Surgical Management of Large Bronchial Collateral Arteries with Pulmonary Stenosis or Atresia

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SUMMARY

Single or multiple large bronchial collateral arteries may provide all or some of the pulmonary arterial blood flow in patients with proximal atresia of the pulmonary artery, and even in patients with only pulmonary stenosis. At the time of corrective surgery such arteries must be ligated in order to provide favorable operating conditions, to avoid cardiac overdistention during repair, and to prevent left-to-right intrapulmonary shunting postoperatively. Their ligation and control require precise preoperative definition of their number, origin, and course, and special intraoperative methods for their exposure. Associated hypoplasia of the pulmonary arteries may be severe enough to preclude corrective operation, but these hypoplastic arteries may enlarge in response to increase of blood flow through them resulting from a surgically created shunt.

Experience with 14 surgically managed cases of this type forms the basis for the report.

Additional Indexing Words

Left-to-right shunt Pulmonary blood flow Open heart surgery

Among patients having severe tetralogy of Fallot, in which the right ventricular outflow tract or pulmonary trunk may be atretic, are a few having large systemic arteries that arise from the aorta or its major branches and then enter the lungs. These provide pulmonary blood flow in parallel to that supplied via the true pulmonary arteries. They may communicate directly with those arteries or may be the only source of blood flow to portions of the lungs in which no true pulmonary arteries are present. The surgical management of such cases poses unique problems which we address here.

(Our interest is not directed toward two other groups of patients having bronchial collateral blood flow: those with diffuse small subpleural and hilar bronchial collateral vessels that have developed in response to cyanosis and inadequacy of pulmonary blood flow, and those with complete absence of pulmonary arteries, in whom the only arteries entering the lungs are bronchial collateral arteries. The former, encountered usually because of conditions in which pulmonary blood flow is reduced, pose no unique problem in management; and the latter we do not presently consider as candidates for surgical correction.)

These arteries of systemic origin that supply the lung have been variously called "true bronchial arteries,"2 "large aortcopulmonary collateral arteries,"3 and "large systemic arteries."4 It has been pointed out that the term "bronchial arteries" is embryologically and anatomically incorrect;4 "aortcopulmonary" may be inaccurate, since not infrequently these vessels do not originate from the aorta but from one of its branches such as the subclavian artery; and since all branches of the aorta are considered to be "systemic arteries," this terminology also seems deficient. We find, therefore, the term "large bronchial collateral arteries" to be the most exactly descriptive term available.

Clinically, the presence of large bronchial collateral arteries demands special attention. First, to leave them uncontrolled during operation would be perilous. During cardiomyotomy, intracardiac return of blood would be markedly excessive — amounting perhaps to the major portion of blood returning from the heart-lung machine — with resulting underperfusion of the patient and flooding of the surgical field. Overdistention of the heart, and especially of the left ventricle, might occur before and after cardiomyotomy when myocardial tone would be impaired as a result of hypothermia or ischemia, or both. Such overdistention could result in sarcomere disruption or subendocardial underperfusion, or both. If the patient survived operation despite these hazards, his postoperative course would be handicapped by the large left-to-right shunt of the bronchial collateral blood flow.
Table 1

Clinical Data from 14 Cases of Large Bronchial Collateral Arteries with Pulmonary Stenosis or Atresia Managed Surgically

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>Hemoglobin (g/dl)</th>
<th>Art. O2 sat (%)</th>
<th>QP/QS</th>
<th>Aortic arch</th>
<th>Associated features</th>
<th>Bronchial collaterals No.</th>
<th>Origin</th>
<th>Date surg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>4, M</td>
<td>17.2</td>
<td>79</td>
<td>R</td>
<td></td>
<td>Prey Blalock-Taussig shunt</td>
<td>3</td>
<td>Upper desc Ao</td>
<td>6-71</td>
</tr>
<tr>
<td>2</td>
<td>5, M</td>
<td>16.0</td>
<td>81</td>
<td>L</td>
<td>PDA</td>
<td></td>
<td>3</td>
<td>1, anom R subel 2, desc Ao</td>
<td>2-72</td>
</tr>
<tr>
<td>3</td>
<td>12, F</td>
<td>14.6</td>
<td>86</td>
<td>1.9</td>
<td>L</td>
<td>PDA</td>
<td>1</td>
<td>Upper desc Ao</td>
<td>5-73</td>
</tr>
<tr>
<td>4</td>
<td>12, F</td>
<td>18.8</td>
<td>77</td>
<td>0.6</td>
<td>L</td>
<td></td>
<td>2</td>
<td>Desc Ao</td>
<td>7-73</td>
</tr>
<tr>
<td>5</td>
<td>26, F</td>
<td>18.7</td>
<td>87</td>
<td>L</td>
<td></td>
<td></td>
<td>1</td>
<td>Desc Ao</td>
<td>8-73</td>
</tr>
<tr>
<td>6*</td>
<td>6, M</td>
<td>16.8</td>
<td>82</td>
<td>L</td>
<td></td>
<td>Hypoplasia of pulm arteries</td>
<td>4</td>
<td>Upper desc Ao</td>
<td>9-73</td>
</tr>
<tr>
<td>7</td>
<td>13, M</td>
<td>17.2</td>
<td>86</td>
<td>1.6</td>
<td>R</td>
<td></td>
<td>6</td>
<td>Upper desc Ao</td>
<td>9-73</td>
</tr>
<tr>
<td>8*</td>
<td>27, F</td>
<td>16.1</td>
<td>86</td>
<td>1.8</td>
<td>L</td>
<td>Waterston shunt; Mobitz II</td>
<td>4</td>
<td>Desc Ao</td>
<td>9-73</td>
</tr>
<tr>
<td>9</td>
<td>16, M</td>
<td>17.9</td>
<td>82</td>
<td>1.0</td>
<td>L</td>
<td></td>
<td>3</td>
<td>2, desc Ao 1, abd Ao</td>
<td>10-73</td>
</tr>
<tr>
<td>10</td>
<td>28, F</td>
<td>16.0</td>
<td>88</td>
<td>2.2</td>
<td>L</td>
<td>Potts shunt</td>
<td>1</td>
<td>Desc Ao</td>
<td>11-73</td>
</tr>
<tr>
<td>11</td>
<td>23, F</td>
<td>20.4</td>
<td>86</td>
<td>1.6</td>
<td>L</td>
<td>ASD, PDA, severe AI (prior SBE)</td>
<td>3</td>
<td>Desc Ao</td>
<td>12-73</td>
</tr>
<tr>
<td>12</td>
<td>3, F</td>
<td>18.9</td>
<td>74</td>
<td>1</td>
<td>R</td>
<td>Extreme hypoplasia pulm arteries, left PDA (R arch)</td>
<td>3+</td>
<td>Desc Ao</td>
<td>1-74</td>
</tr>
<tr>
<td>13</td>
<td>32, M</td>
<td>15.4</td>
<td>91</td>
<td>L</td>
<td></td>
<td></td>
<td>4</td>
<td>Desc Ao</td>
<td>2-74</td>
</tr>
<tr>
<td>14</td>
<td>3, M</td>
<td>17.7</td>
<td>65</td>
<td>0.3</td>
<td>R</td>
<td>Hypoplasia of pulm arteries</td>
<td>1</td>
<td>Desc Ao</td>
<td>3-74</td>
</tr>
</tbody>
</table>

*See text for brief case report.

Abbreviations: AI = aortic insufficiency; Ao = aorta; ASD = atrial septal defect; CO = cardiac output; Hgb = hemoglobin; LV = left ventricle; PDA = patent ductus arteriosus; PT = pulmonary trunk; QP/QS = pulmonary/systemic flow; RPA = right pulmonary artery; RV = right ventricle; SBE = subacute bacterial endocarditis; SVC = superior vena cava.

Second, to ligate the large bronchial collateral arteries via median sternotomy may be difficult because they usually originate in or course through the posterior mediastinum and often are multiple.

Third, not uncommonly the presence of this type of bronchial collateral artery is associated with hypoplasia of the true pulmonary arteries, the latter being too small to carry the normal pulmonary blood flow after repair at pressures compatible with survival.

We have reviewed our experience with this disorder to analyze the anatomic characteristics of the arteries, the methods used in management, and the problems encountered.

Clinical Material and Routine Management

The 14 cases are summarized in table 1. All of the patients were symptomatic because of effort intolerance and dyspnea of various degrees. All had continuous murmurs due to flow through the bronchial collateral arteries and — in cases 1, 8, and 10 — through surgically created shunts. Cyanosis was evident in most, but usually mild.

All were subjected to cardiac catheterization and angiography, the latter always including multiple injections into the thoracic aorta or selective injections into the large bronchial collateral arteries. Often these multiple injections were necessary to confirm
the presence of true pulmonary arteries and to define accurately the number, origin, and course of the large bronchial collateral arteries (figs. 1, 2 and 3). The intracardiac anatomy of each patient was similar to that seen in severe tetralogy of Fallot except for atresia of the pulmonary valve in 12 of the 14, extending various distances into the main pulmonary trunk of eight patients.

Exposure for Control
To provide exposure for isolating and ligating the bronchial collateral arteries, we preferred, where feasible, median sternotomy and dissection through the posterior pericardium or, via the pleural space, through the perihilar mediastinum. However, in six of the 11 cases of corrective repair it seemed that the bronchial collateral arteries would be inaccessible from this anterior approach, so a complementing lateral thoracotomy was performed on the side of the descending aorta.

This simultaneous approach from the front and from the side was first described by Doty et al. We have simplified it by positioning the patient on the operating table with a small sandbag under the hip and shoulder to elevate one side of the body some 30 to 45 degrees from the operating table. The arm was suspended on the drape support above the head. The whole of the anterior and lateral surface of the patient was then prepared and draped. Sufficient exposure was obtained for a lateral thoracotomy simply by rotating the table. The level of the incision was planned according to the level of the origins of the large bronchial collateral arteries depicted in the thoracic aortogram. The bronchial collateral arteries

<table>
<thead>
<tr>
<th>Approach for control bronchial collaterals</th>
<th>Atresia</th>
<th>Pulmonary outflow reconstr</th>
<th>Postop syst press (mm Hg)</th>
<th>Postop complication</th>
<th>Early result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Med sternotomy; bet. RPA &amp; L. atrium</td>
<td>None (patent)</td>
<td>Pericardial patch graft</td>
<td>75 105 40</td>
<td>Immediate thoracotomy for control bronchial art., low CO</td>
<td>Good</td>
</tr>
<tr>
<td>Med sternotomy; R pleural space &amp; post. mediastinum</td>
<td>Valve &amp; prox PT</td>
<td>Homograft Ao &amp; valve</td>
<td>58 110 38</td>
<td>Early low CO</td>
<td>Good; frequent resp infections</td>
</tr>
<tr>
<td>Med sternotomy; lateral to SVC</td>
<td>Valve &amp; PT</td>
<td>20-mm valved Dacron conduit</td>
<td>55 100 20</td>
<td>Infiltration R upper lobe</td>
<td>Good</td>
</tr>
<tr>
<td>Med sternotomy; post. mediastinum (branch to L lung not ligated)</td>
<td>Valve &amp; prox PT</td>
<td>20-mm valved Dacron conduit</td>
<td>65 90 40</td>
<td>Loud cont murmur due to small unligated bronchial art.</td>
<td>Good (but see text)</td>
</tr>
<tr>
<td>Simultaneous L thoracotomy</td>
<td>Valve</td>
<td>Pericardial patch graft</td>
<td>50 115 25</td>
<td>Left pleural effusion</td>
<td>Good</td>
</tr>
<tr>
<td>None: created aorto-R pulm shunt (Dacron)</td>
<td>Valve &amp; PT</td>
<td>None</td>
<td>None</td>
<td>Good</td>
<td></td>
</tr>
<tr>
<td>Simultaneous R thoracotomy</td>
<td>Valve &amp; prox PT</td>
<td>Pericardial patch graft</td>
<td>70 100 40</td>
<td>Mild cardiac failure</td>
<td>Good</td>
</tr>
<tr>
<td>Ligation at earlier L thoracotomy (2-73)</td>
<td>Valve</td>
<td>Pericardial patch graft</td>
<td>55 85 55</td>
<td>Pacemaker for bradycardia (Mobitz II)</td>
<td>Good</td>
</tr>
<tr>
<td>Simultaneous thoracotomy</td>
<td>Valve &amp; PT</td>
<td>20-mm valved Dacron conduit</td>
<td>85 115 23</td>
<td>Left pleural effusion</td>
<td>Good</td>
</tr>
<tr>
<td>Simultaneous thoracotomy</td>
<td>Valve</td>
<td>Pericardial patch graft</td>
<td>45 110 25</td>
<td>None</td>
<td>Good</td>
</tr>
<tr>
<td>Simultaneous L thoracotomy</td>
<td>Valve</td>
<td>Pericardial patch graft</td>
<td>65 90</td>
<td>None</td>
<td>Mod AI persists after repair perforated Ao cusp</td>
</tr>
<tr>
<td>None: modified R Blalock-T shunt (Dacron extension of subclavian)</td>
<td>None (patent)</td>
<td>None</td>
<td>None</td>
<td>Good</td>
<td></td>
</tr>
<tr>
<td>Simultaneous L thoracotomy (only 3 ligated)</td>
<td>Valve &amp; prox PT</td>
<td>25-mm valved Dacron conduit</td>
<td>35 95 22</td>
<td>Transient fever of uncertain origin; continuous murmur</td>
<td>Good</td>
</tr>
<tr>
<td>None: created R aorto-pulm shunt (Dacron)</td>
<td>Valve &amp; PT</td>
<td>None</td>
<td>None</td>
<td>Good</td>
<td></td>
</tr>
</tbody>
</table>
were isolated by incising the mediastinal pleura along the aorta and carefully dissecting up and down the aorta. The vessels were encircled temporarily by heavy silk ligatures.

Primary Surgery

The operating table then was rotated so that the anterior surface of the patient was horizontal. A conventional median sternotomy was performed and the heart was prepared for cardiopulmonary bypass. Once bypass was established, the table was rotated again; and via the thoracotomy incision the large bronchial collateral arteries were doubly ligated. Any additional surgically created shunts were closed by conventional techniques. Standard repair was then carried out: the ventricular septal defect was closed with a patch, and the pulmonary outflow tract was reconstructed with use of either a large onlay of pericardium (six cases) or a conduit of aortic homograft4 or of Dacron6 with an incorporated glutaraldehyde-preserved porcine semilunar valve.*

In three cases the true pulmonary arteries were considered too hypoplastic to allow corrective repair, and in them a palliative shunt operation was performed.

Problems of Management

Failure to Ligate

In addition to the above-mentioned intraoperative difficulties which would be imposed by failure to control flow through these arteries, the effect of the postoperative large left-to-right shunt and consequent burden to the heart is illustrated by a brief case description.

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*Hancock Laboratories.
PULMONARY ATRESIA AND BRONCHIAL ARTERIES

Figure 3

Cases 5 (left) and 7 (right). Note direct communication between bronchial collateral artery and pulmonary artery, at arrows. See also figure 7 A.

Case 1

A 4-year-old boy had an accurate diagnosis of severe tetralogy and three large bronchial collateral arteries originating just beyond the aortic isthmus of the right-sided aorta (fig. 4 A and B), with a functioning left Blalock-Taussig anastomosis. Through a median sternotomy incision, dissection between the left atrium and the right pulmonary artery permitted isolation and ligation of three supposedly separate bronchial collateral arteries. The left subclavian artery was also ligated. The ventricular septal

Figure 4

Case 1. A) Thoracic aortogram showing origin of three large convoluted bronchial collateral arteries from aortic isthmus. At initial operation, artery running to right was ligated twice — by mistake — and large artery running to left lung field remained open. B) Diagram showing three bronchial collateral arteries and left Blalock-Taussig anastomosis. C) Emergent aortogram made immediately after first operation, showing continued patency of largest bronchial artery. Note large surgical drainage tube in pericardial space and anterior mediastinum.
defect was closed and a pericardial patch reconstruction relieved the outflow tract obstruction.

Intracardiac return of blood was excessive throughout the repair, and pulmonary arterial hypertension was present after repair. The heart was overactive, and began to fail during wound closure. It was suspected that a bronchial collateral artery remained patent. Immediately after wound closure, emergency aortography confirmed the persistence of flow through the largest of the bronchial collateral arteries (fig. 4 C). The patient was returned directly to the operating room. Right posterolateral thoracotomy was performed; and the patent bronchial collateral artery was found and ligated, with prompt improvement in hemodynamics. The child then proceeded through a normal convalescence.

In retrospect it was clear that via the anterior approach one convoluted bronchial collateral artery had been ligated twice, giving rise to the mistaken impression that all vessels had been controlled. Had the dissection and adhesions related to the earlier shunt operation not occurred, the missed bronchial collateral artery could have been approached most easily through the median sternotomy via a wide anterior entry into the left pleural space.

**Cases 4 and 13**

The importance of ligation of each large bronchial collateral artery is emphasized further by experience in cases 4 and 13. In the former (fig. 5 left), one of the large bronchial collateral arteries proved to be inaccessible via the median sternotomy incision and so was not ligated; but the operative result is not as favorable as the others. The patient required assisted ventilation for 72 hours after operation, whereas all the others were extubated within 48 hours after surgery. Her post-repair right ventricular-to-left ventricular systolic pressure ratio (RV/LV) was among the least favorable of the group (table 1), and when discharged from the hospital she required digitals for control of mild congestive cardiac failure. She still has a continuous murmur heard over the back. It may become necessary to consider thoracotomy for control of this residual bronchial collateral artery.

In case 13 (fig. 5 right), initially only three of the four large bronchial collateral arteries could be found and ligated via the left thoracotomy incision. After cardiotomy, there was so much intracardiac return of blood as to complicate the repair and it was necessary to institute profound hypothermia and lowering of the perfusion rate. Immediately after repair, a second and more thorough dissection of the aorta revealed the remaining patent collateral originating from its least accessible right posterior aspect, and ligation finally was accomplished. However, a continuous murmur was heard posteriorly after operation, and an aortogram showed only subtotal ligation of the artery that had been difficult to expose.

**Selection of Approach to Large Bronchial Collateral Arteries**

Three possible approaches for ligation of these arteries are considered.

1) If the target artery or arteries lie directly behind the posterior pericardium, cephalad to the left atrium, where they can be exposed by incising the posterior pericardium, one may approach via the median sternotomy incision and pericardial space.

2) If the artery or arteries traverse at or cephalad to the hilus of either lung, they can often be exposed for ligation — again via the median sternotomy — by incising the anterior mediastinal pleura and dissecting the hilus while retracting lung and mediastinum. If a right descending aorta is present, even bronchial collateral arteries arising from the aorta caudal to the hilus can be exposed by anterior retraction of the lung via an incision in the anterior mediastinal pleura.

3) Combined thoracotomy (on the side of the descending aorta) and median sternotomy were re-
quired most often in our experience. The level of the thoracotomy is determined by the circumstances of the case. The pleura and adventitia overlying the aorta are incised, and dissection is carried around the aorta superiorly and inferiorly until each artery demonstrated by preoperative study is identified and prepared for ligation.

Relief of Pulmonary Atresia

If the atretic segment of the pulmonary artery is short — a fibrous cord extending no more than 1 to 1.5 cm from the infundibular space proximally to patent pulmonary trunk distally — we have preferred to relieve the obstruction by patch-graft reconstruction rather than by a valved conduit. An incision is carried from the arteriotomy site to the ventriculotomy site, cutting into the fibrous core of the arterial segment so that the epicardial edges of the incision can separate by a centimeter or so. Then a wide and long pericardial patch is sutured to the entire length of the combined cardiomyotomy-arteriotomy, being stitched to the epicardial edges of the incised fibrous core. This repair was possible in five of our ten patients who had atresia at the origin of the pulmonary trunk. If the distance between the right ventricular infundibulum and patent pulmonary artery is too great, we prefer to insert a valved woven Dacron conduit. We also prefer a conduit if there is significant elevation of pulmonary vascular resistance.

Hypoplasia of Pulmonary Arteries

Among patients having large bronchial collateral arteries can be found the entire range of pulmonary artery development, from effective absence (agenesis or atresia), previously known as type IV truncus arteriosus, through hypoplasia to normality. Because up to some point along this spectrum the hypoplasia is uncorrectable (as in cases 6, 12, and 14), consideration must be given to the problem. For the present this point of transition is a matter of judgment, and guidelines are vague. But to illustrate future hope in cases where the pulmonary arteries are clearly too hypoplastic to allow successful corrective operation, one of our patients is of unique interest.

Case 8

This 27-year-old woman had undergone limited right heart catheterization at another institution when she was eight, and had received a diagnosis of tetralogy of Fallot. Because of increasing restrictions on her activity, cardiac catheterization had been repeated (again elsewhere) when she was 20. That resulted in a diagnosis of pulmonary trunk atresia, and surgery was undertaken to form a shunt from the superior vena cava to the right pulmonary artery (Glenn anastomosis). However, the right pulmonary artery was found to be diminutive — less than 5 mm in diameter — and very large bronchial collateral arteries were seen in the hilus of the right lung. So an anastomosis was performed between the ascending aorta and the right pulmonary artery. The surgeon noted immediate enlargement of the right pulmonary artery to 1 cm in diameter. The patient’s symptomatic status improved dramatically, but over the next few years it deteriorated again.

She was referred to the Mayo Clinic. Repeat cardiac catheterization and angiocardiography showed normal-sized right and left pulmonary arteries, atresia of the pulmonary valve, and the functioning shunt established previously. In addition there were four large bronchial collateral arteries arising from the descending thoracic aorta (fig. 6 A, B, C).

Surgical correction in two stages was planned. In the first, with left thoracotomy, the four large bronchial collateral arteries were ligated. The patient recovered from this operation, but noted some decrease in her exercise tolerance. She returned seven months later for definitive repair, which consisted of closure of the communication between the aorta and right pulmonary artery, pericardial patch repair of the right pulmonary artery at this site, closure of the ventricular septal defect, and reconstruction of the pulmonary outflow tract with pericardium. Recovery was rapid. The only complication was bradycardia due to a Mobitz II heart block that had been present intermittently for six years. A permanent pacemaker was implanted, and since then she has done well from the cardiovascular standpoint. (Six months postoperatively, a right axillary lymph node was found to contain metastatic carcinoma, primary site unknown.)

The experience with this patient, whose hypoplastic pulmonary arteries enlarged to normal size in response to a large shunt, prompted the deliberate adoption of a similar staged procedure for three more recently encountered children (cases 6, 12, and 14) with similarly hypoplastic pulmonary arteries.

Case 6

A boy 6 years old when first examined here had suffered repeated attacks of pneumonia. Cyanosis and clubbing were noted, and cardiac catheterization revealed arterial desaturation and high cardiac output. Angiocardiography showed a ventricular septal defect and pulmonary arterial atresia. Pulmonary blood flow was derived mainly from a large bronchial collateral artery that arose just beyond the aortic isthmus and communicated directly with the left pulmonary artery. In addition, there were three large bronchial collateral arteries arising from the descending thoracic aorta. The right and left pulmonary arteries were seen to be confluent but hypoplastic and multiple peripheral stenoses were present, the most severe of which was just beyond the first branch of the right pulmonary artery (fig. 7 A and B).

In view of the experience gained from study in case 8 (prior to operation in this case), where the pulmonary arteries enlarged after a systemic-to-pulmonary shunt, it was decided to create a shunt between the aorta and the distal right pulmonary artery. Operation consisted of insertion of a knitted Dacron tube, 6 mm in diameter, from the ascending aorta to the right pulmonary artery. The distal end-to-side anastomosis was made across the entire severely stenosed segment, which had a diameter of 2 mm, thus enlarging the stenosis itself by the orifice of the graft. Flow in the graft was found to be 2 L/min. Temporary occlusion of the right pulmonary artery beyond the graft showed that 80% of this flow was to the left lung and to the right upper lobe, while 20% was to the middle and lower lobes on the right.

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Case 8. A) Demonstration of normal-sized right pulmonary artery by contrast medium injected through catheter passed via Waterston anastomosis (ascending aorta to right pulmonary artery, side-to-side). At original operation seven years earlier, this artery had been recorded as less than 5 mm diameter (hypoplastic). B) Demonstration of large bronchial collateral artery by selective injection from descending aorta. C) Diagram of central arteries.

Postoperatively the patient did well, except for mild cardiac failure that necessitated digitalization.

In the future, angiocardiography will be repeated to determine whether the pulmonary arteries have dilated and grown. If so, definitive repair will be effected and the bronchial collateral arteries ligated.

The problems and management in cases 12 and 14 have been closely similar (figs. 8 and 9). In case 10, a Potts anastomosis had been created (fig. 10).

Comment

Differentiation from Small, Diffuse Collaterals

Interesting differences exist between the two types of bronchial collateral arteries, namely the large arteries that are the topic of this report and the small, diffuse arterial collaterals. The former apparently originate during embryogenesis, since they may be fully developed in childhood (our cases 1, 6, 12, 14) or even infancy (four months of age in the series of

Case 6. A) Hypoplasia of pulmonary arteries, which filled directly from bronchial collateral artery, at arrow (compare communication in figure 3). B) Aortogram, showing also peripheral stenosis in right pulmonary artery.
PULMONARY ATRESIA AND BRONCHIAL ARTERIES

Figure 8

Case 12. Hypoplasia of pulmonary arteries (non-confluent), of which left arises from long patent ductus that originates from left subclavian artery (right aortic arch).

Jefferson et al.⁴), whereas the latter develop in response to cyanosis and inadequacy of pulmonary blood flow after birth and progress with time. The large bronchial collateral arteries represent persistence and enlargement of the primitive (fifth-week) intersegmental arteries, which originate from the dorsal aorta and connect with the vascular spaces of the lung bud before the pulmonary arteries develop from the sixth aortic arches.⁴ The abnormal persistence of these intersegmental bronchial collateral arteries in embryos where the pulmonary arteries fail to develop centrally, or to develop at all, could be a response to the lack of normal patency of the pulmonary arterial tree, or could be a separate concomitant developmental fault. The site of origin and course of these bronchial collateral arteries seem to be entirely random, with no similarity among any of our patients.

Injection of contrast medium into specific large bronchial collateral arteries demonstrates three basic patterns of blood flow. Some (pattern 1) anastomose with the patent pulmonary arteries at the hilus and thus opacify the central and thence peripheral pulmonary arterial tree. Bronchial collateral arteries of this type may then continue beyond the anastomoses to terminal branching in the pulmonary parenchyma, thus effecting a circulation parallel to the pulmonary distribution. The other patterns appear when contrast medium injected into a large bronchial collateral artery does not opacify pulmonary arteries at the hilus. This means the collateral artery does not supply blood directly to the central pulmonary arteries, but rather distributes it to segments or lobes directly — either in parallel to true pulmonary arteries (pattern 2) or as sole provider of a segment or lobe if there are no pulmonary arterial branches to that segment or lobe (pattern 3).

Each of these patterns was demonstrated in this experience, and more than one pattern was often en-
countered in a given patient. It is of interest that in case 13 the collateral artery not identified before perfusion was one not anastomosing with the pulmonary artery, and that the excessive flow of blood during cardiacotomy and pulmonary arteriotomy was into the heart via the pulmonary veins and left atrium, rather than into the pulmonary artery.

Preoperative Studies and Planning

This experience allows formulation of a plan for management of patients in whom large bronchial collateral arteries are demonstrated. First, precise mapping of the number, origin, course, and size of each such artery must be accomplished by preoperative opacification of the aortic arch and descending thoracic aorta, at the least, and preferably by selective injection of all large bronchial collateral arteries, or certainly all obscure ones.

Second, adequacy of the central and peripheral pulmonary arteries for reconstruction by the procedure described above must be assessed with regard to lumen size. A tentative and inadequately tested rule of thumb is that if the right and left main pulmonary arteries have a total combined cross-sectional area greater than about one-half the cross-sectional area of the aorta just above the diaphragm, they probably are adequate to receive cardiac output after repair without excessive right ventricular pressure.

If the pulmonary arteries appear to be too hypoplastic for corrective operation, we prefer to insert a Dacron tube graft (6 mm in diameter for children of about 1 square meter body surface area or more, and 5 mm if smaller) from ascending aorta to right pulmonary artery, end-to-side, thus creating a large but controlled shunt in the hope that progressive development of the pulmonary arterial tree will result.

There are patients in whom no true pulmonary arteries may be identified, even after appropriate extensive study, including descending aortography (which achieves a high concentration of contrast material in the aortic blood) and selective injection of large bronchial collateral arteries. Such patients are not included in this review, for preoperative study showed identifiable pulmonary arteries in all 14 patients, as illustrated in the figures.

Third, if the pulmonary arteries are adequate for complete repair, a plan of approach for control of the large bronchial collateral arteries must be devised after study of their exact locations, using either left or right thoracotomy in conjunction with median sternotomy, or the latter alone, as described above.

Addendum

In the period following the series of patients here reported, the authors have cared for an additional seven similar patients, whose ages ranged from 7 years to 20. A left thoracotomy was required in two. All had more than one large bronchial collateral artery; and a successful result was achieved in each, further substantiating the remarkably favorable outlook for patients of this type.

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