Pulmonary Valve Obstruction During Cardiac Catheterization

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The clinical and hemodynamic responses to acute pulmonary valve obstruction during cardiac catheterization in patients with severe pulmonary valve stenosis are presented to illustrate the serious nature of this complication.

Obstruction of a stenotic pulmonary valve by the exploring cardiac catheter, a serious and not infrequent complication of cardiac catheterization, has received little attention in the literature.1-4 Experiences in this laboratory indicate that pulmonary valve obstruction can occur both in a manifest, symptomatic form and in a concealed, asymptomatic form in patients with severe pulmonary stenosis. As catheterization studies are extended more generally to the infant age group and performed more often without anesthesia, the high incidence reported in this study may be more generally observed.

The purpose of this communication is to present clinical and physiologic observations during pulmonary valve obstruction and to derive from these a concept of the precarious hemodynamic status that exists in severe congenital pulmonary valvular stenosis.

Material and Methods

The data presented are derived from an analysis of 334 consecutive catheterization studies performed during an 18-month period in the Cardio-pulmonary Laboratory of the Children's Medical Center, Boston. Pulmonary valve stenosis with intact ventricular septum was considered to be present on the basis of clinical and catheterization findings in 31 of these 334 patients. The ages of the patients ranged from 8 months to 15 years.

Right heart catheterization was performed on patients of all ages under mild sedation. Parenteral premedication was administered approximately 1 hour before catheterization in a dosage of 1 ml. per 30 pounds of body weight (meperidine 25 mg., phenothiazine 0.25 mg., and chlorpromazine 6.25 mg. per ml.).

Multiple blood samples for oxygen analysis were obtained from the superior vena cava, right atrium, right ventricle, and pulmonary artery. Whenever possible blood samples were procured simultaneously from the right ventricle or pulmonary artery and the exposed brachial or femoral artery. A 5F (1.6 mm. diameter) or 6F (2.0 mm. diameter) Lehman catheter was used to obtain intracardiac blood samples and pressure measurements. The oxygen saturation and oxygen capacity were determined by the spectrophotometric method of Gordy and Drabkin.5 Pressure measurements were obtained with an elecromanometer (Sanborn) or strain-gage manometer (Statham P23D) and recorded on a direct-writing oscillograph (Sanborn). The oxygen consumption was determined from measurements of the oxygen content and volume of the expired gas. Systemic and pulmonary blood flows were calculated by means of the Fick principle, and the pulmonary valve area was estimated from the Gorlin formula.6 The calculated systemic and pulmonary blood flows and pulmonary valve areas are not necessarily accurate, since certain assumptions have been necessarily extended. The oxygen consumption, when not measured, has been estimated at 180 ml. per minute per M.7 for infants and children. Furthermore, it has been assumed that the oxygen consumption remains unchanged during the period that the catheter is in the pulmonary orifice. Finally, in the cyanotic patients the calculated pulmonary blood flow is a minimum value, it does not include any collateral bronchial blood flow, and, in fact, represents flow across the pulmonary valve.

Results

In the course of diagnostic cardiac catheterization studies on 31 patients with pulmonary stenosis and intact ventricular septum, 8 patients (25 per cent) presented definite evidence that the cardiac catheter was ob-
ARTERIAL SYSTEM

Fig. 1. Cardiovascular deterioration recorded 4 minutes after a 6F Lehman catheter was passed into the stenotic pulmonary valve of a 19-month-old infant.

Restricting blood flow through the pulmonary valve. Acute symptoms were experienced by 6 of these 8 patients when the cardiac catheter entered the stenotic pulmonary valve orifice.

Sudden restlessness was uniformly the earliest indication that the pulmonary orifice was obstructed and pulmonary blood flow reduced. Restlessness was rapidly followed by crying in infants and apprehension in older children. Increasing cyanosis was usually detected and bradycardia or tachycardia was often recorded. Finally, some neurologic reaction was frequently manifested, ranging from evanescent focal myotonic contractions in a single extremity to one instance of unilateral paresis of several days' duration. These reactions were observed between 4 and 13 minutes after the catheter had entered the pulmonary artery.

The acute, extreme cardiovascular deterioration that can occur is illustrated in figure 1. The first section of the tracing (left), obtained immediately before the catheter was advanced from the right ventricle into the pulmonary artery, shows normal cardiac rhythm, normal systemic blood pressure, and abnormally elevated right ventricular pressure. Approximately 4 minutes after the catheter had passed into the pulmonary artery the infant suddenly became restless and the previous mild cyanosis was observed to be intense. The catheter was immediately withdrawn to the right ventricle and the second section of the tracing (right) was recorded. The arrhythmia, bradycardia, and systemic hypotension persisted for several minutes and then the previous compensated hemodynamic status rapidly returned. This was the only instance of severe cardiac arrhythmia and cardiovascular deterioration that was observed.

Several hemodynamic changes were noted concurrent with pulmonary valve obstruction. The 31 patients with pulmonary valvular stenosis may be divided into 2 groups on the basis of the change in the oxygen saturation of the mixed venous blood before and after the cardiac catheter has entered the pulmonary orifice in each patient (fig. 2). It may be seen that no significant change in mixed venous blood oxygen saturation occurred in
PULMONARY VALVE OBSTRUCTION DURING CATHETERIZATION

FIG. 2. Changes in per cent of mixed venous blood oxygen saturation (ordinate) based on the analysis of blood samples obtained before (right ventricle) and after (pulmonary artery) the cardiac catheter has been manipulated through the pulmonary valve. Stippled area, mean ± 2 S.D. change of per cent saturation in the 22 patients with normal resting arterial saturation.

those patients with normal arterial saturation at rest (open circle). In marked contrast, a striking decrease in mixed venous blood oxygen saturation occurred after the catheter was manipulated into the pulmonary artery in 8 of the 9 patients with resting arterial unsaturation (solid circle) due to an interatrial right-to-left shunt.

Similar observations were made on the systemic arterial oxygen saturation. The 22 patients with normal resting arterial saturation showed no change, but the 9 patients with arterial unsaturation had significant decreases in arterial oxygen saturation in response to acute obstruction of the stenotic pulmonary orifice. The most marked response resulted in a fall of the mixed venous oxygen saturation (pulmonary artery) from 39 to

8 per cent and a simultaneous fall in systemic arterial saturation from 51 to 18 per cent.

The pulmonary valve blood flow in the 31 patients with pulmonary stenosis was calculated on the basis of mixed venous blood samples obtained from the right ventricular outflow tract before the catheter entered the pulmonary artery (fig. 3). As expected, the patients with normal resting arterial saturation (open circles) had a higher pulmonary valve blood flow (mean 4.8 L. per minute per M.²) than the group with resting arterial unsaturation (mean 2.1 L. per minute per M.²).

No evidence of valve obstruction and no
change in pulmonary valve blood flow were observed in any of the 22 patients with pulmonary stenosis and normal arterial saturation. In contrast, clinical or hemodynamic signs of pulmonary valve obstruction were observed in 8 of the 9 patients with resting arterial unsaturation, and once the catheter entered the pulmonary orifice the pulmonary valve blood flow was further decreased from values already considerably below normal (fig. 4).

In order to assess the quantitative significance of these observations the calculated pulmonary valve areas of the patients with catheter obstruction have been compared with the valve areas of the nonobstructed group and with normal pulmonary valve area measurements. The normal pulmonary valve area

was calculated from measurements of the heart valve circumference in infants and children succumbing to an acute noncardiac death. It may be seen from figure 5 that the pulmonary valve area increases from about 0.5 cm$^2$ at birth to about 4.0 cm$^2$ at age 16 years. The 8 patients with pulmonary stenosis who manifested obstruction of the pulmonary valve uniformly had valve areas less than 0.15 cm$^2$. These valve areas are smaller than the average newborn pulmonary valve area and even less than the premature infant valve area. The patients with pulmonary valvular stenosis, normal resting arterial saturation and no obstruction with the cardiac catheter, all had pulmonary valve areas of 0.2 cm$^2$ or greater (fig. 6).

**DISCUSSION**

In our experience pulmonary valve obstruction during diagnostic catheterization has been a serious and not infrequent complication. The following sequence of events probably occurs in a patient with severe pulmonary stenosis and an interatrial communication when the cardiac catheter, passing into the pulmonary valve, further significantly reduces the orifice area. The right ventricle, already functioning under an
extreme load in the presence of severe pulmonary stenosis, is unable to increase significantly the mean systolic ejection pressure and thus fails to maintain the pre-obstruction pulmonary valve blood flow. The right-to-left shunt already present at the atrial level now increases and results in a marked fall in arterial oxygen saturation, increased cyanosis, and perhaps a central nervous system response to the anoxia. In this manner, the central nervous system reactions observed may be similar to the familiar cyanotic spells observed in patients with tetralogy of Fallot.

The analysis presented has been restricted to pulmonary stenosis with intact ventricular septum. A similar reaction in pulmonary valve obstruction might be anticipated in other congenital heart lesions if a critical pulmonary valve or infundibular stenosis and a proximal right-to-left shunt were present.

Indeed, an episode of obstruction was observed in our laboratory in a 5-year-old patient with tetralogy of Fallot.

The inability of the right ventricle further to meet the demands caused by the catheter obstruction of the pulmonary valve can be analyzed in terms of the pressure gradients required to maintain an adequate blood flow through various valve areas. The vertical axis in figure 6 represents the mean systolic ejection pressure gradient, $\Delta P$ (expressed in mm. Hg), necessary to force a given stroke volume of blood through a given stenotic valve orifice.

Application of the Gorlin hydraulic formula indicates that an increase in right ventricular pressure must occur in order to maintain the same stroke volume after the pulmonary orifice has been narrowed by the exploring cardiac catheter. The calculated
The pulmonary valve areas of our patients with pulmonary stenosis are represented on the horizontal axis and range from 0.4 to 0.04 cm². If a 6F catheter (area of catheter tip is 0.03 cm²) is manipulated into a severely stenosed pulmonary valve of 0.10 cm² area or less, the right ventricle must provide a markedly increased pressure gradient (3 × ΔP and 4 × ΔP) to maintain the stroke volume at the pre-obstruction level. Thus, if a patient with a calculated pulmonary valve area of 0.4 cm² were to maintain the same right ventricular stroke volume before and after a 6F catheter was introduced into the valve, the mean systolic ejection pressure gradient across the pulmonary valve would increase from the observed 112 mm. Hg to the calculated 131 mm. Hg, a not improbable increase. If the same catheter were introduced into a stenotic pulmonary orifice of 0.06 cm², the mean systolic ejection pressure gradient would have to increase from the observed 73 mm. Hg to 291 mm. Hg, a highly improbable increase.

The relative significance of the obstructing area of the exploring catheter in different valve areas can be easily visualized. In figure 6 the large open circles represent the pulmonary valve areas drawn to scale and the central solid circles represent the area occupied by a 6F cardiac catheter. It appears significant that a correlation is evident between the theoretical, calculated mean systolic ejection pressure curves and the clinical and catheterization data of the 31 patients under analysis. The 8 patients who had pulmonary valve obstruction during cardiac catheterization are cyanotic at rest and have pulmonary valve areas (0.15 cm² or smaller), which correspond to the steeply rising portions of the pressure curve (fig. 6). In contrast, the patients with normal arterial saturation have valve areas (0.20 cm² or larger), which correspond to the more horizontal portion of the pressure curve, and this group manifested no evidence of pulmonary valve obstruction.

If the complications of pulmonary valve obstruction during cardiac catheterization are to be prevented, continuous clinical alertness and electrocardiographic observations are essential. Accurate oximetric monitoring of arterial saturation can provide an early warning of impending difficulties if anesthesia is utilized. Sudden restlessness, increasing cyanosis, or a significant change in the cardiac rate should lead without delay to withdrawal of the cardiac catheter from the stenotic pulmonary orifice.

**SUMMARY**

In the course of catheterization studies performed on 31 patients with pulmonary stenosis and intact ventricular septum, 6 (20 per cent) experienced acute symptoms and hemodynamic changes after the cardiac catheter had entered the stenotic pulmonary orifice. Two additional patients had only hemodynamic evidence that the cardiac catheter was obstructing blood flow through the pulmonary valve.

Pulmonary valve obstruction in our experience has occurred only when severe pulmonary stenosis has been associated with a right-to-left shunt. The 8 patients presented with clinical symptoms or hemodynamic findings of acute pulmonary valve obstruction all had arterial unsaturation at rest and calculated pulmonary valve areas of 0.15 cm² or less.

Sudden restlessness, increased cyanosis, and tachycardia occurring after the exploring cardiac catheter has entered a stenotic pulmonary orifice are early signs that the pulmonary blood flow has been reduced.

Continuous clinical alertness, oximetric monitoring of arterial saturation, and electrocardiographic observation are essential, particularly in patients with resting arterial unsaturation, if the complications of pulmonary valve obstruction during cardiac catheterization are to be prevented.

**SUMMARIO IN INTERLINGUA**

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tiones de obstruction del valvula pulmonar durante catheterisation cardia voе esser prevenite.

REFERENCES


The author presents an analysis of phlebograms recorded in various types of constrictive pericarditis. On the basis of the relative size of a and c waves, and the degree and the timing of systolic and diastolic collapse (x and y) waves, 4 different patterns can be distinguished that are attributable to predominant involvement of the right or left chambers by the constriction. The 4 basic patterns were found in particular in localized forms of pericardial constriction, while the more extensive processes cause combination patterns or curves modified by alterations attributable to venous congestion. Regression and recurrence of pericardial constriction is exemplified by serial phlebograms recorded in a patient with tuberculous pericarditis who temporarily was treated by cortisone.

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Circulation. 1958;18:53-59
doi: 10.1161/01.CIR.18.1.53

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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