Axillary Arteriovenous Fistula

A Means of Supplementing Blood Flow through a Cava-Pulmonary Artery Shunt

By WILLIAM W. L. GLENN, M.D., AND JOHN E. FENN, M.D.

SUMMARY
A right axillary A-V fistula has been made in a 13-year-old patient with tricuspid atresia who, after undergoing two unsuccessful attempts in infancy to establish a left Blalock shunt and having had a superior vena cava-right pulmonary artery (SVC-RPA) shunt at age 3, showed increasing disability and profound cyanosis (HCT 80%). One year following creation of the fistula there is marked increase in exercise tolerance, a decrease in HCT to 56%, and an increase in pO₂ from 57 to 105 mm Hg.

Creation of an axillary A-V fistula in patients with an SVC-RPA shunt is an effective means of supplementing blood flow to the right lung by increasing the flow through the shunt and by giving it a pulsatile character, and, eventually, by decreasing the blood’s viscosity. Axillary A-V fistula may be required by many patients with a cava-pulmonary shunt who have inadequate perfusion of the contralateral lung and clinical deterioration.

Additional Indexing Words:
Superior vena cava-right pulmonary artery shunt SVC-RPA Cava-pulmonary artery anastomosis Tricuspid atresia

AFTER AN INITIAL period of clinical improvement following a cava-pulmonary artery shunt, usually lasting 4–8 years, signs and symptoms of hypoxia have usually reappeared. Factors have been the decrease in blood flow to the contralateral lung following spontaneous closure of an intracardiac defect (atrial and ventricular septal); closure of a patent ductus or surgically created ductus; obstruction of the pulmonary outflow tract; a decrease in flow to the ipsilateral lung from expansion of collateral veins to the inferior vena cava following elevated pulmonary vascular resistance; or an increase in viscosity of the blood due to hemoconcentration.

The treatment of hypoxemia has usually been the creation of a systemic artery-pulmonary artery shunt to the contralateral lung.1,2 Occasionally total repair of the underlying defect has been possible, leaving the cava-pulmonary artery shunt intact.3 Those cases in which there was already a systemic artery-pulmonary artery shunt to the contralateral lung and the shunt had partially or completely closed presented a particular problem.

Some years ago, investigating other shunting procedures in the laboratory in anticipation of this problem, we made a carotid artery-jugular vein fistula in five animals who had undergone cava-pulmonary artery anastomosis 14–30 months earlier. Flow to the right lung through the cava-pulmonary artery shunt, measured at intervals following opening of the fistula, was 2.3–3.7 times greater 1–1½ years later. Severe congestive failure developed in one animal with a large (10 mm) fistula. This failure disappeared when the fistula was closed.
Closure of the fistula caused a decrease in flow to the lung but, temporarily at least, flow through the lung was still greater than before the fistula, suggesting a diminished pulmonary vascular resistance in response to the fistula. In none of the animals was an increase in collateral circulation between the cavae observed on gross examination. Since one would expect expansion of these veins in the presence of an A-V fistula, this point deserves further investigation.

The procedure was applied in a 13-year-old girl with tricuspid atresia who at the age of 3 had had a caval-pulmonary artery shunt.

**Case Report**

G.P., no. 54-35-47, Yale-New Haven Hospital, a white female, age 13, was admitted to Yale-New Haven Hospital in October, 1960, at 2½ years of age with tricuspid atresia. She was noted to be cyanotic at birth. At 2 months of age she developed the first of many cyanotic spells characterized by tachypnea, limpness, and eye rolling. Physical milestones were grossly retarded and the child was extremely cyanotic at rest. At 13 months of age cardiac catheterization and angiography at another medical center revealed tricuspid atresia, normal position of the great arteries, and a small ventricular septal defect. The hematocrit level was 78%, and she was having four severe hypoxia attacks per month. A systemic artery-pulmonary artery end-to-end anastomosis was made on the left side. The pulmonary artery measured only 8 mm in diameter and no continuous murmur was ever heard. Cyanosis remained severe and she could neither crawl nor walk.

At 2½ years of age she was referred to Yale-New Haven Hospital for consideration for a superior vena cava-right pulmonary artery shunt. The left subclavian artery-pulmonary artery anastomosis was first explored to determine if circulation could be reestablished. At operation both vessels appeared extremely small and the anastomosis was thrombosed. After excision of the thrombosed segment a new anastomosis was made, but again no continuous murmur was heard postoperatively and the patient's clinical condition remained unchanged.

Accordingly, at 3 years of age a cava-pulmonary artery anastomosis was made. The patient was cooled to about 33°C. To obtain a pulmonary artery larger than the right branch, the main pulmonary artery was divided at its origin from the right ventricle and pulled under the superior vena cava and there anastomosed end-to-side to the cava at the surgically enlarged site of origin of the azygos vein. The cava was then doubly ligated above the right atrium (fig. 1).

There was marked improvement. Within 3 months she was crawling, within 4 months walking, color improved, and the hematocrit dropped to 52% in 6 months compared to 72% preoperatively. Postoperative angiograms done at 1 year and at 5 years showed a normally patent cava-pulmonary artery anastomosis with growth commensurate with the adjacent vessels. But she developed slowly, maintaining about a 25 and a 10 percentile level for her age in height and weight, respectively. Physical exercise grew more difficult with age. Cyanosis gradually increased. The hematocrit rose to 63% at age 10, reaching 80% at age 13. She became severely incapacitated by intense fatigue and recurrent headaches. Improvement in oxygenation was mandatory. After ruling out a shunt between the descending aorta and a peripheral branch of the left pulmonary artery, we decided to create an A-V fistula on the right side to increase the blood flow.

**Figure 1**

Patient G.P. Diagram of the cardiopulmonary circulation following anastomosis of the superior vena cava to the right pulmonary artery. The obliterated left subclavian artery-pulmonary artery anastomosis is also illustrated.
to the right lung through the cava-pulmonary artery shunt. This was done in April, 1971.

The fistula was made between the axillary artery and vein as they lay side by side, well anterior to the brachial plexus, in the loose areolar tissue just caudal to the midpoint of the clavicle and just beneath the clavicular head of the pectoralis major muscle. An 8-mm long incision was used for the anastomosis (fig. 2). *(In a smaller child a 6-mm incision would probably be more judicious. If a shunt is too large, it would be relatively simple to reopen the wound and close down the size of the fistula through a transvenous approach after cross clamping the artery and vein.)

Upon removal of the clamps there was a prominent continuous murmur and thrill, which unfortunately disappeared after a few minutes. The vessels were reclamped, the anterior suture line was cut out, and a small clot was removed from the anastomosis site. Heparin was administered intravenously (5000 units) and the anastomosis again completed. This time the shunt remained patent. As a precaution the heparin was not reversed, but despite free drainage of the wound a large hematoma had to be removed later that evening. There is a loud continuous murmur and thrill over the site of the fistula which the patient can appreciate but does not find objectionable.

There has been progressive improvement in both her clinical condition and laboratory findings. Her color is markedly improved. She has gone to school daily, has been on the student council, and only rarely experiences a headache. Digitalis was begun postoperatively because of a tachycardia of 100–110 beats/min. The rate is now consistently below 100. There has been no increase in heart size or change in the ECG pattern since operation. The pulmonary vasculature in the right lung has increased moderately.

The results of the laboratory studies are tabulated in tables 1 and 2. Following the creation of the fistula there has been a progressive fall in the hematocrit and rise in arterial oxygen tension coinciding with an improved clinical condition.

Lung scans with 131I-MAA showed a relative increase in the number of particles trapped in the right lung following operation when compared with the left lung. The particles were more evenly distributed to the right lung after the fistula was made than before. Venous pressure increased in both arms but less so in the left arm.

Discussion

It was apparent early in the development of the cava-pulmonary artery shunt that it was a low-pressure, nonpulsatile, low-flow shunt, having flow characteristics similar to those of pulmonary stenosis.7 Despite the low flow, the long-term benefits of the cava-pulmonary artery shunt, particularly when there is reasonably good flow to the other lung, have been highly satisfactory in most cases. Sixty-three patients with complex cardiac deformities have had anastomosis of the superior vena cava to the pulmonary artery in our clinic since 1958. Fifty-six of these survived operation and there have been only two late deaths, both in adults. In neither case was death related to the failure of the cava-pulmonary artery shunt. On the basis of this excellent palliation we continue to recommend the procedure for patients with uncorrectable malformations of the right side of the heart and diminished blood flow to the lungs, the principle one being tricuspid atresia. Patients with certain other malformations where correction is clearly not possible now, nor thought to be in the future, may benefit from this palliative procedure. Examples include corrected transposition of the great vessels with pulmonary stenosis, certain cases of pulmonary atresia, transposition of the great vessels with a hypoplastic left ventricle and

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*We are grateful to Dr. Emile Holman for his encouragement in the clinical application of this operation.

**Figure 2**

*Patient G.P. Anatomic location of the right axillary artery and vein and the site selected for making an A-V fistula. The technic used to join the artery and vein is illustrated.*

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pulmonary stenosis, and other complex anomalies of the right side of the heart.

In addition to the usual supplemental operations to improve oxygenation, i.e. widening of the intraatrial communication and systemic artery-pulmonary artery shunt to the other lung, some, possibly many, patients with cava-pulmonary artery shunts will eventually require another operation. As demonstrated in the patient reported here, the creation of an A-V fistula between the right axillary artery and vein is an effective means of improving oxygenation. It increases the blood flow through the cava-pulmonary artery shunt and gives the flow a pulsatile character and, eventually, decreases the viscosity of the blood.

At this point in time it is our opinion that a patient with a cava-pulmonary artery shunt who does not maintain adequate oxygenation of the blood as shown by an arterial oxygen tension of less than 60 mm Hg while breathing 100% oxygen for 10 min, a hematocrit level above 70%, and in whom improvement in oxygenation by other operative procedures cannot be expected, needs an axillary A-V fistula.

We do not, of course, know the long-term effects of the fistula. Subacute bacterial endarteritis or endocarditis, congestive heart failure, and expansion of venous collaterals between the superior and inferior vena cavae are particularly to be feared. Should the fistula need to be made smaller due to the development of high-output failure of the left ventricle it could be reduced in size by suturing, using a transvenous approach to the fistula.

### Table 1

Pre- and Post-A-V Fistula Studies on Arterial Oxygen Tension, Hematocrit Level, and Upper Extremity Venous Pressure

<table>
<thead>
<tr>
<th>Date of study</th>
<th>Arterial pO2 (mm Hg)</th>
<th>Hematocrit (% packed rbc)</th>
<th>Venous pressure (mm saline)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Rt arm</td>
</tr>
<tr>
<td>4/22/71</td>
<td>57</td>
<td>80</td>
<td>172</td>
</tr>
<tr>
<td>4/27/71</td>
<td>Right axillary A-V fistula</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5/12/71</td>
<td>64</td>
<td>61</td>
<td>330</td>
</tr>
<tr>
<td>10/13/71</td>
<td>80</td>
<td>57</td>
<td>350</td>
</tr>
<tr>
<td>3/16/72</td>
<td>105</td>
<td>56</td>
<td>—</td>
</tr>
</tbody>
</table>

*At rest breathing 100% O₂ for 10 min.

### Table 2

Lung Scans with ¹³¹I MAA: Ratio of Radioactive Counts

<table>
<thead>
<tr>
<th>Date of study</th>
<th>Right lung (upper : lower)</th>
<th>Both lungs (right : left)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4/22/71</td>
<td>1:2</td>
<td>6:1</td>
</tr>
<tr>
<td>4/27/71</td>
<td>Right axillary A-V fistula</td>
<td>—</td>
</tr>
<tr>
<td>5/12/71</td>
<td>—</td>
<td>10:1</td>
</tr>
<tr>
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</tr>
<tr>
<td>3/16/72</td>
<td>—</td>
<td>13:1</td>
</tr>
</tbody>
</table>

*The decreased perfusion of the right lung through the cava-pulmonary artery shunt, pre-fistula, was coincident with a fall in arterial pO₂ and rise in hematocrit. Similarly, the increase in perfusion of the right lung after the fistula was made corresponds to improvement in the same parameters. There is also better perfusion of the right upper lung field after the fistula was made.

### Addendum

On September 13, 1972, 16 months postoperatively, the patient was restudied and the following results were obtained.

Arterial blood sample (breathing 100% O₂): pO₂ 110 mm Hg; pCO₂ 34 mm Hg; hematocrit 53%; hemoglobin 17.6 g.

Venous pressure: right arm 215 mm saline; left arm 180 mm saline.

Chest X-ray: increased vascularity of the right lung; no increase in heart size following creation of A-V fistula.

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