Arterial Hypoxemia Following Acute Myocardial Infarction

By A. Valencia, M.D., and J. H. Burgess, M.D., F.R.C.P.(C)

SUMMARY
Arterial hypoxemia was common in 53 patients following acute myocardial infarction. It was most marked in patients with evidence of left ventricular failure. Arterial oxygen tension, however, was reduced in many of the patients who were without evidence of failure.

An increase in arterial oxygen tension following three deep breaths suggested maldistribution of ventilation. Right-to-left shunting of blood was demonstrated by 100% oxygen studies. There was a significant correlation between reduced arterial oxygen tension and low pulmonary diffusing capacity, but a poor correlation between the former and a low cardiac index. We suggest that the hypoxemia following acute myocardial infarction is the result of abnormalities in small airways as well as of vascular congestion in the lungs.

The patients with the lowest arterial oxygen tension at the time of study subsequently had more marked arrhythmias and a higher mortality rate.

Additional Indexing Words:
Maldistribution of ventilation Diffusing capacity Cardiac index

Reduced arterial oxygen tension (Pao₂) is commonly found after acute myocardial infarction. In several series this arterial hypoxemia has been associated with low cardiac output and evidence of left ventricular failure suggesting that pulmonary congestion and edema are the major mechanisms. However, hemodynamic abnormalities are not the sole consequence of acute myocardial infarction, and investigators have found evidence of maldistribution of ventilation. This is a well-known cause of arterial hypoxemia. The Pao₂ is usually markedly reduced in cardiogenic shock and is associated with a right-to-left shuntlike effect.

We have previously found that the arterial hypoxemia resulting from experimental hemorrhage shock may be in large part prevented by periodic hyperinflation of the lung, suggesting that small airway abnormalities are important as well as vascular changes.

The present study is designed to investigate further the mechanisms of the abnormalities in respiratory gas exchange following acute myocardial infarction.

Methods

We studied 61 patients admitted to the Coronary Monitoring Unit of the Montreal General Hospital. Patients with evidence of preexisting chronic lung disease were excluded. The patients were divided into three clinical groups (table 1).

Group I: Coronary Insufficiency

Eight patients were diagnosed as suffering from coronary insufficiency. They all had characteristic pain suggestive of cardiac ischemia. Their electrocardiograms showed nonspecific ST-T ab-
Table 1

Results of Cardiorespiratory Studies

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Ht. (cm)</th>
<th>Wt. (kg)</th>
<th>BSA (m²)</th>
<th>Pao₂ (mm Hg)</th>
<th>Paco₂ (mm Hg)</th>
<th>pH (units)</th>
<th>Pao₂ (mm Hg)</th>
<th>Paco₂ (mm Hg)</th>
<th>pH (units)</th>
<th>DLCO</th>
<th>Obs (ml/min/mm Hg)</th>
<th>Pred (%)</th>
<th>% pred</th>
<th>RA O₂ sat (%)</th>
<th>CI (L/min/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40</td>
<td>M</td>
<td>183</td>
<td>81.5</td>
<td>2.04</td>
<td>83</td>
<td>37</td>
<td>7.438</td>
<td>118</td>
<td>25</td>
<td>7.550</td>
<td>505</td>
<td>34</td>
<td>7.478</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>M</td>
<td>158.5</td>
<td>47.5</td>
<td>1.46</td>
<td>60</td>
<td>35</td>
<td>7.455</td>
<td>77</td>
<td>30</td>
<td>7.528</td>
<td>460</td>
<td>34</td>
<td>7.465</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>54</td>
<td>M</td>
<td>167.5</td>
<td>65.5</td>
<td>1.74</td>
<td>84</td>
<td>38</td>
<td>7.410</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>51</td>
<td>M</td>
<td>180</td>
<td>72.5</td>
<td>1.92</td>
<td>70</td>
<td>42</td>
<td>7.422</td>
<td>114</td>
<td>26</td>
<td>7.569</td>
<td>484</td>
<td>33</td>
<td>7.454</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>52</td>
<td>M</td>
<td>183</td>
<td>91</td>
<td>2.12</td>
<td>59</td>
<td>39</td>
<td>7.465</td>
<td>60</td>
<td>35</td>
<td>7.508</td>
<td>415</td>
<td>45</td>
<td>7.465</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>52</td>
<td>M</td>
<td>167.5</td>
<td>72.5</td>
<td>1.88</td>
<td>66</td>
<td>44</td>
<td>7.560</td>
<td>78</td>
<td>34</td>
<td>7.652</td>
<td>425</td>
<td>45</td>
<td>7.585</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>63</td>
<td>M</td>
<td>177</td>
<td>77</td>
<td>1.94</td>
<td>93</td>
<td>37</td>
<td>7.485</td>
<td>106</td>
<td>30</td>
<td>7.563</td>
<td>500</td>
<td>25</td>
<td>7.618</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>63</td>
<td>M</td>
<td>167.5</td>
<td>65.5</td>
<td>1.75</td>
<td>80</td>
<td>35</td>
<td>7.510</td>
<td>92</td>
<td>28</td>
<td>7.565</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>53.1</td>
<td>173</td>
<td>71</td>
<td></td>
<td></td>
<td>74.4</td>
<td>38.4</td>
<td>7.468</td>
<td>92.1</td>
<td>29.7</td>
<td>7.562</td>
<td>465</td>
<td>36</td>
<td>7.510</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>7.4</td>
<td>8.99</td>
<td>12.8</td>
<td></td>
<td></td>
<td>12.4</td>
<td>3.2</td>
<td>0.09</td>
<td>21.6</td>
<td>3.77</td>
<td>0.04</td>
<td>38</td>
<td>7.74</td>
<td>0.07</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Group I: coronary insufficiency

Group II: myocardial infarction without left ventricular failure

Creation, Volume XL, November 1969

VALENCIA, BURGESS
### ARTERIAL HYPOXEMIA

**Group III: myocardial infarction with left ventricular failure**

| 51 | 64 | M | 162.5 | 63.5 | 1.69 | 30 | 40 | 7.445 | - | - | - | 51 | 48 | 7.425 | - | - | - | - |
| 52 | 64 | F | 156 | 56 | 1.54 | 58 | 38 | 7.404 | 96 | 25 | 7.478 | 535 | 34 | 7.382 | - | - | - | - |
| 53 | 63 | M | 166 | 61 | 1.69 | 52 | 35 | 7.410 | 65 | 36 | 7.400 | 220 | 38 | 7.380 | 14.2 | 18.8 | 75 | - | - |
| 54 | 61 | M | 165 | 72 | 1.80 | 59 | 38 | 7.445 | 78 | 34 | 7.480 | 415 | 32 | 7.530 | 20.5 | 24.8 | 82 | - | - |
| 55 | 53 | M | 170 | 68 | 1.80 | 69 | 37 | 7.490 | 87 | 32 | 7.530 | - | - | - | 28.8 | 27.8 | 96 | - | - |
| 56 | 47 | M | 170 | 71 | 1.90 | 72 | 37 | 7.500 | - | - | - | 21.7 | 21.7 | 100 | - | - | - | - |
| 57 | 49 | M | 173 | 79 | 1.94 | 60 | 38 | 7.475 | 63 | 36 | 7.485 | 280 | 28 | 7.575 | 20.2 | 20.4 | 99 | - | - |
| 58 | 39 | M | 170 | 63.5 | 1.72 | 65 | 31 | 7.450 | 73 | 30 | 7.455 | - | - | - | 14.3 | 18.5 | 77 | - | 2.64 |
| 59 | 81 | M | 177 | 77 | 1.98 | 42 | 40 | 7.340 | 47 | 38 | 7.390 | - | - | - | 13.0 | 27.6 | 67 | 44 | 1.50 |
| 60 | 68 | M | 170 | 75 | 1.86 | 55 | 35 | 7.537 | 62 | 35 | 7.560 | - | - | - | 17.6 | 24.4 | 72 | - | 1.04 |
| 61 | 50 | M | 170 | 58 | 1.94 | 69 | 34 | 7.562 | 71 | 32 | 7.590 | - | - | - | 21 | 24.2 | 87 | - | - |

**Mean**

| 58 | 168.5 | 69.5 | 1.80 | 56.4 | 36.6 | 7.489 | 71.3 | 33.1 | 7.485 | 300 | 36 | 7.458 | 19.0 | 23.1 | 84 | 44 | 1.72 |

**Sd**

| 11.77 | 5.79 | 7.65 | 0.13 | 12.5 | 2.69 | 0.06 | 14.5 | 3.91 | 0.06 | 185 | 7.61 | 0.08 | 4.92 | 3.48 | 12.2 | 0.82 |

**Abbreviation**: RA O₂ sat (%) = right atrial oxygen saturation.
normalities, and serial measurements of serum SGOT and LDH remained within normal limits.

Group 2: Myocardial Infarction Without Left Ventricular Failure

Forty-two patients had a history of characteristic ischemic cardiac pain associated with segmental ECG changes typical of acute myocardial infarction. They did not develop basal rales, a third heart sound, or evidence of an increase in jugular venous pressure. Chest x-rays showed no evidence of pulmonary vascular congestion.

Group 3: Myocardial Infarction with Left Ventricular Failure

Eleven patients fulfilling the same criteria for acute myocardial infarction as the patients in group 2 were considered to be in left ventricular failure. The diagnosis of left ventricular failure was based on the finding of moist rales on auscultation of the lung bases and in addition, either a third heart sound at the cardiac apex or elevation of jugular venous pressure. Chest x-rays showed evidence of pulmonary vascular congestion in nine of the 11 patients in this group. The chest x-rays of the remaining two patients were considered normal. One patient in this group was in cardiogenic shock and died within 24 hours of admission.

Procedures

All measurements were made with the patient resting in bed at 45 degrees. No supplementary oxygen, analgesia, or sedation was given for at least 1 hour prior to the studies. We inserted a fine polyethylene catheter percutaneously into a brachial artery during the first 24 hours following admission and collected blood samples for arterial oxygen tension (Pao2), arterial carbon dioxide tension (Paco2), and pH. The samples were taken under the following experimental conditions: (1) while the patient was breathing room air; (2) immediately following three voluntary deep breaths; and (3) in 17 cases, after breathing 100% oxygen through a low resistance valve and rubber mouthpiece with the nose occluded, for 15 minutes. The blood samples were analyzed within 30 minutes of collection with a blood gas analyzer (Instrumentation Laboratories, Model IL-113-52).

We measured the pulmonary diffusing capacity (DLCO) in the same position by a single-breath carbon monoxide technic. The lung volume was obtained simultaneously by the single-breath neon dilution technic. The expired gas samples were analyzed with a gas chromatograph (Hewlett-Packard, Model 700) within 1 hour of collection. Values for hemoglobin were within normal limits in every case and no correction in calculating the diffusing capacity was necessary. We expressed the results in ml/min/mm Hg, and also as percentage of the predicted normal value based on the lung volume at which the measurement was made according to the following formula:

\[ DLCO = V_a \times STPD \times 4.83 + 8.1 \]

where \( V_a \) STPD is alveolar volume at standard conditions of temperature and pressure, and 4.83 and 8.1 are constants obtained as a result of measurements on 100 normal subjects in our laboratory. The normal subjects were studied in a sitting position rather than resting at a 45-degree angle in bed. However, studies in several normal subjects confirmed that the effect of the different position was minimal as the calculation allowed for any difference in alveolar volume.

In 22 patients, we passed a PE 60 polyethylene catheter percutaneously from an antecubital vein to the right atrium. The right atrial position was achieved by advancing the catheter until occasional atrial premature beats were seen and then withdrawing it slightly to a stable position. Pressures were measured with a Statham P23Db strain gauge and recorded on an Electronics for Medicine IR 4 recorder. The reference point for all pressures was taken as 5 cm below the sternal angle. Cardiac outputs were measured by the injection of a weighed quantity of indocyanine green into the right atrium and the aspiration of arterial blood from the brachial artery catheter through a Gilford densitometer (Model 103 IR), the output of which was fed to a Texas Instrument recorder. The dilution curves obtained were calibrated after each study by aspirating four dilutions of dye in previously drawn blood through the densitometer. In 10 patients, blood samples were collected from the right atrium for measurement of Pao2 and pH. The oxygen saturation was calculated from the hemoglobin dissociation curve.

In 31 patients, we measured serial blood gas tensions and DLCO at weekly intervals until discharge.

Results

The patient's vital statistics and results of cardiorespiratory studies are given in table 1. Our lower limit of normal Pao2 in patients without cardiorespiratory abnormalities resting quietly at 45 degrees in bed is 70 mm Hg. We found a normal mean Pao2 in the eight patients fulfilling the criteria for coronary insufficiency although three had a Pao2 of less than 70 mm Hg. While the Pao2 was lowest in those patients with myocardial infarction who were in clinical left ventricular failure, 30 of 42 patients without clinical evidence of left ventricular failure also had reduced Pao2 (fig.

*Circulation, Volume XL, November 1969*
The arterial oxygen tension value for each patient in the three clinical groups. We have taken 70 mm Hg as the lower limit of normal for the PaO₂ of a bedridden patient. The open circles represent the means for each group and the vertical lines and bars indicate 1 standard deviation. The mean PaO₂ for the patients with coronary insufficiency is normal, although three patients have arterial hypoxemia. The patients with coronary insufficiency are not included in the subsequent figures. The mean PaO₂ for the myocardial infarction group without left ventricular failure (LVF) is significantly lower than the mean of those patients with coronary insufficiency (P < 0.01). The patients with myocardial infarction and clinical left ventricular failure had more severe arterial hypoxemia than those patients in whom failure was not detected (P < 0.01).

In 17 cases studied while breathing 100% oxygen, there was evidence of a right-to-left shuntlike effect as shown by a PaO₂ of less than 550 mm Hg (fig. 2). In nearly all cases, PaO₂ increased significantly following three deep breaths (fig. 3). In a group of 12 patients without evidence of cardiopulmonary disease studied at a 45 degree angle while in bed, the mean increase in PaO₂ following three deep breaths was 12 mm Hg (s.d. 4.9). The mean increase following this maneuver in the coronary insufficiency group was 19 mm Hg (s.d. 14.9), in the myocardial infarction without failure group, it was 16 mm Hg (s.d. 9.2), and in the myocardial infarction with failure group, it was 12 mm Hg (s.d. 11.3). Although the patients without clinical evidence of failure tended to show a larger increase in PaO₂ following three deep breaths, the differences between these means are not significant (P > 0.05). Although the patients with extremely low cardiac indices usually had the most severe hypoxemia, there was not a
significant correlation between these two measurements (fig. 4). The right atrial pressures were normal in every case studied. Five of the 10 patients with measurements of right atrial O₂ saturation had values of 60% or more and a cardiac index of less than 2.5 L/min/m². There was a significant correlation between Pao₂ and D_LCO (fig. 5). Although part of the reduction in D_LCO may be due to a reduction in alveolar volume (fig. 6), the correlation is still highly significant when D_LCO is expressed as a percentage of predicted normal based on the lung volume at which the measurement was made (fig. 7). If only the diffusing capacities of those patients in whom we also measured the cardiac index are considered, there is still a significant correlation between Pao₂ and D_LCO (r = 0.51; P < 0.05). We found a poor correlation between cardiac index and D_LCO (r = 0.21; P > 0.05). In 31 patients, serial measurements showed an increase in Pao₂ and lung volume at 1 week as compared to admission values, but no further change at the 2 and 3-week periods (fig. 8).

We reviewed the subsequent incidence and type of arrhythmias in all patients to see whether these were related to the degree of arterial hypoxemia at the time of the initial study. In the patients without clinical left ventricular failure, there was no relation between arrhythmias and Pao₂. Nearly all patients had premature ventricular contractions. One patient with normal Pao₂ subsequently developed ventricular fibrillation. Of the patients with left ventricular failure, four

![Figure 4](http://circ.ahajournals.org/)

**Figure 4**

The relationship between Pao₂ and cardiac index in the 23 patients with acute myocardial infarction in whom we measured the latter. While the patients with a very low cardiac index usually have the most severe hypoxemia, there is not a significant correlation between these two measurements (r = 0.25; P > 0.05). The line in this and subsequent figures is drawn by the method of least squares.

![Figure 5](http://circ.ahajournals.org/)

**Figure 5**

Pao₂ and pulmonary diffusing capacity in acute myocardial infarction. There is a significant correlation between arterial hypoxemia and reduced pulmonary diffusing capacity (r = 0.54; P < 0.01) in 42 patients with myocardial infarction.

![Figure 6](http://circ.ahajournals.org/)

**Figure 6**

Pao₂ and alveolar volume in acute myocardial infarction. Alveolar volume measured by a single-breath neon dilution was reduced in many patients. There is a significant correlation between this measurement and the degree of hypoxemia (r = 0.43; P < 0.01).
**Arterial Hypoxemia**

\[ \text{Pao}_2 \text{ and pulmonary diffusing capacity expressed as a percentage of predicted normal based on the lung volume at which } D_{LCO} \text{ was measured. This shows that the low diffusing capacity is not solely the result of a reduced lung volume.} \]

**Figure 7**

Patients with failure had more severe arrhythmias (five, had multifocal premature ventricular contractions; one had third degree atrioventricular block, and one had left bundle-branch block) than those without failure. Their mean Pao2 was 62 mm Hg.

**Discussion**

Figure 9 is a schematic representation of the four possible pathophysiologic mechanisms of arterial hypoxemia in a patient breathing ambient air at sea level.

**Alveolar Hypoventilation**

Total alveolar hypoventilation is characterized by an increased arterial carbon dioxide tension and arterial hypoxemia. We have excluded this mechanism in our patients by the finding of normal or reduced carbon dioxide tension in every case. Most of our patients had mild alkalosis. In some, this was due to alveolar hyperventilation and respiratory alkalosis, while in others there was metabolic alkalosis. The latter was usually related to diuretic therapy.

**Ventilation-Perfusion Inequality**

The most common mechanism of arterial hypoxemia is decreased ventilation of some
<table>
<thead>
<tr>
<th></th>
<th>Admission</th>
<th>1st week</th>
<th>2nd week</th>
<th>3rd week</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PaO2 (mm Hg)</td>
<td>PaCO2 (mm Hg)</td>
<td>pH (units)</td>
<td>Dlco (% pred)</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
<td>39</td>
<td>7.465</td>
<td>100</td>
</tr>
<tr>
<td>6</td>
<td>66</td>
<td>44</td>
<td>7.560</td>
<td>98</td>
</tr>
<tr>
<td>8</td>
<td>80</td>
<td>35</td>
<td>7.510</td>
<td>84</td>
</tr>
<tr>
<td>Mean</td>
<td>68.3</td>
<td>39.3</td>
<td>7.511</td>
<td>96.9</td>
</tr>
<tr>
<td>SD</td>
<td>10.6</td>
<td>4.5</td>
<td>12.7</td>
<td>255</td>
</tr>
</tbody>
</table>

**Group 1: coronary insufficiency**

- Mean: 68.3, SD: 10.6

**Group 2: myocardial infarction without left ventricular failure**

- Mean: 66.7, SD: 6.7

---

**Table 2**

Follow-Up Measurements: Arterial Blood Gases, Diffusing Capacity, and Lung Volume
alveolar units with respect to their perfusion. In our patients, we were able to show a rise in arterial oxygen tension following three deep breaths comparable to, or greater than, that found in a group of normal subjects. There is a mild degree of maldistribution of inspired air even in normals and periodic full inflations of the alveoli are necessary to preserve a low-surface tension and prevent collapse. The increase in transpulmonary pressure produced by this maneuver may increase air flow to previously underventilated alveoli or open collapsed units. This observation does not distinguish between underventilation and complete nonventilation of perfused alveolar units, but these two mechanisms may be a matter of degree both arising from abnormalities in small airways.

Our results are consistent with those previously published by Pain and his co-workers who showed maldistribution of ventilation after acute myocardial infarction by using a nitrogen washout technic. They also agree with Higgins who found that an increase in tidal volume produced a rise in arterial oxygen tension.

**Shunt**

If an alveolar unit is completely nonventilated (that is, as a result of total obstruction of the airway and resulting collapse), then as far as respiratory gas exchange is concerned, it will behave as an anatomic right-to-left shunt. This represents the most severe form of ventilation-perfusion inequality. One hundred per cent oxygen breathing overcomes the possible effects of alveolar hypoventilation, decreased ratio of ventilation to perfusion, and decreased diffusing capacity. A resulting arterial oxygen tension level below expected values (550 mm Hg) can be ascribed to a right-to-left shuntlike effect. This mechanism has been demonstrated after acute myocardial infarction and is again confirmed by our 100% oxygen studies.1, 7, 8

**Diffusing Defect**

Pulmonary diffusing capacity measurements in patients following acute myocardial infarction have not been previously published. This

---

### Table: Single Breath Mean Diffusion

| Group 1: Myocardial Infarction with Left Ventricular Failure | Group 2: Normal
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>-----</td>
<td>----</td>
</tr>
<tr>
<td>63</td>
<td>8</td>
</tr>
<tr>
<td>52</td>
<td>38</td>
</tr>
<tr>
<td>74</td>
<td>58</td>
</tr>
<tr>
<td>55</td>
<td>36</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>55</td>
<td>27</td>
</tr>
</tbody>
</table>

**Notes:**
- Long vol. = lung volume measured from the single breath mean dilution (ml STPD)
- DlCO is expressed as a percentage of predicted normal

---

**Circulation, Volume XL, November 1969**
measurement depends on the transfer of carbon monoxide across the pulmonary membrane into the pulmonary capillaries and its combination with hemoglobin in the red cells. The overall diffusing capacity will be decreased by a decrease in lung volume. This explains part of the reduction observed in our patients. However, when we express the diffusing capacity as a percentage of predicted normal based on the lung volume at which it was measured, the results are still below expected values. Therefore, the reduction cannot be completely explained by loss of ventilation to small lung units.

The effect of left ventricular failure on the diffusing capacity is variable. Pulmonary venous congestion resulting in an increase in transcapillary pressure would be expected to cause an increase in the diffusing capacity by increasing the pulmonary capillary blood volume. Interstitial edema or fluid-filled alveoli, or both, would be expected to decrease the rate of carbon monoxide transfer. The effect of left ventricular failure on the diffusing capacity may thus depend in part on its duration. We found a low cardiac index in 10 patients in the absence of clinical signs of left ventricular failure. The cardiac output may be decreased in some of these patients in the absence of a significant rise in left ventricular end-diastolic pressure. These considerations could explain the lack of correlation observed between cardiac index and diffusing capacity.

Goldman and co-workers have previously assessed the use of the right atrial oxygen saturation as an indication of cardiac failure following acute myocardial infarction. However, these authors did not measure cardiac output or pressures and relied only on clinical evaluation as an indication of failure in their cases. Our observations of right atrial oxygen saturation suggest that a low cardiac output may be present with values above 60%.

While it seems reasonable to assume that most of the abnormalities in respiratory gas exchange may be produced by decreased pulmonary capillary blood flow or changes in pulmonary transcapillary pressure due to left ventricular failure, our finding of a lack of correlation between arterial oxygen tension and cardiac index suggests that this is not the only factor. Another important mechanism probably is that these patients are sick, bedridden, and have chest pain, all resulting in a lack of periodic deep breaths. A lack of periodic pulmonary hyperinflation has been shown both experimentally and clinically to result in diffuse micro-atelectasis with accompanying abnormalities in respiratory gas exchange, particularly hypoxemia. Left ventricular failure resulting in an elevation of pulmonary venous pressure and causing congestion and edema of small airways and alveolar septa would also produce maldistribution of ventilation with respect to perfusion. We therefore suggest that the arterial hypoxemia that so frequently follows acute myocardial infarction results from a combination of small airway abnormalities and hemodynamic changes in small vessels.

The results of our follow-up studies show that the impairment of respiratory gas exchange persists for at least several weeks following acute myocardial infarction (table 2). These findings agree with those previously reported. The combination of arterial hypoxemia and reduced diffusing capacity would be compatible with thickening of the pulmonary membrane, produced by the organization of transudates. This could reduce the compliance of the involved alveolar units and also result in a decrease in ventilation with respect to perfusion. Such changes could take several weeks to regress.

One of the major achievements of coronary care units has been the early detection and treatment of arrhythmias. The incidence of these was reviewed in our patients to study any possible relation to the arterial hypoxemia. We found that most of our patients suffered from some type of arrhythmia. We also found that the mortality rate was much higher in those patients with left ventricular failure who were most severely hypoxemic. These results agree with those of McNicol and associates who found higher mortality in the patients with a lower arterial oxygen tension.
ARTERIAL HYPOXEMIA

It may be that those patients who suffered a more severe infarction also developed more severe arrhythmias and the hypoxemia was a secondary factor. In any case, we believe that routine measurements of arterial blood gases in patients with myocardial infarction will help in detecting hypoxemia which could be a significant factor in the development of further complications.

Conclusions

We have shown that arterial hypoxemia is common after acute myocardial infarction. Although the hypoxemia is most severe in the presence of clinical left ventricular failure, there is also a significant reduction in arterial oxygen tension in its absence. The degree of hypoxemia is poorly correlated with a low cardiac index, but is associated with abnormalities in the distribution of ventilation and a reduced pulmonary diffusing capacity.

We conclude that the fall in arterial oxygen tension following acute myocardial infarction is the result of abnormalities in both the small airways and the capillaries in the lungs.

Acknowledgment

We are grateful to Miss Olga Panyszak for technical assistance and for the cooperation of the nursing staff of the Coronary Monitoring Unit of the Montreal General Hospital.

References

during general anesthesia with controlled
ventilation: A concept of atelectasis. New Eng
20. McNicol, M. W., Kirby, B. J., Bhoola, K. D.,
Fulton, P. M., and Tattersfield, A. E.: Changes in pulmonary function 6-12 months
after recovery from myocardial infarction.

Emotion and the Pulse Rate
Nature Does Nothing in Vain
Galen 2nd Century A. D.

Delicate and difficult as was the task of measuring the pulse in Galen’s time, he
was clever enough to anticipate by seventeen centuries some of the tests which modern
psychologists have urged should be applied in criminal trials. He detected the fact
that a female patient was not ill but in love by the quickening of her pulse when
someone came in from the theater and announced that he had seen Pylades dance.
When she came again the next day, Galen had purposely arranged that someone
should enter and say that he had seen Morphus dancing. This and a similar test on
the third day produced no perceptible quickening in the woman’s pulse, but it bounded
again when on the fourth day, Pylades’ name was again spoken.—From Lynn Thorn-
dike’s History of Magic and Experimental Science. (c. 1923) vol. 1, page 144.
Arterial Hypoxemia Following Acute Myocardial Infarction
A. VALENCIA and J. H. BURGESS

_Circulation_. 1969;40:641-652
doi: 10.1161/01.CIR.40.5.641

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1969 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/40/5/641