Pulmonary valve stenosis without ventricular septal defect: results of surgery

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It is 18 years since the first successful pulmonary valvotomies were carried out by Sellors (1948) and Brock (1948) in patients with the tetralogy of Fallot, and little less since Brock applied the closed transventricular technique to the treatment of pulmonary valve stenosis without ventricular septal defect. It is nevertheless probably true to say that no other cardiac condition is treated in so many different ways at the present time, due to ignorance of the natural prognosis of mild and moderate pulmonary valve stenosis and to the varying emphasis placed on four important facets of the problem: these are the small but unsatisfactory mortality of most current procedures; the desirability or otherwise of resection of hypertrophic infundibular muscle; the importance of closure of a foramen ovale and its relation to post-operative cyanosis; and the short- and long-term effects of any pulmonary incompetence which may be produced. Few authorities agree on the significance of these problems. The factors responsible for post-operative cyanosis have already been considered (Oakley, Braimbridge, Bentall, and Cleland, 1964).

MATERIAL AND PRE-OPERATIVE FINDINGS

The series consists of the 56 cases of pulmonary valve stenosis operated upon between April 1958 and January 1963 by Mr. W. P. Cleland or Professor H. H. Bentall at the Hammersmith Hospital and Brompton Hospital and followed for at least two years after operation. The patients’ ages ranged from 2 to 46 years, with a mean of 18.5 years. Thirty were male and 26 female. An associated secundum atrial septal defect was present in 15 patients (27%) and cyanosis either at rest or on exercise in 18 patients (32%), nine of whom had an atrial septal defect and nine a valvular foramen ovale. The pre-operative resting right ventricular pressure averaged 122 mm. Hg (range 60 to 300 mm. Hg) in the acyanotic patients and 125 mm. Hg (range 60 to 230 mm. Hg) in the patients with arterial desaturation.

Eighteen patients were entirely asymptomatic and 17 patients complained only of mild shortness of breath or fatigue. Twenty-one patients had more severe disability. This was due to shortness of breath in 16; eight of these were cyanosed at rest and three had squatted in infancy. Syncope was the major symptom in three and ‘lightheadedness’ or ‘giddiness’ on effort in another four. Angina was uncommon and was described by only four patients. The average right ventricular pressure at rest was 153 mm. Hg in the 21 patients with important symptoms, 30 mm. Hg above the average for the whole series. While syncope might be attributed to a restricted cardiac output on effort and angina to inadequate blood flow to the hypertrophied right ventricle, dyspnoea was unexpected in eight patients who had severe right ventricular obstruction but who had no reduction in arterial oxygen saturation even on effort.

Physical retardation was unusual and was seen in only two patients. Four patients may have been infected by maternal rubella, as two had nerve deafness and two had a patent ductus. No patients had associated pulmonary branch stenosis. Two cyanosed patients were hemiplegic. Forty-seven patients showed abnormally large venous ‘a’ waves. An ejection click introduced the stenotic murmur in only seven patients, all of whom had a resting right ventricular pressure below 90 mm. Hg and a normal pulmonary artery pressure. Pulmonary valve closure was delayed to between 0.05 and 0.12 sec. after aortic valve closure in all the 44 patients in whom it could be recorded on the phonocardiogram. No patient was or had been in cardiac failure.

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Thorax (1966), 21, 164.
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The electrocardiogram was abnormal in every patient. In patients whose right ventricular pressure was at or above the systemic pressure the record resembled that in the tetralogy of Fallot, but in those with more severe stenosis T wave inversion characteristically extended beyond V1 to V3 and V4 and there was usually a tall P wave. Two patients had right bundle branch block preoperatively, but in neither case was there an associated atrial septal defect.

Three patients had atypical electrocardiograms with a mean frontal axis between −90° and 180° and a dominant S wave in the three standard and all precordial leads except V4R (Fig. 1). Although endocardial cushion defects with pulmonary stenosis were suspected (Scott, Hauck, and Nadas, 1962) all were found to have ordinary secundum type atrial septal defects in association with severe pulmonary valve stenosis. No explanation for this variant could be found in two patients, but in one

![Fig. 1](http://example.com/fig1.png)

FIG. 1. Electrocardiogram from a 4-year-old boy whose resting right ventricular pressure was 120 mm Hg and who became desaturated on effort. The mean frontal axis is bizarre (−150°), and dominant S waves are seen in all leads except aVR and V4R. Similar atypical electrocardiograms were seen in three patients with severe pulmonary valve stenosis and a secundum atrial septal defect.

![Fig. 2](http://example.com/fig2.png)

FIG. 2. Electrocardiogram from a 28-year-old cyanotic man. Hypoplasia of the right ventricle was suspected, and this seemed to be confirmed at operation. See text.
(Fig. 2), who was deeply cyanosed despite a resting right ventricular pressure of only 60 mm. Hg, a hypoplastic right ventricle was suspected and seemed to be confirmed at operation, when severe pulmonary valve stenosis was relieved and a large fenestrated secundum atrial septal defect was closed.

Radiographs of the chest showed post-stenotic dilatation of the pulmonary artery in all but two patients and a left-sided aortic arch in every case. The radiograph was not useful in the determination of severity, as cardiac enlargement was rare and usually attributable to right atrial dilatation. Selective angiography, carried out in conjunction with right heart catheterization, gave a clear indication of the degree of stenosis and associated infundibular hypertrophy and was performed in all except the early patients of the series.

**SELECTION OF PATIENTS FOR OPERATION**

No one feature determined the decision to operate, although the height of the resting right ventricular systolic pressure was necessarily the main yardstick. Patients whose right ventricular pressures were below 80 mm. Hg were not operated upon unless there was other evidence of severity such as the presence of definite or newly acquired symptoms, the presence of cyanosis or its development on exercise, or disproportionate right ventricular hypertrophy on the electrocardiogram. A significantly reduced cardiac output or subnormal pulmonary artery pressure at catheterization together with angiographic evidence of more important pulmonary valve stenosis than would be expected from the height of the right ventricular pressure alone was also an indication.

When it became apparent that the operative risk was low and the results gratifying, the criteria for operation were broadened a little. Recently it has become clear that right ventricular function may remain impaired when pulmonary valvotomy is delayed until adult life, and valvotomy is now advised for patients with a right ventricular pressure exceeding 70 mm. Hg irrespective of symptoms.

**OPERATIVE TECHNIQUE**

The detailed technique of pulmonary valvotomy passed through several phases. The Melrose/N.E.P. pump oxygenator was used throughout the series initially with the normothermic heart arrested with potassium citrate. The right ventricle was opened longitudinally, the pulmonary valve inverted into the ventricle and incised, and the hypertrophied muscle of the outflow tract was resected. Subsequently elective cardiac arrest was abandoned.

More recently valvotomy has been carried out through the pulmonary artery with the heart beating and at normal temperatures, and this is current policy. The degree of infundibular hypertrophy is assessed by passing a finger down into the ventricle, after which bypass is discontinued and the right ventricular systolic pressure is measured. If there is little infundibular hypertrophy, judged by a satisfactory fall in right ventricular pressure and minimal constriction around the finger, no further action is taken. If the right ventricular pressure remains high and the finger has been tightly gripped, bypass is re-established, the ventricle is opened transversely, and a wide muscular excision is performed.

With the passage of time and greater experience, infundibular resection has been employed less and less. Only localized severe infundibular hypertrophy and unaltered high right ventricular pressures after valvotomy are considered indications for myocardial resection at the present time.

**RESULTS**

**MORTALITY** One patient died (1.8%). She was a cyanosed, epileptic, 18-year-old girl, who died in status epilepticus on the fifteenth day. The other 55 patients survived.

**RAISED JUGULAR VENOUS PRESSURE** Most patients have a raised jugular venous pressure for a time after the relief of pulmonary valve stenosis, and it is difficult to decide when to call this right heart failure. 'Congestive failure' has here been defined as a 'v' wave in the jugular pulse over 5 cm. in height, combined with a large tender liver, with discharge from hospital delayed on this account. A 'high jugular venous pressure' is defined as a persistent 'a' wave above 3 cm. but without an enlarged liver and with a normal time of discharge from hospital.

Using these definitions, three patients were in congestive failure post-operatively. One had had an infundibular resection and a satisfactory reduction of right ventricular pressure from 175 to 50 mm. Hg with no pulmonary incompetence, but months later the 'v' wave was still 8 cm. The second had had a valvotomy through the pulmonary artery, the right ventricular pressure dropping from 180 to 100 mm. Hg. Post-operative supraventricular tachycardia and a post-perfusion
syndrome (Smith, 1964) contributed to her congestive state. The third had had a transarterial valvotomy, her post-operative right ventricular pressure was 20 mm. Hg, and there was no pulmonary incompetence. She remains in right ventricular failure with gross cardiomegaly, still requires diuretics, and is one of only two patients who have had poor long-term results.

Seven patients had 'high jugular venous pressures' after operation. Four had had ventricular incisions and their initial right ventricular systolic pressures of 160, 140, 75, and 70 mm. Hg had been reduced to 100, 80, 45, and 45 mm. Hg respectively. The first of these four patients had residual infundibular obstruction as well as pulmonary incompetence, and the second had a small valve ring with incomplete relief of obstruction.

There was no obvious reason for the raised venous pressure of the remaining two. Three of the seven patients with high jugular venous pressure did not have ventricular incisions, and their right ventricular pressures of 140, 140, and 120 mm. Hg before valvotomy were 140, 90, and 80 mm. Hg after valvotomy. The first of these three had a post-pericardiotomy syndrome, the second was still cyanosed because a patent foramen ovale had not been closed, and the third had a small valve ring. There was little difference in the post-operative course between the four patients who had had infundibular resections and the three who had not.

HYPERTROPHIC INFUNDIBULAR STENOSIS

Hypertrrophic infundibular stenosis was estimated by pre-operative angiographic criteria or as digitally palpable subvalvular obstruction. Nine (16%) had no hypertrophy, 29 (52%) had moderate hypertrophy, 15 (27%) severe, and three (5%) gross hypertrophy. These subdivisions were necessarily inexact, but served a purpose in enabling some correlation to be made between anatomical and physiological data. The right ventricular systolic pressures in this assessment are those taken at operation, with appreciation that they do not always correspond exactly to pre-operative pressures at catheterization. They are used because not all patients had pre-operative cardiac catheterization and because they allow comparison with post-operative pressures on the operating table.

Infundibular resection was not performed in the group of nine patients judged to have no significant hypertrophy (Table I). No pressure measurements were taken in one patient, but only one of the remaining eight had a post-operative pressure above 80 mm. Hg.

<table>
<thead>
<tr>
<th>Degree of Hypertrophy</th>
<th>No. in Group (54)</th>
<th>Infundibulum Resected</th>
<th>Mean Pressures (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pre-operative</td>
</tr>
<tr>
<td>Nil</td>
<td>8</td>
<td>0</td>
<td>84</td>
</tr>
<tr>
<td>Moderate</td>
<td>28</td>
<td>14</td>
<td>112</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td></td>
<td>116</td>
</tr>
<tr>
<td>Severe</td>
<td>15</td>
<td>14</td>
<td>118</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td></td>
<td>140</td>
</tr>
<tr>
<td>Gross</td>
<td>3</td>
<td>0</td>
<td>125</td>
</tr>
</tbody>
</table>

There were 29 patients judged to have moderate hypertrophy, of whom one had no pressure measurements taken, leaving 28 for analysis. Fourteen had infundibular resections and 14 did not. The average pre-operative pressures were similar (112 and 116 mm. Hg), and nine of the resected and 10 of the unresected group had initial right ventricular pressures over 100 mm. Hg with an average reduction to approximately equal levels (69 and 66 mm. Hg). There was no obvious difference in the immediate post-operative course of either group.

There were 15 patients with severe hypertrophy. All but one had infundibular resections. The average pre-operative right ventricular systolic pressure in the resected group was 118 mm. Hg, which fell to much the same level as in the previous groups (61 mm. Hg).

In the last group of three patients with gross hypertrophy the valve orifice of one patient (aged 4) was 4 mm., of another (aged 7) 8 mm., and of the third (aged 18) 1 cm. Infundibular resection was probably beneficial, since an immediate fall in pressure was obtained from an average of 125 mm. Hg to 58 mm. Hg.

POST-OPERATIVE HAEMODYNAMIC STUDIES

Late post-operative haemodynamic studies were carried out in 18 patients who were selected either because there had been a poor initial drop in right ventricular pressure at the completion of operation or because of persistent clinical or electrocardiographic abnormalities which suggested some residual stenosis. Five patients were restudied because of arterial oxygen desaturation either at rest or on effort and in the absence of signs of residual obstruction; they showed evidence of right ventricular dysfunction, and two were the only patients in the series whose symptoms were not relieved. These five patients are described in a separate report (Oakley et al., 1964). These post-operative
HYPERTROPHIC INFUNDIBULAR STENOSIS: PARTIAL AND POOR REGRESSION GROUPS (5)

<table>
<thead>
<tr>
<th>Anomalous coronary artery</th>
<th>Residual Right Ventricular Pressure</th>
<th>Site of Gradient</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 +</td>
<td>62/15 Infundibular</td>
<td>Infundibular</td>
</tr>
<tr>
<td>3 +</td>
<td>41/7 Infundibular and valvar</td>
<td>Infundibular and valvar</td>
</tr>
<tr>
<td>2 +</td>
<td>0/5 Infundibular</td>
<td>Infundibular</td>
</tr>
<tr>
<td>3 +</td>
<td>105/5 Infundibular</td>
<td>Infundibular</td>
</tr>
</tbody>
</table>

We are indebted to Dr. H. I. Whitelocke, of the Nuttall Medical Centre, Kingston, Jamaica, who very kindly restudied one of these patients for us.

65 mm. Hg, which fulfilled the criterion of Tandon, Nadas, and Gross (1965), but not ours, for an excellent result. In these four patients residual right ventricular systolic pressures of 115, 100, 90, and 80 mm. Hg had dropped after an average interval of 1-8 years to 62, 57, 55, and 41 mm. Hg respectively. The first and last of these four patients had had infundibular resections. The first was a patient with an anomalous coronary artery running deep in the outflow tract muscle and itself imposing an obstruction. The second patient had a very high initial pressure (230 mm. Hg). She was recatheterized one year post-operatively and may still be improving. Two other very young patients had small pulmonary valve rings. These patients were judged to have fair but not excellent results. The fifth patient represents the second poor result in the series. Valvotomy was limited by a grossly hypoplastic valve ring. The right ventricular systolic pressure two years after operation was unchanged (105 mm. Hg). Symptomatic benefit was considerable, however, due to the relief of cyanosis by closure of an atrial septal defect.

The other patients who were restudied for other indications all showed complete relief of right ventricular outflow tract obstruction with normal right ventricular systolic pressure.

ELECTROCARDIOGRAPHY Because right ventricular hypertrophy must be considerable before it exerts a detectable influence on the normal left ventricular dominance of the adult cardiogram, it was no surprise to find the electrocardiogram a relatively insensitive guide to the completeness of relief of right ventricular outflow obstruction. Like Gilbert, Morrow, and Talbert (1963), we noticed that complete electrocardiographic resolution was the rule except in those patients with right bundle branch block. A normal electrocardiogram neither denied a small residual gradient nor did it exclude post-operative impairment of right ventricular function (Oakley et al., 1964). Complete right bundle branch block developed after operation in 12 patients and hindered interpretation of the electrocardiogram. As would be expected, it was more common after the transventricular operation (10 out of 22 cases), only one patient who had not had a ventriculotomy showing any conduction defect after operation.

PULMONARY INCOMPETENCE Any early diastolic murmurs, however short and soft, were considered to be due to pulmonary incompetence. Twenty-three patients were found to have pulmonary incompetence after operation (42%). Only five of 33
patients (15%) whose valvotomies had been performed through the pulmonary artery developed pulmonary incompetence, whereas 18 of 22 patients who had had transventricular valvotomies inverting the valve were left with incompetence (82%).

No patient with pulmonary incompetence has subsequently developed cardiac failure, nor has there been any detectable deterioration in exercise tolerance or in electrocardiographic and radiological appearances.

Four patients with pulmonary incompetence have been recatheterized (Fig. 3). The pulmonary arterial pressure curves were almost identical with those in the right ventricle, and the right ventricular end-diastolic pressures were raised except in the one patient whose valvotomy was performed through the pulmonary artery.

Studies on patients who had had a ventriculotomy without the production of pulmonary incompetence showed that the end-diastolic pressures were only raised in those with residual obstruction or when there was demonstrable right ventricular dysfunction.

**Discussion**

**Mortality** The mortality of pulmonary valvotomy has always been low. The closed transventricular approach, in which the valvotome and dilator were passed through the apex of the beating right ventricle, has been largely superseded, but not because of its immediate risk, which varies from zero in smaller groups of cases (Humphreys, Powers, Fitzpatrick, and Lanman, 1954; Gadboys, Kyle, and Glover, 1959; Dilley, Longmire, and Maloney, 1963) to the 12% of Brock’s large series (Brock, 1961). The latter figure included the first cases to be operated upon in the world, and if these early severe cases were excluded his mortality would be 7%, a figure similar to the 8% of 86 cases from Johns Hopkins (Hosier, Pitts, and Taussig, 1956) (Table IV).

The transarterial approach, in which special scissors were introduced through the pulmonary artery, never became widely used. Open valvotomy with inflow occlusion at normal temperatures was introduced in 1951 (Varco, 1951). The normothermic technique has been developed by Lam and Taber (1959) in America and by Edwards (1960) in Britain with a 2% mortality.

The addition of moderate hypothermia was accompanied by a mortality of 5% (Blount, van Elk, Balchum, and Swan, 1957), 6% (Brock, 1961), and 19% (Brom and Kalsbeek, 1959).

Extracorporeal circulation was used by Gerbode, Ross, Harkins, and Osborn (1960), who reported a 10% mortality in 29 cases, largely due to trouble with the pump oxygenator in the early cases. Dilley et al. (1963) had no mortality in 22 cases. Tandon et al. (1965) recently reported a 2.77% mortality in 108 patients.

There was one death (1.8%) in the series reported here. The use of extracorporeal circulation can therefore carry a smaller risk than hypothermia or closed valvotomy.

**Hypertrophic Infundibular Stenosis** Poor relief of right ventricular hypertension may be due to a hypoplastic valve ring, an inadequate valvotomy, or hypertrophic infundibular stenosis.

Inadequate enlargement of the valve orifice was usually blamed for poor results when closed valvotomy was the only practicable procedure (Swan, Cleveland, Mueller, and Blount, 1954). Direct vision of the valve by means of open techniques showed that an adequate valvotomy...
was often done (Dilley et al., 1963), and that complete relief of the stenosis did not necessarily cause an immediate drop in gradient.

Hypertrophic infundibular stenosis was recognized by Connolly, Lev, Kirklin, and Wood in 1953 and independently by Brock in 1955. Removal of the valvar obstruction allows the hypertrophied walls of the outflow tract to meet in systole and to cause an obstruction that may be little less severe than the previous one at valve level (Johnson, 1959). There is then no immediate relief of the right ventricular hypertrophy (McGoon and Kirklin, 1958; Gerbode et al., 1960).

The incidence of infundibular hypertrophy depends upon how it is defined. McGoon and Kirklin (1958) defined it as an infundibular gradient of 20 mm. Hg and reported an incidence of 77%. Johnson (1959) called it 'an infundibular gradient after valvotomy of 20 mm. Hg or more' and found that it followed 51% of closed and 77% of open valvotomies. Brock (1961) defined it as a residual right ventricular systolic pressure of 100 mm. Hg, although he included a few patients with over 80 mm. Hg also, and reported it in 37% of his whole series, 45% after closed and 30% after open valvotomies. In our patients it was assessed by angiocardiography or by palpation of the outflow tract at operation, and 52% had moderate hypertrophy, 27% severe, and 5% gross hypertrophy.

It is a common experience of cardiac surgery that a major operation producing no physiological improvement is likely to be fatal, and the immediate post-operative course of a patient with unrelieved obstruction of the right ventricle may be expected to be stormy. Johnson (1959) reported that 20% of patients with right ventricular systolic pressures above 100 mm. Hg after valvotomy died within 24 hours and 60% had evidence of cardiac failure. Nine per cent. of Brock’s patients whose residual pressures were over 80 mm. Hg died (Brock, 1961). The first two post-operative days were critical.

Excision of the hypertrophied muscle was therefore a logical extension of closed pulmonary valvotomy, but removal of the long smooth tunnel of muscle was more difficult and time-consuming than resection of the short fibro-muscular ring of Fallot’s tetralogy and isolated infundibular stenosis. A punch inserted blindly by the closed technique found no ridge on which to bite and might not relieve the infundibular gradient. The mortality of such a resection could be 29% (Brock, 1961).

The same was true of operations using moderate hypothermia. Ten minutes was too short a time for adequate muscle excision and closure of the ventricle, and the mortality could be 30% (Brock, 1961). If infundibular resection was undertaken it had to be extensive because relief of right ventricular strain had to outweigh the deleterious effects of the ventricular incision. In this series, between 20 and 60 minutes of perfusion were necessary for valvotomy and proper infundibular muscular excision.

The most interesting patients in the series were those with moderate hypertrophy, half of whom had had infundibular resections and half of whom had not. The average pre-operative pressures and the numbers who had residual right ventricular systolic pressures over 100 mm. Hg were similar. Being comparable groups it was possible to assess the value of infundibular resection.

The average right ventricular pressures of both groups were reduced to an approximately equal level, the unresected patients having if anything a better reduction of pressure, and there was no discernible difference in the immediate or later post-operative course of either group.

The patients assessed as having severe hypertrophy were numerically similar to each of these groups, and their average pre-operative right ventricular systolic pressure was also similar. All but one had infundibular resections with a similar fall in average right ventricular pressure, and only one more patient had a residual pressure over 100 mm. Hg. Although the hypertrophy was assessed as severe rather than moderate in this group of patients, they behaved similarly to the previous two groups, and it is possible that infundibular resection was again unnecessary, although it could well be argued that it was the muscular resection that had allowed such a result.

Those with gross hypertrophy were too small a group to allow any deductions to be drawn, but infundibular resection appeared to be beneficial because an immediate and excellent fall in pressure was obtained.

An analysis of these patients has therefore made out no convincing case for infundibular resection unless the hypertrophy is gross. This study was originally undertaken to demonstrate the value of infundibular resection in the treatment of pulmonary valve stenosis, and the finding has come as a surprise.

If infundibular resection is unnecessary, then, it could be argued, extracorporeal circulation may also be unnecessary. Yet the mortality in this series is lower than that for the majority of series
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using moderate hypothermia. Coronary perfusion with oxygenated blood during the period of inflow occlusion may be essential to a hypertrophied ventricle, which may then be able to tolerate a high post-operative gradient more easily. Finally, perfusion allows time for an accurate anatomical valvotomy.

Long-term effects It is usually stated that the hypertrophied infundibulum eventually involutes if patients survive the immediate post-operative period (Engle, Holswade, Goldberg, Lukas, and Glenn, 1958), but Campbell (1959) showed that complete regression of the electrocardiogram was unlikely over the age of 20, if infundibular hypertrophy was severe, and Harkins (1960) and Johnson (1959) reported persistently raised right ventricular pressures even years after complete valvotomies. Brock (1961) found that the incidence of incomplete resolution was 22% in patients left

FIG. 4. Pre- and post-operative angiograms of a patient with severe infundibular hypertrophy. The pre-operative right ventricular systolic pressure in this 22-year-old patient was 150 mm. Hg. The pre-operative angiogram is shown on the upper line. From left to right: antero-posterior view of right ventricular angiogram, lateral view during systole showing domed valve with jet through the stenosed orifice, and lateral view during diastole. The lower line shows the post-operative views when the right ventricular systolic pressure was 55.1. Note good but not complete infundibular regression.
with a residual right ventricular systolic pressure above 80 mm. Hg.

An extensive infundibular resection might be expected to lower the incidence of incomplete involution. Using the same criterion of infundibular hypertrophy as Brock, the group whose post-operative right ventricular systolic pressures were over 80 mm. Hg were selected, and over two-thirds were studied. Of these patients, a third still had infundibular gradients with residual right ventricular systolic pressures over 35 mm. Hg. Infundibular resections made no apparent difference to the resolution, as half of both the satisfactory and unsatisfactory groups had had infundibular resections.

Failure of complete resolution of hypertrophic infundibular stenosis was in this series most often associated with small pulmonary valve rings and therefore with residual obstruction at the pulmonary valve. The only remedy would be extremely radical infundibular resection and insertion of a patch across the valve. Very severe infundibular narrowing (Fig. 4), shown by pre-operative angiography, may be an indication for infundibular resection (Gilbert et al., 1963). The need for infundibular resection appears to us to be the finding at operation of fibrous infundibular narrowing which might not therefore resolve, or such failure of the right ventricular pressure to fall after valvotomy that immediate post-operative survival might be in doubt.

**PULMONARY INCOMPETENCE**

Post-operative pulmonary incompetence is usually regarded as unimportant. The conclusions from studies of experimental pulmonary incompetence have been contradictory. If the pulmonary valve of a dog is excised the heart enlarges, the right ventricular volume and systolic pressure rise, the diastolic pressure and electrocardiogram are unchanged, and the exercise tolerance is normal up to three years later (Kay and Thomas, 1954; Fowler, Mannix, and Noble, 1956; Ratcliffe, Hurt, Belmonte, and Gerbode, 1957). An assessment of the significance of these changes depends on whether exercise tolerance or heart size is the criterion, but a lifelong follow-up of patients with post-operative pulmonary incompetence will be needed before its importance is really known.

Inaccurate commissural division was inevitable with the closed techniques. Campbell (1959) reported the incidence of subsequent incompetence to be 11%, although other writers reported it after 20 to 40% of operations (Hosier et al., 1956; Hanson, Ikkos, Crafoord, and Ovenfors, 1958).

![FIG. 5. Technique of transarterial pulmonary valvotomy: (a) Separation of adherent valve tissue from the pulmonary artery wall; (b) accurate commissural division after mobilization of the valve.](http://thorax.bmj.com)
Hypothermia bypass improved but differed.

The incidence of failure of valve incompetence was associated with hypoplasia of his valve but is asymptomatic, and one patient has right ventricular failure unassociated with either residual stenosis or valve incompetence.

The hypertrophied infundibular muscle was sometimes resected and sometimes not. Infundibular resection appeared to make little difference to the residual right ventricular systolic pressures either on the operating table or at post-operative cardiac catheterization. Failure of complete resolution of raised right ventricular systolic pressures was associated either with extreme initial stenosis or hypoplastic valve rings.

Pulmonary incompetence was five times as common after transventricular as after transarterial valvotomy. Operation through the pulmonary artery appears to be obligatory if incompetence is to be reduced to a minimum.

Table V. Campbell considered that on the evidence available it was better to have a gradient of 20 to 30 mm Hg with no incompetence than no gradient with incompetence.

In this series the high overall incidence of incompetence was due to the early practice of transventricular valvotomy after inverting the valve into the ventricle. Following this manoeuvre, the incidence of pulmonary incompetence was five times that when the transarterial route was used. Valvotomy through the pulmonary artery therefore appears to be preferable. The extra time allowed by the use of extracorporeal circulation was valuable in this respect, because the adherent commissures could be separated from the artery wall and accurately divided (Fig. 5). Complete relief of the stenosis without the production of incompetence was then possible.

The immediate clinical post-operative course of patients with and without incompetence did not differ. Exercise tolerance, electrocardiographic and radiological appearances have not subsequently deteriorated, but it is a clinical impression that those with pulmonary incompetence have not improved as much as those without it, who are normal in every way.

Post-operative cardiac catheterization demonstrated slightly raised right ventricular end-diastolic pressures in all patients who had had ventriculotomies, but particularly when pulmonary incompetence had also been caused. The raised end-diastolic pressure could give rise to reversal of a shunt through a foramen ovale and thus limit the improvement that might otherwise be expected from the operation.

SUMMARY

A series of 56 patients with pulmonary valve stenosis, in whom there was no ventricular septal defect, were treated by valvotomy using extracorporeal circulation. One patient died, a mortality of 1.8%. The long-term results are excellent or fair in all but two patients; one of these has moderately severe residual pulmonary stenosis associated with hypoplasia of his valve but is asymptomatic, and one patient has right ventricular failure unassociated with either residual stenosis or valve incompetence.

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doi: 10.1136/thx.21.2.164

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