Blood-Gas and Hemodynamic Responses to Oxygen in Acute Myocardial Infarction

By Robert M. Davidson, M.D., Barry W. Ramo, M.D., Andrew G. Wallace, M.D., Robert E. Whalen, M.D., and C. Franklin Starmer, Ph.D.

SUMMARY

Blood-gas (Pao₂) and hemodynamic responses to the inhalation of oxygen were studied in 60 patients with acute myocardial infarction. Patients who were not in heart failure on admission and did not develop signs of heart failure within the next 5 days achieved the same Pao₂ level while breathing 100% oxygen as did patients without acute myocardial infarction. Patients with pulmonary edema or cardiogenic shock had a very poor Pao₂ response to oxygen inhalation. Patients in mild heart failure at the time of study and patients who developed heart failure subsequent to the study had a Pao₂ response intermediate between the other two groups. This rise of Pao₂ with oxygen correlated with the cardiac index and right atrial oxygen prior to inhalation of oxygen. Uncomplicated patients responded to inhalation of oxygen with a decrease of heart rate, cardiac index, stroke index, and cardiac work, and an increase of peripheral resistance. Patients in pulmonary edema or cardiogenic shock or with a low cardiac index or low Pao₂ responded with only a slight increase in peripheral resistance. The Pao₂ achieved while breathing oxygen appeared to determine the type of hemodynamic response to oxygen. Administration of oxygen to patients with acute myocardial infarction is useful in identifying latent heart failure and in predicting the subsequent clinical course of these patients.

Additional Indexing Words:
Heart failure  Cardiac catheterization  Cardiac output  Coronary artery disease

The Administration of oxygen to patients with acute myocardial infarction has long been an accepted mode of therapy. Until recently, however, there were remarkably little data concerned with the physiologic effects of oxygen in such patients, and several reports demonstrated a very wide range of changes in blood gases and hemodynamics in response to the inhalation of oxygen.¹⁻¹⁹ This variability among patients with acute myocardial infarction in their response to oxygen is in distinct contrast to normal subjects who exhibit a consistent increase of arterial Pao₂ (Pao₂) and decrease of heart rate and cardiac output.⁵ ¹⁰⁻¹⁸ Several apparent phenomena might account for the variable response to oxygen among patients with acute myocardial infarction or the difference between patients with acute infarction and normal subjects (i.e., the presence of pulmonary edema, the level of arterial oxygen concentration, and the response of the myocardium to a change in oxygen tension).

This study was undertaken to examine the effects of oxygen administration in a large group of patients with acute myocardial infarction and to further elucidate the factors which determine the hemodynamic and blood gas response. It was hoped that this information might provide a basis for selecting patients in whom the potentially beneficial effects of administration of oxygen could be tested. Furthermore, responses to the inhalation of oxygen were analyzed to determine whether or not the observed changes could be of value in predicting the subsequent clinical course of patients with acute myocardial infarction.

Methods

Sixty patients with acute myocardial infarction were studied within 1 hour of admission to the hospital and generally within 24 hours after the onset of symptoms.
There were 42 males and 18 females, ranging in age from 36 to 85 years. All patients who were included in this study were considered to have had definite acute myocardial infarction on the basis of satisfying at least two of the following three criteria: a typical clinical history, electrocardiographic changes of transmural infarction (Q waves and S-T changes), and typical serial changes of serum creatine phosphokinase and either glutamic oxaloacetic transaminase or lactic acid dehydrogenase.

The patients were assigned to one of four groups according to their clinical status prior to the hemodynamic study. Class I included 28 patients who had no clinical evidence of heart failure at the time of admission to the hospital. Clinical criteria for heart failure included $S_3$ gallop and/or wet rales and/or shock or pulmonary edema. Thirteen of these 28 patients subsequently developed one or more of the physical findings of heart failure and will be referred to as class IB. The remaining 15 patients did not develop congestive heart failure and will be referred to as class IA. Class II consisted of 19 patients with findings on admission of mild-to-moderate congestive heart failure (crepitant rales and/or an $S_3$ gallop). Class III consisted of nine patients with overt pulmonary edema, and class IV included four patients with cardiogenic shock (systolic blood pressure less than 90 mm Hg, urine output of less than 20 cc/hour, cold extremities, and disturbed sensorium). For the purpose of statistical analysis, patients in classes III and IV were considered as one group (III/IV).

In addition to these 60 patients, nine others were included in our study to serve as controls. These patients were suspected of having acute myocardial infarction at the time of admission but were subsequently found to lack objective evidence for this diagnosis. Six had arteriosclerotic heart disease, and three were not thought to have heart disease. None of the control patients had heart failure; their mean age was 49 years. No patient was included in this study if he had evidence of parenchymal or obstructive lung disease, an oral temperature greater than 38° C, or a hematocrit of less than 35%. No patient received medication within 30 min prior to study. Informed consent was obtained from each patient or from his family.

The patients were studied in the catheterization laboratory on the Myocardial Infarction Research Unit. A no. 7 Zucker catheter (U. S. Catheter and Instrument Corporation, Glens Falls, New York) was passed through a basilic or femoral vein under local anesthesia and positioned in the high right atrium under fluoroscopic control. A no. 16 Teflon needle (Longwell, Becton-Dickson Corporation, Rutherford, New Jersey) was placed in the radial or femoral artery. Arterial and right atrial pressures were measured using as zero reference a point 10 cm above the surface of the catheterization table. Pressures were measured with Hewlett-Packard transducers (no. 1280C) and recorded on a Hewlett-Packard data acquisition system. Cardiac output was estimated by the dye dilution technic. Indocyanine green dye was injected into the right atrium, and blood was withdrawn continuously from the radial or femoral artery through a Waters densitometer (XP-302). Dye curves were calibrated using the patient’s blood to which a known amount of dye was added; and cardiac output was calculated by the method of Thompson et al. Samples of arterial and right atrial blood were placed in ice immediately after being drawn, and blood gases were measured within 30 min. Arterial $P_{O_2}$ was measured with an Instrumentation Laboratories Blood Gas System (11352). Oxygen saturation was measured on a reflection oximeter (American Optical Company model 1824). The oximeter was calibrated using standards supplied with the machine and by the method of Van Slyke and McNeal. The oximeter was linear when the oxygen saturation was between 37 and 100%. A linear regression equation derived from the correlation between the oximeter and Van Slyke values was developed to correct the oximeter reading. The equation was: oxygen saturation = $-13.692 + 1.137$ (oximeter reading) ($r = 0.997$; see 1.78).

Each patient was studied while breathing room air and after 10 min of breathing 100% oxygen. Oxygen was delivered through a rebreathing-type plastic face mask at a flow rate of 8-10 liters/min. This method of administering oxygen was chosen because of its common use in the clinical setting. The quantitative results of our study cannot, however, be extrapolated to situations where other methods of administering oxygen are employed. Each patient was studied in the recumbent or near-recumbent position. Total peripheral resistance (TPR, in units) was calculated according to the formula:

$$\text{TPR} = \frac{\text{mean aortic pressure} - \text{right atrial pressure}}{\text{cardiac output}}$$

Cardiac work (in kg-m/min) was calculated according to the formula:

$$\text{Work} = \frac{\text{mean arterial pressure} \times \text{cardiac output} \times 13.6}{1000}$$

(where pressures are in mm Hg, and cardiac output is in liters/min). Analysis of variance was used to test the difference of a selected variable across patient classes. The paired t test was used to test differences within a patient class while breathing room air or oxygen. The scheffé contrast was used to indicate pairs of patient classes which were different.

**Results**

The mean arterial $P_{O_2}$ ($P_{A_{O_2}}$) while breathing room air was found to correlate with the clinical class of the patient at the time of admission to the hospital. Highest values were observed in control patients, and progressively lower values were observed in patients with acute infarction and with increasing severity of heart failure (table 1). There was considerable overlap, however, between the $P_{A_{O_2}}$ values of individual patients in different classes; the difference between mean $P_{A_{O_2}}$ in adjacent classes was not statistically significant.
During inhalation of oxygen the difference between classes became much more apparent than during inhalation of room air, as shown in figure 1. The response of the arterial PO$_2$ to inhalation of oxygen was virtually identical in control and class I A patients and differed markedly from the responses observed in classes II and II patients. Patients who were either class III or IV had an even greater reduction of the PaO$_2$ response to inhalation of oxygen. The difference between these three groups (control and IA, IB and II, and III/IV) with respect to the change of PaO$_2$ produced by oxygen was highly significant (P < 0.01).

The individual PaO$_2$ values of each class I patient are shown in figure 2 (while breathing oxygen). The mean PaO$_2$ of class IA patients was significantly higher than class IB patients (P < 0.025). Although there was overlap of values in the range from 200 to 300 mm Hg, only three of the class IA patients had a PaO$_2$ of less than 270 mm Hg and only three of the class IB patients had a PaO$_2$ of greater than 270 mm Hg. From these observations, we have concluded that the PaO$_2$ while breathing oxygen correlates better with clinical class, and with the subsequent development of heart failure, than does the PaO$_2$ while breathing room air.

To elucidate further the possible relation between PaO$_2$ and the degree of heart failure, correlations between PaO$_2$ and either cardiac index or right atrial oxygen saturation were examined. In the group of patients with a cardiac index of less than 2.5 liters/min/m$^2$ the PaO$_2$ while breathing oxygen was 185 ± 27 mm Hg, while in the group with a cardiac index of 2.5 liters/min/m$^2$ or greater the PaO$_2$ was 261 ± 21 mm Hg (P < 0.05). A similar difference was noted when patients were separated on the basis of their right atrial oxygen saturation while breathing room air. In the group with a right atrial oxygen saturation of less than 60% the PaO$_2$ while breathing oxygen was 182 ± 24 mm Hg, while in those with a right atrial oxygen saturation of 60% or greater the PaO$_2$ was 263 ± 16 mm Hg.

Table 1

<table>
<thead>
<tr>
<th>Patient group</th>
<th>No. of patients</th>
<th>PaO$_2$ (mean ± se, mm Hg)</th>
<th>O$_2$-air diff</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>9</td>
<td>84 ± 4</td>
<td>306 ± 27</td>
</tr>
<tr>
<td>Acute myocardial infarction:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>28</td>
<td>72 ± 2</td>
<td>268 ± 11</td>
</tr>
<tr>
<td>Class IA</td>
<td>15</td>
<td>76 ± 3</td>
<td>307 ± 15</td>
</tr>
<tr>
<td>Class IB</td>
<td>13</td>
<td>67 ± 3</td>
<td>218 ± 18</td>
</tr>
<tr>
<td>Class II</td>
<td>16</td>
<td>65 ± 5</td>
<td>243 ± 24</td>
</tr>
<tr>
<td>Class III/IV</td>
<td>13</td>
<td>50 ± 3</td>
<td>111 ± 14</td>
</tr>
<tr>
<td>Cardiac index (liters/min/m$^2$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥ 2.5</td>
<td>18</td>
<td>66 ± 4</td>
<td>261 ± 21</td>
</tr>
<tr>
<td>&lt; 2.5</td>
<td>15</td>
<td>58 ± 4</td>
<td>185 ± 27</td>
</tr>
<tr>
<td>Right atrial O$_2$ sat (%):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥ 60</td>
<td>25</td>
<td>70 ± 3</td>
<td>262 ± 16</td>
</tr>
<tr>
<td>&lt; 60</td>
<td>22</td>
<td>58 ± 4</td>
<td>182 ± 24</td>
</tr>
</tbody>
</table>

Figure 1

*Each hatched area represents the mean and se of PaO$_2$ values for each group of patients while breathing room air (left) and 100% oxygen (right). The responses of the three groups (IA and control, IB and II, and III/IV) were distinctly different.*
OXYGEN IN ACUTE MYOCARDIAL INFARCTION

Figure 2

Each point represents the $P_{A02}$ value for an individual patient while breathing 100% oxygen. The points on the left represent values of class IA patients. The points on the right are the values for class IB patients. The bars represent the mean for each group. The dashed line at 270 mm Hg represents the $P_{A02}$ value which best separates the majority of patients in each group.

$p < 0.01$. Thus, to the extent that a reduced cardiac index and/or right atrial oxygen saturation reflects a defect in tissue perfusion, this defect appears to correlate with the ability of patients to respond to the inhalation of oxygen with an appropriate rise in $P_{A02}$.

The changes of mean arterial pressure and heart rate in response to the inhalation of oxygen were analyzed in 47 patients; the change of right atrial pressure in 50 patients and the changes of cardiac index, stroke index, peripheral resistance, and cardiac work in 34 patients. The hemodynamic alterations consequent to the inhalation of oxygen in these patients are presented in Table 2. In class IA patients the inhalation of oxygen was accompanied by a significant increase in peripheral vascular resistance and decrease in cardiac index, heart rate, stroke index, and cardiac work. These changes are comparable to those which have been observed when normal subjects inhale oxygen.7, 21-23 In contrast to class IA patients, class IB patients failed to show a significant increase of peripheral resistance or decrease of cardiac index, heart rate, stroke index, or cardiac work. The absence of significant changes in these hemodynamic variables in class IB patients was similar to the response of patients with overt heart failure at the time of study, i.e., classes II and III/IV.

To clarify further the possible relation between the presence or absence of heart failure and the hemodynamic response to inhalation of oxygen, patients were again divided into those with a cardiac index above or below 2.5 liters/min/m² while breathing room air. Patients with a baseline cardiac index of greater than 2.5 liters/min/m² showed a significant decrease of cardiac index and heart rate and an increase of peripheral resistance when given oxygen. In patients with a cardiac index of less than 2.5 liters/min/m² there was no significant change of cardiac index or heart rate and only a borderline increase of peripheral resistance. Similarly, when patients were divided into two groups based on whether or not they achieved a $P_{A02}$ of 200 mm Hg or greater while breathing oxygen, those who raised their $P_{A02}$ to at least 200 mm Hg showed an increase of peripheral resistance and decrease of cardiac index and heart rate. Patients who failed to increase their $P_{A02}$ to at least 200 mm Hg failed to change their peripheral resistance, cardiac index, and heart rate. From these observations, we have concluded that the major determinant of the hemodynamic response to inhalation of oxygen in patients with acute myocardial infarction is the rise in arterial $P_{A02}$.

Discussion

Previous studies have demonstrated that patients with acute myocardial infarction have arterial hypoxia and depressed hemodynamics while breathing room air and that the degree of these abnormalities is related to the clinical state of the
Table 2

Hemodynamic Responses to Oxygen (Mean ± se)

<table>
<thead>
<tr>
<th>Patient group</th>
<th>AP (mm Hg)</th>
<th>HR (beats/min)</th>
<th>CI (liters/min/m²)</th>
<th>SI (ml/beat/m²)</th>
<th>TPR (units)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Air</td>
<td>O₂</td>
<td>Air</td>
<td>O₂</td>
<td>Air</td>
</tr>
<tr>
<td>Class IA</td>
<td>104 ± 4</td>
<td>105 ± 4</td>
<td>78 ± 4</td>
<td>73 ± 4</td>
<td>3.4 ± 0.4</td>
</tr>
<tr>
<td>% change</td>
<td>+1</td>
<td>-6</td>
<td>-14</td>
<td>-14</td>
<td>+21</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.003</td>
<td>&lt;0.003</td>
<td>&lt;0.003</td>
</tr>
<tr>
<td>Class IB</td>
<td>98 ± 8</td>
<td>102 ± 8</td>
<td>85 ± 5</td>
<td>83 ± 5</td>
<td>2.8 ± 0.2</td>
</tr>
<tr>
<td>% change</td>
<td>+4</td>
<td>-2</td>
<td>-3</td>
<td>-3</td>
<td>+5</td>
</tr>
<tr>
<td>P value</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Class II</td>
<td>98 ± 4</td>
<td>97 ± 4</td>
<td>89 ± 5</td>
<td>85 ± 5</td>
<td>2.7 ± 0.2</td>
</tr>
<tr>
<td>% change</td>
<td>-1</td>
<td>-4</td>
<td>-4</td>
<td>-3</td>
<td>+6</td>
</tr>
<tr>
<td>P value</td>
<td>NS</td>
<td>&lt;0.02</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Class III/IV</td>
<td>87 ± 7</td>
<td>93 ± 9</td>
<td>101 ± 5</td>
<td>101 ± 4</td>
<td>2.0 ± 0.2</td>
</tr>
<tr>
<td>% change</td>
<td>+7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+12</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>CI (liters/min/m²):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥2.5</td>
<td>101 ± 4</td>
<td>103 ± 4</td>
<td>84 ± 4</td>
<td>79 ± 5</td>
<td>3.4 ± 0.2</td>
</tr>
<tr>
<td>% change</td>
<td>+1</td>
<td>-6</td>
<td>-9</td>
<td>-7</td>
<td>+12</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>&lt;0.002</td>
<td>&lt;0.02</td>
<td>&lt;0.05</td>
<td>&lt;0.04</td>
</tr>
<tr>
<td>&lt;2.5</td>
<td>96 ± 6</td>
<td>101 ± 7</td>
<td>90 ± 5</td>
<td>88 ± 4</td>
<td>2.0 ± 0.1</td>
</tr>
<tr>
<td>% change</td>
<td>+5</td>
<td>-2</td>
<td>0</td>
<td>-4</td>
<td>+11</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Pao₂ (mm Hg):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥200</td>
<td>101 ± 3</td>
<td>102 ± 3</td>
<td>84 ± 3</td>
<td>79 ± 3</td>
<td>3.1 ± 0.2</td>
</tr>
<tr>
<td>% change</td>
<td>+1</td>
<td>-6</td>
<td>-10</td>
<td>-8</td>
<td>+15</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>&lt;0.005</td>
<td>&lt;0.005</td>
<td>&lt;0.005</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>&lt;200</td>
<td>91 ± 5</td>
<td>97 ± 6</td>
<td>94 ± 5</td>
<td>94 ± 5</td>
<td>2.3 ± 0.2</td>
</tr>
<tr>
<td>% change</td>
<td>+7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+6</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: AP = arterial pressure; HR = heart rate; CI = cardiac index; SI = stroke index; TPR = total peripheral resistance; NS = not significant.

Patients², 3, 7, 8, 10–12, 15, 18, 19, 27–33 Several studies have also suggested that the changes of arterial Pao₂ and cardiac output which result from the administration of oxygen are abnormal.¹, 7, 8, 10, 11, 16, 19, 32 The present study provides additional data which are in general agreement with those observations. Furthermore, our data suggest that examining blood-gas and hemodynamic responses to oxygen can be of value in predicting the subsequent development of clinical heart failure in patients who do not have obvious signs of heart failure at the time of admission to the hospital.

Arterial Pao₂ values correlated with clinical class as well as with cardiac index and right atrial oxygen saturation. Differences of mean values of Pao₂ were small between clinical classes while breathing room air, but these differences were magnified during the inhalation of oxygen. While breathing 100% oxygen, patients could be clearly separated into three groups. Maximal Pao₂ was noted in the control and class IA patients, and the lowest Pao₂ values were observed in class III/IV patients. The classes IB and II patients had an intermediate response. Regardless of clinical class, patients with a low cardiac index or a low right atrial oxygen saturation had a much smaller increase of Pao₂ when given oxygen than patients with a higher baseline cardiac index and right atrial oxygen saturation. These observations suggest that patients with acute myocardial infarction, but with minimal or no reduction of cardiac output, resemble normal subjects in their response to inhalation of oxygen. However, patients with acute myocardial infarction and objective evidence of reduced ventricular performance show a reduced ability to oxygenate their arterial blood when they are given oxygen. The fact that the response of arterial Pao₂ to inhalation of oxygen correlates better with hemodynamic measurements than it does with clinical class, and that class IB patients show a significantly different response than class IA patients, supports this view.

Several mechanisms have been postulated to explain the arterial hypoxia which is observed in many patients with acute myocardial infarction.

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These include ventilation-perfusion abnormalities, anatomic right-to-left shunting, reduced cardiac output, atelectasis, and diffusion abnormalities. The existence of ventilation-perfusion abnormalities in patients with myocardial infarction has recently been demonstrated by Kazemi and co-workers. Their data suggest that decreased perfusion of the lung bases may result, at least in part, from an elevated pulmonary venous pressure and interstitial edema. Improvement of PaO₂ after the administration of a diuretic supports this thesis. Pulmonary arterial pressure was not measured in the present series of patients, but recent studies have shown that patients with acute myocardial infarction and elevated pulmonary artery diastolic or wedge pressures are more hypoxemic than patients with normal pressures.

It has also been suggested that a reduced cardiac output per se may contribute to arterial hypoxia and a decrease in the oxygen saturation of mixed venous blood. The association of a low cardiac output and a low right oxygen saturation with a poor PaO₂ response to oxygen was demonstrated in the present study. The cardiac output and/or the right atrial oxygen saturation may be important in determining the PaO₂ response to inhalation of oxygen, but a causal relation between these variables is difficult to prove.

The hemodynamic responses to oxygen also can be related to clinical class, to cardiac index, and to arterial oxygen saturation. In previous studies, patients who appeared clinically and hemodynamically to be uncomplicated responded to the inhalation of oxygen with a decrease of heart rate and cardiac output. Patients in cardiogenic shock or those with a very low cardiac output or marked hypoxemia responded to oxygen with only a slight increase in cardiac output and in arterial pressure. In the present study, uncomplicated patients demonstrated a decrease in heart rate, cardiac index, and cardiac work, and an increase in peripheral resistance. Patients in acute pulmonary edema or cardiogenic shock or those with a low cardiac index or moderate to severe hypoxemia showed only a slight increase of peripheral resistance with inhalation of oxygen and no other significant hemodynamic changes. The ability to achieve a PaO₂ of 200 mm Hg or greater during inhalation of oxygen appeared to be the most important factor determining the type of hemodynamic response.

Patients with acute myocardial infarction and normal subjects appear to differ in the mechanism of the reduction of cardiac output during the inhalation of oxygen. In normal subjects, the reduction of output is due entirely to a decrease in heart rate, since there is no accompanying reduction in stroke volume. This is apparently a reflex mechanism since it can be prevented with atropine. In subjects with uncomplicated myocardial infarction there is a decrease in stroke volume as well as cardiac output. At the present time there is no evidence in man that arterial PaO₂ values in the range observed in this study exert a direct inotropic action on the heart. The possibility of a negative inotropic action of oxygen should be considered, however, because at least two reports have shown that oxygen at hyperbaric pressures exerts a direct suppressive action on myocardial contractility in experimental animals.

The finding of most immediate clinical interest in this study was that patients who appeared uncomplicated at the time of admission, but who later developed clinical evidence of heart failure (class IB) had a distinct difference in their arterial Pao₂ response to oxygen when compared to patients who remained free of heart failure (class IA). Our data would suggest that it is possible to identify patients who are in subclinical heart failure by measuring their PaO₂ after breathing oxygen for 10 min. The early identification of such patients using this test may be a convenient and reliable way to predict heart failure, and thus to identify a group in whom the efficacy of therapeutic interventions including oxygen could be evaluated.

References


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