An Experimental Study of the Effect of Chronic Atelectasis on Pulmonary and Bronchial Blood Flow

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Four dogs and one patient with uncomplicated atelectasis of an entire lung were studied. Arterial oxygen saturations were nearly normal, indicating diminished flow to the atelectatic lung. Angiography confirmed this finding. Vinyl plastic injections showed no evidence of increased bronchial collateral circulation and pathological examination of the atelectatic lung did not demonstrate any bronchiectasis.

Studies of the effects of acute and chronic atelectasis of one lung in dogs have demonstrated that blood flow to the collapsed lung promptly diminishes. In the acute experiments this decrease was due to increased resistance in the vascular bed of the atelectatic lung. This increased resistance is not due solely to the local anoxia or hypercapnia. Anoxia causes increase in resistance but pulmonary venous hypercapnia is associated with diminished pulmonary vascular resistance. This suggests that the mechanical collapse contributes significantly to the diminished flow.

Liebow et al. have shown that following ligation of the pulmonary artery in dogs and in patients with bronchiectasis, pulmonary stenosis, tuberculosis, carcinoma and emphysema, there is enlargement of the bronchial arteries and their anastomoses with pulmonary arteries. It is postulated that the extensive communication between the high pressure bronchial system and the pulmonary artery leads to reversal of flow in the pulmonary artery and this helps to shunt blood from the diseased areas of lung.

In all the conditions in which enlargement of bronchial arteries has been described in humans, either chronic infection or ischemia could be the causative factor. Ellis occluded the apical lobe bronchi of dogs' lungs with umbilical tape and in some dogs also occluded the bronchial circulation. None of these animals developed increased bronchial collateral except one who developed bronchiectasis. In atelectasis produced by transection of one main bronchus, both infection and ischemia can be avoided but decreased perfusion has been demonstrated. In the experiments to be reported in dogs with chronic unilateral atelectasis, the bronchial collateral was studied by the method of Liebow and distribution of pulmonary flow from the arterial oxygen saturations and by angiography. The findings in a patient with a traumatic rupture of the bronchus corroborated the experimental results.

Methods

Mongrel dogs were anesthetized with pentobarbital, endotracheal intubation was carried out, and a left or right thoracotomy was performed. A Starling pump was used to give positive pressure controlled respiration. The left or right main stem bronchus was isolated and transected and both ends closed with end-over sutures of 4-0 silk. It was possible with some experience to do this in such a manner that the bronchial arteries did not have to be ligated. The chest was closed and the dogs were given penicillin for 4 to 6 days.

Five to nine months after operation the dogs were anesthetized with pentobarbital and the pulmonary vascular system outlined by intravenous angiography. Femoral arterial blood was drawn from the dogs before administering the pentobarbital prior to angiography. The arterial oxygen saturation was determined photometrically by the Hickam method, the pH with a Cambridge glass electrode pH meter at room temperature and corrected to body temperature by subtracting 0.0147 pH units per degree of difference in temperature, and the plasma carbon dioxide with a VanSlyke manometric apparatus. The partial pressure of carbon dioxide was calculated from the latter 2 determinations by the Henderson-Hasselbaleh equation. The dog was then killed and vinyl plastic was injected into the pulmonary artery, pulmonary
veins and bronchial arterial system and trachea according to the method used by Liebow. For controls a dog whose pulmonary artery had been tied for 4 months, and a normal dog, had similar plastic injections. At the time of killing the dog sections of the atelectatic lung were taken for microscopic study.

RESULTS

The arterial blood gas results are shown in table 1. In other studies done on many normal unanesthetized dogs in this laboratory the oxygen saturations range between 94 to 97 per cent, so that only one of these dogs (102) has definite depression of the oxygen saturation. The levels of carbon dioxide tension are also within the limits found on awake, normal dogs in this laboratory. In previous studies by one of us (R.M.P.) we have shown that dogs treated in a similar manner had markedly diminished flow to the atelectatic lung. These high oxygen saturations confirm the fact that these dogs also have only a small portion of the blood going to the atelectatic lung.*

* The percentage of total cardiac output going to each lung in these experimental animals can be calculated from a modification of the method of Comroe. The formula used is:

\[ S_{aO2} \cdot QA + S_{vO2} \cdot (1-QA) = S_{aO2} \]

where \( S_{aO2} \) = saturation of blood coming from normal lung

\( S_{vO2} \) = saturation of blood coming from atelectatic lung

\( S_{aO2} \) = systemic arterial saturation

\( QA \) = fraction of total cardiac output going to atelectatic lung

\( S_{aO2} \) has been measured. The saturation of blood coming from the normal lung was assumed to be the same as arterial blood from normal dogs, i.e., 97 per cent for this laboratory. The saturation of blood from the atelectatic lung is the same as mixed venous and was estimated in this study to be 75 per cent. This is a high figure for dogs who were struggling. If the estimate is too high the calculated flow to the atelectatic lung would be too great. No pulmonary artery samples were drawn because this demands anesthesia and might depress respiration and therefore invalidate the assumption of full saturation of blood draining the atelectatic lung unless 100 per cent oxygen was used. The mean systemic saturation in these dogs was 93.8 and the calculated mean flow to the atelectatic lungs was 15 per cent of the total cardiac output. In our previously reported studies we have found that 45 per cent of the total flow goes to the left lung in normal dogs.

The angiography demonstrated in all animals that there was marked decrease in the volume of the lung on the side of the transected bronchus (fig. 1). This occurred promptly following surgery and the degree of collapse varied little with time. The resulting extra space in the hemithorax was filled by some counterclockwise rotation, displacement of the heart toward the involved side and overexpansion of the opposite lung. In the animals whose left main bronchus had been transected, the right apical and intermedidate diaphragmatic lobes revealed compensatory overexpansion. In the dog with transection of the right main bronchus, the apical and diaphragmatic lobes of the left lung revealed compensatory overexpansion. The caliber of the main pulmonary artery was normal in every case, while the caliber of the main branch to the transected side was decreased. There was a marked decrease in the number of third and fourth order branches from the pulmonary artery visualized in the atelectatic as compared to the normal side.

At autopsy the aerated lung filled most of the thoracic cage. The collapsed lung appeared uninfected and there were no adhesions or only a few along the parietal pleural suture line. There was no evidence of any aeration of the experimental lung in any animal.

The plastic injections showed essentially identical (figs. 2 and 3) bronchial arterial supply to both lungs and comparable to the normal control. The dog with a ligated pulmonary artery (fig. 4), on the other hand, had massive increase in the bronchial collateral. The only other significant finding on these plastic injections was an apparent increase in

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**TABLE 1.—Femoral Arterial Blood Studies at Time of Killing**

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Duration of Atelectasis</th>
<th>Cont. Cap. vol. (%)</th>
<th>Sat. (mEq./L.)</th>
<th>CO2 (mm. Hg)</th>
<th>pH</th>
<th>PCO2 (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>100 L.</td>
<td>0 mo.</td>
<td>20.4</td>
<td>21.5</td>
<td>96</td>
<td>22.5</td>
<td>7.34</td>
</tr>
<tr>
<td>102 R.</td>
<td>5 mo.</td>
<td>23.9</td>
<td>26.2</td>
<td>91</td>
<td>23.6</td>
<td>7.42</td>
</tr>
<tr>
<td>106 L.</td>
<td>5 mo.</td>
<td>16.8</td>
<td>17.9</td>
<td>94</td>
<td>21.3</td>
<td>7.28</td>
</tr>
<tr>
<td>107 L.</td>
<td>6 mo.</td>
<td>18.7</td>
<td>20.0</td>
<td>94</td>
<td>20.4</td>
<td>7.35</td>
</tr>
</tbody>
</table>
UNILATERAL CHRONIC ATELECTASIS

FIG. 1. Angiocardiogram of a dog 6 months after ligation of the left main stem bronchus. The right main pulmonary artery is 2 or 3 times the normal size while the left is not visible because of the shift and rotation of the heart. Note the small left descending pulmonary vessel (arrow) seen through the cardiac shadow, and compare with the one on the right, which is two or three times larger.

the size of all elements of the normal with a decrease in the size of these in the collapsed lung.

Histologic preparations of the atelectatic lung revealed that the bronchioles were widely dilated and completely filled with opaque basophilic mucus. The epithelium was well preserved as ciliated cuboidal structures in some areas and pseudostratified columnar cells in others. The muscular layer was not remarkable. Particularly good examples of bronchial arteries were present and these showed no tendency to medial hypertrophy, dilatation or collateral network formation. Except for a few rare subpleural collections, the alveoli were totally collapsed. There was no evidence of hemorrhage, exudative reaction, necrosis, scar formation or bronchiectasis. The vascularity and lymphoid tissue were not remarkable.

CLINICAL

At the time these animal studies were being made a 15 year old white boy was admitted to the North Carolina Memorial Hospital who 6 months before had been run over by a loaded trailer. He suffered a rupture of the left main bronchus about 0.5 cm. distal to the carina, which was completely healed when he was studied here. A cardiac angiogram (fig. 5) demonstrated a normal appearing main pulmonary artery. The right main pulmonary artery appeared enlarged and all of its branches were wider than usual. The left main pulmonary artery, in contrast, was small measuring less than half the size of the right and its major branches were proportionately smaller than the corresponding branches on the right. Bronchial arteries were not seen. Arterial blood was drawn at rest and during exercise. There was a moderate depression of the oxygen saturation as shown in table 2 but calculations similar to those used in the dog experiments show flow through the atelectatic lung was less than 20 per cent of total cardiac output. A pneumonectomy was done and no enlargement of the bronchial arteries and their anastomoses were seen at operation or pathological examination.

DISCUSSION

These studies have demonstrated again that atelectasis causes a marked decrease in pulmonary blood flow to the affected lung.

<table>
<thead>
<tr>
<th>Date</th>
<th>Condition</th>
<th>Cont. vol. (%)</th>
<th>Cap. vol. (%)</th>
<th>Sat.</th>
<th>CO2 (mEq/L)</th>
<th>pH</th>
<th>PO2 (mm.Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 mo. post-injury</td>
<td>Rest</td>
<td>18.2</td>
<td>19.7</td>
<td>92</td>
<td>26.4</td>
<td>7.29</td>
<td>53</td>
</tr>
<tr>
<td>6 mo. post-injury</td>
<td>Exercise</td>
<td>18.4</td>
<td>20.1</td>
<td>91</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 wk. P.O.</td>
<td>Rest</td>
<td>15.9</td>
<td>17.0</td>
<td>93</td>
<td>28.9</td>
<td>7.40</td>
<td>46</td>
</tr>
<tr>
<td>1 yr. P.O.</td>
<td>Rest</td>
<td>16.7</td>
<td>17.7</td>
<td>94</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 yr. P.O.</td>
<td>Oxygen</td>
<td>18.6*</td>
<td>17.7</td>
<td>100+</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Determined gasometrically with the VanSlyke-Neill apparatus.
Fig. 2 Left. Plastic injection study of dog's lung several months after ligation of the left main bronchus just distal to the carina and viewed posteriorly. The pulmonary arteries (gray) are markedly decreased in size on the left as compared to the normal side. The left main pulmonary artery can be seen (straight arrow) just below the carina and is much smaller than the right (curved arrow). The aorta and its branches are black and the tracheobronchial tree is white. There is no increase in bronchial arteries.

Fig. 3 Middle. Plastic injection study of dog's lung several months after transection of the left main bronchus (arrow) and viewed from the right posteriorly. Many pulmonary arteries (gray) are filled on the left but all are smaller than the anatomically comparable vessels on the right. The aorta is black and the tracheobronchial tree is white. There is no increase in bronchial arteries.

Fig. 4 Right. Plastic injection study of dog's lung several months after ligation of the left main pulmonary artery and viewed posteriorly. The descending aorta and its branches (black) have been partially removed, and most of the remaining vessels in the left hilus represent bronchial arteries, larger and more numerous than usual. The bronchial arteries can be traced well into the periphery of the lung, best seen in the upper lobe region on the left. On the right the bronchial arteries are normally inconspicuous. The pulmonary arteries are gray and the tracheobronchial tree is white.

Staudacker\textsuperscript{13} reported a study in which chronic atelectasis was produced in dogs by constricting a bronchus in one series and in another constricting a bronchus and stripping the bronchial arteries from the bronchus. In the uncomplicated series the findings were similar to ours. With bronchial stripping the diminution of flow to the atelectatic lung was less marked. Their findings on angiogram are similar to ours. Unfortunately, no postmortem findings are included and apparently no studies were made of the bronchial arterial system. Tying the bronchus and stripping the bronchial arteries from it may lead to some necrosis or other anatomic alteration which could explain the difference in blood flow. Ellis found that the bronchial arteries did regenerate in similar experiments but increased collateral was not encountered. His morphologic findings were very similar to ours but no blood flow studies were made.

Unilateral nitrogen breathing causes a decrease in the flow and increased vascular resistance of the anoxic lung but of a lesser de-
UNILATERAL CHRONIC ATELECTASIS

Fig. 5. Representative film of venous angiocardiogram of the patient described. There is cardiac shift and rotation following atelectasis of the left lung. The normal main pulmonary artery can be seen as well as the slightly enlarged right main pulmonary artery and the smaller left main pulmonary artery and its main descending branch.

degree than that seen in atelectasis. In dogs, ventilation of one lung with a gas mixture containing high concentrations of carbon dioxide does not cause a decrease in flow. For this reason it is unlikely that the greater fall in flow with atelectasis in animals is due to the local hypercapnia which is present in atelectasis and absent in low oxygen breathing. Rosenberg in Liebow's laboratory found that wrapping one lung in a constricting bag caused a diminished pulmonary flow to this lung without increased bronchial collateral. He points out that in his study the diminution in flow to the constricted lung may be caused by inflow stasis due to the constricting bag. Since the atelectatic lungs can be readily reexpanded as demonstrated by Webb and Burford and Benfield in dogs and in numerous clinical cases, this cannot be the mechanism in atelectasis. The failure to demonstrate any increased bronchial collateral means that Liebow's theory that back pressure through bronchopulmonary arterial collaterals is not the mechanism by which pulmonary artery flow is locally decreased.

Local constriction of the pulmonary artery bed seems the best explanation. The local constriction is probably greater in the presence of collapsed alveoli since the diminution of flow is greater in degree in atelectatic than anoxic lungs. Benfield et al. reconstituted the stenosed bronchi in their dogs and found that the amount of blood passing through unventilated lung fell from an average of 14 to 1 per cent of cardiac output. There were no morphologic changes in chronically atelectatic lungs or the reinflated lungs. These findings are similar to ours and those of Webb and Burford and Adams. However, in preliminary findings Benfield reported that function does not return completely to normal after re inflation of the chronically atelectatic lung. This abnormality is apparently due to increased pulmonary vascular resistance and if the normal lung is removed, pulmonary hypertension results.

The findings in one clinical case reported here are similar to those in dogs. Before any definite recommendations can be made about the proper treatment for patients with chronic atelectasis the ultimate function of lungs after reconstitution of the bronchial continuity must be clarified. It will be important to determine if the arterial changes described by Benfield are reversible.

SUMMARY

In a series of dogs and in one clinical case the effect of chronic atelectasis due to transection of one bronchus was studied. The blood flow to such a lung diminishes to 10 to 15 per cent of the total cardiac output. No increase in bronchial collateral was noted in these lungs. This diminution in flow is the result of local constriction of the pulmonary artery bed. The constriction is not solely the result of local anoxia but is probably enhanced by collapse of the alveoli.

SUMMARY IN INTERLINGUA

In un serie de canes e in un caso clinic, le effecto de chronic atelectasis causate per transsecction de un broncho eseva studiate. Le fluxo de sanguine verso un tal pulmon se reduce a inter 10 e 15 pro cento del total rendimento cardiac. Nulle augmento del collateral
bronchial esseva notate in iste pulmones. Le diminution del fluxo es le resultato del constriction local in le vasculatura pulmonar. Le constriction non es exclusivamente le resultato de anoxia local. Illo es probabilemente promovite per un collaps del alveolos.

REFERENCES


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