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*Chest* 1971;60:352-355  
DOI 10.1378/chest.60.4.352

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A M E R I C A N C O L L E G E O F



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# Effects of Low-Flow Oxygen on the Hemodynamics and Left Ventricular Function in Patients with Uncomplicated Acute Myocardial Infarction\*

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Cardiac output, arterial, central venous and left ventricular pressures and systolic time intervals were measured in patients with uncomplicated acute myocardial infarction during room air breathing and administration of oxygen at 6 L/min. With oxygen cardiac output decreased and arterial pressure increased. These changes were of small magnitude. Measurement of left ventricular pressures, stroke work, and the ratio of pre-ejection period to left ventricular ejection time failed to show evidence of impaired ventricular performance resulting from oxygen administration. Blood gas measurements revealed a high incidence of hypoxemia which was easily corrected by administration of low-flow oxygen. From these data we conclude that low-flow oxygen does not significantly impair the hemodynamic status or left ventricular performance of patients with uncomplicated acute myocardial infarction. Since hypoxemia is often present, low-flow oxygen should be used routinely.

Administration of oxygen (O<sub>2</sub>) has become a standard practice in the management of patients with acute myocardial infarction (AMI).<sup>1</sup> In patients with pulmonary edema or shock hypoxemia may be severe and O<sub>2</sub> therapy appears to be justified.<sup>2</sup> In patients with uncomplicated AMI, however, the value of routine O<sub>2</sub> therapy has not been established. Arterial hypoxemia is present in most patients with AMI,<sup>3-5</sup> and the degree of hypoxemia correlates well with functional severity and ultimate prognosis.<sup>5-6</sup> Although hypoxemia following AMI probably reflects the degree of myocardial damage and circulatory impairment, it

may also contribute to the development of arrhythmias and may adversely affect cardiac performance.

On the other hand, studies by Thomas and associates<sup>7</sup> and Sukumalchantra and co-workers<sup>8</sup> have shown that administration of 8 to 10 L/min of O<sub>2</sub> to patients with AMI often results in a decreased cardiac output and increased arterial pressure, which is most likely due to O<sub>2</sub> induced arteriolar constriction. If this effect were of significant magnitude, O<sub>2</sub> therapy might, in itself, adversely affect ventricular performance.

Our studies were designed to assess the effects of low-flow O<sub>2</sub> (6 L/min by mask or nasal cannula) on cardiac performance in patients with uncomplicated AMI.

## METHODS

Patients admitted to the coronary care unit of the Cook County Hospital with a diagnosis of AMI were evaluated. Informed consent was obtained from each patient prior to the study. Patients with shock, clinical congestive failure or hemodynamically significant arrhythmias were excluded. Also

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Supported in part by grant HE-08834 from the National Heart and Lung Institute, National Institutes of Health, Bethesda, Maryland.

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excluded were patients in whom a diagnosis of acute myocardial infarction (characterized by history, serial ECG and serial enzyme changes) could not be substantiated.

Studies were conducted in the coronary care unit. Mean arterial pressure (MAP) was measured from an indwelling catheter or plastic cannula introduced percutaneously into the right brachial or radial artery. Central venous pressure (CVP) was measured through a standard intracatheter advanced from a superficial arm vein into the superior vena cava or right atrium. Left ventricular end-diastolic pressure (LVEDP) was measured in seven patients from a No. 5 Teflon end-hole catheter advanced under fluoroscopic control into the left ventricle following percutaneous insertion into the right brachial or femoral artery. All catheters were connected to Statham 23 Db transducers. Mean pressures were derived electronically.

Cardiac output (CO) was measured by averaging the values derived from two or more indicator-dilution curves obtained following injection of indocyanine green dye into the central circulation and sampling arterial blood by continuous withdrawal through a Waters densitometer through the use of Harvard constant infusion-withdrawal pump. Cardiac index (CI) was calculated by dividing the CO by the body surface area derived from the nomogram of DuBois. Stroke volume (SV) was calculated by dividing CO by the heart rate (HR) determined from a continuously monitored ECG. Systemic vascular resistance (SVR) was calculated as:

$$SVR = \frac{MAP - CVP}{CO}$$

Left ventricular stroke work (LVSW) was estimated from the formula:

$$LVSW = \frac{SV \times (MAP - LVEDP) \times 13.6}{1000}$$

Systolic time intervals were measured according to the method of Weissler and co-workers,<sup>9</sup> using a Hewlett-Packard No. 62-1500-C16 dynamic microphone for recording of heart sounds and a Hewlett-Packard No. 21051D pulse wave pick-up for recording the external carotid pulse. The  $QS_2$  is the time from the q wave of the ECG to the first high frequency component of the second heart sound. Left ventricular ejection time (LVET) was measured from the onset of the external carotid pulse to its incisura. In some patients the central aortic pressure pulse measured directly was substituted for the external carotid pulse. This substitution has been shown not to significantly influence the results.<sup>10</sup> The pre-ejection period (PEP) was derived by subtracting the LVET from the  $QS_2$ .

Blood gas and pH determinations were done by use of the Instrument Laboratory, Inc. 113 pH/blood gas analyzer.

Statistical analysis was performed by the Student T test for paired observations. A p value of .05 or less was considered to indicate statistical significance.

#### PROCEDURE

Control data were obtained after the patient had been breathing room air for at least 30 minutes. Oxygen was then administered either by mask or nasal cannula at a flow rate of 6 L/min. All measurements were repeated after 20 or more minutes (mean 51 minutes) from the time  $O_2$  was started. In two patients adequate control measurements were not obtained prior to starting  $O_2$  but were obtained 15 and 30 minutes after  $O_2$  had been stopped. In some patients the study was repeated on the second or third day following admission.

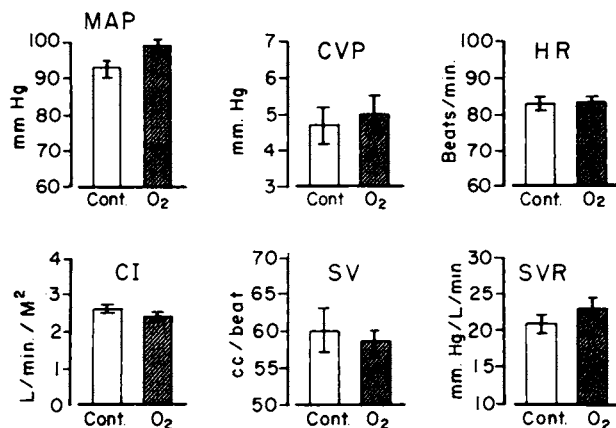


FIGURE 1. Hemodynamic values during room air breathing (cont), and administration of low-flow oxygen ( $O_2$ ). Mean values  $\pm$  one standard error are shown. (See text for abbreviations.)

#### RESULTS

A total of 46 studies were conducted in 31 patients. No patient was studied more than once in a single day. There were 21 men and 10 women. The average age was 60 and ranged from 37 to 82 years. Twenty-four patients had transmural infarction (pathologic q waves) and seven patients had nontransmural infarction (ECG and enzyme changes of AMI without pathologic q waves). There was no mortality or significant morbidity associated with the study, and all patients were discharged from the CCU to convalesce on the general medical wards.

#### Hemodynamic Responses to $O_2$ (Fig 1)

During low-flow  $O_2$  MAP increased from  $93 \pm 2$  to  $99 \pm 2$  mm Hg (mean  $\pm$  SEM) ( $p < .01$ ); CVP changed from  $4.7 \pm 0.5$  to  $5.0 \pm 0.5$  mm Hg (NS) and HR was unchanged, being  $83 \pm 2$  and  $83 \pm 2$  per min; cardiac index (CI) fell from  $2.6 \pm 0.1$  to  $2.4 \pm 0.1$  L/min/M<sup>2</sup> ( $p < .01$ ); SV fell from  $60 \pm 3$  to  $57 \pm 3$  ml (NS), and systemic vascular

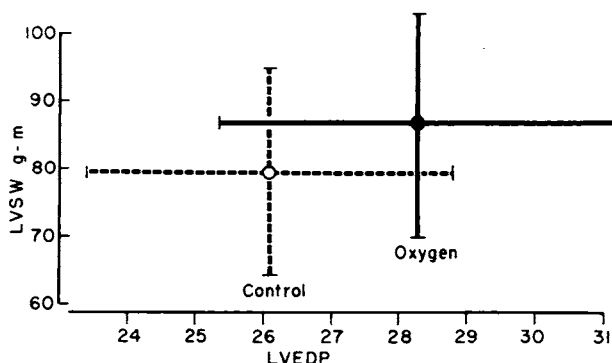


FIGURE 2. The relationship between LVSW and LVEDP is shown during room air breathing and administration of low-flow  $O_2$ . The mean  $\pm$  one standard error is shown for both LVSW and LVEDP.

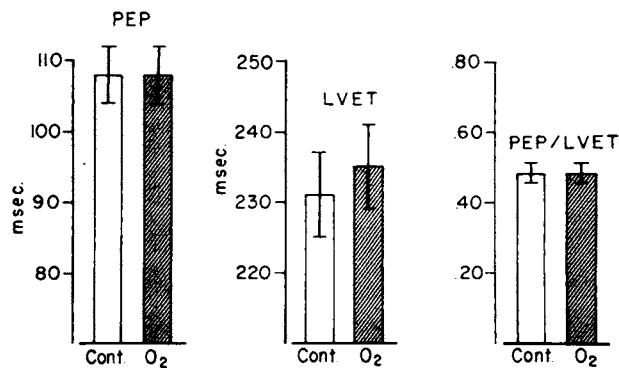


FIGURE 3. Systolic time intervals during room air breathing and administration of low-flow O<sub>2</sub>. resistance (SVR) increased from  $20.7 \pm 1.2$  to  $22.9 \pm 1.2$  mm Hg/L/min ( $p < .01$ ).

#### Left Ventricular Pressure-Work Responses to O<sub>2</sub> (Fig 2)

LVEDP and LVSW were measured in seven patients. During low-flow O<sub>2</sub> LVEDP increased from  $26.1 \pm 2.7$  to  $28.3 \pm 2.9$  mm Hg, while LVSW increased from  $79.6 \pm 15.2$  to  $86.9 \pm 16.9$  g-m.

#### Systolic Time Interval Responses to O<sub>2</sub> (Fig 3)

PEP, LVET and the PEP/LVET ratio were determined on 31 occasions in 22 patients. During low-flow O<sub>2</sub> PEP did not change from the control value of  $108 \pm 4$  msec, while LVET increased from  $231 \pm 6$  to  $235 \pm 6$  msec. The PEP/LVET ratio was  $.48 \pm 0.25$  and  $.48 \pm 0.24$  during both the control and low-flow O<sub>2</sub> periods.

#### Arterial Blood Gases (Fig 4)

During low-flow O<sub>2</sub> arterial Po<sub>2</sub> increased from  $68 \pm 1$  mm Hg (room air) to  $142 \pm 8$  mm Hg. At the same time, arterial Pco<sub>2</sub> remained unchanged at  $34 \pm 1$  mm Hg and pH increased slightly ( $7.433 \pm .006$  to  $7.438 \pm .006$ ).

#### DISCUSSION

Although hypoxemia following AMI is most marked in patients developing shock or pulmonary

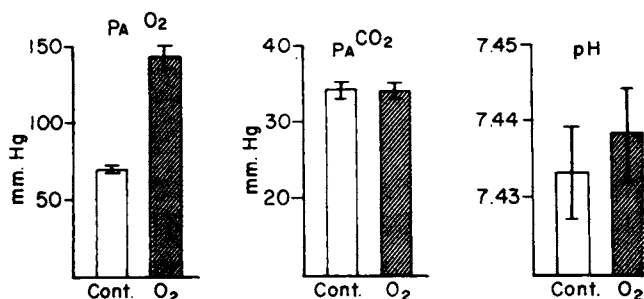


FIGURE 4. Arterial blood gas and pH values during room air breathing and administration of low-flow O<sub>2</sub>.

edema,<sup>5-6</sup> a significant number of patients with apparently uncomplicated AMI have Po<sub>2</sub> values under 70 mm Hg.<sup>3-5</sup> Values for Po<sub>2</sub> of less than 70 mm Hg during room air breathing were present in 16 of our 31 patients and in five the Po<sub>2</sub> was under 60 mm Hg.

Although correction of mild hypoxemia is not of proved benefit in patients with uncomplicated AMI, the routine administration of O<sub>2</sub> to such patients is widely practiced and, unless proved harmful, appears to be justified on the theoretical grounds that increased O<sub>2</sub> delivery to ischemic myocardium might prevent arrhythmias, improve contractility and reduce the amount of permanent myocardial damage.

Previous studies<sup>5,7,8</sup> have shown that O<sub>2</sub> administration (in somewhat higher concentrations than we employed) to patients with AMI results in a reduction in CO and a rise in MAP. We have found similar hemodynamic responses to low-flow O<sub>2</sub> in our patients. We would stress, however, that the magnitude of the changes we observed was so small that they are of doubtful clinical significance.

In order to better evaluate the effects of low-flow O<sub>2</sub> on left ventricular performance, we have measured changes in LVEDP and LVSW. Small increases in both (averaging 12 percent and 10 percent, respectively) occurred, suggesting that ventricular performance was not altered during low-flow O<sub>2</sub>. This conclusion is supported by the results obtained from the measurement of systolic time intervals. Neither PEP nor PEP/LVET ratio were affected by low-flow O<sub>2</sub>. The PEP/LVET ratio has been correlated with left ventricular ejection fraction<sup>11</sup> and tends to increase as myocardial performance worsens.<sup>9</sup>

The blood gas measurements confirm the high incidence of hypoxemia present in patients with uncomplicated AMI. It also becomes clear that in nearly all such patients correction of hypoxemia is possible with low-flow O<sub>2</sub>. In fact, considering the potential toxic effects of O<sub>2</sub> on the lung,<sup>12</sup> it would seem advisable to administer O<sub>2</sub> at the minimal concentration necessary to maintain a normal hemoglobin oxygen saturation.

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## A Symptom Connoting Many Causes and Sequels

It was in the middle of high mass on a Sunday when the youthful socialite entered the church. Her attractive dress, exuberant vigor and prodigious feminine loveliness would have drawn prompt attention anywhere. They failed to do so at this time because the eyes of worshippers were fixed on their prayer books. As she was walking down the aisle, however, the silence of devotion was broken and all eyes turned toward her, provoked by her sharp staccato cough. Then she took her place in the front pew, with pious satisfaction. Obviously, coughing served impulsively her aggressive self-assertiveness. Another kind of inappropriate psychogenic cough results from conversion of mental or emotional stress or anguish. Thus cough may become a symbolic attempt to escape from a challenging problem, to mask, reduce or release suppressed tension or to give alternate expression of a sense of frustration. Subconsciously, the patient resorts to this maneuver so as to enable him to seek medical attention for his externalized, disguised anxiety. Such sham cough may appear at frequent intervals, last for hours and may result in physical exhaustion or even in occupational disability. Relative to cardiovascular etiology, in mitral stenosis and to a lesser extent in mitral insufficiency, cough is predicated upon pulmonary venous hypertension. In left ventricular failure due to hypertension, arteriosclerosis or aortic valve disease, paroxysmal nocturnal cough with sudden distressing dyspnea may awaken the patient. The disturbing episode of cardiac asthma occurs about 3-4 hours after going to bed. In addition to shift of blood from dependent parts of the body to the pulmonary vasculature, functional impairment of the lung and elevated levels of carbon dioxide are likely to contribute to such attacks. In postmyocardial infarction syndrome, cough may be a conspicuous symptom without evidence of heart failure. Uncommon causes of cough are on record, such as that due to osteophytes of the cervical spine, aneurysm of the ascending palatine artery, vascular ring syndrome. In one instance, spasmodic cough appeared whenever the patient turned his head. Its cause was a neurilemmoma of the vagus nerve behind the lower portion of the sternomastoid. Cough and expectoration may be the chief complaint in intralobar sequestration (Rokitansky's

lobe). Cough has been reported in 33 per cent of patients with diverticulum of the esophagus. Invariably, audiences received with much amazement the case report in which volitional cough of the patient projected a slender pedunculated esophageal lipoma into his open mouth. No doubt, excessive cough of long duration exerts adverse influence upon the individual, in general, and upon the cardiorespiratory organs, in particular. Of the long list of such possibilities, only a few will be mentioned. Tussive syncope is thought to be attributable to pronounced decrease in the systemic arterial pressure associated with an increase in the right ventricular pressure which may reach as high as 300 mm Hg during paroxysms of coughing (McCann, W S et al, *Arch. Int. Med.* 84:845, 1949). The consequent intrapulmonary pressure which may reach 200 mm Hg over and above atmospheric pressure may activate a vago-cortical reflex and result in loss of consciousness and/or convulsions. Severe coughing of prolonged duration causes trapping of blood in the pulmonary circulation, throws undue strain on the right ventricle and may be instrumental in the development of cor pulmonale. Pulmonary interstitial emphysema, pneumomediastinum and subcutaneous emphysema may result from strenuous coughing when perivascular alveoli rupture and air passes from the lung along vascular sheaths to the mediastinum. It is a rational assumption that the integrity of alveolar septa may be undermined by microbial toxins, noxious inhalants, and inadequate blood supply through diseased blood vessels and consequently the septa may rupture under unduly high intraalveolar pressure. When some of the alveoli are involved by fibrosis, atelectasis or accumulation of exudate, adjacent alveoli are subject to uneven stresses during cough. Also, check-valve mechanism of connecting bronchioles may add to sustained increase in alveolar pressure during cough. Even during the expulsive phase of cough there is "retropressure" or jet effect in the alveoli; the thrust at the closed end of a tube is equal to the thrust of gas escaping at the restricted open end. The combined effect of these factors is likely to exert its damaging effect upon the lung parenchyma and contribute to the development of emphysema.

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**This information is current as of November 17, 2009**

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