Studies on Anomalous Collateral Systemic-Pulmonary Circulation

Report of Four Cases

By G. J. Tammeling, M.D., J. Nieveen, M.D., and H. J. Sluiter, M.D.

SUMMARY
The circulatory status of four patients with anomalous systemic-pulmonary circulations has been studied by bronchopulmonary circulation, cardiac catheterization, angiography, and dye-dilution techniques. In three of these patients the affected lung was smaller than normal, and bronchography showed no abnormalities. On the affected side angiography failed to show filling of the pulmonary artery, and the oxygen uptake appeared to be zero or minimal. In two of the patients bronchopulmonary circulation revealed an effective collateral circulation, whereas the dye-dilution curves were almost normal. In the other patient there was anatomic evidence of both transpleural systemic-pulmonary arterial communication and occlusive disease of the pulmonary veins. Here the dye-dilution curves revealed considerable shunting of blood, whereas no effective collateral circulation could be demonstrated. The fourth patient has extensive unilateral bronchiectasis and illustrates the condition wherein neither pulmonary nor systemic blood flows through the pulmonary capillary bed.

The pathogenesis of the various pathways of collateral circulation is discussed, as well as some methodological and technical problems.

Additional Indexing Words:
Bronchopulmonary circulation
Obstruction of pulmonary veins
Bronchospirometry

Transpleural arterial anastomoses
Absence of pulmonary artery
Pneumoangiography
Dye-dilution curves

In recent years the number of reports on anomalous systemic-pulmonary circulation has steadily increased, and several aspects of anatomic and functional pathology have been studied.1-6 Anomalous communications between bronchial and pulmonary circulation have been described in organizing pulmonary disease,6-11 congenital atresia, or acquired obliteration of the pulmonary artery.12-15 As not only bronchial vessels, but also other systemic arteries may be involved,16,17 the diagnostic label has been appropriately changed from collateral bronchopulmonary circulation to collateral systemic-pulmonary circulation.

By means of special techniques the various possibilities of anomalous collateral systemic-pulmonary circulation can be differentiated.12, 13, 18-21 Total collateral circulation is usually estimated by dye-dilution curves,22,23 the effective portion of it by bronchospirometry,2 the difference between these two being the ineffective portion. Cardiac catheterization and angiography provide information on the location of vascular anastomoses and occlusions and on the direction of blood flow.24,25 A few years ago, several of these techniques were reviewed and discussed at the Fifth Annual Aspen Conference.26 Until now, these techniques have been scarcely used for patients.

The present paper reports the results in four patients. It could be demonstrated that the actual circulatory status was different in each of these patients. In one of them the...
physiological findings were verified by the anatomic study of the resection specimen.

Methods

Technical details of spiography and residual volume determination can be found elsewhere.27 Total ventilation (VT) and physiological dead-space-ventilation (Vdphys) were measured with the patients breathing room air in an open circuit. The expiratory gases were collected in a Douglas bag, and their volume was measured by a calibrated Wright respirometer.* Gases were analyzed in duplicate with the Scholander apparatus.† Carbon dioxide tension of the arterial blood (Paco2) was determined either by the Astrup micromethod,‡ or by a Severinghaus-type membrane-electrode.§ Arterial oxygen saturation (SaO2) was measured by the reflection method according to Brinkman and Zijlstra.** During cardiac catheterization the oxygen saturation was measured using a CC-oximeter.†† Oxygen tension (Pao2) was determined with a Clark-type-membrane electrode.‡‡ When both oxygen tension and saturation are mentioned, the saturation was calculated from the oxygen tension and the standard disso- 
ciation curve.

A-a gradient for oxygen was calculated from direct oxygen tension measurements and estimation of the mean alveolar oxygen tension from the alveolar gas equation. Right-to-left shunt, while the patient was breathing pure oxygen, was estimated from the arterial oxygen tension.

Bronchospirometry §§ was performed with a Carlen’s catheter after local anesthesia with 0.5% tetracaine solution. Volume and oxygen uptake of each lung were measured with both spirometer systems containing an oxygen-rich gas mixture. The expiratory CO2 tension (Pco2) of the individual lungs was continuously recorded by an infra-red gas analyzer.***

Effective portion of the collateral systemic-pulmonary blood flow was measured by the direct Fick method, introduced by Bloomer and co-workers2 for measurement of collateral circulation of the lung, with a modified Fishman technique.18 The diseased lung was connected to an oxygen-filled bronchospirometer system, and the collateral lung to a bag, containing about 12% oxygen in nitrogen. Expired gases were collected in a Douglas bag. The effective portion was calculated using the Fick equation:

\[ Q = \frac{V_{O2}}{C_{CO2} - C_{O2}} \]

where:

\[ Q \] = the effective collateral blood flow through the affected lung (liters per minute).

\[ V_{O2} \] = oxygen uptake of the affected lung in milliliters per minute (STPD).

\[ C_{CO2} \] = oxygen content of end-capillary blood of ventilated alveoli (ml per liter of blood).

The oxygen content was calculated from the oxygen capacity of hemoglobin and the physically-dissolved oxygen, assuming that the end-capillary oxygen tension (Pco2) equals alveolar oxygen tension, on breathing an oxygen-rich mixture. It should be pointed out that the error in the estimation of Pco2 has little effect on the final results.18

\[ C_{O2} \] = oxygen content (ml per liter of blood) of systemic blood as sampled from the brachial artery. The oxygen content was calculated from the oxygen capacity and oxygen saturation of the hemoglobin, allowance being made for the physically-dissolved oxygen.

Estimation of the effective portion by the above-mentioned equation is valid only when the pulmonary capillaries of the affected lung are exclusively perfused by systemic blood. The effective portion does not include systemic blood by passing the pulmonary capillary bed or blood flow through capillaries of nonventilated alveoli.

Cardiac catheterization was performed via an antecubital vein. Dye-dilution curves were recorded by the cuvette-reflection method, using indocyanine green.28 The dye was injected into the pulmonary artery and sampled at the brachial artery. The shape of the curve permits estimation of the cardiac output and of left-to-right and right-to-left shunts. An abnormally early recirculation of as little as 5% of the total output of the right ventricle can be detected by this method.28

Angiography was performed by injection of 60% Isopaque into the pulmonary artery or via Ó’dman catheters, introduced by the Seldinger technique into the femoral artery, the tip lying

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†Godart, De Bilt, The Netherlands.
‡Type AME-1, Radiometer, Copenhagen, Denmark.
§Type E 5031, Radiometer, Copenhagen, Denmark.
‡‡Type E 5041, Radiometer, Copenhagen, Denmark.
***Capnograph, type CG/58003, Godart, De Bilt, The Netherlands.
in the aortic roof.* Exposures were made on separate films; in one patient cineangiocardiography was used.

In one patient pneumonectomy was performed. The resected specimen was Formalin-fixed and sent to Dr. Liebow's laboratory (Yale University, New Haven, Connecticut) for further study. Here, the vessels were injected with radiopaque vinyl plastic and the bronchi with nonradiopaque white plastic. A large number of blocks for histological studies were obtained. Finally, the specimen was digested in 20% potassium hydroxide. Films were made at various stages.

Results

The patients are described in the sequence in which they were admitted to the hospital. Patients 1, 2, and 3 have many common aspects, but they had basically different circulatory states; patient 4 had very extensive, mainly unilateral bronchiectasis and was included in the series mainly for discussion purposes.

The results of the special investigations are summarized in four tables: lung volumes (table 1), additional pulmonary function data (table 2), data on arterial blood gases, gas exchange in the individual lungs and effective portion of collateral systemic-pulmonary circulation (table 3), and data obtained during cardiac catheterization (table 4).

Report of Cases

Case 1

W.S. Mo. (case history, 38297), a 17-year-old male, was admitted to the hospital because of an abnormal chest roentgenogram, discovered during a mass radiography survey. As a child he had bronchitis once or twice, he coughed occasionally but raised no sputum and otherwise had no complaints. The posteroanterior roentgenogram of the chest (fig. 1) revealed displacement of the heart and mediastinum to the right, with an elevated right diaphragm and a large anterior mediastinal hernia. The right lung was smaller than the left, and showed a lace-like pattern, whereas the pulmonary artery contours were absent. Deep inspiration produced marked mediastinal swing to the right; during expiration no overswing to the left was noted. Electrocardiogram, bronchograms, and bronchoscopy were normal.

Assessment of pulmonary function revealed a restrictive lung disease with slight reversible obstruction of the airway. Oxygen uptake of the affected lung was zero, while the patient was breathing either room air or a gas mixture with a high oxygen concentration. Some carbon dioxide, however, was evolved in this lung (fig. 2, table 3). The effective portion of the collateral systemic-pulmonary circulation was calculated to be 0.88 L per minute.

During cardiac catheterization, the catheter could not be passed into the right pulmonary artery. Angiography failed to show the filling of the right branch (fig. 3). Dye-dilution curves were normal (fig. 4).

From these data it was concluded that the functioning capillary bed of the right lung is exclusively perfused by systemic blood. Anatomic verification of the systemic-pulmonary anastomoses could not be performed, since surgical treatment was not indicated in this patient. A scheme of the possible systemic-pulmonary circulation is shown in figure 5.

Case 2

R. Di. (case history, 37356), a woman, 19 years old, was admitted because of chest x-ray abnormalities discovered during a routine investigation. She had no complaints. Family history was negative for chest disease and congenital anomalies. The chest roentgenogram was very similar to that of patient 1 (fig. 6). A loud systolic murmur was noted over the apex of the right lung. Electrocardiogram, bronchograms, and bronchoscopy were normal.

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*Elema Schölander AOT, type 841-1, with Gidlund injection syringe.

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Figure 1

Roentgenogram of the chest of patient 1 (W.S. Mo.).
Exploration of the pulmonary function showed a restrictive pattern and a slight, reversible obstruction of the airway. Arterial blood gases were normal at rest, but hypoxemia and hypercapnia were present on exertion. Oxygen uptake of the right lung was very low (10 ml per minute) and did not increase during artificially induced severe systemic hypoxemia (table 3). End tidal carbon dioxide tension in the diseased lung was clearly lower than in the contralateral lung (fig. 2). During cardiac catheterization the catheter could be passed into the right pulmonary artery, where oxygen saturation was significantly higher than at the corresponding level in the left branch. Cineangiography failed to show contrast medium entering the right pulmonary artery. Aortography revealed the filling of the right internal mammary artery originating from the right innominate artery, with subsequent opacification of the right upper lung field, and right and left pulmonary arteries (fig. 7).

Figure 2

Records of the expiratory carbon dioxide tensions (PeCO₂) of every single lung during the breathing of room air. Note that the polarity of the recorder has been the reverse in patient 3 (subject 38631). Subject 38297 is patient 1; 37356, patient 2; 1461, patient 4.
The dye-dilution curve showed a significant abnormally early recirculation, calculated to be about 50% of the output of the right ventricle (fig. 4).

Because of this large shunt and because the blood gases were severely disturbed during exercise, pneumonectomy was performed by Dr. J. Homan van der Heide. The resected specimen revealed the following abnormalities (Dr. A. A. Liebow):

**Figure 3**

Angiogram of patient 1 (W.S. Mo.). The right pulmonary artery is not opacified (1% sec).

**Figure 4**

Dye-dilution curves, recorded from the brachial artery (the dye was injected into the pulmonary artery).

**Figure 5**

Schematic presentation of the relationship between systemic and pulmonary blood flow through the diseased lung. The schemata are modified according to Heimburg.\(^{30}\) SA = systemic artery; SV = systemic veins; BA = bronchial artery; PA = pulmonary artery; PC = pulmonary capillary bed; PV = pulmonary veins.
The pulmonary veins were remarkable for their small size and their small lumina, not only at the hilum, but also within the lung. The major trunks seemed for the most part uninterrupted. In the thickened pulmonary septa, the pulmonary veins showed marked, intimal thickening and some of the smallest were occluded by partly recanalized connective tissues; collateral venous channels were observed especially in the upper lobe. Most remarkable was the immense size of some of these collateral veins found between and accompanying the pulmonary arteries and bronchi. Collateral veins also were present in the pleura.

At the hilum, the pulmonary arteries had relatively thin walls resembling those of pulmonary veins. These arteries communicated with large transpleural arterial connections in the upper part of the lung (fig. 8). These are interpreted to represent the connections with the internal mammary artery. Smaller transpleural connections were found in the middle and lower lobes. The pleural arterial vessels had the structure of systemic arteries. The pulmonary arteries were patent everywhere.

It is noteworthy that the histological sections provided no evidence of enlargement of bronchial arteries within the parenchyma. Considerably enlarged bronchial veins emerged at the

![Figure 6](image)

*Figure 6*

Roentgenogram of the chest of patient 2 (R. Di.)

![Figure 7](image)

*Figure 7*

Aortogram of patient 2 (R. Di.). (Left) A prominent artery, originating from the right innominate artery running downward to the lung (1½ sec). (Right) Consequent filling of the left branch of the pulmonary artery (2½ sec).
hilum. The cast of the bronchial tree showed no gross abnormalities. The lung parenchyma showed interstitial fibrosis close to the septa, which in many places were greatly thickened, due to immense increase in the number of lymphatic channels and of abundant bundles of smooth muscle (fig. 9).

**Comment**

The veno-occlusive and veno-restrictive disease helps to explain the small oxygen uptake of the right lung. The systemic arterial inflow did not reach, or only reached the minor part of the capillaries of the right lung, but rather was shunted into the pulmonary artery of the contralateral lung. This means that the pulmonary circulation of the left lung is overloaded, even at rest, nearly three times as compared to normal. This probably explains the increased pressure in the pulmonary artery and the abnormal systemic arterial blood gases on exertion. The latter is supported by the fact that about 9 months after operation the systemic arterial oxygen and carbon dioxide tensions during exercise were normal. Although there is no direct anatomic evidence for large connections between systemic arteries and pulmonary or systemic veins, the possibility that the systemic blood is, at least partly, shunted directly along this pathway cannot be excluded. The enlarged bronchial veins at the hilum may point in this direction. A schematic presentation of the systemic-pulmonary relationship is shown in figure 5.

**Case 3**

B. Re.-H. (case history, 38631), a woman, 41 years of age, was admitted to the hospital because of fatigue, pain in the left hemithorax that increased in severity during exercise but was not typically anginal in character, chronic cough, and expectoration with occasional blood-stained phlegm. She had suffered from several episodes of pneumonia. She could not manage her household duties. The chest roentgenogram was very similar to those of patients 1 and 2, but in this patient the lesions were located on the left side. Electrocardiogram was normal and so were

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**Table 1**

Spirometric and Bronchospirometric Data on Pulmonary Volumes (ATPS)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Lung volumes*</th>
<th>Both lungs</th>
<th>Actual value</th>
<th>Diseased lung (%)</th>
</tr>
</thead>
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<tr>
<td></td>
<td></td>
<td>Predicted</td>
<td>Actual</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>TLC</td>
<td>6.50</td>
<td>5.78</td>
<td>24</td>
</tr>
<tr>
<td>W.S.Mo.</td>
<td>VC</td>
<td>5.20</td>
<td>4.58</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>FRC</td>
<td>2.26</td>
<td>2.20</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>FEV$_{1.0}$</td>
<td>&gt; 4.10</td>
<td>2.96</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>V$_T$ (L/min)</td>
<td>—</td>
<td>6.4</td>
<td>46</td>
</tr>
<tr>
<td>2</td>
<td>TLC</td>
<td>4.40</td>
<td>3.40</td>
<td></td>
</tr>
<tr>
<td>R.Di.</td>
<td>VC</td>
<td>3.52</td>
<td>2.50</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>FRC</td>
<td>1.54</td>
<td>1.32</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FEV$_{1.0}$</td>
<td>&gt; 2.81</td>
<td>1.80</td>
<td></td>
</tr>
<tr>
<td></td>
<td>V$_T$ (L/min)</td>
<td>—</td>
<td>4.5</td>
<td>31</td>
</tr>
<tr>
<td>3</td>
<td>TLC</td>
<td>4.26</td>
<td>3.63</td>
<td>28</td>
</tr>
<tr>
<td>B.Re.-H.</td>
<td>VC</td>
<td>3.15</td>
<td>2.53</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>FRC</td>
<td>1.66</td>
<td>1.70</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>FEV$_{1.0}$</td>
<td>&gt; 2.20</td>
<td>1.79</td>
<td></td>
</tr>
<tr>
<td></td>
<td>V$_T$ (L/min)</td>
<td>—</td>
<td>5.2</td>
<td>26</td>
</tr>
<tr>
<td>4</td>
<td>TLC</td>
<td>6.30</td>
<td>4.79</td>
<td>29</td>
</tr>
<tr>
<td>H.Ev.</td>
<td>VC</td>
<td>4.66</td>
<td>2.62</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>FRC</td>
<td>2.45</td>
<td>2.91</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>FEV$_{1.0}$</td>
<td>&gt; 3.26</td>
<td>1.13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>V$_T$ (L/min)</td>
<td>—</td>
<td>11.0</td>
<td>48</td>
</tr>
</tbody>
</table>

*Abbreviations: TLC = total lung capacity; VC = vital capacity; FRC = functional residual capacity; FEV = forced expiratory volume; V$_T$ = total ventilation.

For predicted values: see reference 27.
the bronchograms with the exception of some dilated mucous cysts in the left main bronchus; bronchoscopy was not performed.

Studies of pulmonary functions revealed a restrictive disturbance with some reversible obstruction of the airway. On breathing room air

Figure 8

Cast from the resected lung (patient 2, R. Di.), demonstrating various transpleural arterial communications. B = bronchial tree; PA = pulmonary arteries; CV = plexus of collateral venules; arrow 1 indicates bronchial veins; arrow 2, pulmonary arteries connected to branches of intercostal arteries; arrow 3, terminal ends of pulmonary arteries connected with mammary arteries; arrow 4, pulmonary arteries probably connected with diaphragmatic vessels. (Courtesy Dr. A. A. Liebow.)
or pure oxygen, the oxygen uptake of the left lung was zero; a slight but unmistakable carbon dioxide excretion, however, was noted (fig. 2). The effective portion of the collateral circulation was calculated to be 0.85 L per minute. The oxygen saturation at the half-way point in the left pulmonary artery was higher than at the corresponding site in the right. The pulmonary artery pressure was slightly raised. Dye-dilution curves revealed only a small, abnormally early recirculation (5 to 10% of the right ventricle output) (fig. 4). Angiography failed to reveal a left pulmonary artery.

These data indicate that in this patient the functioning capillary bed of the left lung is perfused exclusively by systemic blood. Reversal of blood flow in the pulmonary artery is evident. The exact anatomic status, and particularly, the source of the systemic blood is unknown. A scheme of the possible circulation is given in figure 5.

Case 4

H. Ev. (case history, 1461), a man, 51 years of age, had suffered from childhood from a productive cough with copious purulent, sometimes bloodstained sputum and from repeated bouts of pneumonia. Bronchography, performed in 1944, revealed diffuse cylindrical and saccular bronchiectasis, most pronounced in the left lung. He was admitted to the hospital several times for acute exacerbations of his chronic bronchitis, often being in respiratory failure. He was treated repeatedly with antibiotics, corticosteroids, and physical therapy. In the course of the years he started complaining of gradually increasing dyspnea on exertion. The electrocardiogram showed marked clockwise rotation with some suggestion of right ventricular hypertrophy. A chest roentgenogram showed extensive bronchiectasis with fibrotic and emphysematic changes in the left lung; similar changes in the right lung were much less pronounced.

Lung function assessment indicated a restrictive pulmonary disease with fairly pronounced, only partly reversible, airway obstruction. The oxygen uptake of the left lung was zero, quite independent whether the right lung breathed room air, oxygen, or a 12.6% oxygen gas mixture. Carbon dioxide excretion was virtually absent: the end-expiratory carbon dioxide tension was only 6 mm Hg. Cardiac catheterization revealed an abnormally high pressure in the pulmonary artery. Oxygen saturation in the left branch (not in wedge position) was identical to the systemic arterial oxygen saturation. Dye-

Figure 9

Histological structure of the lung (patient 2, R. Di.) showing interstitial fibrosis and septal thickening. The pulmonary veins are indicated by black arrows, the other vessels are predominantly lymphatics. Note the large number of lymphatics and the abundant bundles of smooth muscle. (Courtesy Dr. A. A. Liebow.)
Table 2

<table>
<thead>
<tr>
<th>Lung function studies</th>
<th>Units</th>
<th>Predicted</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_D$ Phys./$V_t$ ratio</td>
<td></td>
<td>$&lt;0.30$</td>
<td>$0.51$</td>
<td>$0.57$</td>
<td>$0.67$</td>
<td></td>
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<tr>
<td>$S_{aO_2}$ at rest</td>
<td>%</td>
<td>97</td>
<td>97</td>
<td>96</td>
<td>94-98</td>
<td>80-89</td>
</tr>
<tr>
<td>$S_{aO_2}$ during exercise</td>
<td>% (watt)</td>
<td>97</td>
<td>97 (100)</td>
<td>84 (125)</td>
<td>92 (100)</td>
<td>91 (100)</td>
</tr>
<tr>
<td>$P_{aco_2}$ at rest</td>
<td>mm Hg</td>
<td>40</td>
<td>34-37</td>
<td>42</td>
<td>33-35</td>
<td>46-59</td>
</tr>
<tr>
<td>$P_{aco_2}$ during exercise</td>
<td>mm Hg (watt)</td>
<td>40</td>
<td>58 (125)</td>
<td>36 (100)</td>
<td>50 (100)</td>
<td></td>
</tr>
<tr>
<td>A-a gradient for oxygen</td>
<td>mm Hg</td>
<td>$&lt;10$</td>
<td>—</td>
<td>10</td>
<td>10</td>
<td>32</td>
</tr>
<tr>
<td>$P_{ao_2}$ (during $O_2$</td>
<td>mm Hg</td>
<td>$&gt;-640$</td>
<td>600</td>
<td>620</td>
<td>550</td>
<td></td>
</tr>
<tr>
<td>Right-to-left shunt ($O_2$</td>
<td>% RV output</td>
<td>$&lt;5$</td>
<td>—</td>
<td>5</td>
<td>4</td>
<td>7.5</td>
</tr>
</tbody>
</table>

*For abbreviations: see “Methods.”
†Lode-Lanooy bicycle ergometer.

Discussion

Patients 1, 2, and 3 have the following characteristics in common: (1) One lung is smaller than normal both anatomic and functionally; (2) the normal vascular contours of the homolateral pulmonary artery are absent, whereas the lateral pulmonary arteries are present; (3) the findings of the anteroposterior radiographs is apparent from the anatomic diagnosis of the homolateral pulmonary artery; (4) the finding that patient 1 has not been established definitely which systemic arteriovenous fistula was present. In patients 2 and 3, the finding that in the laterals pulmonary artery could be accepted as conclusive evidence for the diagnosis of pulmonary artery atresia. The syndrome of a normal bronchial supply of a vessel was recognized in the atresia demonstrated by angiography in the bronchial tree. The bronchial angiography fails to reveal the absence of the homolateral artery. In patient 1, the finding that in the homolateral pulmonary artery could be accepted as conclusive evidence for an additional homolateral pulmonary artery is apparent from the bronchial angiography.

References

1. TAMMELING ET AL. Circulation, Volume XXXV, March 1967

2. Circulation, Volume XXXV, March 1967


5. Circulation, Volume XXXV, March 1967
### Table 3

**Data on Arterial Blood Gases, Respiratory Gas Exchange of the Individual Lungs, and Effective Collateral Systemic-Pulmonary Circulation**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Right lung</th>
<th>Gas mixtures supplied to</th>
<th>Left lung</th>
<th>Systemic arterial blood</th>
<th>Right lung (ml/min)</th>
<th>Left lung (ml/min)</th>
<th>Effective portion of systemic-pulmonary collateral blood flow (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Air</td>
<td>Air</td>
<td>Air</td>
<td>PaO₂ 98 (mm Hg) SaO₂ (%)</td>
<td>Vo₂ 0 20</td>
<td>296 190</td>
<td>——</td>
</tr>
<tr>
<td>W.S. Mo.</td>
<td>O₂</td>
<td>O₂</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>——</td>
</tr>
<tr>
<td></td>
<td>O₂</td>
<td>12.4% O₂</td>
<td>—</td>
<td>82.5</td>
<td>44</td>
<td>—</td>
<td>—— 0.88</td>
</tr>
<tr>
<td>2</td>
<td>Air</td>
<td>Air</td>
<td>O₂</td>
<td>PaO₂ 96.5 (mm Hg) SaO₂ (%)</td>
<td>Vo₂ 10 10</td>
<td>215 —</td>
<td>——</td>
</tr>
<tr>
<td>R. Di.</td>
<td>O₂</td>
<td>12.0% O₂</td>
<td>—</td>
<td>71.5</td>
<td>—</td>
<td>—</td>
<td>——</td>
</tr>
<tr>
<td>3</td>
<td>Air</td>
<td>Air</td>
<td>O₂</td>
<td>PaO₂ 104 (mm Hg) SaO₂ (%)</td>
<td>—</td>
<td>0 6</td>
<td>——</td>
</tr>
<tr>
<td>B. Re.-H.</td>
<td>O₂</td>
<td>O₂</td>
<td>—</td>
<td>98</td>
<td>—</td>
<td>—</td>
<td>——</td>
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<tr>
<td></td>
<td>O₂</td>
<td>12.4% O₂</td>
<td>—</td>
<td>185</td>
<td>—</td>
<td>27 —</td>
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<td>4</td>
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<td>O₂</td>
<td>O₂</td>
<td>PaO₂ 90 (mm Hg) SaO₂ (%)</td>
<td>—</td>
<td>0 —</td>
<td>——</td>
</tr>
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<td>H. Ev.</td>
<td>O₂</td>
<td>O₂</td>
<td>—</td>
<td>78</td>
<td>—</td>
<td>0</td>
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<tr>
<td></td>
<td>O₂</td>
<td>12.4% O₂</td>
<td>—</td>
<td>78</td>
<td>—</td>
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</tr>
</tbody>
</table>

For abbreviations: see “Methods.”

### Table 4

**Data Obtained During Cardiac Catheterization**

<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PaO₂ (mm Hg)</td>
<td>SaO₂ (%)</td>
<td>PaO₂ (mm Hg)</td>
<td>SaO₂ (%)</td>
</tr>
<tr>
<td>Superior caval vein</td>
<td>3</td>
<td>73-79</td>
<td>7</td>
<td>63</td>
</tr>
<tr>
<td>Inferior caval vein</td>
<td>3</td>
<td>82</td>
<td>6</td>
<td>70</td>
</tr>
<tr>
<td>Pulmonary artery (trunk)</td>
<td>23/8</td>
<td>73-79</td>
<td>30/15</td>
<td>72</td>
</tr>
<tr>
<td>Right branch</td>
<td>—*</td>
<td>—*</td>
<td>41/34†</td>
<td>92</td>
</tr>
<tr>
<td>Left branch</td>
<td>23/8</td>
<td>77-72</td>
<td>38/17</td>
<td>69</td>
</tr>
<tr>
<td>Brachial artery</td>
<td>97</td>
<td>97</td>
<td>97</td>
<td>92</td>
</tr>
<tr>
<td>Right ventricle output (L/min)</td>
<td>—</td>
<td>8.8</td>
<td>5.8</td>
<td>—</td>
</tr>
</tbody>
</table>

*Catheter cannot be passed into the right branch.
†“Arterial” curve in wedge position.
arteries communicate with the pulmonary vascular bed. The similarity of the roentgenological findings in the first three patients suggests, that in patients 1 and 3 the pulmonary vascular bed also communicates via transpleural arterial connections with the internal mammary artery or intercostal arteries, and that the bronchial arteries are not primarily involved. On the other hand, the effective collateral systemic pulmonary blood flow in patients 1 and 3 may indicate that there is no veno-restrictive or veno-occlusive disease. Venous changes of this kind are not present when there are "accessory pulmonary arteries," that is, vessels coming directly from the aorta which have the distribution of pulmonary arteries and which replace branches of the normal pulmonary artery.

A major problem is whether the anatomic disorders in patient 2 are congenital or acquired. The fact that there is no enlargement of true bronchial arteries within the lung strongly suggests that the process is developmental rather than acquired.

Patient 4 shows a fairly well established picture which, following the classical work of Liebow and co-workers,7 has been described repeatedly.10 Here the systemic blood probably comes from bronchial arteries. We included this patient to illustrate the condition wherein neither pulmonary nor systemic blood flows through the capillary bed of ventilated alveoli.

The finding that one of the lungs has no measurable oxygen uptake, whereas the systemic arterial oxygen saturation is normal, will occur under two conditions: (condition a) when there is no functioning capillary bed, either because the alveolocapillary structures are completely destroyed or because existing capillaries are not perfused, and (condition b) when the functioning capillary bed is perfused by fully or nearly fully oxygenated blood. These conditions can easily be differentiated by bronchospirometry: in condition (a), no carbon dioxide output is found, whereas in condition (b) carbon dioxide is excreted. Under conditions of artificially induced systemic arterial hypoxemia, in condition (a), a measurable oxygen uptake will not be found, whereas in condition (b), oxygen uptake has to be demonstrated.

Estimation of the effective portion of the systemic-pulmonary collateral circulation with the direct Fick method is only valid when the functioning capillary bed (that is, pulmonary capillaries of ventilated alveoli) of the affected lung is perfused exclusively by systemic arterial blood.2,18,20 The latter condition is present when the affected lung has a measurable carbon dioxide output with absent, or undetectable small, oxygen uptake (patients 1 and 3). When the systemic arterial oxygen saturation is normal, even a small oxygen uptake (for example, 10 ml per minute, as in patient 2) cannot be explained by systemic collateral circulation, unless the latter amounts to several liters per minute.

In the case published by Landrigan and associates20 under the diagnosis "absent left pulmonary artery," the oxygen uptake of the affected lung amounted to 36 ml per minute when both lungs were breathing oxygen and the systemic arterial oxygen saturation was 100%. This can only indicate that the affected lung is not being exclusively perfused by systemic arterial blood. In this patient, calculation of effective collateral circulation therefore was impossible.

Recently, Oakley and associates21 described a patient ("congenital absence of a pulmonary artery") in whom the oxygen uptake of the affected lung was 27 ml per minute, whereas the systemic arterial oxygen saturation amounted to 94.8%. This patient had no oxygen excretion in the diseased lung, when this lung breathed helium and the contralateral lung breathed pure oxygen. The authors attributed this phenomenon to a high diffusion gradient for oxygen in the affected lung. The explanation seems difficult to reconcile with the undoubted oxygen uptake under normal bronchopulmonary conditions. In our patient 1 we carried out a similar investigation: The diseased lung excreted 35 ml of oxygen per minute while this lung breathed pure nitrogen and the contralateral lung pure oxygen. In consequence, there is, at least in
our patient, no evidence that diffusion of oxygen in the affected lung was impeded by an abnormal diffusion barrier.

A question arises as to why, in the patients 1 and 3, in whom the effective collateral circulation was calculated to be about 0.9 L per minute, the dye-dilution curves failed to show this considerable collateral circulation, although this technique claims to permit detection of abnormally early recirculation of as little as 5% of the right ventricle output. Two explanations are possible: (1) The systemic blood drains via precapillary anastomoses, pulmonary capillaries, and systemic veins to the right heart. (2) The systemic blood drains, via the pulmonary capillaries and veins, directly to the left heart but, due to a marked inequality of perfusion of the affected lung, the recirculation peak is flattened to an extent that the dye concentration at the brachial artery is too low to permit detection. Better results may be obtained by injection of the indicator into the ascending aorta and simultaneous sampling from both brachial artery and left atrium (via the transseptal approach). This method has, however, not been applied in our patients.

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References

Coronary Care Units

I am determined to scrutinize even a not too serious suggestion that such statistically acceptable answers must be sought and found before sensible decision can be made to proceed with development of intensive care units for patients with acute myocardial infarction. . . .

So long as development of intensive care units is without threat to survival and comfort of those for whose care they are designed, reason for their existence is self-evident. It was self-evident when, by his intervention, the physician first accomplished return of effective cardiac function in the arrested or fibrillating ventricles of a patient who was restored thereby to extended life of acceptable quality.

. . . An intensive care unit by its very name declares devotion to a self-evident goal. Intensified care of the ill is not inevitably equivalent to improved care but breaches in the identity are commonly achieved only by the stupid and the doctrinaire. Moreover, assiduous pursuit of a goal oftentimes reaps astonishingly unexpected gains.

Studies on Anomalous Collateral Systemic-Pulmonary Circulation: Report of Four Cases

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