Regression after Open Valvotomy of Infundibular Stenosis Accompanying Severe Valvular Pulmonic Stenosis

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Three patients operated upon under hypothermia for severe valvular pulmonic stenosis had right ventricular pressures in excess of 100 mm. Hg after open valvotomy. The residual obstruction, localized by pressure measurements to the subvalvular region, appeared to be due to greatly hypertrophied musculature in the outflow tract of the ventricle. Infundibular resection was not attempted. Electrocardiographic signs of right ventricular hypertrophy gradually disappeared, and cardiac catheterization about 1 year after surgery showed normal or nearly normal right ventricular pressures. Postoperative improvement is attributed to regression of hypertrophy of the right ventricle consequent to relief of obstruction at the valve.

Open pulmonary valvotomy through the pulmonary artery in the hypothermic patient was adopted at The New York Hospital in January 1956, because the relief of valvular stenosis afforded by this technic was reported to be more complete than by closed methods.13 It was anticipated that correction of the stenosis by an adequate valvotomy would be evidenced in the operating room by a prompt reduction of right ventricular pressure to nearly normal levels. However, within the first year of experience with this operation, 3 patients with severe valvular pulmonic stenosis and intact cardiac septa were encountered who still had a right ventricular pressure in excess of 100 mm. Hg after completion of the procedure. An area of obstruction to pulmonary blood flow remained which, by pressure measurements obtained by needle puncture through the ventricular wall, was localized to the subvalvular portion of the right ventricle.

When this situation was first encountered, we were confronted with a difficult decision.

To open the right ventricle and resect the obstructing tissue was deemed unwise, because of the risk of inducing uncontrollable ventricular fibrillation in the hypothermic heart. But to do nothing further and leave the patient with an obstruction sufficient to produce a right ventricular pressure greater than 100 mm. Hg also caused us concern. There seemed to be less risk in the second choice; so it was decided to see how much benefit would be derived from relief of the stenosis at the pulmonary valve. Should additional surgery on the infundibular area be necessary, it could be done with the aid of extracorporeal circulation. There was hope that this obstruction was due to hypertrophied musculature that would decrease in size after valvotomy lessened the work load on the right ventricle; but there was little evidence at the time to indicate such a fortunate outcome ever happened.

Observations on these 3 patients are reported below. The pressure measurements are summarized in table 1. Cardiac catheterization was performed within the few weeks preceding operation and from 10 to 15 months after valvotomy. In the operating room pressures were measured through direct needle puncture of the right ventricle and pulmonary artery with a Statham P23D strain-gage transducer and were recorded on a Sanborn Polivisso recorder. Measurements were obtained immediately preceding circulatory occlusion. They were again recorded after valvotomy and...
infundibular exploration, when the circulation had been restored, the incision in the pulmonary artery sutured, and the electrocardiogram, pulse rate, and systemic blood pressure had returned to pre-occlusion levels.

**Case Reports**

*Case 1.* E. L., 563942, was 5½ years old at the time of surgery. She was born prematurely at The New York Hospital in 1950, after a pregnancy complicated by exposure of the mother to German measles during the second month. A twin was macerated. A systolic murmur was detected at birth, and there was cardiac enlargement. For the first 3 months the baby was cyanotic. She then did well until about 4 years of age when she could not keep up with other children at play. The heart enlarged progressively to a cardiothoracic ratio of 59 per cent (fig. 1), and electrocardiograms showed progressively more marked right ventricular hypertrophy with the development at age 5 of right ventricular “strain.” She was aecyanotic, but had a left parasternal bulge and a systolic thrill and murmur in the pulmonary area. The pulmonary component of the second heart sound was diminished.

The findings were those of severe valvular pulmonary stenosis with intact cardiac septa. However, 2 features raised the question of accompanying infundibular stenosis: first, the wide radiation of the systolic murmur along the left sternal border from the first through the fourth interspaces and second, the absence of much poststenotic dilatation of the main pulmonary artery (fig. 1).

At cardiac catheterization (table 1) the pressure changed abruptly at the level of the pulmonary valve from 16/7 mm. Hg in the pulmonary vein to 178/7 mm. Hg in the right ventricle (fig. 1). Table 1 shows that right ventricular pressures fell to 170/0 mm. Hg before valvotomy and to 125/0 mm. Hg after valvotomy. The poststenotic dilatation of the main pulmonary artery was minimal (fig. 1).

**Table 1.**—Changes in Right-Sided Pressures (mm. Hg) before and after Valvotomy

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yrs.)</th>
<th>Preoperative catheterization (time)</th>
<th>Operating room</th>
<th>Postoperative catheterization (time)</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. L.</td>
<td>5 6/12</td>
<td>(8 d.) RV 178/7 PA 16/7</td>
<td>Before valvotomy After valvotomy</td>
<td>170/0 125/0 29/4 (14 mo.) 23/4</td>
</tr>
<tr>
<td>E. R.</td>
<td>27</td>
<td>(2 wk.) RV 158/8 PA 12/5</td>
<td>160/5 155/10 20/10 21/5</td>
<td>20/6 (15 mo.)</td>
</tr>
<tr>
<td>B. R.</td>
<td>19/12</td>
<td>(2 mo.) RV 162/3 PA —</td>
<td>175/15 100/20 35/6 10 mo.</td>
<td>31/16 20/4</td>
</tr>
</tbody>
</table>

**Fig. 1.** Case 1. Roentgenograms in frontal (*left*) and right anterior oblique (*right*) projections show cardiac enlargement, in particular of the right atrium and right ventricle, and absence of poststenotic dilatation of main pulmonary artery. Peripheral lung fields hypovascular. (Reprinted with permission from Pediatrics 19: 1144, 1957.)
artery to 178/7 mm. in the right ventricle. No shunt was demonstrable in either direction.

A week later, on January 20, 1956, the patient was cooled to 29 C. and pulmonary valvotomy was performed under direct vision. Right ventricular and pulmonary arterial pressures measured prior to interruption of the circulation agreed closely with those at cardiac catheterization. A small, cone-shaped valve with an orifice approximately 2 mm. in diameter was grasped and incised to the valve ring. To our surprise, the pressures measured after restoration and stabilization of the circulation showed only a partial reduction of the right ventricular pressure to 125/0 mm. Hg (fig. 2). Pressures recorded at different levels of the right ventricle were consistently elevated but in the distal portion of the outflow tract, just proximal to the pulmonic valve, the pressure was 40/6, a few mm. higher than that in the pulmonary artery. The muscle here was unusually thick, judged by the depth to which the needle had to be introduced to record a cavity pressure. It was believed that the valvular stenosis had been relieved but that there was co-existent subvalvular stenosis. Ventriculotomy and infundibular resection under hypothermia were considered contraindicated.

The postoperative course was one of steady improvement to the point of unlimited exercise without symptoms. During the first 6 months the electrocardiographic evidence of right ventricular “strain” disappeared and the pattern of right ventricular hypertrophy regressed, to be replaced by an rsR’ complex of normal amplitude in the right precordial leads' (fig. 3).

Cardiac catheterization 14 months after the operation demonstrated a slight gradient of only 6 mm. Hg across the pulmonic valve, but the right ventricular pressure was normal. With exercise the gradient during systole increased to 14 mm. There was no shunt, and the cardiac output was normal (table 1).

Case 2. E. R., 237581, was operated upon at the age of 27 years. She was first seen at the New York Hospital when 10 years old because of a heart murmur known since the age of 1 year. Ordinary activity produced no symptoms, but she became fatigued and short of breath with strenuous exercise. There was a loud, harsh systolic murmur maximal in the pulmonary area. The heart was not enlarged. The 3-lead electrocardiogram showed right axis deviation with abnormally large amplitude of S, and Rs deflections, indicative of right ventricular hypertrophy. By the age of 13, changes in the T waves that are associated with right ventricular “strain” (deeply inverted T3 and biphasic T4) were present.

After adolescence she continued to live comfortably within the restricted activities advised by physicians. She did not return to the hospital until age 27, this time because of infertility since her marriage 6½ years earlier.

Her blood pressure was 120/70 and the pulse was 80. She was not cyanotic, and there was no clubbing. Cardiac findings included a slight left parasternal bulge, a coarse systolic thrill, and a long, loud, harsh systolic murmur maximal in the first and second interspaces and audible over the precordium, in the neck vessels, and the lung fields. The pulmonic component of the second heart sound at the base was inaudible. Diastole was clear. The neck veins were flat, and the liver was not enlarged or pulsatile. Peripheral pulses were normal.

Roentgenograms and fluoroscopy of the chest in frontal and oblique projections showed a cardiothoracic ratio of 45 per cent and slight enlargement of the right ventricle. There was normal prominence of the main pulmonary artery (fig. 4). Pulsations were diminished in the branches, and pulmonary vascularity in the peripheral lung fields was slightly decreased. An electrocardiogram showed right ventricular hypertrophy and “strain” with T-wave abnormalities more marked than at age 13. The blood count was normal.

The impression was severe valvular pulmonic stenosis with intact cardiac septa. The absence of poststenotic dilatation of the main pulmonary artery raised the question of accompanying infundibular stenosis.

At cardiac catheterization the pressure change from a pulmonary artery pressure of 12/5 mm. to a right ventricular pressure of 158/8 mm. Hg occurred abruptly at the valvular region on re-
repeated withdrawals of the catheter (fig. 5). On exercise the right ventricular pressure rose to 195/11 mm. of Hg. Thus catheterization studies supported the diagnosis of severe isolated valvular pulmonic stenosis.

Pulmonary valvotomy was performed under direct vision on June 15, 1956, with the patient’s temperature reduced to 28 C. Pressures measured in the right ventricle and pulmonary artery prior to occlusion of the circulation were in agreement with the readings obtained at catheterization. During 3½ minutes of circulatory occlusion the pulmonary artery was opened, and a cone-shaped, fused pulmonic valve with an orifice about 2 mm. in diameter was incised on each side out to the valve ring. The surgeon introduced his finger through the opened valve into the right ventricle and noted some narrowing of the outlet of the ventricle.

After the occluding tapes were released and regular sinus rhythm and systolic blood pressure had stabilized at the pre-occlusion values, pressures were again measured and found to be unchanged. It was thought that 2 incisions in what should normally be a 3-cusped pulmonic valve might have been inadequate. Therefore, the circulation was occluded for an additional 3½ minutes while the pulmonary valve and infundibular region were re-explored. The pulmonary valve was seen to be opened fully to the valve ring; nevertheless, another incision was made in it. The surgeon was convinced that it was opened to its fullest extent. Visual and digital inspection of the right ventricle indicated that there was a marked narrowing of the
outflow portion. During diastole the finger could readily be inserted, but this area contracted tightly about the finger during systole. When the finger was removed, the subvalvular portion of the ventricle could be seen to open and then to close tightly like a sphincter with each contraction of the heart.

After the circulation was restored and the incision in the pulmonary artery was closed, a third set of pressure readings was obtained. They differed very little from the prevalvotomy determinations (fig. 6). Pressure in the right ventricle just below the pulmonary valve ring was similar to that in the pulmonary artery.

The postoperative course was complicated by a delayed febrile reaction with chest pain and pleural and pericardial effusions, interpreted as the postpericardiotomy syndrome.

Within the next 6 months the patient was able to exercise normally and vigorously without fatigue or dyspnea. She now realized the limitations she had minimized preoperatively. By the sixth month the electrocardiographic pattern of the right ventricular "strain" and hypertrophy had disappeared. Instead, there was normal left ventricular dominance and an rsR' pattern in V6 and V1; the amplitude of the deflections was normal (fig. 7). Her heart size was normal, the second sound in the pulmonic area was easily heard, and at the left base there was a soft systolic murmur followed by a short, soft diastolic murmur of pulmonic insufficiency.

By this time she was 3 months' pregnant and on the first anniversary of her operation she gave birth to a healthy girl. She tolerated the pregnancy and delivery without difficulty.

Three months later cardiac catheterization was repeated. The cardiac output, previously restricted, was greater than normal and the pressures were normal. The systolic gradient between the right ventricle and pulmonary artery had been completely abolished (fig. 5 and table 1).

Case 3. B. R., 740274, was an infant 21 months old at the time of valvotomy. She fatigued somewhat more quickly than others of her age but otherwise seemed healthy.

She was normal in size and showed neither cyanosis, clubbing, nor chest deformity. A systolic thrill was palpable over the pulmonary area and a harsh systolic murmur was maximal there. It was transmitted over the heart to the lung fields and to the neck. The second heart sound was
diminished at the left base. There was no venous distention, and the liver was not palpable.

Fluoroscopy and roentgenograms of the chest disclosed a cardiothoracic ratio of 59 per cent with enlargement of the right atrium, right ventricle, and main pulmonary artery. The peripheral lung fields were excessively clear (fig. 8). An electrocardiogram showed right atrial enlargement, right ventricular hypertrophy, and "strain." Blood counts were normal.

The impression was that she had severe valvular pulmonic stenosis with intact cardiac septa. Despite the few symptoms surgery was advised because of the radiologic and electrocardiographic evidence of severe pulmonary stenosis. Preoperative cardiac catheterization (table 1) recorded a right ventricular hypertension of 162/3 mm. Hg. The catheter tip could not be advanced through the pulmonary valve into the pulmonary artery. No shunt was demonstrable. The arterial oxygen-hemoglobin saturation was 93.6 per cent.

Operation on the pulmonary valve under direct vision was performed on November 30, 1956. Pressures in the operating room prior to circulatory occlusion at a temperature of 23 C. measured 175/15 in the right ventricle and 23/18 mm. Hg in the pulmonary artery. Three incisions were made in the stenotic valve out to the valve ring. A clamp passed through the valve into the right ventricle met no fixed obstruction when it was spread. The circulation was interrupted for 4 1/2 minutes. After the incision in the pulmonary artery was sutured and the heart rate and systemic blood pressure had returned to their previous levels, pressures were again measured. A somewhat damped right ventricular tracing showed a pressure of 100/20 mm. Hg in the right ventricle and a reading of 31/16 in the pulmonary artery. Though there remained a high pressure in the right ventricle, no additional procedures were undertaken because the valvular stenosis was considered relieved and because by this time the marked postoperative improvement in the first patient had become evident.

She too showed progressive improvement in the
FIG. 9. Case 3. Electrocardiograms before and 10 months after surgery show marked improvement in evidence of right atrial enlargement and right ventricular hypertrophy and "strain."

FIG. 10. Section through pulmonary artery, pulmonary valve, and right ventricle of 5-year-old child with severe valvular pulmonic stenosis and intact ventricular septum. Note bulge of thick muscular wall beneath the valve.

electrocardiogram, with regression of right ventricular "strain" and hypertrophy and the appearance of an rsR' pattern over the right ventricle (fig. 9). The heart size decreased (fig. 8). Cardiac catheterization performed 10 months postoperatively revealed a nearly normal pressure in the right ventricle of 35/6 and in the pulmonary artery of 20/4 mm. Hg (table 1).

DISCUSSION
This form of infundibular pulmonic stenosis accompanying severe valvular pulmonic stenosis is attributed to the marked hypertrophy of the right ventricular musculature. The great muscular thickening of the wall of the right ventricle that is found in patients with extreme valvular pulmonic stenosis and intact ventricular septum is well known. The manner in which the muscle mass bulges beneath the fused valve leaflets is illustrated in figure 10 by a section made through the pulmonary artery, pulmonary valve, and adjac-
cent right ventricle of a 5-year-old boy who died in heart failure from this congenital anomaly. The tremendously hypertrophied muscle fibers and fibrous tissue in this region are shown in figure 11.

In addition to the uniform hypertrophy of the wall of the right ventricle, there are 2 muscle bundles in the infundibulum that are particularly enlarged in this malformation. Both Brock and Kirklin and associates have called attention to the importance of these bands in narrowing the outflow tract of the right ventricle. One, the crista supraventricularis, extends from the pulmonary valve down to the anterior wall of the right ventricle. The other bundle, composed of parietal and septal bands, extends from the pulmonary valve down along the ventricular septum.

The thickening of the muscle wall and of these bundles was sufficient to produce significant narrowing of the outflow tract in 4 of 6 specimens of marked pulmonic stenosis with intact ventricular septum reviewed by Kirklin. This was true also in 6 specimens with this malformation available for re-examination from the autopsy files at the New York Hospital. These 6 patients died as a result of severe valvular pulmonic stenosis at the ages of 7 and 15 days, and 2, 4½, 5½, and 30 years.

To evaluate obstruction of the subpulmonary tract, Bing and his co-workers made a paraffin cast of the cardiac chambers and pulmonary artery of a man who died with pulmonic stenosis and atrial septal defect. They stated that although muscular hypertrophy may exist in this condition, it did not result in narrowing of the right ventricular outflow tract. We believe it may have been the inability to evaluate by this technic the dynamic role of muscular contraction that led to their opinion.

It is difficult to apply to the living patient the information obtained from postmortem specimens. Though these indicate co-existent
infundibular narrowing in some patients with marked valvular stenosis, a better estimate of the situation during life may be obtained by visualization of this region during systole and diastole. Angiocardiographic studies by Kjellberg and co-workers and by Campeti did not show a fixed infundibular obstruction in patients with valvular pulmonic stenosis. These pictures showed no constriction early in systole, but they graphically portrayed the narrowing imparted to this region at the end of systole by forceful contraction of the crista supraventricularis, hypertrophied muscle bands, and ventricular wall.

Rodbard et al. described pressure changes at cardiac catheterization in 10 patients in whom he postulated that muscular contraction in the infundibular region served as a mechanism of pulmonic stenosis. He demonstrated the development of a gradient between the ventricle and the pulmonary artery that was greater in late than in early systole. In one of these patients a muscular ring was found at surgery.

Exaggeration of the infundibular narrowing by myocardial contraction was appreciated in case 2 of this report by the surgeon as he felt the muscle tighten about his finger. On withdrawal of the finger, the subvalvular portion of the ventricle could be seen to close like a sphincter.

Of interest in regard to infundibular muscular hypertrophy as a mechanism of obstruction of pulmonary blood flow is the description by Gasul and his co-workers of the development of infundibular stenosis in infants with ventricular septal defect. They had increased pulmonary blood flow and no evidence of pulmonary stenosis on first catheterization. Though the mechanism responsible for this development is probably different, similar anatomic and functional changes may occur in the outflow tract of the right ventricle in these 2 situations, one with increased and the other with impeded pulmonary flow.

The size of the pulmonary valve ring may also contribute to the obstruction to pulmonic blood flow that was found after valvotomy. This is difficult to evaluate because no measurements of the valve ring were made in the 3 patients, though the area appeared smaller than normal. Less attention has been paid in the literature to the circumference of the valve ring in this anomaly than to the size of the opening in the fused cusps. However, there are a number of reports in which the ring of the pulmonic valve was found to be small when the stenosis of the valve was marked. For example, Ordway et al. found the circumference of the pulmonary valve ring to be 4 cm. in a 25-year-old man whose aortic ring measured 6 cm. The central opening in the stenosed pulmonary valve was 2.5 mm. in diameter. The pulmonic valve ring of a 39-year-old man reported by Selzer and collaborators measured 5 cm. in circumference, in comparison to the aortic ring, 7.2 cm. The tiny orifice in the pulmonary cone was 2 mm. across. One of the specimens reviewed by us was that of a 30-year-old woman, whose pulmonary valve was 4.5 cm. and aortic valve 8 cm. in circumference. The diameter of the opening in the diaphragm-like pulmonary valve was 2 mm. According to Saphir the circumferences in the normal adult are 8.5 cm. for the pulmonic and 7.5 cm. for the aortic valve.

Calculations of the pressure-flow relationships across a valve of this limited circumference indicate that the area enclosed by the ring is not small enough to cause more than a mild obstruction. It is possible, however, that the slight systolic pressure gradient observed at postoperative catheterization in patients 1 and 3 may have been due to a smaller valve ring than is normal.

In our 3 patients, it is puzzling why only 1 area of stenosis was detected by preoperative cardiac catheterization if 2 areas were found to exist at operation. In case 2, repeated withdrawals of the catheter from pulmonary artery to right ventricle gave a sharp pressure change at the region of the pulmonary valve, with no indication of an intermediate zone (fig. 5). In case 1, a few ven-
tricular premature beats occurred as the catheter entered the ventricle from the pulmonary artery, so that the pressures for these few beats were more difficult to evaluate. Nonetheless, the change to a high pressure seemed to take place at the level of the valve. In the third patient, the tip of the catheter appeared to hang in the region of a pulmonary valve. The probable explanation for the paradoxical appearance of a second area of obstruction after valvotomy was suggested by Brock. When the valve was intact, he thought that the high pressure in the ventricle distended the chamber so that the thick muscular walls of the infundibulum were held apart. Relief of the obstruction at the valve might lower the pressure enough for the walls to come together and produce a subvalvular stenosis with hypertension proximal to the block.

Although this explanation indicates that the infundibular stenosis becomes significant after valvotomy, it is of interest that clinically 2 patients (E. L. and E. R.) had features that preoperatively suggested associated infundibular stenosis: absence of poststenotic dilatation so common in valvular pulmonary stenosis, and in the first child, wide radiation of the systolic murmur along the left sternal border rather than maximal localization in the second interspace.

Cineangiography or selective angiography with frequent exposures correlated with the electrocardiogram might help in the preoperative distinction between a fixed subvalvular obstruction, rigid during all phases of the cardiac cycle, and one due to contraction of thick muscle.

One may speculate on the reason for continued improvement in these patients who were left with an important area of obstruction proximal to the one that was relieved surgically. The work of the heart was lessened by the decreased resistance at the valve. Decrease in size of the muscle fibers, in response to the decreased work, would cause less and less obstruction during infundibular contraction. Thus there would be progressively decreasing work and concomitantly increasing lumen in the outflow tract of the ventricle as the thick muscle mass melted away. Electrocardiographic evidence indicated that most of this regression of hypertrophy took place in the first 6 months after surgery but continued even up to 20 months.

Preliminary observations on regression of hypertrophy in the first patient were published in June 1957. One month later Himmelstein and his co-workers reported a patient, aged 14, in whom they believed there had been some reduction in hypertrophy of the musculature of the right ventricular outflow tract following closed transventricular valvotomy. The pressure in the right ventricle was reduced at operation from 180/5 to 130/5 mm., but 6 months after operation the pressure had fallen to 60/7 mm. She too was said to show a decrease in electrocardiographic evidence of right ventricular hypertrophy.

Regression after valvotomy of accompanying infundibular stenosis may have occurred in a patient reported by Kirklin and in one by Campbell and Brock. Kirklin and associates reported a patient in whom cardiac catheterization studies 5 months after a closed pulmonary valvotomy showed a fall in right ventricular pressure, despite persistence of subvalvular stenosis after operation. Right ventricular pressure was 125/5 before and 110/5 mm. after valvotomy. Five months later it was 65/8 mm. of Hg. In this patient, as well as Himmelstein's, studies later than the sixth postoperative month might have shown even a more striking reduction in right ventricular hypertension.

Case V78 reported by Campbell and Brock in 1955 showed an improvement in right ventricular pressure at postoperative cardiac catheterization that was not evident immediately following a closed valvotomy. Right ventricular pressures in the operating room were 97/5 before and 94/9 mm. after valvotomy. Catheterization at an unspecified time after operation revealed a pressure of 31/0 in the right ventricle and 19/6 mm. in the pulmonary artery.

Manning and Mahoney have recently
studied 3 patients similar to ours who showed regression of infundibular stenosis following open pulmonary valvotomy. A report on their observations is in progress.

We believe that open valvotomy has an advantage over closed procedures when this situation is encountered in the operating room. The surgeon can then be confident that the stenosis at the valve has been completely relieved. Repeated manipulations of the valve are unnecessary. Inspection of the subvalvular region can then be carried out by direct vision into the empty heart, by finger palpation, or by the introduction of an instrument that can be spread open inside the chamber. If no fixed, rigid infundibular obstruction, such as a diaphragm or ring is encountered, then experience from these 3 patients implies that no further surgical procedures are indicated. Though infundibular resection has been recommended if a significant systolic gradient remains after valvotomy, we believe that the type of infundibular stenosis due to muscular hypertrophy is better and more safely treated by giving it the opportunity to regress after valvotomy.

**Summary and Conclusions**

Three patients, an infant, a child, and an adult, with severe valvular stenosis were observed to have a right ventricular systolic pressure greater than 100 mm. Hg following valvotomy performed under hypothermia through an opening in the pulmonary artery. The obstruction that remained was localized by pressure measurements to the outflow tract of the ventricle. Intracardiac exploration of this region indicated that the obstruction was not rigid but appeared to be due to contraction of the hypertrophied muscle. During 6 to 20 months postoperatively electrocardiographic signs of right ventricular hypertrophy disappeared, and cardiac catheterization 10 to 15 months after operation disclosed pressures within normal limits in the right ventricle with only slight or no transvalvular gradients.

It appeared that the severe valvular stenosis was responsible for marked hypertrophy of the wall of the right ventricle, sufficient by itself to narrow the outflow tract once the obstruction at the valve was relieved. Restoration of valve function by valvoplasty reduced the work of the ventricle so that the hypertrophied myocardial fibers returned to a more nearly normal size and was accompanied by complete regression of this secondary form of infundibular stenosis.

The surgical implication from these observations is that if the valve has been opened fully and no fixed obstruction, such as a diaphragm or ring, is found within the right ventricle, then additional attempts to treat the muscular stenosis surgically are unnecessary.

**Summario in Interlingua**

In tres patientes—un infante, un puero, e un adulto—con sever stenosis valvular, le pression systolica dextero-ventricular esseva plus que 100 mm de Hg post valvotomy effectuate sub hypothermia a transverso un apertura in le arteria pulmonar. Le mesuration de pressiones permitteva le localisation del remanente obstruction in le via de effluxo ab le ventriculo. Le exploration intracardiae de iste region indicava que le obstruction non esseva rigide. Illo pareva esser le effecto de un contraction del musculo hypertrophia. In le curso de inter 6 e 12 menses post le operation, le signus cardiological de hypertrophia dextero-ventricular dispareva, e catheterisations cardiac effectuate inter 10 e 15 menses post le operation revelava pressiones intra le limites normal in le ventriculo dextere e solmente leve o nulle gradientes transvalvular.

Il pareva que le sever stenosis valvular esseva responsabile pro le marcate grades de hypertrophia in le pariete del ventriculo dextere e que iste hypertrophia sufficeva a restringer le via de effluxo post que le obstruction valvular habeva esse pace alleviata. Le restauration del function valvular per valvoplastia reduceva le labor del ventriculo de manera que le hypertrophia fibras myocardial retornava a dimensiones plus proixe
al norma con le effecto de un complete regression de iste forma secundari de stenosis infundibular.

Ab le puncto de vista chirurgic, le conclusion a derivar ab iste observationes es le sequente: Quando le valvula ha esiste aperite completamente, nulle effortio additional a corriger le stenosis muscular per medios chirurgic es necessari, excepte in casos in que il existe un obstruction fixe como per exemplo un diaphragma o un anulo.

REFERENCES
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Hence, since a man may make experiment in many places, it appears that the function of the portal in the veins is the same as that of the Signoides, or three pointed portals, which are made in the orifice of the aorta or vena arteriosa, to wit that they may be closely shut up, lest they should hinder the blood to return back again.—William Harvey. De Motu Cordis, 1628.
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