Distribution of Pulmonary Blood Flow after Myocardial Ischemia and Infarction

By Homayoun Kazemi, M.D., Edward F. Parsons, M.D., Laercio M. Valença, M.D., and Denise J. Strieder, M.D.

SUMMARY

Regional distribution of pulmonary blood flow and ventilation was determined with the $^{133}$Xenon technic in the erect position at the bedside in 15 patients an average of 6 days after uncomplicated myocardial infarction (MI) and in five patients with severe angina in the same coronary care unit. Follow-up studies were repeated within 3 to 25 weeks on six of the patients with MI.

There was marked reduction in perfusion to the lung base after myocardial infarction. Patients with severe angina showed some underperfusion of the lower lung zones, but to a much less degree than those with acute MI. The pattern of pulmonary perfusion reverted toward that seen in angina in the follow-up studies of patients with MI. Distribution of ventilation was normal in all patients.

The results of the study suggest that there are probably chronic changes in the pulmonary vasculature of patients with arteriosclerotic heart disease which lead to redistribution of pulmonary blood flow toward the apex, and that the marked underperfusion of the lung base demonstrated following acute myocardial infarction reflects an acute increase in the pulmonary venous and interstitial pressures most likely due to occult left ventricular failure.

Additional Indexing Words:
Angina
Left ventricular failure
Pulmonary perfusion
$^{133}$Xenon

Heart disease as well as lung disease may cause marked changes in the regional distribution of ventilation and blood flow in the lungs. If clinical examination fails to reveal these changes, then their significance may remain obscure.

Measurements of regional pulmonary blood flow using radioactive gases have shown that the basal segments of the lung are relatively underperfused in patients with mitral stenosis$^{1,2}$ and left ventricular failure.$^{3}$ These changes are attributed to increased pulmonary venous and arterial pressures, interstitial pulmonary edema, increased pressure in the perivascular space, or irreversible vascular changes in the dependent parts of the lungs. Since myocardial infarction is capable of changing cardiac output and intravascular pressures, it may also alter regional distribution of pulmonary perfusion. This study was undertaken to determine the regional distribution of pulmonary perfusion and ventilation in patients with uncomplicated acute myocardial infarction and patients with severe angina pectoris hospitalized in the same coronary care unit. The results show a relative hypoperfusion of the lung bases in both patient groups, the abnormality being much more marked after myocardial infarction.

Methods

Patient Selection

Twenty patients (16 male and four female)
were studied in the Coronary Care Unit at the Massachusetts General Hospital. Fifteen were proven to have had definite myocardial infarction by serial electrocardiographic and serum enzyme changes, and they were studied 3 to 11 days after infarction (mean, 6 days). The other five had severe chest pain and ischemic changes in the electrocardiogram consistent with the diagnosis of angina pectoris, and they were studied 3 to 7 days after hospital admission (mean, 5 days). Ages ranged from 41 to 76 years. All patients selected were asymptomatic at the time of study and gave no history of complicating disorders, including lung disease, and they had been at rest in bed prior to the study. They showed no clinical evidence of congestive heart failure and experienced no cardiac arrhythmias during the course of the study. Nine of the 15 patients with myocardial infarction gave a history of smoking cigarettes as well as four of the five in the angina group. To assess the degree of obstructive airway disease, vital capacity, timed vital capacity, and peak flow rate measurements were made in all patients at the bedside at the time of study.

Patients were receiving a variety of antiarrhythmic drugs, particularly procainamide by mouth and lidocaine (Xylocaine) given intravenously; but no morphine, nitrites, or catecholamines at the time of study. Six of the patients with myocardial infarction were taking digitalis prior to admission. Other medications employed were anticoagulants and sedatives.

Six patients in the myocardial infarction group had the studies repeated from 3 to 25 weeks after infarction in the laboratory, at which time they had been ambulatory for days or weeks.

**Distribution Studies**

Regional distribution of pulmonary perfusion and ventilation were studied with $^{133}$Xe with the patient seated on the side of his bed. Each study was done from 20 to 30 min after the patients had assumed the sitting position. The technic used was similar to that reported by Ball and associates, and the basic methodology has been presented previously.

Six scintillation counters, three for each lung, were positioned behind the chest of the seated subject. The upper edge of the apical counter was placed at the level of the clavicle in the midclavicular line and the center of the basal counter at the level of the fifth anterior intercostal space. The middle counter was placed equidistant between the two. The average distance between the upper edge of the apical counter and the lower edge of the basal counter was 25 cm. The medial edge of each counter was placed 3 cm lateral to the mid-spinal line with a divergent angle of 5 to 15° between each horizontal pair. Counters consisted of sodium iodide (TI) crystal, 50 mm in diameter and 20 mm in height, collimated with divided lead, 7.5 cm deep. A low-level discriminator was set at 60 kev, to cut off scattered radiation and allow for direct counting of 81 kev gamma rays of $^{133}$Xe. Data were stored on a seven-channel magnetic tape recorder and were later read off through a count rate meter and printed out on an X-Y recorder by a PDP-7 computer.

The subject breathed in a Collins 9-L spirometer, equipped with a CO$_2$ absorber and filled with approximately 2 L of oxygen. Constant volume was maintained in the spirometer by adjusted continuous flow of oxygen into the system. Distribution studies started with the injection of 3 mc of $^{133}$Xe in 5 ml of normal saline through a no. 18 polyethylene catheter in the antecubital vein. Simultaneously the subject held his breath at the end of a tidal inspiration. Because of the very low solubility of xenon in blood, approximately 90% of the injected dose escaped through the pulmonary capillary bed into the alveolar air on the first passage, where it remained in the gas phase during breath holding. Therefore, plateaus of radioactivity were recorded for 10 to 12 sec, the heights of which were proportional to regional pulmonary blood flow. The subject then breathed in the spirometer until even distribution of xenon was achieved in the closed system formed by the lung and spirometer. This point was reached when constant levels of activity were recorded by all the counters. Breath holding was then repeated for 10 sec at the end of a tidal inspiration and plateaus of activity were recorded which were proportional to volume of the aerated lung seen by each counter. The subject then was switched to breathing room air, and open system washout of xenon was recorded as a progressive fall in radioactivity. In the meantime, 3 mc of $^{133}$Xe was added to the spirometer. After complete washout, the subject inhaled one breath of xenon-enriched air from the spirometer and again held his breath for 10 sec. Plateaus of radioactivity were then recorded which represented the regional distribution of ventilation. The study was completed in 8 to 15 min in most subjects.

The plateaus of activity recorded during each breath-holding period were measured in millimeters of graph height, proportional to counts per minute, above background levels. Total activity from the six counters was summed up in turn for perfusion, volume, and ventilation, and then the activity seen by each counter was expressed as a percentage of total counts recorded at each breath-holding period. Indices of regional distribution of function were then presented as ratios of ventilation to volume ($V/\text{unit volume}$), perfusion to volume ($Q/\text{unit volume}$), and

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ventilation to perfusion (V/Q). For example an area having the same relative percentage of ventilation, volume, and perfusion would have indices of 1 for the three measurements.

Mean normal values for xenon distribution studies shown in figures 1 to 3 are from five normal subjects (mean age, 60 years). These values are identical to those from a larger group of normals (22 subjects) with an average age of 32 years, whose values are used as the normal for this laboratory and are comparable to those from other institutions.7,8

Results

Biometric data and results of simple ventilatory tests for all the patients are given in table 1. The myocardial infarction and angina groups were similar in age and sex distribution and ventilatory function. There was evidence of minimal obstructive airway disease in some of the patients in both groups, although the mean FEV1.0 and peak flow rate, as compared to predicted normal values, were normal. The vital capacity was normal or slightly reduced in individual patients in both groups.

Regional distribution of pulmonary ventilation/unit volume was normal in the immediate postinfarction period in all patients (fig. 1). In contrast, regional distribution of perfusion/unit volume was abnormal in all patients, but more so in those with myocardial infarction (fig. 2). The MI patients showed relative underperfusion of the bases of the lungs. This was in contradistinction to the perfusion gradient in normal subjects, which while they were in the erect position, in-

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**Table 1**

<table>
<thead>
<tr>
<th></th>
<th>Myocardial infarction</th>
<th>Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>Age, mean (yr)</td>
<td>56</td>
<td>58</td>
</tr>
<tr>
<td>Sex</td>
<td>12M: 3F</td>
<td>4M: 1F</td>
</tr>
<tr>
<td>FEV1.0, mean &amp; range (% VC)</td>
<td>76% (64-92)</td>
<td>72% (56-83)</td>
</tr>
<tr>
<td>VC, mean &amp; range (% predicted)</td>
<td>79% (62-92)</td>
<td>82% (70-94)</td>
</tr>
<tr>
<td>Peak flow rate, mean &amp; range (% predicted)</td>
<td>88% (74-111)</td>
<td>90% (78-97)</td>
</tr>
</tbody>
</table>

Abbreviations: FEV1.0 = forced expiratory volume; VC = vital capacity.

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**Figure 1**

Regional distribution of pulmonary ventilation per unit volume of aerated lung (V/unit volume) in the upper, middle, and lower lung zones of five normal subjects (average age, 60 years), 15 patients after acute myocardial infarction (average age, 56 years), and five hospitalized with angina (average age, 58 years). Vertical bars are ±2 sd of the mean.

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**Figure 2**

Regional distribution of pulmonary perfusion per unit volume of aerated lung (Q/unit volume) in the three lung zones of normal subjects, 15 patients after myocardial infarction, and five with angina. Vertical bars are ±2 sd of the mean.
Follow-up distribution studies in six patients were carried out between the third and 25th weeks after myocardial infarction, and regional distribution of perfusion/unit volume for these six patients in the acute and follow-up periods are shown in figure 4. There was only partial return of distribution pattern toward normal so that none of the patients had a normal pattern of pulmonary blood flow at the time of the follow-up study.

**Discussion**

Because it is a low pressure system, the pulmonary circulation is influenced by gravity. In the normal lung in the erect position, there are increasing gradients of perfusion and ventilation from apex to base, the perfusion gradient being more marked than that for ventilation. Distribution of pulmonary perfusion depends on the relation of pulmonary arterial to alveolar and pulmonary venous pressures, and therefore, the lung has been divided into zones determined by the relative magnitude of these pressures. The present study shows that following myocardial infarction the gradient of perfusion from top to bottom of the lung is significantly reduced (fig. 2) and remains reduced for at least 3 to 25 weeks thereafter (fig. 4). The perfusion gradient is reduced in patients with angina as well, but the pattern of distribution is much closer to normal (fig. 2).

Diminished perfusion of lung base after myocardial infarction in our patients is similar to that seen in mild to moderate mitral stenosis and probably is brought about by similar mechanisms. In mitral stenosis the basal hypoperfusion in the erect position has been attributed to increased pulmonary interstitial pressure in the dependent parts of the lung, causing constriction of vessels greater than 100 microns and to perivascular fibrosis and thickening of adventitia which predominate in the lower lung zones. These changes are due to increased pulmonary venous pressure, leading to perivascular edema which first appears around the larger pulmonary vessels. The underperfusion of the lung base at follow-up in MI patients and those with angina suggests that chronic changes are present in the pulmonary vascular bed of patients with arteriosclerotic heart disease and that myocardial infarction adds acute changes consistent with elevated pulmonary venous pressure and interstitial edema even in those patients in whom left ventricular failure is not demonstrable clinically. Unfortunately, it was not feasible to obtain direct intravascular pulmonary pressures in our subjects.

Other factors may also lead to relative underperfusion of the lung base but were not
PULMONARY BLOOD FLOW

present in these patients. Aging has been reported to be associated with reduction of blood flow to the lung base, which becomes significant after the age of 65 years. However, patients in this study were compared with normal subjects whose average age was 60 years. Rest in bed prior to the initial study could not have caused hypoperfusion of the lung base because (1) the anginal patients had also been in bed for several days and were treated in the same manner as those with infarction, but they showed a more normal pattern of pulmonary perfusion; (2) significant hypoperfusion was also present in the six patients with MI who returned for follow-up studies in the laboratory and had been ambulatory for sometime before then; and (3) all patients had been upright for more than 20 min before being tested. Reduced lung volume should also be mentioned, but in view of the normal or minimally reduced vital capacity of our patients (Table 1) a major role cannot be ascribed to this mechanism. In addition, since the breath was held at the same relative lung volume during the measurements of regional blood flow in all the patients and the normal controls, the underperfusion of the lung base in the patients was not artificially created by breath holding at a lower lung volume as compared to controls.

Hypoxemia, a known complication of acute myocardial infarction, causes pulmonary vasoconstriction, which in turn brings about more uniform distribution of pulmonary blood flow. Acute hypoxemia was not present at the time of the study, since the measurements were carried out while the patients breathed oxygen. However, chronic hypoxemia after myocardial infarction cannot be ruled out as a possible contributing factor to the abnormal pattern of pulmonary perfusion seen in some of these patients, particularly those in the follow-up studies. Changes in regional ventilation may affect regional blood flow. Such mechanism is effectively ruled out in these patients since the regional distribution of ventilation was normal with a gradient of 1:2 from apex to base (Fig. 1).

In summary, this study indicates a redistribution of blood flow in the lungs of patients with recent myocardial infarction with increased perfusion to the apex and decreased perfusion to the base. These changes are more marked than those observed in patients with angina or after recovery from myocardial infarction. This pattern of perfusion abnormality probably reflects the effects of acute increase in pulmonary venous and interstitial pressures in the days following myocardial infarction.

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