Muscular Subaortic Stenosis

Initial Left Ventricular Inflow Tract Pressure in the Assessment of Intraventricular Pressure Differences in Man

By E. Douglas Wigle, M.D., Yves Marquis, M.D., and Pierre Auger, M.D.

SUMMARY

Two types of intraventricular pressure difference within the left ventricle of man are described. The first type was encountered in eight consecutive patients with muscular subaortic stenosis in whom the outflow tract pressure distal to the stenosis was low and equal to the aortic systolic pressure, whereas all ventricular pressures proximal to the stenosis, including that just inside the mitral valve (the initial inflow tract pressure) were high. The second type was encountered in five of 10 patients with nonobstructive cardiomyopathy when a cardiac catheter was advanced to the left ventricular wall where it became entrapped or imbedded in cardiac muscle in systole and recorded a high ventricular pressure that was believed to reflect subendocardial intramyocardial tissue pressure. In this second type of intraventricular pressure difference, the initial inflow tract pressure, as well as all truly intracavitary pressures, were low and equal to the aortic systolic pressure.

By defining two types of intraventricular pressure difference, the recent controversy as to the nature of such pressure differences in man may be resolved, and the obstructive nature of muscular subaortic stenosis reaffirmed.

Additional Indexing Words:
Cardiomyopathy
Hypertrophic subaortic stenosis

Intraventricular Pressure difference of the left ventricle may be defined as that state in which the systolic pressure in one portion of the ventricle exceeds the systolic pressure in another portion of this chamber. Characteristically, intraventricular pressure differences are encountered in patients with subaortic stenosis (muscular or fibrous) in which the systolic pressure in the left ventricle proximal to the stenosis exceeds the systolic pressure distal to the stenosis (and proximal to the aortic valve) (fig. 1 left). In the present considerations we are not concerned with the intraventricular pressure difference caused by fibrous subaortic stenosis.

Intraventricular pressure differences have also been recorded in the normal1-13 and hypertrophied14-17 canine left ventricle at rest,1-4 and during administration of agents with a positive inotropic effect on the myocardium,6, 7, 9, 11, 12, 14, 15 stellate ganglion stimulation,13 or hemorrhagic shock.5, 8, 10 A number of authors6, 7, 9, 14 have suggested that these pressure differences were also the result of muscular obstruction to left ventricular outflow (muscular subaortic stenosis). More recently,
Figure 1

(Left) In muscular subaortic stenosis because the obstruction to left ventricular outflow (arrow) is caused by systolic apposition of the ventricular septum and anterior leaflet of the mitral valve, the intraventricular pressure distal to the stenosis (and proximal to the aortic valve) is low (+), whereas all ventricular pressures proximal to the stenosis including the one just inside the mitral valve (the inflow tract pressure) are elevated (++). (Right) When an intraventricular pressure difference is recorded due to catheter entrapment from cavity obliteration, the elevated ventricular pressure is recorded only in the area of entrapment (++). The intraventricular systolic pressure in all other areas of the left ventricular cavity, including that in the inflow tract just inside the mitral valve is low (+) and equal to aortic systolic pressure. The three areas of the left ventricle represented by the +’s in each of these diagrams are, from above downward, the outflow tract just below the aortic valve (subaortic region), the inflow tract just inside the mitral valve, and the left ventricular apex.

however, a growing amount of evidence has accumulated to indicate that intraventricular pressure differences in dogs are not the result of obstruction to left ventricular outflow, but rather are consequent upon the catheter recording the high ventricular pressure being entrapped or imbedded in cardiac muscle in systole.8, 10–15 The tip of a catheter that is located at the left ventricular apex may become entrapped in cardiac muscle when this portion of the ventricle becomes devoid of blood (obliterated) in systole,8, 10, 11 Martin8, 10 and White11 and their associates have demonstrated this phenomenon by cineangiography in dogs in hemorrhagic shock8, 10 and during infusion of isoproterenol.11 Others have demonstrated that a cardiac catheter may become entrapped in trabeculae carneae of the left ventricle other than at the apex.12, 13, 16 Studies carried out in this laboratory15 demonstrated that when intraventricular pressure differences due to catheter entrapment occurred in dogs during infusion of norepinephrine, an elevated systolic pressure was recorded at the apex of the left ventricle, but the ventricular systolic pressure in the inflow tract was low and equal to the systolic pressure in the outflow tract and aorta (fig. 1 right). This latter situation (fig. 1 right) is incompatible with obstruction of the outflow tract (fig. 1 left).

Recently, it has been suggested that the intraventricular pressure difference in patients with muscular subaortic stenosis may not be the result of obstruction to left ventricular outflow but rather may be the result of catheter entrapment.19 Having demonstrated in dogs that the left ventricular inflow tract pressure was not elevated in the presence of an intraventricular pressure difference due to catheter entrapment15 (fig. 1 right) and knowing that it should be elevated and equal to the left ventricular apex pressure in muscular subaortic stenosis (fig. 1 left) we considered assessment of left ventricular inflow tract pressure in patients diagnosed clinically to have muscular subaortic stenosis of great importance. This report describes the detailed results of such a study carried out in eight consecutively seen patients with muscular subaortic stenosis. The principles on which this work was based have been previously reported,15 as have similar studies carried out by Ross and associates.18

The purpose of this report is to demonstrate (1) that in muscular subaortic stenosis the inflow tract pressure is elevated above aortic systolic pressure and this portion of the left ventricle is blood-filled during systole and (2) that the position of intracardiac catheters when recording the intraventricular pressure difference in muscular subaortic stenosis locate the site of obstruction to the outflow tract of the left ventricle. In addition, the features of intraventricular pressure differences due to catheter entrapment in man, as
encountered in patients with nonobstructive cardiomyopathy, are described.

**Terminology and Methods**

**Terminology**

In this study the term “left ventricular inflow tract” refers to the area of the left ventricle just below the mitral valve. The outflow tract is the area below the aortic valve bound anteromedially by the ventricular septum and posterolaterally by the anterior leaflet of the mitral valve. The obstruction to left ventricular outflow in muscular subaortic stenosis is believed to be caused by systolic apposition of the ventricular septum and anterior leaflet of the mitral valve, that is, the obstruction is in the outflow tract. The apex of the left ventricle is the third area of this chamber referred to (fig. 1).

The *initial* left ventricular inflow tract pressure is the first ventricular pressure recorded on entering the left ventricle via the mitral valve with an *end-hole* transseptal catheter. The usual transseptal catheter has an end hole and four side holes in the centimeter proximal to the tip, and in crossing the mitral valve the pressure recorded is frequently partially atrial and partially ventricular in origin. By using an end-hole catheter, however, a sharp and well-defined pressure change occurs on entering the left ventricle from the left atrium.

The term, “catheter entrapment,” has been used to describe the situation wherein cardiac muscle enfolds a catheter situated in a portion of the ventricular cavity that is emptied of its blood content in systole (obliterated). For this situation to develop, a catheter must first be advanced toward the ventricular wall, and secondly this portion of the ventricle must empty in systole. It thus becomes a moot point whether one should speak of catheter entrapment (implying the heart muscle comes up to meet the catheter) or possibly catheter imbedding, implying that the catheter is advanced into the interstices (trabeculae carneae) of the ventricular wall to the point where it will become enfolded in cardiac muscle during systole. Because we feel both catheter advancement to the ventricular wall and cavity obliteration may play a variable role from case to case, or from time to time in any one case, we have chosen to use the terms “catheter imbedding” and “catheter entrapment” interchangeably in this report.

**Group Studies**

The eight patients with muscular subaortic stenosis on whom these studies were carried out were diagnosed clinically as having this condition by currently accepted criteria.

In addition, 10 patients diagnosed clinically as having nonobstructive cardiomyopathy underwent hemodynamic investigation.

**Methods**

All 18 patients underwent combined retrograde aortic and transseptal left heart catheterization. Left ventricular cineangiograms were obtained in at least one projection in every instance. In six of the eight patients with muscular subaortic stenosis left ventricular-aortic pressure differences were recorded during the left ventricular cineangiograms to ascertain whether or not the catheter recording the high ventricular pressure remained within the opacified portion of the ventricle during systole.

The end-hole transseptal catheter was used in each patient with muscular subaortic stenosis to assess the initial left ventricular inflow tract pressure. This catheter was passed from left atrium to left ventricle and vice versa up to 10 or more times in each direction. When this part of the study was completed, this catheter was replaced by an ordinary transseptal catheter by leaving the spring guide wire in the left atrium in order to change the catheters without having to puncture the atrial septum a second time. In three of the patients with muscular subaortic stenosis, two retrograde arterial catheters were introduced percutaneously. The first of these catheters had both end and side holes, while the second had only an end hole. The latter catheter was used with the transseptal catheter to ascertain the intracardiac position of catheters when recording inflow tract and outflow tract pressures. These catheter positions were photographed on numbered 70-mm film and the pressure recordings in these catheter positions were correspondingly numbered.

In the 10 patients with nonobstructive cardiomyopathy accurate assessment of the initial left ventricular inflow tract pressure was also carried out. In addition the transseptal catheter was advanced to the left ventricular wall in order to ascertain whether this catheter would become entrapped or imbedded in cardiac muscle in systole. Catheter positions in these patients were similarly photographed on numbered 70-mm film to correspond with the various intraventricular pressures recorded.

**Results**

**Muscular Subaortic Stenosis**

Figures 2 to 5 demonstrate the findings in the eight cases of muscular subaortic stenosis.
MUSCULAR SUBAORTIC STENOSIS

Figure 2A

Figure 2 A, B, and C are from the same surgically proven case of muscular subaortic stenosis. (A) The aortic pressure recording was continuous, while the end-hole transseptal catheter was repeatedly introduced into, and withdrawn from, the left ventricle via the mitral valve. On passing the catheter from left atrium to left ventricle and vice versa, the first ventricular pressure on entering the left ventricle (the initial inflow tract pressure) and the last ventricular pressure on withdrawal from the left ventricle were elevated above aortic systolic pressure.

Figure 2B

Catheter placement and corresponding intracardiac pressures in the same case of muscular subaortic stenosis as figure 2A. The left panel indicates the position of the transseptal catheter just proximal to the mitral valve when recording left atrial pressure. The position of the aortic catheter is also shown. The center panel indicates the position of the transseptal catheter when recording the elevated left ventricular inflow tract pressure just inside the mitral valve. The right panel shows that advancing the transseptal catheter further into the left ventricular cavity does not alter the ventricular pressure. The catheters in this and succeeding catheter placement pictures have been outlined with dashed lines for clarity of illustration.
Without exception in the eight cases, no matter how often the end-hole transseptal catheter traversed the mitral valve, the first recorded pressure on entering the left ventricle (the initial inflow tract pressure) and the last recorded pressure on withdrawing from the left ventricle were elevated above the systolic pressure in the aorta or in the outflow tract of the left ventricle distal to the stenosis (figs. 2A, 3A, and 4). It is considered an anatomic impossibility for an end-hole transseptal catheter to become entrapped in cardiac muscle when recording the initial inflow tract pressure on entering the left ventricle or the last pressure on being withdrawn from the ventricle. This belief that the elevated inflow tract pressure was not the result of catheter entrapment was enhanced by the fact that when the end-hole transseptal catheter was withdrawn from the left ventricle during systole, the last recorded portion of ventricular pressure was elevated above aortic systolic pressure (fig. 3A lower right) or

**Figure 2C**

In the left panel, the end-hole retrograde catheter records the left ventricular outflow tract (L.V.O.T.) pressure distal to the stenosis, while the end-hole transseptal catheter records the elevated left ventricular inflow tract (L.V.I.T.) pressure proximal to the stenosis. Note that these two ventricular pressures decline simultaneously. In the right panel the inflow tract pressures recorded via each of these catheters were precisely superimposed, providing strong evidence that the elevated ventricular pressures were being recorded from a blood-filled, high pressure area of the ventricle. The systolic pressure difference measured between the two catheters in the left panel or between the position of the retrograde catheter in the left and right panels locate the obstruction to the area of the outflow tract of the left ventricle.
Muscular subaortic stenosis above the outflow tract pressure distal to the stenosis (fig. 4).

Figures 2B and 5 demonstrate the end-hole transseptal catheter position just proximal to the mitral valve, when left atrial pressure was being recorded and the position of this catheter when the elevated left ventricular inflow tract pressure was being recorded. It can be seen that this transseptal catheter is just inside the mitral valve while recording this latter pressure. Figures 2B and 5 also demonstrate that the elevated ventricular pressure remains unchanged when the transseptal catheter is advanced further into the left ventricular cavity. With the transseptal catheter in these positions (figs. 2B, 3B, and 5), blood could be withdrawn from the proximal end of the catheter in systole as well as in diastole. If the proximal end of the catheter were left open, a jet of blood shot out in systole. The tip of the transseptal catheter could be seen to move freely during the different phases of the cardiac cycle, and when radiopaque dye was manually injected through the catheter, the dye was immediately ejected from the ventricle. These observations provide evidence that the catheter tip was lying free in a blood-filled area of the left ventricle in systole and

---

**Figure 3A**

Another surgically proven case of muscular subaortic stenosis. Continuous aortic pressure recordings while the end-hole transseptal catheter entered the left ventricle from the left atrium (top) and was withdrawn from the left ventricle to the left atrium (bottom) in diastole (left) or in systole (right). The inflow tract pressure just inside the mitral valve was invariably elevated above aortic systolic pressure. The flattened transseptal pressure recordings in the beat prior to entering the ventricle (top) were believed the result of closure of the mitral leaflets on the end of this catheter. Note the ventricular pressure declined at the time of the dicrotic notch in aortic pressure.

*Circulation, Volume XXXV, June 1967*
was not entrapped in cardiac muscle when recording these elevated left ventricular systolic pressures. The elevated left ventricular systolic pressure declined at or before the dicrotic notch in aortic pressure or simultaneously with the lower ventricular pressure recorded in the outflow tract distal to the site of stenosis (figs. 2 to 5).

Figures 2C, 3B, and 5 demonstrate the positions of the end-hole retrograde catheter when recording the outflow tract pressure distal to the stenosis and when recording an elevated inflow tract pressure identical to that recorded by the transseptal catheter. The site of the change in systolic pressure and hence the obstruction causing this difference in pressure must lie between these two positions of the retrograde catheter or between the transseptal catheter position and the retrograde catheter position, when the latter records the outflow

---

**Figure 3B**

*Same case as figure 3A. Simultaneous aortic and left ventricular outflow tract and inflow tract pressure recordings (top center) with corresponding retrograde and transseptal catheter positions in the posteroanterior (top left) and left anterior oblique (top right) views. The aortic catheter is just at the aortic valve. In the lower half of the figure, both the end-hole retrograde and transseptal catheters are in the inflow tract, their positions being shown in the posteroanterior (left) and left anterior oblique (right) views. These pictures of catheter placement, with their corresponding pressure recordings, locate the site of the intraventricular pressure difference, and hence the obstruction, to the outflow tract of the left ventricle. Note that inflow tract pressure declined simultaneously with outflow tract pressure or with the dicrotic notch in aortic pressure. The dashed lines outline the silhouette of the left ventricle.*

_Circulation, Volume XXXV, June 1967_
MUSCULAR SUBAORTIC STENOSIS

tract pressure distal to the stenosis. In each of these figures, whether viewed in the posterolateral (figs. 2C, 3B, and 5) or left anterior oblique (fig. 3B) projections, the site of the systolic pressure difference and hence the obstruction is located in the outflow tract of the left ventricle. The fact that two (or more) catheters recorded an identically elevated left ventricular inflow tract pressure (figs. 2C, 3B, and 5) and that the transseptal catheter could be moved about in this same area without altering the pressure recorded (figs. 2B and 5) provided strong additional evidence that a high pressure, blood-filled cavity existed in the inflow tract area of the left ventricle throughout systole in muscular subaortic stenosis. When a catheter was situated in this area during left ventricular cineangiography, it remained within the angiographic silhouette while recording an elevated left ventricular systolic pressure.

Nonobstructive Cardiomyopathy

In all 10 patients in this group the transseptal catheter was advanced to the left ventricular wall to ascertain whether this positioning would result in this catheter becoming entrapped in cardiac muscle in systole. In five of the 10 patients left ventricular systolic pressure became elevated when the catheter was in contact with left ventricular myocardium (figs. 6 and 7). In one of these five cases, elevated left ventricular systolic pressure was readily recorded with catheter advancement toward the ventricular wall. In this case cineangiographic obliteration of the left ventricular apex was marked in systole. In the other four cases in which cineangiography did not demonstrate a great degree of systolic obliteration of the left ventricular cavity, the transseptal catheter had to be pushed against the ventricular wall in order to record an elevated systolic pressure from imbedding of the catheter in the myocardium (fig. 7).

In these cases when an elevated left ventricular systolic pressure was recorded from the transseptal catheter being imbedded or entrapped in cardiac muscle in systole, the initial left ventricular inflow tract pressure was invariably low and equal to the systolic pressure in the outflow tract and aorta (figs. 1, 6, and 7). The elevated left ventricular pressure from catheter entrapment usually declined after the dicrotic notch in aortic pressure (figs. 6A and B, and 7) or after the left ventricular cavity pressure (fig. 6C). Blood could not be withdrawn from the proximal end of the entrapped catheter in systole providing evidence that the tip was surrounded

Figure 4

Continuous recording, in a case of muscular subaortic stenosis, of left ventricular outflow tract (L.V.O.T.) pressure, while an end-hole transseptal catheter was withdrawn from the left ventricular inflow tract (L.V.I.T.) to left atrium (L.A.) in mid-systole. Note that L.V.O.T. and L.V.I.T. pressures decline simultaneously.
by muscle not blood. If the proximal end of the catheter were left open, blood did not come out in systole. Cinefluoroscopy of the catheter revealed that the tip did not move freely during the cardiac cycle but appeared relatively immobile from being entrapped in muscle. Manual injection of 3 to 6 ml of radiopaque dye through the catheter in one case resulted in injection of some of the dye into the myocardium.

As is evident from figure 6A and C, the degree of catheter entrapment determined the degree of elevation of left ventricular systolic pressure, as well as the delay in decline in this pressure, both on advancing (fig. 6A) and on withdrawing (fig. 6C) the catheter from the left ventricular wall. It was possible to record bizarre intraventricular pressures by pushing the transseptal catheter against the left ventricular wall too forcefully. On withdrawing the entrapped catheter from the wall, one could feel a definite tug on the catheter in systole, and when a sharp pressure change occurred on withdrawing the catheter from the entraped position (fig. 6B), a feeling that the catheter was "snapping" back into a free intracavitary position was experienced. As indicated in fig. 7, a catheter need not be at the left ventricular apex to become entrapped in cardiac muscle in systole.

![Catheter placement pictures and corresponding pressure recordings from a case of muscular subaortic stenosis.](http://circ.ahajournals.org/)

**Figure 5**

*Catheter placement pictures and corresponding pressure recordings from a case of muscular subaortic stenosis. In the left panel both the end-hole transseptal and retrograde catheters are in the inflow tract proximal to the stenosis. In the second panel the retrograde catheter has been withdrawn to the outflow tract distal to the stenosis. In the third panel the retrograde catheter is unchanged in position, but the end-hole transseptal catheter has been withdrawn to being just inside the mitral valve. In the fourth panel the transseptal catheter records left atrial pressure just proximal to the mitral valve. Note that all ventricular pressures decline simultaneously and that the inflow tract pressure is equally elevated whether recorded just inside the mitral valve (third panel), further in the ventricle (second panel) or by two different catheters (first panel).*
Having demonstrated that catheter entrapment may occur in nonobstructive cardiomyopathy, we attempted to imbed the transseptal catheter in cardiac muscle in several cases of muscular subaortic stenosis. Figure 8 demonstrates a continuous pressure recording in such an instance using an end-hole transseptal catheter. In the first three cardiac cycles (fig. 8 top) the catheter is entrapped in cardiac muscle and the "entrapped" pressure tracing falls after the outflow tract pressure distal to the stenosis. From the fourth cardiac cycle the transseptal catheter records left ventricular cavity pressure proximal to the stenosis as the catheter is withdrawn into the left atrium. This figure demonstrates that catheter entrapment is possible in muscular subaortic stenosis. However, the elevated inflow tract pressure (fig. 8) provides evidence that the intraventricular pressure difference is not due to catheter entrapment, but rather to outflow tract obstruction.

The act of inspiration has been shown to decrease or abolish the intraventricular pressure difference in muscular subaortic stenosis \(^{28}\) (fig. 9 top). When recording an intraventricular pressure difference due to catheter entrapment, inspiration resulted in either an

**Figure 6A**

*Figure 6A, B, and C shows intraventricular pressure difference due to catheter entrapment in a patient with nonobstructive cardiomyopathy in whom cineangiography demonstrated marked systolic emptying of the left ventricular apex with cavity obliteration.*

*In the left panel (above) with the transseptal catheter in the inflow tract just inside the mitral valve, there was no systolic pressure difference between the left ventricle (L.V.) and the aorta. In the center panel with the transseptal catheter advanced to the L.V. wall, there was a small late systolic pressure difference between the left ventricle and the aorta. With further advancement of the transseptal catheter to the L.V. apex (right panel) a large left ventricular-aortic systolic pressure difference developed due to entrapment of the transseptal catheter at the left ventricular apex. Note that left ventricular pressure declined after the dicrotic notch in aortic pressure. The elevated left ventricular systolic pressure was believed to reflect, to a variable degree, subendocardial intramyocardial tissue pressure (see text).*
increase in this pressure difference (fig. 9 bottom) or the recording of bizarre, obviously factitious, pressures, presumably from excessive imbedding of the catheter in the left ventricular wall.

**Discussion and Conclusions**

These studies are believed to provide evidence that two types of pressure difference may be encountered in the left ventricle of man. The first type was encountered in patients with muscular (or fibrous) subaortic stenosis in whom the ventricular systolic pressure distal to the stenosis (and proximal to the aortic valve) was low and equal to aortic systolic pressure, whereas all ventricular pressures proximal to the site of stenosis including that just inside the mitral valve were elevated (fig. 1 left). In patients with muscular subaortic stenosis, when an end-hole transseptal catheter was repeatedly drawn back and forth across the mitral valve, the first ventricular pressure on its entrance into the left ventricle (the initial inflow tract pressure) and the last ventricular pressure on its withdrawal from the left ventricle were elevated above the systolic pressure in the aorta or in the subaortic region of the ventricle.

![Figure 6B](http://circ.ahajournals.org/)

**Figure 6B**

Catheter entrapment in same case of nonobstructive cardiomyopathy as figure 6A and C. In the left panel (control) a small left ventricular-aortic systolic pressure difference was present with the transseptal catheter imbedded (entrapped) in cardiac muscle in a different position from that shown in figure 6A. With development of a spontaneous tachycardia (right panel) the pressure difference increased dramatically, presumably from greater systolic emptying of the left ventricle with a resulting increase in catheter entrapment. When the transseptal catheter was withdrawn to the left ventricular inflow tract just inside the mitral valve, the systolic pressure difference between the left ventricle and aorta vanished, that is, the inflow tract pressure was not elevated, and hence the pressure difference was not caused by obstruction to left ventricular outflow. When withdrawing the transseptal catheter, one could feel a systolic tug on the catheter until it snapped back into the inflow tract (see text). A similar left ventricular apex to inflow tract withdrawal pressure recording was obtained during infusion of isoproterenol with identical results.
MUSCULAR SUBAORTIC STENOSIS

is believed to be an anatomic impossibility for such an end-hole catheter to become entrapped in cardiac muscle when recording the initial left ventricular inflow tract pressure in this manner.

In muscular subaortic stenosis blood could be withdrawn from the proximal end of the transseptal catheter recording the elevated inflow tract pressure in both systole and diastole. If the proximal end of the catheter was left open, blood shot out of this end during systole. Cineangiograms revealed that the catheters recording the elevated inflow tract pressure remained within the angiographic silhouette during systole. The elevated ventricular pressure fell at or before the dicrotic notch in aortic pressure or simultaneously with the lower ventricular pressure distal to the stenosis. When more than one catheter was positioned proximal to the stenosis, identically elevated ventricular pressures were recorded, and these catheters could be moved about in the inflow tract without altering the pressure recorded. These observations in patients with muscular subaortic stenosis are believed to provide evidence that the elevated left ventricular systolic pressure in this condition was recorded from a high pressure, blood-filled area of the left ventricle proximal to the stenosis. The outflow tract of the left ventricle was identified as the site of the obstruction by the location of the intracardiac catheters at the time of recording the intraventricular pressure gradient. This site corresponded to the site of septal encroachment on the outflow tract visualized during left ventricular cineangiography.

The second type of intraventricular pressure difference in man was encountered when an elevated left ventricular systolic pressure was recorded as a consequence of a catheter

Figure 6C

Same case of nonobstructive cardiomyopathy as figures 6A and B. Simultaneous recordings of left ventricular cavity pressure and the pressure recorded via a transseptal catheter entrapped at the cardiac apex (left) as the latter catheter was withdrawn to a free intracavitary position in the inflow tract just inside the mitral valve (right). With the transseptal catheter entrapped in the left ventricular wall (left), the pressure elevation occurred in late systole and this pressure declined after the intracavitary pressure. When both catheters were free in the cavity (right), the pressures recorded by them rose and declined simultaneously. On withdrawal of the entrapped transseptal catheter, the recorded pressure declined gradually, whereas in figure 6B the transition from the catheter being entrapped in muscle to being free in the cavity was abrupt.

Circulation, Volume XXXV, June 1967
being entrapped or imbedded in cardiac muscle in systole. Under these circumstances the inflow tract pressure was low and precisely equal to the systolic pressure in the outflow tract as well as to the aortic systolic pressure. Blood could not be withdrawn in systole from the proximal end of the catheter recording this elevated pressure because the tip was surrounded by muscle (not blood). The elevated ventricular systolic pressure in these circumstances frequently declined after the dicrotic notch in aortic pressure or following intracavitary pressure. Catheters recording an elevated ventricular systolic pressure due to entrapment in muscle could not be moved without altering the degree of entrapment and hence the recorded pressure. Catheter entrapment most readily occurred when there was exaggerated systolic emptying of the left ventricle with resultant cavity obliteration (fig. 1 right). To become entrapped, a catheter must be advanced toward, or to, the ventricular wall. Although this entrapment phenomenon was demonstrated in patients with nonobstructive cardiomyopathy in this study, we believe it may occur in normal hearts or in other cardiac conditions. Intraventricular pressure differences due to catheter entrapment in man and in dogs during pharmacological stimulation of the left ventricle have in common a low left ventricular inflow tract pressure.

The reason for an entrapped or imbedded catheter recording an elevated systolic pressure is not entirely clear. It would appear entirely possible, however, that a catheter entrapped in left ventricular muscle would reflect, to a variable degree, subendocardial intramyocardial tissue pressure which has been demonstrated to exceed intracavitary or aortic systolic pressure, and to decline after

![Catheter placement pictures and intracardiac pressure recordings from a patient with nonobstructive cardiomyopathy. Transseptal catheter positions when recording left atrial pressure (left panel), left ventricular inflow tract pressure (second panel), and when the catheter recorded an elevated left ventricular systolic pressure from being entrapped in cardiac muscle (third and fourth panels). The catheter position was essentially the same when recording the pressures in the third and fourth panels, but in the fourth panel the catheter has been pushed more against the left ventricular wall and the ventricular pressure falls after the dicrotic notch in aortic pressure. Even when a catheter became imbedded in muscle some distance from the left ventricular apex (third and fourth panels), a low inflow tract pressure could be recorded (second panel).](image_url)
intracavitary pressure or following the dicrotic notch in aortic pressure.13, 29, 30

By reference to figure 1 it can be appreciated that the differentiation of an intraventricular pressure difference due to muscular subaortic stenosis from that due to catheter entrapment may not be possible on using a left ventricular apex to aortic withdrawal pressure recording. In each of these two situations such a withdrawal would record a high, then a low intraventricular pressure, and subsequently aortic pressure. The recording of initial left ventricular inflow tract pressure, as herein described does, however, permit one to distinguish between the two types of intraventricular pressure differences (fig. 1).

The problem in clinical diagnosis presented by a patient with evidence of left ventricular

![Figure 8](http://circ.ahajournals.org/)

*Figure 8*

*Intraventricular pressure recordings in a case of muscular subaortic stenosis during infusion of isoproterenol (1 µg/min). The left ventricular outflow tract (L.V.O.T.) pressure distal to the stenosis was recorded continuously. In the first three cardiac cycles (top left) the end-hole transseptal catheter was imbedded in the left ventricular wall (note late decline in recorded pressure). From the fourth cycle in the top recording, the transseptal catheter recorded left ventricular cavity pressure proximal to the stenosis; the pressure was elevated above, and declined simultaneously with, the L.V.O.T. pressure. The last recorded pressure from the inflow tract (L.V.I.T.) prior to the transseptal catheter entering the left atrium (L.A.) was elevated above the L.V.O.T. pressure. When the end-hole transseptal catheter was imbedded in the myocardium, the top of the recorded pressure was frequently flattened (top left). It is possible that if a catheter with end and side holes had been used, the imbedded pressure recording might have exceeded left ventricular cavity pressure proximal to the stenosis.*

*Circulation, Volume XXXV, June 1967*
hypertrophy and an apical systolic ejection murmur may indeed be difficult, as it was in several of the patients in both groups herein reported. Usually the problem is that of differentiating muscular subaortic stenosis from a nonobstructive cardiomyopathy with secondary mitral insufficiency or from the hyperkinetic heart syndrome. In either of the latter conditions an intraventricular pressure difference due to catheter entrapment could be encountered if a cardiac catheter were advanced to the left ventricular wall or exaggerated systolic emptying of the left ventricle occurred or if both occurred. It is our present view that the accurate assessment of left ventricular inflow tract pressure will permit hemodynamic differentiation between muscular subaortic stenosis and the two nonobstructive cardiac conditions referred to.

In the present study and in that reported by

![Figure 9](http://circ.ahajournals.org/)

**Figure 9**

(Top) Simultaneous left ventricular inflow tract (L.V.I.T.) and aortic pressure recordings in a case of muscular subaortic stenosis demonstrating a decrease in the systolic pressure gradient with inspiration and an increase with expiration. (Bottom) Pressure recordings in a patient with nonobstructive cardiomyopathy via the transseptal catheter entrapped in left ventricular (L.V.) wall and via a retrograde catheter lying free in the left ventricular (L.V.) cavity. The act of inspiration (arrow) resulted in a marked increase in the intraventricular pressure difference due to catheter entrapment. This effect could be related to the anatomic alterations caused by inspiration that imbedded the catheter further into the ventricular wall or to physiological alterations causing a smaller systolic ventricular volume or to both. Expiration reversed this effect.
Ross and associates,18 during left ventricular cineangiography in muscular subaortic stenosis, a catheter situated in the left ventricular inflow tract remained within the angiographic silhouette in systole and diastole. Conversely, with intraventricular pressure differences due to catheter entrapment, the entrapped catheter was outside of the angiographic silhouette in systole.11,16 The latter was particularly notable when there was marked systolic emptying of the left ventricular apex and cavity obliteration. Figure 10 indicates that a catheter may be outside of the angiographic silhouette in systole and yet may record an elevated left ventricular systolic pressure that was not due to catheter entrapment. In this particular case (fig. 10), the patient had surgically proven valvular aortic stenosis with a left ventricular-aortic systolic pressure difference of 65 mm Hg. The fact that the catheter was outside of the angiographic silhouette in systole was merely a manifestation of the catheter position and degree of systolic emptying of the ventricle. Similarly, in muscular subaortic stenosis, if a catheter was advanced to the left ventricular apex, such a catheter might appear outside of the angiographic silhouette in systole, but this finding would not necessarily indicate that the intraventricular pressure difference was due to catheter entrapment. Emptying of the left ventricular apex in muscular subaortic stenosis may be particularly marked because of the late onset of mitral insufficiency, which almost invariably accompanies this type of outflow tract obstruction. Recent observations in this laboratory suggested that the mitral insufficiency in muscular subaortic stenosis was secondary to the outflow obstruction in that pharmacological abolition of the stenosis also abolished the mitral leak.25 We have also observed catheters outside the angiographic silhouette in systole in patients in whom no intraventricular pressure difference was recorded. These observations have led us to stress the importance of the initial left ventricular inflow tract pressure in the assessment of intraventricular pressure differences. The fact that Ross and associates18 have made identical observations using a polyethylene, rather than a transseptal end-hole catheter lends further support to the significance of inflow tract pressure measurements in the assessment of intraventricular pressure differences.

The differentiation of an intraventricular pressure difference due to muscular subaortic stenosis from that due to catheter entrapment is of obvious importance in selecting the proper therapy in these two conditions. The ventriculomyotomy operation has been demonstrated to be of value in cases of muscular subaortic stenosis33-35 but would not be indicated in patients with an intraventricular pressure difference due to catheter entrapment. The fact that this operation has invariably shortened left ventricular ejection time as well as reduced or abolished the pressure gradient in patients with muscular subaortic stenosis is believed to be incontrovertible evidence of the obstructive nature of the lesion.
of the lesion, as well as further evidence as to the effectiveness of this type of surgery.36

Acknowledgment
The authors would like to express their gratitude to Misses Jean McMeekan, B.Sc., Neive Gonzalez, and Rose Marie Cseplo, and to Mr. Donald Mills for their excellent technical assistance, to Mr. Frederick Lammerich of the Department of Art as Applied to Medicine, University of Toronto, and to the Department of Medical Photography, Toronto General Hospital.

References
MUSCULAR SUBAORTIC STENOSIS


The Fruit and the Seed

Everyone who enjoys thinks that the principal thing to the tree is the fruit, but in point of fact the principal thing to it is the seed. Herein lies the difference between them that create and them that enjoy.—FRIEDRICH WILHELM NIETZSCHE, Maxims.
Muscular Subaortic Stenosis: Initial Left Ventricular Inflow Tract Pressure in the Assessment of Intraventricular Pressure Differences in Man
E. DOUGLAS WIGLE, YVES MARQUIS and PIERRE AUGER

Circulation. 1967;35:1100-1117
doi: 10.1161/01.CIR.35.6.1100

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1967 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/35/6/1100

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/