Exchange of Blood Between Pulmonary and Systemic Circulations via Bronchopulmonary Anastomoses

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The existence of bronchopulmonary anastomoses has led to the general belief that the blood from the aorta and bronchial arteries can reach the pulmonary vessels and left atrium. There is reasonable evidence to suggest that the reverse can occur. Three observations pertain to the flow of blood from the pulmonary artery to the bronchial veins and right atrium: (a) In 1931 Berry and Daly1 perfused the entire pulmonary arterial and bronchial vessels of the dog and noted that in two dogs, about 1 per cent of the pulmonary arterial blood finds its way to the right atrium. (b) More recently, an isolated right lung preparation was perfused simultaneously through the pulmonary and bronchial vascular systems.2 In each of three dogs, the maximal contribution of pulmonary arterial blood to right atrial flow were 2, 8, 7.3, and 7.5 per cent of the total pulmonary blood flow. (c) The heart-lung preparation was modified so that the bronchial arterial flow was maintained and bronchial venous flow was collected.3 The total bronchial venous flow was reduced by 30 to 50 per cent when bronchial arteries were occluded. This suggests that under the conditions of these experiments, about 50 to 70 per cent of bronchial venous flow arose from blood in the pulmonary circulation.

The demonstration of pulmonary to bronchial shunt as reported above is based on temporary interruption of bronchial circulation in dogs in which the central nervous system and systemic circulation have not been maintained. The pulmonary and bronchial circulations were supplied by perfusion pumps in two studies (a and b) and by the animal's own heart in one (c). The experiments reported herein pertain to anesthetized dogs with intact central nervous system and systemic circulation to test the existence of pulmonary to bronchial shunt without interruption of bronchial circulation. The dye-dilution technique originally applied by Cudkowicz et al.4 for measuring bronchial to pulmonary flow was modified to allow measurements of exchange of blood in either direction, i.e., bronchial to pulmonary and pulmonary to bronchial.

Methods

Mongrel dogs were anesthetized with morphine sulfate (2 mg./Kg.) and chloralose (70 mg./Kg.). The trachea was cannulated to allow artificial respiration by means of a Starling Ideal pump. The following catheters were inserted: (a) Azygos vein: a polyethylene catheter (1.5 mm. diameter) was introduced through one jugular vein and guided until its tip was in the distal portion of the main pulmonary artery, (b) Pulmonary artery: a Cournand catheter was inserted into the carotid artery and guided manually until the tip was in the azygos vein about 1 cm. from its junction with the superior vena cava; the chest was opened via a right fifth intercostal incision to allow the insertion of such catheter as well as of the next one. (c) Left atrium: a second thoracotomy incision was performed (fourth left intercostal) to insert a polyethylene catheter (1.5 mm. diameter) through the atrial appendage. The tip had multiple holes to assure uninterrupted sampling of blood. (d) Left ventricle: another Cournand catheter was inserted into the carotid artery and guided manually until

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Supported by U. S. Army Medical Research and Development Command, Department of the Army, under Contract DA-49-193-MD-2093.

Dr. Aramendia is a Fellow of the Consejo Nacional de Investigaciones Cientificas y Tecnicas, Argentina.

Dr. Martinez is a Research Fellow under U. S. Public Health Service Training Grant No. 20-474.

Received for publication June 22, 1962.
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its tip was inside the left ventricle. (e) Femoral arteries: each artery contained a polyethylene catheter; one for sampling of arterial blood, the other for continuous recording of systemic blood pressure by a Statham strain gauge. Both chest incisions were closed to allow spontaneous respiration.

Cardio-green was injected in a volume of 1 ml. containing 2.5 mg. of the dye and was flushed with 5 ml of saline. The continuous withdrawal of blood was maintained by a Harvard pump at the rate of 16 ml./min. The blood was then passed through a Waters cuvette densitometer (XC-250A) for continuous recording of dye concentration on a direct, rectilinear writer system (Sanborn Polysio). The baseline was set at the top of the record, and the increasing concentration of the dye caused a downward deflection of the record. Nevertheless, in the following sections, the point of maximal concentration of the dye will be referred to as peak. The cardiac output was calculated by the method devised by Hamilton and applied by Wood to the densitometer.5

Four groups of dogs were used: (1) normal anesthetized dogs; (2) dogs subjected to inhalation of steam for 30 seconds to induce thermal injury to the lungs6; (3) dogs with ligation of the left pulmonary artery performed eight to nine months previously under pentobarbital anesthesia, 20 mg./Kg., I.V. (performed by Dr. G. Peskin, Harrison Department of Surgical Research, University of Pennsylvania); (4) dogs which had received four to five months previously an injection of 5 Gm. of glass beads (300 μ diameter) through a catheter in the main pulmonary artery introduced by fluoroscopic guidance. These dogs were under pentobarbital anesthesia and were allowed to recover.

Results

PULMONARY TO BRONCHIAL FLOW

The detection of shunt from the pulmonary artery to the bronchial vein was attempted by injection of Cardio-green into the main pulmonary artery while withdrawing continuously a sample of blood from the azygos vein, into which the bronchial vein drains. The search was conducted in three steps:

1. Creation of Artificial Shunt Between the Pulmonary Artery and Azygos Vein

In one dog, the proximal portion of one lobar artery was connected to a vein draining into the azygos vein by means of plastic tubing. The injection of dye into the main pulmonary artery resulted in a biphasic curve represented in figure 1 A. There was an initial peak followed by a delayed peak. The former disappeared when the injection was repeated while the artificial shunt was clamped; the delayed peak persisted (fig. 1 B). These results present experimental support for the possibility of demonstrating a pulmonary artery to azygos vein shunt, provided that the peak concentration of the dye transmitted by the shunt can occur sooner than that following the flow of the dye through the existing channels (i.e., pulmonary artery to left side of the heart to aorta to intercostals to azygos vein).

2. Dye-Dilution Curve from Azygos Vein

The next four dogs did not have an artificial shunt, and the normal flow of blood in the azygos vein was sampled by a catheter. The curves of dye concentration in the azygos vein showed only a single peak (table 1). The appearance of a single peak made it impossible to distinguish a shunt similar to the one created artificially. The single peak did not support the existence of a pulmonary to bronchial shunt but did not exclude its existence. If the shunt was functioning, the single peak means that the rate of flow in the shunt was slower than the rate of flow of systemic blood reaching the azygos vein, i.e., the time of appearance of the dye traveling the shunt coincided with the time of appearance of the blood traveling via the usual channels: pulmonary artery to vein to left ventricle to aorta to intercostals to azygos vein.
Dye-Dilution Curve from Azygos Vein in Anesthetized Normal Dogs

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Weight Kg.</th>
<th>All Injections into pulmonary artery except:</th>
<th>Dye-dilution curve from azygos vein</th>
<th>Peak concentration mg./L.</th>
<th>Peak concentration mg./L.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>25</td>
<td>(No artificial shunt)</td>
<td>11 16 0.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>23</td>
<td>(Open artificial shunt)</td>
<td>3 6 0.42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>21</td>
<td>Left ventricle</td>
<td>9 24 1.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>Left ventricle</td>
<td>10 21 2.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>Left ventricle</td>
<td>11 25 1.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6†</td>
<td>17</td>
<td>Left ventricle</td>
<td>12 26 1.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>19</td>
<td>Left ventricle</td>
<td>18 46 1.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left ventricle</td>
<td>12 33 2.4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*See also figure 1.
†See also figure 2.

3. Injection of Dye into the Left Ventricle

The next step was to compare the dye-dilution curve of blood in the azygos vein following the injection into the pulmonary artery with that into the left ventricle. The latter would bypass any pulmonary artery to bronchial shunt so that any difference between the two curves would mean the operation of the shunt. In two dogs, the initial comparison did not show any differences which suggested the participation of a pulmonary to bronchial shunt (dogs 6 and 7 of table 1). The curve from the left ventricular injection appeared three to six seconds earlier than that from the pulmonary arterial injection, but despite this gain in time, the curve failed to show a double peak. One of the two dogs suffered a spontaneous reduction of cardiac output, and this resulted in a difference in the peak level of dye in the blood collected in the azygos vein (fig. 2). The concentration was higher following the injection into the pulmonary artery as compared to that following an injection in the left ventricle. An explanation would be that the latter injection delivers into the azygos the dye conveyed by the bronchial arteries, but the pulmonary arterial injection adds the dye from the pulmonary to bronchial vein shunt, producing a rise in the concentration level. This is the only suggestive evidence that the pulmonary artery blood can reach the azygos vein directly without resorting to artificial interruption of the bronchial arteries.

BRONCHIAL TO PULMONARY BLOOD FLOW

The measurement of blood flow from the bronchial artery to the pulmonary vessels was performed essentially as described by Cudkowicz et al. The method originally consisted of injection of Evans blue dye into the upper portion of the aorta and the collection of consecutive blood samples from the left atrium and a peripheral artery. The dye-dilution curve from the peripheral artery showed a single peak representing the flow of blood in the aorta, or aortic blood flow. The curve from the left atrium showed a double peak, the initial one representing bronchopulmonary flow and the latter the recirculation of the dye via the systemic circulation. The broncho pulmonary flow (Qbp) can be calculated by the following formula:

\[ Q_{bp}/Q_{Ao} = S_{bp}/S_{Ao} \]

where \( Q_{Ao} \) means aortic blood flow; \( S_{Ao} \) means the area of the dye-dilution curve from a sys-
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FIGURE 2

Copies of dye-concentration curves of blood drawn from the azygos vein following injection of Cardio-green into left ventricle and pulmonary artery. Each pair of curves was drawn so that the initial slope would coincide in time. Note that the upper pair of curves did not show a difference in area; the lower pair of curves, taken about an hour later, with lower cardiac output, shows a larger area for the curve from the pulmonary artery.

Our own modifications of the above method consisted of substituting Cardio-green dye and using a continuously recording densitometer to sample blood alternately between the left atrium and femoral artery. The dye was injected into the left ventricle rather than into the upper portion of the aorta, since it was felt that the former served as a better mixing chamber. The coronary blood flow was excluded as a source of interference with the bronchopulmonary flow because Marchioro et al. had previously shown that the dye-dilution curve from the coronary sinus was superimposed in time with that from the venous return to the right atrium. Thus, the first peak of the dye-concentration curve should represent blood that reaches the left atrium through a shorter pathway such as the bronchopulmonary anastomoses. In one dog, the double peak following left ventricular injection was more conspicuous than that following an aortic injection of the dye (fig. 3). It is possible that this was caused by a lack of mixing of the dye.

The injection of Cardio-green into the left ventricle with sampling of blood from the femoral artery allowed the calculation of left ventricular output. A comparison of the area of the initial peak of the dye-concentration curve from the left atrium with the area of the single peak curve from the systemic artery gave a percentage figure for bronchopulmonary flow. The last calculation consisted of expressing the bronchopulmonary flow as L./min. from the original cardiac output figures. All of the results are summarized in tables 2 and 3.

Normal Dogs

The five normal anesthetized dogs showed a remarkable consistency in bronchopulmonary flow ranging from 6 to 9 per cent of cardiac output, with a mean value of 8.2 per cent. In three dogs, duplicate control measurements of bronchopulmonary flow showed an agreement within 4.9, 7.7, and 4.3 per cent of their respective mean values (table 2).

Chronic Ligation of the Left Pulmonary Artery

Three dogs were subjected to ligation of the left pulmonary artery and were observed for nine months. One dog showed fibrosis of the left lung and no detectable bronchopulmonary flow. The two other dogs had lungs that were normally ventilating, and the values for bronchopulmonary flow ranged from 12 to 20 per cent of cardiac output (dogs 8 and 9 of table 3). These values are larger than those described for the group of normal dogs.

Chronic Embolization of the Main Pulmonary Artery

Four additional dogs were observed for
### TABLE 2

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Weight Kg.</th>
<th>Situation</th>
<th>Cardiac output L./min.</th>
<th>Bronchopulmonary flow output X 100 = per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>20</td>
<td>First control</td>
<td>2.07</td>
<td>0.201</td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>First control</td>
<td>2.90</td>
<td>0.246</td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>First control</td>
<td>2.43</td>
<td>0.210</td>
</tr>
<tr>
<td>6</td>
<td>23</td>
<td>Second control</td>
<td>2.39</td>
<td>0.227</td>
</tr>
<tr>
<td>7</td>
<td>20</td>
<td>First control</td>
<td>3.70</td>
<td>0.278</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Second control</td>
<td>3.50</td>
<td>0.283</td>
</tr>
</tbody>
</table>

#### Mean* 27

- First control: 2.07
- Second control: 1.87

*Based on average values for dogs 5, 6, and 7 and on single value for dogs 2 and 4.

### TABLE 3

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Weight Kg.</th>
<th>Situation (chest)</th>
<th>Cardiac output L./min.</th>
<th>Bronchopulmonary flow output X 100 = per cent</th>
<th>Mean aortic mm. Hg</th>
<th>Pulmonary arterial pressure mm Hg</th>
</tr>
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<tbody>
<tr>
<td>8*</td>
<td>17</td>
<td>Ligated: 8 mos. (closed)</td>
<td>1.173</td>
<td>15</td>
<td>120</td>
<td>27</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>1.085</td>
<td>19</td>
<td>120</td>
<td></td>
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<td></td>
<td>1.206</td>
<td>17</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>20</td>
<td>Ligated: 9 mos. (closed)</td>
<td>0.001</td>
<td>12</td>
<td>140</td>
<td>25</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>1.104</td>
<td>20</td>
<td>140</td>
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<td></td>
<td></td>
<td>0.007</td>
<td>15</td>
<td>140</td>
<td></td>
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<tr>
<td>10</td>
<td>23.5</td>
<td>Embolized: 5 mos. (closed)</td>
<td>3.09</td>
<td>6.6</td>
<td>130</td>
<td>15</td>
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<td></td>
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<td></td>
<td></td>
<td>3.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>26</td>
<td>Embolized: 4 mos. (closed)</td>
<td>3.46</td>
<td>9.6</td>
<td>115</td>
<td>13</td>
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<td></td>
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<td></td>
<td></td>
<td>2.22</td>
<td>130</td>
<td>13</td>
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<td></td>
<td>1.68</td>
<td>100</td>
<td>12</td>
</tr>
<tr>
<td>12</td>
<td>25</td>
<td>Embolized: 5 mos. (closed)</td>
<td>3.50</td>
<td>0.9</td>
<td>110</td>
<td>14</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>2.54</td>
<td>77</td>
<td>13</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>2.79</td>
<td>160</td>
<td>15</td>
</tr>
<tr>
<td>13</td>
<td>23.6</td>
<td>Embolized: 5 mos. (open)</td>
<td>1.65</td>
<td>25.0</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>20</td>
<td>Control (closed)</td>
<td>1.85</td>
<td>5.9</td>
<td>150</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Steam inhalation (2 min. after)</td>
<td>2.6</td>
<td>9.1</td>
<td>140</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Steam inhalation (7 min. after)</td>
<td>1.48</td>
<td>3.0</td>
<td>150</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(30 min. after)</td>
<td>0.89</td>
<td>1.6</td>
<td>115</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>17</td>
<td>Aorta clamped (open)</td>
<td>1.49</td>
<td>30.0</td>
<td>105</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Steam inhalation (20 min. after)</td>
<td>1.89</td>
<td>1.26</td>
<td>105</td>
<td></td>
</tr>
</tbody>
</table>

*See also figure 3.

Ligation of the left pulmonary artery. Period of observation (months).
Embolization of the lungs with glass beads. Period of observation (months).

four to five months following the injection of glass beads. Three of these dogs showed the following values for bronchopulmonary flow after closure of the chest: 6.6, 9.6, and 0.9 per cent of cardiac output (dogs 10, 11, and 12 of table 3). One dog failed to breathe spontaneously after closure so that the first measurement of bronchopulmonary flow was performed while the dog was under artificial respiration and with the chest open (dog 13 of table 3). The value for bronchopulmonary flow was 25 per cent of cardiac output. The high value here could be due to embolization alone or to artificial ventilation or both.
Experiments of intentionally ventilating the chest in two other dogs were not helpful in deciding the cause for the high value of bronchopulmonary flow. One dog (no. 11) showed an increase, whereas the other dog (no. 12) showed no change.

Inhalation of Heat

The inhalation of steam was tested in two anesthetized dogs. One dog showed an immediate increase in bronchopulmonary flow which lasted only for a few minutes, and then the flow was reduced to a level below control (dog 14 of table 3). The other dog was intentionally subjected to ligation of the descending aorta to increase the bronchopulmonary flow, and this was in turn reduced by the inhalation of steam (dog 15). The explanation for these changes will be discussed below.

Discussion

The above results are best discussed in terms of their relationship to previous estimations of blood flow in the bronchial circulation of the dog. The various components of the canine bronchial circulation that have been studied are summarized in figure 4.

A. MAJOR BRONCHIAL ARTERY

It is logical to start with the major bronchial artery because the first quantitative measurement of bronchial arterial flow in the intact dog was performed by Bruner and Schmidt in 1947.8 A bubble flow-meter was inserted into the right major (posterior) bronchial artery which was supplied from one common carotid artery. The average normal flow in 50 anesthetized dogs was 4.8 ml./min. (±2.5) with fluctuations from a maximal mean flow of 6.6 to a minimal mean flow of 3.1. This mean flow value for one major bronchial artery did not include the contribution from the minor bronchial arteries. Furthermore, all blood in the major bronchial artery was not distributed exclusively to the lung. To estimate this, Bruner and Schmidt injected erythrocytes labeled with P32 directly into the right bronchial artery, and the dog was killed abruptly for analysis of the radioactive isotope. In six dogs, an average of 69 per cent of the blood flowing through the right bronchial artery was detected in the parenchyma of the right lung and 31 per cent in the mediastinal structures. Bruner and Schmidt have added two other calculations. In 30 normal dogs, the weight of the right lung was found to average 1.28 times the left. Assuming that the bronchial flow into the left lung was proportionate to that into the right in terms of relative lung weight, and assuming that the fractionation of blood in the right bronchial artery is the same as in the other bronchial arteries, the total flow discharged into both lungs would be the observed right bronchial flow times 1.26 [or times the quantity of 0.69 + 0.69 (0.8)]. In six dogs breathing spontaneously, cardiac output was measured simultaneously with right bronchial flow; the average value for total bronchopulmonary flow was 5.2 ml./min. or 0.3 per cent of cardiac output. The maximum value was 1 per cent of cardiac output. Nakamura9 used a similar combination of procedures and reported a mean value for five dogs of 0.75 per cent. None of these estimates includes contributions from the minor bronchial arteries so that the maximal value of 1 per cent of cardiac output represents only blood flow in major bronchial arteries, and total bronchial arterial flow (major plus minor) would be greater than 1 per cent.
B. TOTAL BRONCHIAL ARTERIAL FLOW
The estimation of arterial inflow to include the major as well as the minor bronchial arteries has been performed by the isolation of and the creation of a pouch from the portion of descending aorta from which the bronchials originate. The arteries from this aortic pouch which did not proceed to the lungs were ligated, and only the bronchial arteries remained open. The blood flowing in the ascending aorta was then shunted directly into the descending aorta distal to the pouch, with provisions to supply the pouch with blood and to measure the corresponding blood flow with a rotameter. The values reported by Horisberger and Rodbard, who developed such a method, averaged 12 ml./min. in seven dogs. A second series of 15 dogs showed a mean value of 30 ml./min., and a third series of six dogs showed a mean of 49 ml./min. The difference between the second and third series is that the latter was performed on dogs with an intact autonomic and central nervous system. The mean figure of 30 ml./min. would represent the closest estimation in dogs with an intact autonomic and central nervous system of total bronchial arterial flow, but unfortunately, the cardiac output was not measured in the same group of dogs. From known figures of cardiac output for dogs of equivalent body weight, the total bronchopulmonary flow is certainly more than 1 per cent of estimated cardiac output.

C. BRONCHIAL VENOUS FLOW
The distribution of bronchial arterial inflow into the bronchial venous and bronchopulmonary components has been investigated by separate perfusion of the bronchial and pulmonary vessels. When the perfusion in the latter was stopped and bronchial perfusion continued, about 5 ml./min. were collected in the bronchial veins. This represented 18 to 40 per cent of total bronchial arterial flow in four dogs and 13 to 72 per cent in three dogs. The estimations in two other reports of bronchial venous flow were on dogs without the use of perfusion pump and without interruption of pulmonary blood flow. The bronchial venous flow in a group of 15 dogs averaged 9.6 ml./min. and in a second group of six dogs averaged 21 ml./min. These flows represent about 30 to 70 per cent of total bronchial arterial flow. Since pulmonary blood flow was not interrupted, the blood collected from the bronchial veins represents both the fraction from the bronchial arteries and that from the pulmonary artery. The distinction between the two will be discussed again under (E).

D. BRONCHOPULMONARY FLOW
The measurement of bronchopulmonary flow has been performed largely by interruption of pulmonary flow by ligation of both vessels of a lobe. Shedd et al. inserted a plastic catheter into the ligated segment of the lobar vein and collected blood at a rate of about 6 to 8 drops/min., even though the external tip was elevated at a level about 40 cm. above the vein. Williams and Towbin measured this flow by a similarly placed catheter, but the outside tip was 5 cm. below the level of the pulmonary artery. In five dogs, the bronchopulmonary level was 4.4 to 9 ml./min. In each of these dogs, the flow varied with the height of systemic arterial pressure and gradually diminished with time. Parker and Smith measured flow in four dogs and recorded 1 ml. or less per minute but did not specify the level of the collecting tip of the venous catheter.

The difference in quantity of flow reported among the above experiments can be explained by the location of the collecting catheter (in the lobar artery or vein) and by the level of the outside tip (above or below the lobar vessels). In a group of nine dogs, two catheters were inserted (one into artery and the other into vein), and the outside tips were both at the same level as the lobar vessels. The total flow from both catheters ranged from 2.1 to 8.2 ml./min. with the fraction of the venous catheter about 60 per cent of the total of both. When one of the two collecting catheters was clamped, the resulting outflow from the remaining catheter was equal to the...
combined flow when both were open. These results indicate that in the absence of normal pulmonary arterial flow to the left lower lobe, the total amount of bronchopulmonary blood flow is from 2 to 8 ml/min., and the amount is constant whether collection is from the lobar artery or vein or both.

The results from the dye-dilution technique are much larger than those quoted above for direct measurements of bronchopulmonary flow. Cudkowicz et al. reported in two dogs the following values: 3.6 and 1.9 per cent of cardiac output. The experiments reported above indicate an average of 8.3 per cent in five dogs. This value for bronchopulmonary anastomoses is the highest value reported for bronchopulmonary flow. It is interesting to note that the highest values for bronchopulmonary flow were derived from dogs with closed chest using indirect methods, whereas the lowest values were derived from dogs with open chest using direct methods of flow measurement. The true values in the unanesthetized state probably lie somewhere in between these two extremes.

E. PULMONARY TO BRONCHIAL SHUNT

The accessibility of pulmonary blood to the bronchial veins has been reported previously while interrupting bronchial blood flow. The experiments reported above attempted to detect such a shunt without interruption of bronchial flow by injecting the dye into the pulmonary artery and sampling the blood in the azygos vein. The dye-dilution curve failed to show a double peak which would have made the detection of the shunt simple. However, the lack of a double peak does not exclude the existence of the shunt because it is still possible that the flow through the pulmonary to bronchial shunt is slower than the flow of blood in the bronchial artery to bronchial veins and that a single curve represents both flows. The only evidence from the above experiments which is suggestive of a pulmonary to bronchial venous shunt is the comparison of the dye concentration curves from the azygos vein when equal amounts of the dye are injected either into the pulmonary artery or left ventricle. In one dog, such an injection showed a larger area in the dye-dilution curve following pulmonary arterial injection than that following a left ventricular injection. The difference is probably due to the pulmonary bronchial shunt.

Additional studies are necessary to assess the participation of a pulmonary to bronchial shunt without interruption of the bronchial circulation, and if present, it will be necessary to question the validity of measurements of bronchopulmonary flow by independent perfusion of the pulmonary and bronchial systems and measuring the exchange of blood between the two reservoirs. Salisbury et al. measured the transfer of blood from systemic to the pulmonary reservoirs to be 0.5 to 1 per cent of total systemic flow, and values reported by Auld et al. fall in the same range. This transfer of blood is an underestimation of bronchial to pulmonary flow because the amount transferring from the pulmonary and systemic reservoirs has been subtracted. The results are still valid in that they suggest that the bronchial to pulmonary flow is larger than pulmonary to bronchial flow, but the absolute values of each will have to be measured directly.

The most important feature of the above calculations is the suspicion that the generally accepted figure for bronchial blood flow of 1 per cent of cardiac output may be an underestimation. The extremely high value of 8 per cent for normal dogs by the dye-dilution technique will require confirmation by other methods that are applicable in dogs with closed chest. The even higher values for bronchopulmonary flow in dogs with chronic ligation of one pulmonary artery is in line with previous reports. The absence of a rise following pulmonary embolization may be related to the lack of its severity in interrupting pulmonary flow as much as actual ligation. The initial rise followed by a fall in bronchopulmonary flow during thermal injury to the lung is a new observation. The best explanation for the increase in bronchopulmonary flow is that the inhalation of heat induced a constriction of the bronchial veins.
with a shifting of blood flow toward the bronchopulmonary anastomotic channels. Such an explanation will need further experimentation from three standpoints.

1. **Mechanism for bronchial venous constriction:** The induction of venous constriction by heat is an unusual phenomenon because most known vascular responses are the opposite. It is very possible that the bronchial veins contain a highly developed vasoconstricting mechanism which is lacking in the bronchopulmonary anastomoses. Experiments devoted to a comparison of the reactivity of both channels to chemical agents have been completed and are reported elsewhere.\textsuperscript{8,11}

2. **Mechanism for increase in bronchopulmonary anastomoses:** If this proves to be a passive phenomenon, secondary to bronchial venous constriction, the basic cause for similar observations in chronic lung disease may be related. The adjustment in the bronchial circulation during emphysema, pulmonary embolization, and pulmonary stenosis may actually depend on a primary bronchial venous mechanism with a resulting increase in correction of the bronchial venous constriction.

3. **Relationship of bronchial circulation to pulmonary edema:** The increase in caliber of bronchial veins, accompanied by an increase in bronchial arterial flow, will lead to congestion of the bronchial mucosa. It is very possible that the edema seen following thermal injury to the lungs may be due to bronchial congestion so that appropriate therapy would include relief of bronchial vascular congestion.

**Summary**

In anesthetized dogs, the dye-dilution technique was applied to detect the transfer of blood between the bronchial and pulmonary vessels. The injection of Cardio-green into the pulmonary artery did not show a double peak in the dye-concentration curve of blood in the azygos vein. This does not exclude the possibility of a shunt from the pulmonary artery to the bronchial veins because the flow of the dye may take longer to reach the bronchial veins than does that from the bronchial artery. The injection of the dye into the left ventricle with sampling of the blood in the left atrium showed a double peak in the dye-dilution curve. The latter is interpreted to indicate blood flow from the bronchial arteries to the pulmonary vessels via the bronchopulmonary anastomoses. In five dogs, the mean value for bronchopulmonary flow was 8 per cent of left ventricular output. The corresponding flow values were increased in three dogs with chronic ligation of the left pulmonary artery, not increased in four dogs with pulmonary embolization, and reduced in two dogs subjected to inhalation of heat. All these results suggest that previous estimates of 1 per cent for bronchial flow may be an underestimation, but the maximum value of 8 per cent in anesthetized dogs calculated by the dye-dilution technique will have to be re-evaluated by other methods.

**References**


Book Reviews


The central subject of this monograph is the chemistry of extracorporeal dialysis. It is intended primarily for both the student of medicine and the clinician in need of practical help in evaluating a disease state where dialysis may be required. The following clinical situations are discussed: renal insufficiency, dehydration, and systemic intoxications from salicylate, barbiturate, bromide, diphenylhydantoin, ammonia, and other chemicals. A large amount of space has been devoted to the techniques of the procedure of hemodialysis. The appendices list current manufacturers of dialyzers and associated equipment.


This book is intended to survey the present knowledge of diseases of the heart that are caused by complex pathogenic situations. The following cardiopathies are discussed: hyalinizing, necrotizing, calcifying, arteriosclerotic, suppurating, and superficial. The illustrations are excellent, and the author concludes as follows: "A great deal of work will yet have to be done to determine how, and to what extent, this knowledge can be applied to the problems of clinical medicine."


This is a biography of one of the pioneers in "phthisiology" and the inventor of the stethoscope. It covers Laennec's childhood, through his medical studies, his professorship, and medical practice. The discovery of direct auscultation is recounted in detail. The medical historian will find the bibliography useful.


The authors, who have made important contributions to neurochemistry, have written a short but informative book which describes a great number of physical and chemical methods that they have found useful in the study of the nervous system. It is obviously impossible in a short book to cover the whole field of "practical neurochemistry," and the authors have not attempted this. Rather, they have discussed methods with which they are personally familiar and about which they obviously speak with authority. There are certain omissions, such as gas chromatography, and some subjects could well have been treated more extensively. There is little discussion, for example, of the use of enzymes in analytical procedures. On the whole, however, the book should prove useful for students and investigators concerned with neurochemistry and with tissue metabolism in general.
Exchange of Blood Between Pulmonary and Systemic Circulations via Bronchopulmonary Anastomoses

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*Circ Res.* 1962;11:870-879
doi: 10.1161/01.RES.11.5.870

*Circulation Research* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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