Effects of Acute Hypoxia on the Pulmonary Vascular Bed of Patients with Acquired Heart Disease

With Special Reference to the Demonstration of Active Vasomotion

By Paul N. Yu, M.D., Gerald Glick, M.D., Bernard F. Schreiner, Jr., M.D., and Gerald W. Murphy, M.D.

The effects of breathing low oxygen (acute hypoxia) on the pulmonary circulation in normal subjects and in patients with various cardiopulmonary diseases have been reported by many workers. In general, during the period of acute hypoxia the pulmonary arterial pressure and the pulmonary blood flow usually rise, while the pulmonary wedge or left atrial pressure is unchanged or slightly increased. In most instances there is an increase in the calculated pulmonary vascular resistance, since the magnitude of rise in the pulmonary arterial to left atrial pressure gradient usually is proportionately higher than that in the pulmonary blood flow.

Based upon the augmentation in the pulmonary vascular resistance during acute hypoxia, it has been generally assumed that decreased alveolar oxygen tension invokes active constriction of the pulmonary vessels. Changes in the vascular resistances, however, can result simply from passive distention or narrowing associated with a proportional increase or decrease in both the volume of blood and distending pressure within the vessel. Therefore, the measurement of pulmonary vascular resistance alone may not necessarily reflect a change of caliber of the vessels. Active vasoconstriction of the pulmonary vascular bed in man during acute hypoxia would be more reliably demonstrated if one could show a decrease in pulmonary blood volume with an attendant rise in pulmonary vascular pressures. To our knowledge, no such clinical studies have been reported in the literature except for our preliminary data in five cases included in a previous communication.

The purpose of this paper is to report our further observations on the effects of acute hypoxia on the pulmonary vascular bed and to demonstrate the presence of active vasomotion of the pulmonary vessels in patients with acquired heart disease.

Methods and Clinical Material

All patients underwent right heart and transseptal left heart catheterization, with a catheter placed in the main pulmonary artery and another catheter in the left atrium. The zero level for right heart pressure was taken 6.5 cm. below the sternal angle; for the left heart pressure, 8.5 cm. below the sternal angle. A no.-18 Courand needle was inserted into a brachial artery and connected to a cuvette densitometer. Indicator-dilution curves were inscribed from the brachial artery after indocyanine green (Cardiogreen) was injected sequentially into the pulmonary artery and the left atrium, as described by Milnor and associates. The two injections were usually accomplished within a 2-minute period, and the order of injection was randomized. The sampling rate was 0.7 to 0.8 ml. per second, and each curve was inscribed on a direct writing recorder. All tracings were corrected for "dead space" time from the arterial needle to the densitometer which was approximately 2 seconds.

The "central" blood volume is that volume between the main pulmonary artery and the brachial artery, including all temporarily equidistant...
Values of cardiac indexes derived from the PA curves are plotted against those derived from the corresponding LA curves obtained from the patients of the present series. There was good agreement between the values derived from the two sets of curves, and no systematic error was observed. The solid line indicates identity, and the two dotted lines represent the boundaries of a difference of ±10 per cent.

branches. It was calculated by the Stewart-Hamilton method from the product of the cardiac index and the mean transit time from pulmonary artery to brachial artery.

$$\text{CBV} = \text{CI} \cdot T_{M(PA-BA)}$$

where,

$$\text{CBV} = \text{"central" blood volume (ml./M.2)}$$

$$\text{CI} = \text{cardiac index (ml./M.2/sec.)}$$

$$T_{M(PA-BA)} = \text{mean transit time in seconds from PA to BA}$$

The pulmonary blood volume is the volume within the pulmonary arteries, pulmonary capillaries, and pulmonary veins. It was calculated from the cardiac index multiplied by the mean transit time from main pulmonary artery to left atrium.

$$\text{PBV} = \text{CI} \cdot (T_{M(PA-BA)} - T_{M(LA-BA)}$$

where,

$$\text{PBV} = \text{pulmonary blood volume (ml./M.2)}$$

$$T_{M(LA-BA)} = \text{mean transit time in seconds from LA to BA}$$

The volume within the left atrium, left ventricle, and arterial components, left atrial to brachial arterial volume, was calculated as follows:

$$\text{LA to BA volume} = \text{CI} \cdot T_{M(LA-BA)}$$

As has been previously reported from this laboratory, there was a good agreement between the values of cardiac index derived from indicator-dilution curves following pulmonary arterial injection and those following left atrial injection. In 84 determinations obtained from 40 patients, the correlation coefficient was high (r = 0.90), and there was no systematic error (fig. 1).

In the calculation of blood volume, therefore, the mean values of the cardiac index derived from the paired indicator-dilution curves were used.

The reproducibility of the method has been demonstrated in another series of 16 patients on whom duplicate determinations of pulmonary blood volume were made after a period of 10 to 15 minutes. The standard deviation of the duplicate determinations was 14.9 ml./M.2 (fig. 2). In a given patient, a measurement of pulmonary blood volume differing by 50 ml./M.2 from the control value was considered significant. In the same series of patients the standard deviation of duplicate determinations of central blood volume was 31.4 ml./M.2. Therefore, a difference of 100 ml./M.2 or greater between the control and experimental values was considered significant.

In the early part of our study the arterial blood withdrawn for indicator-dilution curves was not re-infused into the patients. Therefore, when multiple curves were inscribed, the total blood loss during the procedure would be considerable. Recently, the blood has been returned to the patients.
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after inscription of each dilution curve, and the total blood loss has been minimal.

As suggested by Milnor and associates,¹⁵ the mean distending pressure in the pulmonary vessels was calculated by averaging the mean pulmonary arterial and mean left atrial pressures. Since, during inhalation of 12 per cent oxygen, only minimal alterations in the intrapleural pressures have been observed in man,⁷⁻⁸ the change in intravascular distending pressure may be considered a reasonable estimate of changes in “transmural” pressure.

The arterial blood oxygen was analyzed by the manometric method of Van Slyke and Neill.¹⁷

Eighteen patients, 16 with valvular heart disease and two with cardiomyopathy, were included in this study. Among the 16 patients with valvular heart disease, eight had predominant aortic valvular lesions, and eight had predominant mitral valvular lesions. There were 12 men and six women, ranging in age from 17 to 51 years.

During the control period, while the patients were breathing room air, pulmonary arterial, left atrial, and brachial arterial pressures and the electrocardiogram were recorded simultaneously. In most instances pulmonary wedge pressure was also recorded either prior to or simultaneously with left atrial pressure. Dilution curves were inscribed after sequential injections of the indicator into the pulmonary artery and the left atrium. Subsequently, arterial blood samples were obtained for gas analysis.

During the period of acute hypoxia, the patients inhaled 12 per cent oxygen in nitrogen through a mouthpiece for 10 to 14 minutes, and the minute ventilation and respiratory frequency were constantly monitored. Between the eighth and twelfth minutes of inhalation of low oxygen, recording of pressures, inscription of indicator-dilution curves, and sampling of arterial blood were repeated. In five cases simultaneous pulmonary wedge and left atrial pressures were recorded. The above protocol was carried out in 16 patients (cases 1 to 16).

Three patients (cases 12, 17, and 18) were studied after a prolonged period of acute hypoxia (for at least 16 minutes) and after removal of a considerable amount of blood (350 ml. or more).

In three patients (cases 1, 15, and 16) studies were made during acute hypoxia before and during infusion of acetylcholine into the right ventricle through the proximal lumen of a double lumen catheter, while the tip of the catheter was placed in the main pulmonary artery. In addition, a second no.-18 Cournand needle was inserted into the left femoral artery for continuous recording of systemic arterial pressure. During the infusion of acetylcholine the pulmonary arterial, left atrial, and femoral arterial pressures, minute ventilation, and respiratory frequency were continuously recorded. The infusion rate was regulated at 2 to 4 mg. per minute in order to produce an appreciable reduction of the pulmonary arterial pressure without initiating cough or affecting the femoral arterial pressure or ventilatory rate.

Results

The hemodynamic responses and changes in “central” blood volume and in pulmonary blood volume during inhalation of 12 per cent oxygen by 16 patients are summarized in table 1. The following changes were observed during acute hypoxia.

Arterial oxygen saturation declined significantly from a control mean value of 94 per cent to a mean value of 74 per cent (p < 0.001). The changes in pulmonary vascular pressure and pulmonary blood volume were not directly related to the magnitude of decline of systemic arterial oxygen saturation.

The cardiac index increased slightly from 2.75 L./min./M.² to 3.06 L./min./M.². There was a significant increase in the heart rate (p < 0.02), while stroke index showed no consistent change.

The mean pulmonary arterial pressure increased 5 mm. Hg or more in 10 of 16 patients. The increase in mean pulmonary arterial pressure for the whole group was statistically highly significant (p < 0.001). In general, those patients with an elevated resting mean pulmonary arterial pressure manifested a greater rise than those with normal resting pressures. The mean left atrial pressure did not change more than 4 mm. Hg, except in one patient (case 16) who showed a rise of 6 mm. Hg. In the five patients in whom the pulmonary wedge and left atrial pressures were recorded simultaneously, the difference between the two never exceeded 2 mm. Hg. In two, the pulmonary wedge pressures were slightly higher than the left atrial pressure, whereas in three the opposite was noted. The pulmonary distending pressure increased in 13 of 16 patients (p < 0.01), with an increase of 3 mm. Hg or greater in six.

In the entire series no consistent change was observed in the mean brachial arterial
### Table 1: Circulatory Effects of Acute Hypoxia

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<th>Condition</th>
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<th>( V_0 )</th>
<th>Blood flow</th>
<th>Mean pressures (mm Hg)</th>
<th>MTT (sec.)</th>
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<td>21.0 92</td>
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<td>5.9</td>
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The symbol "p" indicates the probability that this observed difference is a chance occurrence. A difference with a chance probability of 0.05 or less is considered to be significant.

The pulmonary vascular resistance (PVR) may be calculated as the ratio of pulmonary arterial pressure (PA) to pulmonary blood flow (Qp).

\[ \text{PVR} = \frac{\text{PA}}{\text{Qp}} \]

Increased pulmonary vascular resistance may be due to increased muscularity or decreased distensibility of the vessels. The resistance may be increased in a variety of conditions, including pulmonary hypertension, pulmonary embolism, and chronic obstructive pulmonary disease.

In this study, the pulmonary vascular resistance was measured in 16 patients using a pulmonary artery catheter. The results showed a significant increase in pulmonary vascular resistance in the hypoxic group compared to the control group.

The table below shows the mean pulmonary artery pressure (PA) and pulmonary vascular resistance (PVR) for each group.

<table>
<thead>
<tr>
<th>Group</th>
<th>PA (mm Hg)</th>
<th>PVR (dyne sec/cm²)</th>
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<td>Hypoxic</td>
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The difference in pulmonary vascular resistance between the control and hypoxic groups was statistically significant (p < 0.05). This suggests that hypoxia may be a contributing factor to increased pulmonary vascular resistance.

The increased resistance may lead to increased pulmonary artery pressure and right ventricular pressure, which can eventually lead to right ventricular hypertrophy and failure.

The pulmonary vascular resistance in this study was measured using a pulmonary artery catheter. The catheter was inserted into the right atrium and advanced through the right ventricle and pulmonary artery into the lung parenchyma. The pressure in the pulmonary artery and right ventricle was recorded continuously during the study.

In conclusion, hypoxia is associated with increased pulmonary vascular resistance, which may lead to increased pulmonary artery pressure and right ventricular pressure, and eventually right ventricular hypertrophy and failure.

The increased pulmonary vascular resistance may be due to increased muscularity or decreased distensibility of the vessels. Further studies are needed to determine the underlying mechanism of increased pulmonary vascular resistance in hypoxia.
Continuously recorded tracings of femoral arterial (FA), left atrial (LA), and pulmonary arterial (PA) pressures and electrocardiogram in case 9. Note that during the period of breathing low oxygen there was a significant rise in pulmonary arterial pressure despite a slightly decreased femoral arterial pressure and an unchanged left atrial pressure. In this case, during acute hypoxia, a significant decrease in the pulmonary blood volume was observed. The arrow indicates the beginning of hypoxia. Numbers at the top represent minutes of hypoxia.
The changes in pulmonary distending pressure ($PA_m + LA_m/2$) are plotted against those in pulmonary blood volume during inhalation of low oxygen. In 13 of 16 patients an increase in pulmonary distending pressure was accompanied by a decreased or unchanged pulmonary blood volume. These discordant changes in the pressure-volume relationship are convincing evidence of active vasoconstriction of the pulmonary vascular bed. Open circles represent control values; black dots represent values during acute hypoxia.

In the three patients who received acetylcholine, the elevated pulmonary arterial pressure induced by acute hypoxia could be promptly lowered by this agent. The pulmonary arterial pressure was significantly lower during combined acute hypoxia and acetylcholine infusion than during acute hypoxia alone. The reduction occurred within the first 2 minutes of infusion. When an acetylcholine infusion was added to hypoxia, the pulmonary blood volume increased significantly in two patients and remained unchanged in the third. In one patient (case 10) when the acetylcholine infusion was terminated but hypoxia continued, the pulmonary arterial pressure...
### Table 2

**Effects of Acetylcholine on Pulmonary Circulation during Acute Hypoxia**

<table>
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<th>Case no.</th>
<th>Diagnosis</th>
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<th>S, O₂ (**)</th>
<th>Blood flow CI, HR, SI</th>
<th>Mean pressures (mm Hg) PA, LA, Po, BA, PVR</th>
<th>MTT (sec.) PA-BA, PA-LA</th>
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A = Control period.  
B = Period of acute hypoxia.  
C = Period of acute hypoxia and acetylcholine infusion.  
D = Period of acute hypoxia.

Other abbreviations and symbols are identical to those used in Table 1.

**Discussion**

Technics employing simultaneous or sequential injections of indicators afford the possibility of nonuniform mixing when the time of injection is the potential errors involved. These have been reviewed in detail elsewhere, i.e., the mixing of indicator in the left atrium and the turbulence within the left atrium. The potential errors must be made with an appreciation of the following two points should be emphasized.

First, there is the possibility of nonuniform mixing of the left atrium and the turbulence within the left atrium. This may be related to the position of the left atrium in the flow rate of the left atrium, the turbulence within the left atrium to the pulmonary artery. The changes occurring exist, and the results must be made with an appreciation of the changes occurring exist.

**Note:**

The calculations of peripheral vascular resistance were obtained by the formula:  

\[ \text{PVR} = \frac{\text{Mean arterial pressure}}{\text{Cardiac output}} \]

The calculation was based on the assumption that the mean arterial pressure was constant during the period of observation. The mean arterial pressure was calculated from the following equations:

\[ \text{Mean arterial pressure} = \frac{\text{Systolic pressure} + 2 \times \text{Diastolic pressure}}{3} \]

where the systolic pressure is the highest pressure observed during the period of observation, and the diastolic pressure is the lowest pressure observed during the period of observation. The cardiac output was calculated as the product of the stroke volume and the heart rate.

**Technics:**

The techniques employed in these studies included the use of the dye dilution method to determine the cardiac output and the pulmonary vascular resistance. The dye was injected into the left atrium, and the dye concentration in the pulmonary artery was measured using a fluorometer. The cardiac output was calculated using the formula:

\[ \text{Cardiac output} = \frac{\text{Dye concentration in pulmonary artery}}{\text{Dye concentration in left atrium}} \times \text{Volume of dye injected} \]

The pulmonary vascular resistance was calculated using the formula:

\[ \text{PVR} = \frac{\text{Mean arterial pressure}}{\text{Cardiac output}} \]

These values were used to calculate the pulmonary vascular resistance in each patient. The results are presented in Table 3.

**In three patients (cases 12, 17, and 18), the changes observed were summarized in Table 2, and the data obtained were summarized in Table 3.**
atrial arrhythmia, etc. Thus, a variable fraction of the left atrial blood volume may be included in the pulmonary blood volume.

Secondly, an unavoidable potential source of error relates to the uneven pulmonary blood flow that is known to occur in the lungs of patients with mitral valvular disease. It is theoretically possible that as the result of poor mixing of indicator in slowly moving pools, or as the result of non-mixing in stagnant blood pools, the estimated pulmonary blood volume may be erroneously high or low.

The results of the present study confirm previous reports by other workers and from this laboratory that short-term hypoxia in man induces an increase in pulmonary arterial pressure and pulmonary vascular resistance with little or no effect on pulmonary wedge, left atrial, and systemic arterial pressures.

The elevated pulmonary distending pressure in the face of a decreased or unchanged pulmonary blood volume is convincing evidence of the presence of active constriction of the pulmonary vascular bed, as a result of augmented tone of the pulmonary vessels. We feel that the evaluation of discordant changes of the pressure-volume relationship in the pulmonary vascular bed is a far better and more reliable index of active vasomotion than the alteration in the calculated pulmonary vascular resistance. This contention is supported by the data obtained from three of our patients (cases 1, 3, and 9) in whom active vasoconstriction occurred during acute hypoxia, despite a slight decrease or no change in calculated pulmonary vascular resistance.

From the present study it is not certain in what segment of the pulmonary vascular bed active vasoconstriction has occurred. As pointed out by other workers, pulmonary capillaries probably are not contractile vessels, since they lack a muscular component. The absence of a pressure gradient from the pulmonary wedge position to the left atrium speaks strongly against any significant sphincter action by pulmonary veins in man.

Nevertheless, since pulmonary veins contain about 53 per cent of the blood in the pulmo-

---

**Table 3**

<table>
<thead>
<tr>
<th>Case</th>
<th>Diagnosis</th>
<th>Age, A.G.</th>
<th>Condition</th>
<th>Amount of blood removed (ml)</th>
<th>Mean of blood flow (ml/min.)</th>
<th>Mean of blood S. O. 2</th>
<th>PA, LA, P.A.L.</th>
<th>C.W.</th>
<th>M.T.R. (sec.)</th>
<th>P.V.R.</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>MS</td>
<td>44 M</td>
<td>A</td>
<td>450</td>
<td>76.0</td>
<td>2.92</td>
<td>12</td>
<td>10</td>
<td>115</td>
<td>1.43</td>
</tr>
<tr>
<td>17</td>
<td>MI</td>
<td>30 F</td>
<td>A</td>
<td>350</td>
<td>76.6</td>
<td>2.81</td>
<td>12</td>
<td>10</td>
<td>118</td>
<td>1.42</td>
</tr>
<tr>
<td>18</td>
<td>MI</td>
<td>52 M</td>
<td>A</td>
<td>350</td>
<td>92.0</td>
<td>2.20</td>
<td>12</td>
<td>10</td>
<td>118</td>
<td>1.42</td>
</tr>
<tr>
<td>12</td>
<td>MS</td>
<td>44 M</td>
<td>B (19)</td>
<td>450</td>
<td>76.0</td>
<td>2.92</td>
<td>12</td>
<td>10</td>
<td>115</td>
<td>1.43</td>
</tr>
<tr>
<td>17</td>
<td>MI</td>
<td>30 F</td>
<td>B (19)</td>
<td>350</td>
<td>76.6</td>
<td>2.81</td>
<td>12</td>
<td>10</td>
<td>118</td>
<td>1.42</td>
</tr>
<tr>
<td>18</td>
<td>MI</td>
<td>52 M</td>
<td>B (19)</td>
<td>350</td>
<td>92.0</td>
<td>2.20</td>
<td>12</td>
<td>10</td>
<td>118</td>
<td>1.42</td>
</tr>
</tbody>
</table>

**A** = Control period

**B** = Prolonged period of acute hypoxia and phlebotomy.

Figures in parenthesis denote duration of hypoxia in minutes.

Other abbreviations and symbols are identical to those used in table 1.
nary vascular bed, it is reasonable to speculate that during acute hypoxia mild contraction of the pulmonary veins may still occur with an appreciable change in the volume but no change in venous pressure. Such constriction would increase venous return to the left atrium, but, in the presence of a compensated heart, the venous pressure would not be expected to rise. It should be pointed out, however, that in cattle and in dogs hypoxia may indeed induce a pressure gradient between the pulmonary wedge position and the left atrium as a consequence of a sphincter mechanism located either within the pulmonary veins or at their junction with the left atrium.

Since the pressures in the left atrium and in the pulmonary wedge position did not change significantly, the bulk of the evidence obtained in the present study indicates that acute hypoxia in these patients produces its most pronounced effect on pressure by its action proximal to the pulmonary arteries. The increase in pulmonary arterial or distending pressure was generally most impressive in those patients who had pre-existing pulmonary hypertension. This augmented change could not be accounted for by any unusual degree of increased pulmonary blood flow. In these patients some degree of pulmonary vasoconstriction probably was already present. During acute hypoxia any additional constriction of the pulmonary arterioles would result in a disproportionately greater increase in the vascular pressure and resistance than in those patients without pre-existing pulmonary hypertension.

Utilizing indicator-dilution curves, other investigators have shown inconsistent changes in "central" blood volume during hypoxia. However, since the volume so determined has included blood within variable portions of the right and left heart, in addition to that in the pulmonary bed, it is conceivable that critical changes in blood volume within the lungs may have been obscured by changes in various cardiac chambers or in the arterial component. Our present studies have shown, in fact, that in several patients the pulmonary blood volume decreased significantly even though the "central" blood volume showed an increase or no appreciable change.

In experimental animals some workers, using radioisotope methods, demonstrated an increase in pulmonary blood volume during acute hypoxia.

Recently, many investigators have reported the effects of acetylcholine on the pulmonary circulation in normal subjects and in patients with cardiopulmonary diseases. It is generally agreed that in normal subjects with increased pulmonary arterial pressure induced by acute hypoxia and in patients with a moderate degree of pulmonary hypertension acetylcholine does have a vasodilating effect. Our recent study of this agent in five patients has provided evidence that it does, indeed, produce active vasodilation of the pulmonary vessels. This active vasodilation was manifested by a significant reduction in the pulmonary arterial and pulmonary distending pressure associated with an increase in the pulmonary blood volume during infusion of this agent into the pulmonary circulation.

The results of the present study have confirmed the earlier studies reported by Fritts and associates and have further demonstrated that acetylcholine may counteract and reverse vasoconstriction of the pulmonary vessels induced by acute hypoxia. When an acetylcholine infusion was added, we noted a considerable reduction of pulmonary arterial and pulmonary distending pressures, despite an increase or no change in pulmonary blood volume. These results strongly suggest that the augmented tone produced by hypoxia was counteracted by the active vasodilation caused by acetylcholine.

With prolonged hypoxia, particularly after removal of a considerable amount of blood, active vasomotion of the pulmonary vessels was no longer demonstrable. Rather, a uniform decrease in both systemic and pulmonary arterial pressures was observed. This reduction was not necessarily due to a severe degree of arterial oxygen desaturation, since none of
these three patients had a saturation of less than 70 per cent during the inhalation of the low oxygen. Almost 20 years ago, Barcroft and Edholm\textsuperscript{38} noted similar changes in man and even vasovagal-like reactions during acute hypoxia combined with phlebotomy. They interpreted these changes on the basis of profound peripheral vasodilation and increased blood flow to the skeletal muscles. In the present study we have demonstrated a decrease in systemic arterial pressure as well as in cardiac index. The calculated peripheral vascular resistance was reduced. Fishman and associates\textsuperscript{4} also noted the tendency of the elevated pulmonary arterial pressure to fall toward the initial levels in many of their patients as breathing at low levels of oxygen was continued. We believe, therefore, that the actively induced rise in pulmonary arterial pressure seen early in the course of acute hypoxia may be superseded by a fall of this pressure when the period of acute hypoxia is prolonged. The reduction in pulmonary arterial pressure is secondary to systemic hypotension, which may supervene when hypoxia is continued too long.

Finally, we wish to emphasize that the interpretation of these data should be restricted to patients with altered pulmonary circulation secondary to acquired heart disease. Although it is possible that similar results would be obtained in subjects with no cardiopulmonary disease, actual verification must await further study.

Summary and Conclusions

The present study was undertaken to investigate the nearly simultaneous changes in pulmonary blood flow, volume, and pressures during acute hypoxia in 18 cardiac patients. Indicator-dilution curves were inscribed from a brachial artery after rapid sequential injections of Cardiogreen into the main pulmonary artery and the left atrium (transseptal technic). Pulmonary blood volume was estimated from the product of cardiac output and mean transit time from the pulmonary artery to the left atrium. The distending or "transmural" pressure was calculated by averaging the mean pulmonary arterial and the mean left atrial pressures.

During acute hypoxia the presence of active vasoconstriction was demonstrated by an increase in the pulmonary distending pressure associated with a decrease or no change in the pulmonary blood volume. The increase in the pulmonary distending pressure was mainly due to an augmented pulmonary arterial pressure, no appreciable alteration in the left atrial pressure being noted.

Acetylcholine infused into the pulmonary circulation may counteract the hypoxic vasoconstriction and cause active vasodilation, despite the continuation of hypoxia. The vasodilating effects were evidenced by a decrease in pulmonary distending pressure and an increase in pulmonary blood volume.

When the period of acute hypoxia was prolonged and a considerable amount of blood was removed, there was a uniform reduction of pressures in both systemic and pulmonary circuits associated with a decrease in pulmonary blood volume. These changes were probably due to peripheral vasodilation, decreased venous return, and diminished cardiac output.

Acknowledgment

We are indebted to Dr. Arthur Dutton, Associate Professor of Radiation Biology and Scientist (Statistics), Atomic Energy Project, University of Rochester, for his help with the statistical aspects of this study.

We would like to thank Miss Mary Ellen Lindsay, Miss Ann Gratiot and Mr. Waddell Johnson for their technical assistance, and Mrs. Paula Robbins and Mrs. Bonnie Sollie for their secretarial aid.

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HYPOXIA AND PULMONARY VASCULAR BED


Giovanni Battista Morgagni, the Founder of Pathologic Anatomy

In dealing with the rhythm of the pulse, Morgagni recognized two kinds of intermittency. One kind was of long duration and of serious nature, which he considered due to hypoxia; this was the kind of disorder with which Lancisi, according to his own testimony, was afflicted. Morgagni also recognized another type of intermittency, in which the intermission was brief; this is illustrated by the ease of the distinguished professor of physic at Bologna, who happened to discover that once in a while his pulse was intermittent. Morgagni's advice was to tell this patient to take his finger off his wrist and not to inquire too anxiously about his condition. The advice was followed, and resulted in a complete recovery.—C. G. Tedeschi, M.D. Giovanni Battista Morgagni, The Founder of Pathologic Anatomy: A Biographic Sketch On the Occasion of the 200th Anniversary Of The Publication Of His “De sedibus et causis morborum per anatomem indagatis.” The Boston Medical Quarterly 12:122, 1961.
Effects of Acute Hypoxia on the Pulmonary Vascular Bed of Patients with Acquired Heart Disease: With Special Reference to the Demonstration of Active Vasomotion

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