palpitations on exertion began in 1963 and progressed until December 1966, at which time she was admitted to another hospital for investigation. Two cardiac catheterizations were carried out, following which the authors were asked to see the patient.

The significant clinical findings were restricted to the cardiovascular system. The arterial pulse was regular and of full volume. The blood pressure was 165/115 and there were grade 2 arteriosclerotic changes in the ocular fundi. The a waves in the jugular venous pulse were 3 cm above the sternal angle. A thrusting left ventricular impulse and an atrial gallop sound were palpable in the midaxillary line. There was no significant right ventricular heave, but a systolic thrill was felt along the upper left sternal border.

A grade IV/VI systolic ejection murmur, which increased in intensity with inspiration, was maximal in the third left intercostal space (fig. 1). This murmur faded toward the aortic area and lower left sternal border, and a faint midsystolic murmur was noted at the cardiac apex (fig. 1).

**Figure 1**

*Phonocardiogram demonstrating the loud systolic murmur (SM) in the pulmonary area and the faint apical systolic murmur. LSB4 = left sternal border, fourth intercostal space; S₁ = first heart sound; S₂ = second heart sound; S₃ = fourth heart sound.*

**Figure 2**

*Electrocardiogram showing evidence of left atrial and ventricular hypertrophy.*

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Posteroanterior chest x-ray showing marked left ventricular enlargement.

The first and second heart sounds were normal. A fourth heart sound was present at the apex.

Continuous pressure recording after isoproterenol infusion as the transseptal catheter was withdrawn from the position of catheter entrapment at the apex of the left ventricle (left), to the left ventricular (L.V.) inflow tract (center), to the left atrium (right). The low L.V. inflow tract pressure strongly suggested that the intraventricular pressure difference recorded at this time was not the result of obstruction to left ventricular outflow.

The electrocardiogram revealed evidence of left atrial and ventricular hypertrophy (fig. 2), and chest x-rays and fluoroscopy showed left atrial and ventricular enlargement (fig. 3). Routine blood studies were normal. Mild impairment of renal function was compatible with chronic

<table>
<thead>
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<th>Right heart pressures* (mm Hg)</th>
<th>Left heart pressures* (mm Hg)</th>
<th>Control</th>
<th>Isoproterenol</th>
<th>Nitroglycerin</th>
<th>After drug stimulation</th>
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</thead>
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<td>R.A. (mean)</td>
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<td>125-140</td>
<td>160-225</td>
<td>235-300</td>
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<tr>
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<td>5</td>
<td>17-25</td>
<td>22-28</td>
</tr>
</tbody>
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*Pressures recorded are systolic unless otherwise indicated.
Abbreviations: R.A. = right atrium; R.V.I.T. = right ventricular inflow tract; R.V.O.T. = right ventricular outflow tract; P.A. = pulmonary artery; R.V.E.D. = right ventricular end-diastolic; L.A. = left atrium; L.V. = left ventricular; I.V. = intraventricular; L.V.E.D. = left ventricular end-diastolic.

Hemodynamics Recorded During First Two Cardiac Catheterizations (December 8 and 15, 1966)
glomerulonephritis. The clinical findings suggested obstruction to right ventricular outflow and nonobstructive cardiomyopathy of the left ventricle.

A summary of the hemodynamic findings from the two heart catheterizations is shown in table 1. Dye curves and blood samples for oxygen content excluded the possibility of a left-to-right shunt. A constant pressure difference was recorded across the right ventricular outflow tract. There was no pressure difference across the left ventricular outflow tract at rest, but intraventricular pressure differences were recorded after the administration of isoproterenol and

### Table 2

**Hemodynamics Recorded During Third Heart Catheterization (November 20, 1967)**

<table>
<thead>
<tr>
<th>Site</th>
<th>Control (mm Hg)</th>
<th>After methamphetamine (mm Hg)</th>
<th>Site</th>
<th>Control (mm Hg)</th>
<th>After methamphetamine (mm Hg)</th>
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<tbody>
<tr>
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<td>16</td>
<td>L.A. (mean)</td>
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<td>46-49</td>
<td>70-95</td>
<td>L.V.I.T.</td>
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<tr>
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<td>29-32</td>
<td>L.V.O.T.</td>
<td>130-190</td>
<td></td>
</tr>
<tr>
<td>P.A.</td>
<td>33-38</td>
<td>29-32</td>
<td>L.V. entrapment</td>
<td>200-350</td>
<td></td>
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<tr>
<td>Gradient</td>
<td>11-16</td>
<td>41-63</td>
<td>Aorta</td>
<td>130-190</td>
<td></td>
</tr>
<tr>
<td>R.V.E.D.</td>
<td>10</td>
<td>15-18</td>
<td>I.V. pressure difference</td>
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<td></td>
<td></td>
<td></td>
<td>L.V.E.D.</td>
<td>17-21</td>
<td></td>
</tr>
</tbody>
</table>

*Pressures recorded are systolic unless otherwise indicated.

Abbreviations are the same as in table 1, except for: L.V.I.T. = left ventricular inflow tract; L.V.O.T. = left ventricular outflow tract; I.V. = intraventricular.
Right heart pressure recordings under control conditions using the double-lumen right heart catheter with the end-hole in the pulmonary artery (P.A.), while the side-hole passes from the right atrium (R.A.) to the right ventricle (R.V.) between the third and fourth beats in the tracing. The first recorded ventricular pressure inside the tricuspid valve (initial right ventricular inflow tract pressure) is elevated above P.A. pressure, indicating that this pressure difference is the result of outflow obstruction and not due to catheter entrapment in the myocardium.

nitroglycerin (table 1). A pressure recording during withdrawal of the transseptal catheter from the apex of the left ventricle to the left atrium revealed that the left ventricular systolic pressure just inside the mitral valve (inflow tract pressure) was not elevated (fig. 4). The findings of a low inflow tract pressure strongly suggested that the pressure difference within the left ventricle resulted from catheter entrapment in the myocardium. Both the clinical and hemodynamic findings were, therefore, compatible with right ventricular outflow tract obstruction and a nonobstructive cardiomyopathy of the left ventricle.

The patient was discharged from hospital in January 1967 and advised to restrict her physical activity. In July 1967 she had two episodes of paroxysmal nocturnal dyspnea. She subsequently began experiencing retrosternal chest discomfort and a further decrease in her exercise tolerance. In November 1967 she was admitted to the Toronto General Hospital for reassessment. Results of physical examination and laboratory findings were unchanged. Combined right heart, retrograde aortic, and transseptal left heart catheterizations were carried out. A no. 8 Courand double-lumen catheter was used for the right-sided studies and an end-hole transseptal catheter was utilized to assess the left ventricular inflow tract pressure.

Figure 6

Continuous aortic pressure recording while the end-hole transseptal catheter (outlined by dashed lines in lower part of figure) is withdrawn from just inside the mitral valve (left ventricular inflow tract) (left) to the left atrium (right). There is no systolic pressure difference between the inflow tract of the left ventricle and the aorta.

Figure 7

The results of the heart catheterization studies are shown in table 2 and figures 5 to 8. At the beginning of the procedure there was a subvalvular systolic pressure difference of 11 to 16 mm Hg across the right ventricular outflow tract (fig. 5). All right ventricular systolic pressures proximal to the outflow obstruction including that just inside the tricuspid valve (initial right ventricular inflow tract pressure) were elevated (fig. 6). Immediately following the introduction of the transseptal catheter into the left atrium the patient had a severe vasovagal attack associated with hypotension, bradycardia, hyperventilation, sweating, nausea, and vomiting. Methamphetamine (Methedrine, 25 mg) and atropine (4 mg) were administered intravenously. Considerable improvement ensued, but the patient’s condition precluded drug studies or cineangiograms.

Following the administration of methamphetamine the systolic pressure difference across the right ventricular outflow tract increased to 41 to 63 mm Hg (fig. 5). At this time there was no pressure difference between the left ventricular inflow tract and the aorta (fig. 7). When both
ventricular cardiomyopathy, the left intraventricular pressure difference being due to catheter entrapment in the myocardium.

**Discussion**

The demonstration that two types of intraventricular pressure difference (subvalvular obstruction to right ventricular outflow and catheter entrapment by left ventricular myocardium) may occur in the same patient is believed of practical significance. Of considerable importance is the differentiation of this situation from that encountered in patients with muscular subaortic stenosis. In the latter condition, intraventricular pressure gradients may occur in both ventricles, both gradients resulting from obstruction to ventricular outflow. In muscular subaortic stenosis all left ventricular pressures proximal to the obstruction, including the initial inflow tract pressure, are elevated above the systolic pressure in the outflow tract distal to the stenosis.1-3 When a left intraventricular pressure difference results from catheter entrapment in the myocardium, as in the present case, the initial inflow tract pressure is not elevated and is precisely equal to the outflow tract pressure, as well as to all other truly intracavitary pressures. Only the catheter entrapmed in the myocardium records an elevated ventricular systolic pressure.1-7 By using initial inflow tract pressure recordings, plus ancillary observations,1,3 it is possible to differentiate an intraventricular pressure difference due to catheter entrapment in myocardium from that due to subvalvular outflow tract obstruction in either the left or right (fig. 6) ventricles.

In patients with muscular subaortic stenosis the murmur is usually maximal at, or just medial to, the left ventricular apex, although a separate murmur may be noted in the pulmonary area in the presence of obstruction to right ventricular outflow.8 In the present case the murmur and thrill arising from the obstruction to right ventricular outflow was maximal in the pulmonary area. The murmur at the left ventricular apex was faint and could have been due to radiation of the murmur from the pulmonary area, or to the

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**Figure 8**

In the left panel both the retrograde aortic and transseptal catheters are located in the left ventricular inflow tract (L.V.I.T.) where they record virtually identical pressures. In the center and right panels the elevated ventricular systolic pressure is recorded by the transseptal catheter, which has been advanced toward the left ventricular apex and has become entrapped in the myocardium during systole, as evidenced by the fact that the elevated pressure at times falls after the L.V.I.T. (intracavitary) pressure (right). Blood could not be withdrawn from the entrapped transseptal catheter in systole (see text).

The retrograde aortic and transseptal catheters were positioned in the left ventricular inflow tract, they recorded virtually identical pressures (fig. 8, left). When the transseptal catheter was advanced toward the left ventricular apex, an elevated systolic pressure was recorded, and an intraventricular pressure difference existed between the pressure recorded by the transseptal catheter near the apex of the left ventricle and the pressure recorded in the inflow (or outflow) tract of the left ventricle via the retrograde catheter (fig. 8, center). With the transseptal catheter in this position, the elevated ventricular pressure frequently declined after the inflow tract pressure (fig. 8, right), blood could not be withdrawn from the proximal end of the catheter in systole (because the tip was entrapped in muscle), and the catheter tip ceased to move freely (as it had in the inflow tract).

The mean left atrial pressure and left ventricular end-diastolic pressures were elevated (table 2), and dye curves done by injecting indocyanine green into the left ventricle and sampling in the left atrium showed a trivial amount of mitral insufficiency. The cardiac output, measured by the Fick technique, was 3.0 L/min/m² of body surface area. These hemodynamic findings were believed to confirm that this patient had muscular subvalvular obstruction to right ventricular outflow and a nonobstructive left

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trivial degree of mitral insufficiency that was present. Thus, although there was gross left ventricular hypertrophy and enlargement (figs. 2 and 3), the murmur over the apex of this chamber was insignificant compared with that in the pulmonary area.

The systemic hypertension in the present case was believed secondary to chronic glomerulonephritis. The hypertension could have contributed to the degree of left ventricular hypertrophy present but was not believed the sole cause, since cardiac enlargement was noted within 2 years of the attack of acute nephritis. During isoproterenol or nitroglycerin stimulation of an intraventricular pressure difference in this patient, the initial inflow tract pressure was not elevated (fig. 4), indicating the absence of outflow tract obstruction, even with appropriate drug stimulation. The presence of hypertension was, therefore, not felt to be preventing obstruction to the left ventricular outflow tract in this patient.

References
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