Late Hemodynamic Complications of Anastomotic Surgery
for Treatment of the Tetralogy of Fallot

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and Melvin H. Evans, M.D.

Seventeen patients with late hemodynamic complications of anastomotic surgery for the tetralogy of Fallot have been studied. Two syndromes have been noted and both seem related to shunts of greater than ideal size. The clinical characteristics of these syndromes are delineated.

Deficient pulmonary blood flow is the basic physiologic abnormality in the classical tetralogy of Fallot, since a large portion of the venous blood returned to the right heart is pumped out the aorta. Surgical anastomosis of a systemic artery to the pulmonary artery increases pulmonary blood flow and brings about great clinical improvement. The Blalock-Taussig operation, in which the end of the subclavian artery is anastomosed to the side of the pulmonary artery, and the Potts operation, in which an anastomosis is created between the aorta and pulmonary artery, have been employed extensively to accomplish this purpose. These operations, and also the anastomosis of the innominate artery to the pulmonary artery, create what is essentially an artificial ductus arteriosus. In contrast to the situation in a naturally occurring patent ductus arteriosus, the blood passing through this artificial ductus contains a substantial proportion of unoxygenated blood that has passed into the aorta from the right heart.

It has been recognized since the early years of anastomotic surgery that the communication must exceed a certain minimal size in order to achieve the desired improvement in cyanosis and exercise tolerance. An anastomosis of adequate size for a child may become too small as the patient becomes an adult or the anastomosis may be partially or completely occluded by thrombosis. The complications that result from an anastomosis which is too large are less frequent. The construction of a shunt of excessive size may result in immediate heart failure and death within a matter of days after operation. Prolonged observation of patients after operation reveal that other consequences of an excessively large anastomosis may appear after many years. The 3 patients to be presented illustrate such late complications of a systemic-pulmonary anastomosis that is too large.

Case 1. JHH 615031, A-90909

This white male was first seen at age 27 because of cyanosis and exertional dyspnea. He had been cyanotic since birth but felt that his exercise tolerance had improved with increasing age. There was a history of squatting in childhood. On physical examination there was cyanosis of the lips. The heart was not enlarged, the rhythm was regular, and a harsh systolic murmur was heard along the left sternal border. The lungs and abdomen were normal. The red blood cell count was 7.2 million per mm3, and the hematocrit was 65 per cent. The electrocardiogram showed right ventricular hypertrophy. Chest x-ray at the time of the first examination is shown on the left of figure 1. Rapid and simultaneous filling of the aorta and pulmonary artery was demonstrated by angiography. The diagnosis of tetralogy of Fallot was established.

A 6.5-mm. Potts anastomosis was constructed between the aorta and the pulmonary artery and a continuous murmur was heard postoperatively. During the first 14 months after operation the continuous murmur was noted to have decreased in intensity and it was not heard after the twenty-
fourth postoperative month. As the murmur decreased, the hematocrit rose from 54 to 74 per cent and thrombosis of the anastomosis was suspected. Exercise tolerance decreased, cyanosis increased, and the patient noted frequent episodes of dyspnea and tachycardia.

Physical examination during the terminal illness 4 years after operation revealed cyanosis, minimal enlargement of the heart, a presystolic gallop rhythm at the apex, and no murmurs. The lungs were clear and the liver tender and enlarged to 5½ fingerbreadths below the costal margin. X-ray of the chest at this time (fig. 1, right) revealed a striking enlargement of the left pulmonary artery that had not been present immediately postoperatively. No pulsation was visible in the artery on fluoroscopy. A cardiac catheter was passed from the right ventricle into the aorta, but the pulmonary artery was not entered. The pressure was essentially the same in the right ventricle and aorta and the oxygen saturation of the aortic blood was 58 per cent.

No further surgery was deemed advisable. A loss of 7 pounds followed treatment with digitalis and thiorermin. The patient was discharged home but returned within 1 month because of increasing dyspnea and cyanosis. The physical findings were essentially unchanged. The patient became apprehensive and increasingly dyspneic and died suddenly on the night of admission.

At autopsy there was valvular and infundibular pulmonary stenosis associated with a ventricular septal defect and overriding of the aorta. The Potts anastomosis was seen to be widely patent (fig. 2), measuring 12 mm. in diameter as opposed to 6.5 mm. measured at operation. In the main pulmonary artery there was gross atherosclerosis, which was more extensive than in the adjacent aorta (fig. 2). Microscopic examination of the lungs revealed extensive changes in the small pulmonary arteries (fig. 3). Some vessels showed concentric narrowing and others showed areas of intimal thickening and occlusion with reanализation.

Comment. Although the pulmonary artery pressure was not obtainable, it is reasonable to assume from the clinical findings and examination of the pulmonary vessels that pulmonary hypertension had developed prior to death. Presumably, a rise in pulmonary resistance occurred, the pulmonary artery pressure rose, the pulmonary artery dilated, and the anastomosis enlarged. As the pulmonary pressure approached that in the aorta the flow through the anastomosis diminished to such an extent that the anastomotic murmur disappeared. Thus, the patient was physiologically deprived of the anastomosis even though anatomically it remained open. The decrease in pulmonary blood flow was followed by a return of cyanosis and a rise in hematocrit.

Case 2. JHH #A-41658

The second patient differs significantly from the first in that 10 years elapsed between operation and the appearance of complications. This patient was first seen as a boy of 6½ years and had been cyanotic since early infancy. His activity was

![Fig. 1. Chest x-rays, case 1. Left, 7/17/52, preoperatively; right, 5/21/56, during terminal illness.](image)
limited by dyspnea on slight exertion, but he had been attending school. On physical examination he was found to be cyanotic and there was slight clubbing of the fingers and toes. There was a faint systolic murmur at the base of the heart. The hematocrit was 81 per cent, the red cell count 9.7 million per mm.\textsuperscript{3}, the arterial oxygen saturation 61 per cent.

At operation Dr. Alfred Blalock found the innominate, carotid, and subclavian arteries to be of unusually large size. The right subclavian was larger than the right pulmonary artery, but a satisfactory end-to-side anastomosis was constructed between these 2 vessels. The postoperative course was complicated by recurrent hemorrhagic pleural effusions. In the immediate postoperative period the erythrocyte count fell to 6.7 million per mm.\textsuperscript{3} and the arterial oxygen saturation rose to 81 per cent. Two years after operation the erythrocyte count was 5.5 million per mm.\textsuperscript{3} and the oxygen saturation was 83.2 per cent. The patient complained of dyspnea only on vigorous exertion.

Five years after operation the patient was leading an essentially normal life but avoided competitive athletics. The chest x-ray taken during the fifth postoperative year is seen on the left in figure 4. At this time the pulmonary vascular markings were prominent.

Ten years after operation, at age 16, he was able to carry on normal activities without fatigue and could climb 2 flights of stairs easily. In addition to the continuous murmur of the anastomosis, which had been heard since operation, the patient developed a blowing, high-pitched descreesendo early diastolic murmur in the second and third left interspaces. The erythrocyte count was 6.47 million per mm.\textsuperscript{3}. On x-ray both pulmonary arteries were prominent.

Eleven years after operation the patient reported several episodes of hemoptysis. On one occasion one half cup of blood was expectorated and on numerous occasions the sputum was blood-streaked. His exercise tolerance was essentially unchanged. The basal diastolic murmur was heard, as was the continuous murmur of the anastomosis. On fluoroscopy the pulmonary conus was prominent and the left pulmonary artery was described as huge ("the size of an orange"). Chest x-ray at this time is shown at the right of figure 4. The massively dilated pulmonary artery filled on angiocardiography, proving that it was not an extravascular shadow (fig. 5). Cardiac catheterization demonstrated the presence of a ventricular septal defect, but unfortunately the pulmonary artery was not entered.

Figure 6 shows the changes in hematocrit and pulmonary artery size during the 11 years following operation. It can be seen that the immediate response was excellent, with a fall in hematocrit that was accompanied by clinical improvement in color and exercise tolerance. The line diagrams at the top of the figure are tracings of the left heart border showing the progressive enlargement of the left pulmonary artery. The mean frontal plane area of the heart increased during the last several years from 122 cm.\textsuperscript{2} to 166 cm.\textsuperscript{2}.
The patient died suddenly during the twelfth postoperative year and autopsy was performed at the Maimonides Hospital of Brooklyn, New York.\textsuperscript{6} Postmortem examination confirmed the original diagnosis of ventricular septal defect and pulmonary stenosis. The pulmonic valve was "completely atretic" and showed "no stoma of communication between the ventricles and the huge pulmonary artery." Infundibular stenosis was also present. There was a ventricular septal defect measuring 2.5 cm. in diameter immediately beneath the overriding aorta.

The main pulmonary artery measured 7 cm. in diameter, as did the primary branches in their maximum dimension. The sublavian-pulmonary anastomosis was open, measuring 2.5 cm. in diameter. There was an aneurysmal sac on the left wall of the main pulmonary artery measuring 4 cm. in average diameter from which a dissecting aneurysm of the pulmonary artery originated. This dissecting aneurysm, measuring 3 cm. in length, extended from a break in the intima 4 cm. distal to the pulmonary valve to an area on the outer surface of the pulmonary artery from which blood leaked on pressure. This area was within the pericardium and was apparently responsible for the 300 ml. of blood found in the pericardium. Extensive intimal fibrosis was present in both medium and small pulmonary arteries.

\textit{Comment.} This man had many features in common with the previous patient. It is probable that he, too, had pulmonary hypertension secondary to pulmonary vascular changes. The continuous murmur was present at the time of his last examination and the anastomosis was open at autopsy, but the rising hematocrit indicated that the volume of the shunt was decreasing.

\textit{Case 3: JHH \#B-16305}

This patient exhibited congestive heart failure and cardiac enlargement, but did not have the striking pulmonary artery enlargement seen in the first 2 patients. This syndrome, which is physiologically distinct from that exhibited by the first 2 patients, appears to be another consequence of a shunt of excessive size.

This patient was first seen at age 23 with a history of cyanosis since birth, diminished cardiac reserve during infancy and gradual improvement during adolescence. Physical examination, laboratory and x-ray studies, confirmed the diagnosis of tetralogy of Fallot. The preoperative chest film is seen at the left of figure 7.

A 7-mm. Potts anastomosis was created between the aorta and pulmonary artery in 1955. Following operation the hematocrit fell from 74 to 49 per cent and the patient was pleased with his symptomatic improvement. One year after operation the transverse diameter of the heart had increased to 60 per cent of the thoracic diameter (figure 7, right), as opposed to a preoperative value of 50 per cent (fig. 7, left).
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Two years after operation the patient complained of epigastric fullness and his liver was found to be enlarged. Five pounds of weight were lost following an injection of a mercurial diuretic. Diuretics and digitalis were required to control abdominal distention and breathlessness. On physical examination there was no cyanosis, a collapsing pulse was seen in the neck, the blood pressure was 168/75 mm. Hg in the right arm, and pistol shot sounds were heard over the femoral arteries. There was a forceful apical impulse with a sustained heave. A loud continuous murmur was heard over the entire chest. The chest x-ray (fig. 7) revealed a cardiothoracic ratio of 60 per cent, but the pulmonary artery was not unusually dilated. The electrocardiogram showed more evidence of left ventricular hypertrophy than had been seen in the preoperative records. Pulmonary pressure was 40/22 mm. Hg at cardiac catheterization, 28 months after operation. A large pulmonary blood flow was indicated by the oxygen saturation of 95 per cent in the pulmonary arteries.

**Comment.** The primary problem here appears to be excessive left ventricular work leading to cardiac enlargement and congestive failure. The physical findings in this third patient suggested that the physiologic prob-
TABLE 1.—Seventeen Patients with Late Hemodynamic Complications after Surgery

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age at operation</th>
<th>Operation</th>
<th>Age</th>
<th>Cardiac output ratio (%)</th>
<th>Pulmonary artery enlargement</th>
<th>Congestive failure</th>
<th>Systemic B.P. mm Hg</th>
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<tr>
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<td>6.5-mm. Potts</td>
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Potts anastomosis constructed between aorta and pulmonary artery.

R B-T, right Blalock-Taussig anastomosis; right subclavian to right pulmonary artery anastomosis.

L B-T, left Blalock-Taussig anastomosis; left subclavian to left pulmonary artery anastomosis.

R I, right innominate to right pulmonary artery anastomosis.

L I, left innominate to left pulmonary artery anastomosis.

There are 8 patients (cases 1, 2, 4-9) with pulmonary artery enlargement of the degree noted in patients 1 and 2. Patients 1 and 2 are the only ones in whom the hematocrit has risen and cyanosis returned. It seems likely that the other 6 patients also have pulmonary hypertension, which has reduced the volume of the shunt, but that the critical level at which cyanosis and polycythemia return has not been reached. Only 2 patients have shown signs of congestive heart failure (patients 1 and 3). Patient 10 is similar to patient 3 in that cardiac enlargement and a wide arterial pulse pressure exist, but patient 10 has shown no signs of congestive heart failure. Progressive increase in heart size is the only indication that the shunt is too large in patients 10 through 17. No meaningful estimate of the incidence of these complications can be made prior to the completion of the survey of all patients who have survived 10 years after operation.

**DISCUSSION**

There is probably a precise relationship between the severity of the original malformation in patients with the tetralogy of Fallot and the magnitude of the shunt necessary for optimal physiologic compensation. If the shunt is smaller than this optimal value and the pulmonary blood flow is not restored to normal levels, cyanosis will not be completely alleviated. If, on the other hand, the shunt is larger than this theoretical optimal value, the cyanosis will disappear and the patient will have an excellent initial result, but late complications may develop. There appear to be 2 physiologic sequences that may follow the creation of too large an anastomotic pathway.

First, part of the left ventricular output is directed through the anastomosis to the pulmonary circulation. Systemic flow must be maintained, and hence the left ventricular output and work must of necessity increase. If this work load exceeds the ability of the ventricular muscle to respond, congestive failure results. In this regard it is possible that either congenital or developmental de-
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fatalities of the myocardium may play a significant role. This sequence of events might be referred to as the arteriovenous fistula failure syndrome. Tachycardia, systolic hypertension, wide pulse pressure, cardiac enlargement, venous hypertension, hepatomegaly, and fluid retention are the salient features of this syndrome. Such was the clinical picture in the third patient.

Second, if pulmonary hypertension develops either due to pre-existing thrombotic occlusions or changes that are secondary to increased flow or pressure, a different physiologic sequence is seen. Pulmonary resistance increases, flow through the anastomosis decreases and, hence, the left ventricular work load is lessened. Thus, pulmonary hypertension might conceivably be considered to offer protection against the arteriovenous fistula failure syndrome. As pulmonary pressure rises, the pressure gradient between the systemic and pulmonary terminals of the anastomosis decreases; as these pressures approach equality, the flow through the anastomosis may cease. Pulmonary stenosis and right ventricular hypertension persist and right ventricular failure may develop, as in patient 1. As the volume of the shunt lessens, arterial oxygen saturation decreases, and polycythemia and cyanosis return. The clinical manifestations of this pulmonary hypertension syndrome are enlargement of the pulmonary artery on x-ray, diminution in intensity of the anastomotic murmur, rising hematocrit, increasing cyanosis, and possibly cough and hemoptysis.

We were not able to record pulmonary artery pressures in either patient 1 or patient 2. The clinical history and the microscopic examination of the lungs constitute the evidence upon which we base the diagnosis of pulmonary hypertension. Pulmonary hypertension has been documented in a similar patient to be reported by Rapaport and Leeds. Elevated pulmonary artery pressure was recorded at thoracotomy in a 46-year-old woman 8 years after a Blalock-Taussig anastomosis.

There are 2 plausible explanations for the pulmonary vascular changes responsible for the increase in pulmonary resistance. First, it is possible that these organized occluded vessels existed prior to operation. Similar changes were described in 1948 by Dr. Arnold Rich in patients with the tetralogy of Fallot who died prior to surgery. The development of pulmonary hypertension after operation might well be related to the extent of the preoperative damage to the pulmonary vasculature. An alternative explanation fits better with the clinical facts in the second patient, who developed pulmonary hypertension 10 years after operation. In this patient it seems likely that the vascular changes developed as a consequence of operation and were related to the increased pulmonary blood flow or pressure, as in other conditions characterized by a left-to-right shunt.

The pathogenesis of the pulmonary hypertension that develops in patients with congenital heart disease and increased pulmonary blood flow is not completely understood. A careful study of pulmonary vascular changes in patients who died after anastomotic operations would be a useful addition to currently available information regarding pulmonary hypertension in these circumstances. Such a study is currently in progress at The John Hopkins Hospital.

Two less likely explanations for the pulmonary vascular changes deserve consideration. It is theoretically possible that areas of stenosis in the pulmonary arteries distal to the anastomosis could be responsible for pulmonary hypertension, but the sequence of early improvement followed by evidence of decreasing shunt could not be explained on this basis. It is also possible that multiple small emboli from the anastomosis could have produced the pulmonary vascular changes, but there was no evidence of thrombus formation on the Potts anastomosis in the first patient.

The last 7 patients in table 1 are included only because of increasing heart size; all are asymptomatic. We suspect that they represent an earlier or less severe stage of the syndrome exhibited by the third patient. The heart size usually increases slightly during the first year after operation in patients with
a satisfactory postoperative course. The increase in size is to be expected because of the increased left ventricular work and also because of the patient's increase in physical activity. The heart size usually becomes stable after 1 year in those patients whose shunt is presumably of optimal size. Progressive increase in heart size beyond the first year in a patient with the tetralogy of Fallot is strong presumptive evidence of too large a shunt.

Consideration of the change in heart size may be useful clinically in the differentiation of a reduction in the shunt due to anatomic closure and a reduction in the shunt due to pulmonary hypertension. If cyanosis returns and the heart size remains small, it is likely that the anastomosis has closed anatomically. If, on the other hand, progressive enlargement of the heart precedes the return of cyanosis, the development of too large a shunt would appear more likely.

Eleven patients in this group of seventeen had end-to-end subclavian to pulmonary anastomoses. In 2 the innominate artery was used, and in 4 an aorta to pulmonary anastomosis was constructed. Thus, the larger anastomoses constitute more than one third of the patients in this group, representing a much higher frequency than in the entire operated population. It appears obvious, therefore, that these complications are more frequent with larger anastomoses. It should be stated in this regard that the Potts anastomoses in 3 of the 4 patients were larger than the usually recommended size, i.e., 6.5, 6.5, and 7 mm. in diameter. It is also worthy of emphasis that the anastomosis in the first patient increased in size from 6.5 to 12 mm. during the 4 years after operation. Increase in size of an anastomosis of smaller initial dimensions has not been proved but might conceivably occur. The greater length of the limiting lumen of a subclavian-pulmonary anastomosis makes it less likely that such an anastomosis would increase in size.

We have no experience with surgical therapy to alleviate these complications in the group discussed, all of whom have had the classic tetralogy of Fallot, but a situation that is physiologically indetical has been treated. A clinical picture similar to that presented by case 3, with cardiac enlargement, hepatic enlargement, and fluid retention, was seen in a 19-year-old boy with pulmonary stenosis and a single ventricle who had an 8-mm. Potts anastomosis. One year after the initial operation the Potts anastomosis was taken down and a left end-to-side subclavian to pulmonary anastomosis constructed. At the time of operation the Potts anastomosis was estimated to be 10 to 12 mm. in diameter. One year after the second operation the heart was still large but exercise tolerance had increased and heart failure was more easily controlled. Obviously, open heart correction of the primary malformation would be the treatment of choice for these complications but was not considered possible in the above-mentioned patient because of the single ventricle. The third case presented is considered a possible candidate for surgical correction of the primary malformation by means of open heart technic.

Summary

Two syndromes may develop as late hemodynamic complications of anastomotic operations for the tetralogy of Fallot. Both can be related to a shunt of greater than ideal size. The first is characterized by cardiac enlargement and congestive failure similar to that seen in patients with arteriovenous fistulae. The second is characterized by pulmonary hypertension, aneurysmal dilatation of the pulmonary artery, decreasing shunt, return of cyanosis, cardiac failure, and death.

Summario in Interlingua

Due syndromes occurre como tardive complicaciones de chirurgia anastomatic in casos del tetralogia de Fallot. Ambes pote esser relationate a un derivation de dimensiones plus que ideal. Le prime es characterisate per allargamento cardiac e disfallimento congestive simile a illo vidite in patientes con fistulas arteriovenose. Le secunde es characterisate per hypertension pulmonar, dilatation
aneurysmal del arteria pulmonar, decrescenza del derivation retorno de cyanose, disfalloimento cardiac, e morte.

REFERENCES

4. HALLE, J. A.; A study of second-sided systemic-pulmonary anastomoses for pulmonary stenosis with cyanosis. To be published.


The physical signs, radiologic findings, electrocardiographic changes and differential diagnosis of atrial septal defects are discussed and various clinical and catheterization data are presented for 100 patients studied by the authors. Of this group the diagnosis was proved by catheterization or necropsy in 66 patients, including 7 with anomalous pulmonary venous drainage and 8 others with pulmonary valvular stenosis in addition. In the other 34 patients the diagnosis seemed certain on clinical grounds. Observation of the course of these patients indicates that after infancy is passed the first 2 decades are relatively normal periods, but the prognosis in the fourth and fifth decades is much less good and only one half of these seen at the hospital are still well at 40 and less than a quarter at 50. Usually the heart is large, often of such size as would generally indicate a poor prognosis, but nearly all patients who survive the first year or so do well without the heart becoming any larger until 25 and more often 35 years of age. Then, in an increasing number, but not in all, the strain of the large right ventricular output produces increasing dyspnea and ultimately right-sided heart failure. In some patients pulmonary arterial pressure rises, reversing the shunt and producing central cyanosis and pulmonary artery thrombosis.
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