CLINICAL PROGRESS

The Clinical Significance of the Pulmonary Collateral Circulation

By Alfred P. Fishman, M.D.

The normal lung contains two distinct circulations of strikingly different origins, proportions, and functions. On the one hand is the huge pulmonary circulation, which originates in the right heart and pours the entire venous cardiac output into the lungs for gas exchange; on the other, is the diminutive systemic circulation, which delivers oxygenated blood to the lungs for the sustenance of its tissues, nerves, vessels, and conducting airways. Vascular connections between the two circulations are sparse and require elaborate injection techniques for their display. Among the most elusive of these communications are the precapillary vessels that join pulmonary to bronchial arteries (fig. 1).

The systemic blood supply of the lung undergoes a remarkable proliferation in various disorders of the heart and lungs: 1–4 old vessels enlarge and become tortuous; new vessels appear and join with the old to form bizarre medusan patterns. And, in contrast to the normal lung, in which precapillary communications between the two circulations are difficult to demonstrate, the enlarged precapillary anastomoses between the pulmonary and systemic circulations are readily apparent.

There are several popular synonyms for the systemic circulation of the lung. In this presentation, two of these—"bronchial circulation" and "pulmonary collateral circulation"—will be used interchangeably. It should be emphasized, however, that even though the designation "bronchial circulation" is retained for the sake of convention, the term is inadequate since (1) the systemic arterial vessels to the lung include arteries other than the bronchial arteries, and (2) the systemic arterial blood is distributed to intrapulmonary structures as well as to the bronchi.

It is generally held that as the pulmonary collateral circulation expands it assumes new functions. In figure 2 are illustrated the functions that may be subserved by the normal and by the expanded pulmonary collateral circulations. Precisely which function the expanded circulation will assume depends on two major conditions: (1) the total quantity of blood transported by the pulmonary circulation per unit time, i.e., the combined rates of pulmonary arterial and pulmonary collateral blood flow and (2) the access of hypoxemic collateral arterial blood to the pulmonary alveoli. For example, when the pulmonary arterial blood flow is normal and the systemic arterial blood is well oxygenated, the increment in total pulmonary blood flow contributed by the collateral arterial circulation may constitute a hemodynamic burden (middle panel); on the other hand, when the pulmonary arterial inflow of venous blood is abnormally low and the collateral arteries deliver hypoxemic blood to the alveolar capillaries, the collateral circulation may participate in oxygen uptake (right panel).

It should be noted that the anatomic peculiarities of the pulmonary collateral circula-

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The pulmonary arteries (A) and the bronchial arteries (B) are joined by fine anastomotic channels (C). After Ruysch (1731)\textsuperscript{7}

Figure 1

The pulmonary arteries (A) and the bronchial arteries (B) are joined by fine anastomotic channels (C). After Ruysch (1731)\textsuperscript{7}

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The remainder of this essay considers the clinical significance of the pulmonary collateral circulation in terms of the three potential functions depicted in figure 2: nutrient, hemodynamic, and respiratory.

The Pulmonary Collateral Circulation as a Nutrient Circulation

In the normal lung, the collateral arterial circulation satisfies the usual criteria for a nutrient circulation: it is small, it carries oxygenated blood, and its blood is distributed to the walls of the tracheobronchial tree, the supporting framework of the lungs, and the adventitial aspect of the pulmonary arteries and veins.\textsuperscript{8}

It seems self-evident that this diminutive circulation lacks the capacity to transport large quantities of blood. This view is consistent with the bulk of the experimental evidence that the normal collateral blood flow is approximately one to two per cent of the cardiac output;\textsuperscript{12-15} but, it is contrary to that of I. de B. Daly and co-workers,\textsuperscript{8} who found in special preparations that the collateral circulation may exert an appreciable hemodynamic effect on the pulmonary circulation. The apparent inconsistency may lie in the nature of Daly's experiments: their design seems more apt to disclose the ultimate potential of the collateral circulation rather than its usual performance either in vivo or during less drastic experimental circumstances.

The nutrient circulation of the lungs has attracted clinical attention on three counts. The first is the observation that occlusion of either a very large or a very small pulmonary artery by ligation or by an embolus is rarely followed by pulmonary infarction. The viability of the pulmonary tissue in the face of pulmonary arterial occlusion (fig. 3) may logically be attributed to the nutrient circulation.

The second reason for clinical interest (fig. 4) is the hypothesis that obliteration of the nutrient circulation may be involved in the genesis of "ischemic pulmonary disease." A clinical example of a consequence of this disorder would be "emphysema."\textsuperscript{10-19} Unfortunately, this plausible hypothesis lacks convic-
Schematic representation of three potential functions of the systemic arterial blood supply to the lungs. For each function is indicated: (1) the relative sizes of the pulmonary arterial (PA) and the bronchial arterial (BA) circulations and (2) a corresponding clinical state.

Figure 2

Note on several accounts: (1) the practical difficulty of deciding if a sparse collateral circulation in clinical “emphysema” is the cause, or merely a consequence, of the intrinsic pulmonary disease; (2) the evidence that in some instances of “emphysema,” the collateral arterial circulation of the lung may be unusually prominent; (3) the ambiguous use of the term “emphysema” in both clinical and anatomic descriptions; and (4) the uncertainty concerning the relevance of animal experiments in which the pulmonary collateral blood supply of the lung is deliberately compromised, to the various types of “emphysema” encountered in clinical medicine. At the present time, the notion of “ischemic pulmonary disease” is intriguing, but without clinical or experimental substance.

The third reason for clinical interest lies in the demonstration that primary carcinoma of the lung—in contrast to metastatic carcinoma—may be deprived of a pulmonary arterial blood supply by thrombosis. An extreme instance of this phenomenon is illustrated in figure 5. In such cases of bronchogenic carcinoma with interrupted pulmonary arterial blood supply, the adjacent bronchial arteries have been found to be unusually prominent. It seems reasonable to implicate the combination of the expanded systemic arterial blood supply and the strategic location of the neoplasm with respect to the tracheobronchial tree in the brisk hemoptysis of patients with bronchogenic carcinoma. Moreover, the blood supply to a pulmonary neoplasm may also determine the success of measures designed to destroy the tumor. For example, the introduction of cancerocidal agents directly into the pulmonary artery for the treatment of a bronchogenic carcinoma presupposes that mixed venous blood has direct access to the tumor, but, as indicated...
Posteroanterior x-rays of the chest in a young woman (O.B.) prior to (1945), and 8 years after (1953), ligation of the left pulmonary artery. Despite the lack of a pulmonary arterial blood supply, the ventilatory performance of the left lung was virtually unimpaired.\(^\text{11}\)

above, the bulk of the evidence is against this view. Finally, the oxygenation of the blood to a pulmonary tumor may influence not only its pattern of growth but also its response to therapeutic agents.

The Hemodynamic Effects of the Pulmonary Collateral Circulation

The arterial and venous portions of the pulmonary collateral circulation need not expand proportionately.\(^\text{22, 28}\) Moreover, the two vascular segments seem to be stimulated to proliferate by entirely different pathologic processes. Consequently, they are considered separately.

Striking enlargement of the collateral arterial circulation has been observed both in chronic fibrosing pulmonary disease\(^\text{24-26}\) and after ligation of one pulmonary artery.\(^\text{27-31}\) In these situations, the normal pulmonary arterial blood flow is supplemented by an increment in blood flow from the collateral arterial circulation. Although some proliferation of the collateral arterial vessels also occurs in the vicinity of healed surgical incisions of the lung and of localized pulmonary lesions such as tuberculosis,\(^\text{3, 32, 33}\) bronchiectasis,\(^\text{24-26}\) bronchogenic carcinoma,\(^\text{3}\) and pulmonary emboli,\(^\text{1, 29}\) it seems reasonable to look to the human subjects with generalized pulmonary inflammatory disease, and to both dogs and humans with ligated pulmonary arteries for maximum hemodynamic effects.

It should, perhaps, first be emphasized that the anatomic display of large collateral arteries provides, per se, no reliable measure of their hemodynamic significance. The uncertainty stems from the lack of information.

Figure 3

Posteroanterior x-rays of the chest in a young woman (O.B.) prior to (1945), and 8 years after (1953), ligation of the left pulmonary artery. Despite the lack of a pulmonary arterial blood supply, the ventilatory performance of the left lung was virtually unimpaired.\(^\text{11}\)

Figure 4

Posteroanterior x-ray of the chest of a 36-year-old man with bullous ("atrophic") emphysema. The patient does not have either chronic bronchitis or other apparent cause for the rarefaction of the lung. The relationship of bullous emphysema to "ischemic pulmonary disease" is unclear.
concerning the behavior of the anastomotic channels: do they serve merely as passive conduits between the high-pressure systemic circulation and the low-pressure pulmonary circulation or do they operate, instead, as high-resistance, sphincteric vessels under the control of systemic vasomotor nerves. In favor of the latter view is the sphincteric construction of the collateral arteries as well as the lack of evidence for either left ventricular enlargement or an abnormally wide systemic pulse pressure in patients with enormous overgrowths of the collateral arterial circulation. On the other hand, Alley and co-workers have shown by angiocardiography that in a lung that has been virtually destroyed by chronic suppurative disease, the anastomotic channels do seem to serve as low-resistance conduits, delivering arterial blood into the pulmonary circulation with sufficient force to divert mixed venous blood away from the anastomotic sites. All-in-all, the available evidence suggests that (1) local expansion of the collateral arterial circulation in the vicinity of local pulmonary disease is apt to be without appreciable hemodynamic effect (this conclusion is consistent with the normal pulmonary hemodynamics that characterize multilobar, but circumscribed, bronchiectasis) (2) generalized expansion of the collateral arterial circulation may affect the distribution of mixed venous blood throughout the lung; this situation is apt to obtain in universal bronchiectasis or in generalized, suppurative disease of the lung, and (3) the largest collateral arterial circulations are smaller than many of the left-to-right shunts encountered in congenital heart disease; this conclusion is supported by the lack of left ventricular enlargement and of pulmonary hypertension in patients with large collateral circulation. It should be noted that the inability of the expanded collateral circulation to affect, per se, the behavior of either the pulmonary circulation or left heart does not exclude the possibility of a secondary role, e.g., in aggravating or producing pulmonary hypertension in patients with restricted pulmonary vascular beds.

The appraisal of the hemodynamic significance of an expanded collateral venous circulation is still in its infancy. The largest collateral venous circulations have been induced experimentally by ligation of lobar pulmonary veins and occur spontaneously in "emphysema." The general uncertainties that attend the indiscriminate use of the term "emphysema" have been mentioned.

### Table 1

"Effective" Collateral Blood Flow in Twelve Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Diagnosis</th>
<th>&quot;Effective&quot; collateral L./min.</th>
<th>&quot;Effective&quot; collateral Total × 100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Permanent occlusion of one pulmonary artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O.B.</td>
<td>11 Years after ligation</td>
<td>.86</td>
<td>8</td>
</tr>
<tr>
<td>A.B.</td>
<td>6 Months after occlusion</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Occlusion of one pulmonary artery by balloon</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J.S.</td>
<td>Carcinoma</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>J.Me</td>
<td>Carcinoma</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>P.C.</td>
<td>Carcinoma</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>J.C.</td>
<td>Bronchiectasis</td>
<td>.32</td>
<td>4</td>
</tr>
<tr>
<td>T.C.</td>
<td>Bronchiectasis</td>
<td>.38</td>
<td>4</td>
</tr>
<tr>
<td>J.G.</td>
<td>Idiopathic clubbing</td>
<td>.35</td>
<td>8</td>
</tr>
<tr>
<td>Congenital atresia of main pulmonary artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J.W.</td>
<td>Tetralogy of Fallot</td>
<td>2.54</td>
<td>100</td>
</tr>
<tr>
<td>W.Y.</td>
<td>Tetralogy of Fallot</td>
<td>1.35</td>
<td>100</td>
</tr>
<tr>
<td>A.K.</td>
<td>Tetralogy of Fallot</td>
<td>3.79</td>
<td>100</td>
</tr>
<tr>
<td>B.C.</td>
<td>Tetralogy of Fallot</td>
<td>5.00</td>
<td>100</td>
</tr>
</tbody>
</table>

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Table 2

<table>
<thead>
<tr>
<th>Clinical state</th>
<th>Clubbed digits</th>
<th>&quot;Effective&quot; collateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic clubbing</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Pulmonary congenital atresia</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Ligated pulmonary artery</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>Bronchogenic carcinoma</td>
<td>+</td>
<td>0</td>
</tr>
</tbody>
</table>

previously. With particular respect to the collateral venous circulation, the published reports fail to establish if the proliferation of the collateral veins is a regular feature of all types of "emphysema," including the rarefied, destroyed lungs of "chronic bronchitis and emphysema," or only of bullous disease of the lung.

In the normal lung, the proximal bronchial veins drain predominantly into the right atrium.22, 23 The arrangement of the alternate pathways, which ordinarily favors the drainage of these vessels into the right atrium, is schematically depicted in the left half of figure 6. Liebow23, 38 has suggested that with the advent of right heart failure, the combination of an elevated right atrial pressure and insufficient bronchial venous valves may re-route the proximal bronchial venous drainage into the left atrium.23, 38 According to this concept illustrated in the right half of figure 6, right heart failure should be self-perpetuating by virtue of the increased systemic arterial hypoxemia and the pulmonary arterial hypertension which it effects.

Although the proposed increment in bronchial venous return is of conceptual interest, its practical meaning is obscure. A major difficulty in interpretation is the complex interplay of initiating and aggravating factors in hypoxemic types of right heart failure so that quantitative measurements of bronchial venous return to the left heart become exceedingly complicated. Recent advances in respiratory methodology, however, have revived the prospect of separating the increase in anatomic venous admixture contributed by the bronchial veins from the "venous-like" admixture that results from inhomogeneity of alveolar ventilation and perfusion.

The Role of the Pulmonary Collateral Circulation in Alveolar-Capillary Gas Exchange

By a series of ingenious and complicated experiments on dogs that had been subjected to ligation of a major pulmonary artery, Bloomer and co-workers10 showed that the expanded collateral circulation was available, at least in part, to participate in alveolar-capillary gas exchange. Since these experiments involved deep anesthesia, the blood that reached the lung by way of the collateral circulation was unsaturated with oxygen, and could therefore take up oxygen from the alveolar gas.

Unfortunately, such a protocol cannot be applied to the study of unanesthetized animals or man, since their collateral arterial blood, which is normally saturated with oxygen, can take up very little additional oxygen from the alveoli (fig. 7). The spirograms, from the right and left lungs, respectively, were obtained by bronchospirometry from a patient who, in 1946, had undergone urgent ligation...
of the left pulmonary artery during the course of an operation designed to close a patent ductus arteriosus. It may be seen that under ordinary conditions of ambient air breathing, the lung with the ligated pulmonary artery barely contributes to oxygen uptake. The reason for this low oxygen uptake is the small diffusion gradient for oxygen (fig. 8). The effect of improving this diffusion gradient, and the demonstration that the lack of oxygen uptake is attributable to the high oxygenation of the collateral arterial blood rather than to the lack of a precapillary pulmonary collateral circulation, is shown in figure 9. This figure illustrates the use of bronchospirometry to induce collateral arterial hypoxemia while maintaining alveolar oxygen tensions in the left lung, at, or above, ambient air values; under this special circumstance, the oxygen uptake by the left lung increased to approximately 60 ml. per minute, indicating that in man, as in the dog, ligation of a pulmonary artery provokes a proliferation of the collateral arterial circulation.

The experiences with two such patients, as well as with the separate measurement of blood flow through each lung, suggested an experimental approach of wider applicability. In each case, a technic was applied that permitted the use of the Fick principle to measure the rate of "effective" collateral arterial blood flow. This approach required (1) the arrest of pulmonary arterial blood inflow and (2) the perfusion of the pulmonary capillaries by hypoxemic collateral arterial blood (fig. 10). In patients with discrete, unilateral pulmonary lesions, the protocols were complicated; they included (1) bronchospirometry for the administration of different inspired gas mixtures to each lung and for the separate collection of the expired gases; (2) cardiac catheterization for the sampling of mixed venous blood and for the inflation of an occlusive balloon in the pulmonary artery to
one lung; and (3) brachial arterial cannulation for the sampling of systemic arterial blood. By way of contrast, in patients whose lungs were perfused solely by systemic arterial blood, e.g., pulmonary atresia (fig. 11), the protocol simply required the measurement of oxygen uptake and the procurement of a blood sample from a peripheral artery.

It should be emphasized that measurements of "effective" flow by the Fick principle, i.e., based on alveolar-capillary gas exchange, can only provide a minimal estimate of the collateral arterial blood flow; by definition, they exclude the "ineffective" flow, i.e., that portion of the total collateral arterial blood flow that bypasses the gas exchanging surface of the lungs. It is also noteworthy that protocols such as those described, which involve the deliberate occlusion of one pulmonary artery, should theoretically augment the collateral flow by decreasing the pulmonary arterial pressure at the outlet of the anastomoses.12,34

The "effective" collateral blood flow was measured in 12 human subjects.11 The clinical description of each subject is related to the value for "effective" collateral flow in table 1. It may be seen that no "effective" collateral blood flow could be detected either in the normal lung or in the lung that contained a bronchogenic carcinoma. By way of contrast, large "effective" flows—approximating normal pulmonary arterial values at rest (and even increasing somewhat during mild exercise)—were measured in the patients with congenital lack of a pulmonary arterial blood supply.43 One of these patients, a young woman with limited exercise tolerance, came to autopsy following an attempt to supplement surgically the pulmonary collateral blood flow through the creation of an additional systemic-pulmonary arterial anastomosis. Unfortunately, the sacrifice of her spontaneous pulmonary collateral circulation (fig. 12) during thoracotomy effected a decrease in the pulmonary blood flow that was not offset by the artificial anastomosis.
**The Role of the Pulmonary Collateral Circulation in the Pathogenesis of Hemoptysis and of Clubbing of the Digits**

There is considerable anatomic evidence to suggest that an abnormal pulmonary collateral circulation may be responsible for hemoptysis in a wide variety of cardiac and pulmonary disorders. For example, the expanded collateral arterial circulation may be responsible for hemoptysis in patients with tuberculous cavitation, bronchiectasis, and bronchogenic carcinoma. In contrast to the relatively high incidence of hemoptysis in bronchogenic carcinoma, the incidence is low in metastatic carcinoma, which lacks an expanded collateral arterial circulation. The collateral venous circulation is generally held to be a major basis for hemoptysis in mitral stenosis. Another cause for hemoptysis in mitral stenosis is pulmonary infarction, which has been ascribed to an excessive collateral arterial inflow into a vascular segment that has been deprived of its pulmonary arterial inflow by a embolus.

In recent years, evidence has been adduced to implicate the pulmonary collateral circulation in the genesis of clubbed digits in certain clinical disorders. But, that this collateral circulation is not the common denominator in all forms of clubbed digits is easily shown. For example, while certain pulmonary diseases commonly associated with clubbing, i.e., bronchiectasis and bronchogenic carcinoma are generally accompanied by an expanded collateral circulation, other pulmonary diseases, such as tuberculosis, with equally prominent collateral arterial circulations are generally not associated with clubbed digits. Moreover, in clinical states such as subacute bacterial endocarditis, in which clubbed digits are common, prominent collateral circulations remain to be identified. Finally, it is beyond the stretch of the imagination to conceive a role for the pulmonary...
collateral circulation in unilateral clubbing.

The possibility that the collateral circulation may, in some instances, be involved in the genesis of clubbed digits has encouraged speculation about which portion of the total collateral arterial inflow could be involved, i.e., the "effective" portion, which participates in alveolar-capillary gas exchange, or the "ineffective" portion, which bypasses the gas-exchanging surface of the lung. Evidence can be mustered from published reports to support either view. Our own experience (table 2) and that of others, would favor the "ineffective" portion. This evidence is predominantly one of exclusion. For example, patient O.B., with the ligated pulmonary artery and a large "effective" collateral blood flow (table 1) had no clubbing of the digits. Similarly, patient A.B. with bronchogenic carcinoma and clubbed digits had no appreciable "effective" flow. On the other hand, the results of others, using more indirect methods, support the view that it is the "effective" rather than the "ineffective" portion of the collateral blood flow that is responsible for clubbing of the digits. These divergent opinions are cited not as a point of departure for resolving a troublesome question, but rather to emphasize that the relationship between clubbed digits and the pulmonary collateral circulation is still an enigma.

**General Comments**

Before closing, three other aspects of the pulmonary collateral circulation warrant mention: (1) the limitations of available methodology, (2) the insights that the pulmonary collateral circulation affords into the growth of collateral circulation in general, and (3) the therapeutic implications of the pulmonary collateral circulation.

It is to be anticipated that a full understanding of the clinical significance of the
The anatomic basis for measuring “effective” collateral blood flow in subjects with either pulmonary atresia and a ventricular septal defect or truncus arteriosus. Since the total blood supply to the lungs derives entirely from the systemic circulation, the Fick principle is easily applied.\(^\text{11}\)

Pulmonary collateral circulation will result from a combination of clinical, physiologic, and anatomic approaches. At the present time, although clinical descriptions may often be inadequate for precise clinicopathologic correlations, and many clinical states remain to be explored by anatomic methods, it is predominantly the physiologic methodologies that are lacking. For example, there is no reliable way, in either intact animal or man, to measure rates of collateral blood flow that are less than 10 to 15 per cent of the cardiac output.\(^\text{11, 13, 15}\) The suspicion that lesser flows may be involved in clinical syndromes, e.g., clubbing of the digits, has driven investigators to indirect methods that can only establish qualitatively the presence of collateral arterial inflow into the lungs. These indirect methods have involved the sampling of highly oxygenated blood from diseased portions of the lung and the recording of distorted pressure pulses from diseased regions.\(^\text{33, 48, 49}\) But except in instances of flagrant anastomoses—such as in the “destroyed lung”—these measurements are too susceptible to technical limitations and to misinterpretation to inspire confidence.

The clinicopathologic studies of the pulmonary collateral circulation have thrown some light on the general principles involved in the development of collateral circulations.\(^\text{50, 51}\) For example, Liebow and co-workers have shown that (1) in accord with the observations of Schlaepfer,\(^\text{31}\) the pulmonary collateral circulation develops more rapidly in the newborn than in the adult;\(^\text{4}\) (2) the decrease in blood pressure beyond the site of pulmonary vascular occlusion is probably a stimulus for the expansion of the collateral circula-
tion; and (3) purely mechanical factors are insufficient to account for the extent or the organization of the expanded collateral circulation. Weibel has extended these studies by demonstrating the sequences of development of the expanded collateral circulation: an initial enlargement of existing vessels followed by a period of angiogenesis. These observations of Schlaepfer, Liebow, and Weibel are all consistent with the demonstration (table 1) that congenital atresia of a pulmonary artery is associated with disproportionately larger pulmonary collateral blood flows than is ligation of a pulmonary artery in adulthood.

Finally, Liebow and co-workers have attempted to turn the pulmonary collateral circulation to therapeutic advantage. They have devised procedures that are designed to relieve myocardial ischemia and to bring oxygenated blood into the aorta of patients with congenital transposition of the great vessels. Up to this time, the practicality of these therapeutic procedures has not been established.

Summary

The clinical significance of the normal and the expanded pulmonary collateral circulation is considered from the point of view of its nutrient, hemodynamic, and respiratory functions.

In the normal lung, the collateral arterial circulation is too diminutive to serve other than a nutrient function. Its roles in preventing pulmonary infarction after embolization, in producing "ischemic lung disease," and in the direct treatment of pulmonary carcinomas are considered.

The expanded collateral arterial circulation associated with localized pulmonary lesions or which occurs after ligation of a pulmonary artery seems to be without appreciable hemodynamic effect on either the pulmonary circulation, the left heart, or the systemic circulation. Only in patients with generalized suppurative disease of the lung has it been shown to affect the pulmonary circulation by diverting mixed venous blood away from diseased areas. The possibility exists that the increment in pulmonary blood flow contributed by the collateral arterial circulation may aggravate pulmonary hypertension in patients with diffuse restriction of the pulmonary vascular bed.

The venous portion of the expanded collateral circulation may conceivably contribute to pulmonary hypertension by aggravating systemic arterial hypoxemia in patients with right heart failure from hypoxemic cor pulmonale.

The expanded collateral arterial circulation may assume a major respiratory function in subjects with systemic hypoxemia and curtailed pulmonary blood flow. In such cases, the "effective" pulmonary collateral blood flow has been shown to equal normal values for pulmonary arterial blood flow.

Different portions of the pulmonary collateral circulation may be responsible for hemothysis in different diseases: in pulmonary disease, the arterial portion; in mitral stenosis, the venous portion; in pulmonary infarction, an imbalance between the two.

The relationship between the pulmonary collateral circulation and clubbing of the digits remains uncertain.

The pulmonary collateral circulation is considered, finally, with respect to (1) limitations of present methodology, (2) principles involved in the development of collateral circulations in general, and (3) therapeutic implications.

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