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ARTERIAL BLOOD GAS MEASUREMENTS IN THE MANAGEMENT OF PATIENTS WITH CHRONIC BRONCHITIS AND EMPHYSEMA

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It is well known that a proportion of patients with severe emphysema develop anoxia and chronic respiratory acidosis (Taquini, Fasciolo, Suarez, and Chiodi, 1947, 1948; West, Baldwin, Cournand, and Richards, 1951; Cohn, Carroll, and Riley, 1954). We have made a particular study of the value of estimating the carbon dioxide tension and oxygen saturation of the arterial blood in the diagnosis, prognosis, and management of patients with chronic lung disease. This paper presents the results of observations made on such patients over a period of five years.

METHODS

Patients were considered to be suffering from heart failure when they had pitting peripheral oedema and the jugular venous pressure was elevated in all phases of respiration. All the patients were at rest when the specimens of blood were taken. The blood was obtained from the brachial artery and stored at room temperature in a stoppered syringe which had been rinsed with a solution of heparin.

The *p*H of the whole blood was measured within 10 minutes of withdrawal using a Marconi *p*H meter (type T.F. 511D) and a glass electrode. The blood was introduced anaerobically into a Stadie electrode vessel of 1.5 ml. capacity surrounded by a water jacket at room temperature. The *p*H of the blood was compared with that of standard buffer solutions and a temperature correction of -0.014 *p*H unit per degree below 38° C. was applied (Rosenthal, 1948). The carbon dioxide and oxygen saturation of the blood were measured by the manometric technique of Van Slyke and Neill (1924). The haematocrit was measured after centrifugation at 3,000 r.p.m. for 30 minutes. The carbon dioxide tension and bicarbonate content of the plasma were then obtained from the observed data using the nomogram of Singer and Hastings (1948).

RESULTS

We have studied 77 patients with chronic pulmonary disease who had not developed heart failure or been treated with diuretics. Sixty-eight of them were suffering from chronic bronchitis and emphysema; the others were asthmatic or had pulmonary fibrosis or bronchiectasis. They have been observed for periods varying from one to 60 months (average 21.2 months). In many instances the arterial blood has been analysed several times and the gas content varied considerably. For the purpose of comparing different patients in the diagrams those results have been used which showed the highest carbon dioxide tension recorded in each individual. In 58 of these 77 patients the maximum recorded carbon dioxide tension was less than 55 mm. Hg. Many of the 19 patients who were found to have higher carbon dioxide tensions presented unusual clinical features. Seven of them subsequently developed heart failure (Fig. 1). One was suffering from one of the most severe bronchiolar infections encountered in the whole series and two other patients died three days after the investigation. One patient was comatose with depressed respiration after 3 grains (180 mg.) of butobarbitone; her carbon dioxide tension had fallen to 46 mm. Hg two weeks later. One patient with status asthmaticus also had normal blood gases one week later. The carbon dioxide tension in the arterial blood of two other patients with severe bronchospasm was less than 50 mm. Hg from one to two years later. The remaining five patients have maintained a high carbon dioxide tension in the arterial blood but have not, so far, developed heart failure. Only five of the 58 patients in whom the carbon dioxide tension was never found to be more than 55 mm. Hg have developed heart failure. These results show that a high tension of carbon dioxide in the arterial blood of a patient with chronic pulmonary disease may be a transient phenomenon associated with severe bronchospasm or pulmonary infection. If the condition persists, the patient is liable to develop heart failure.

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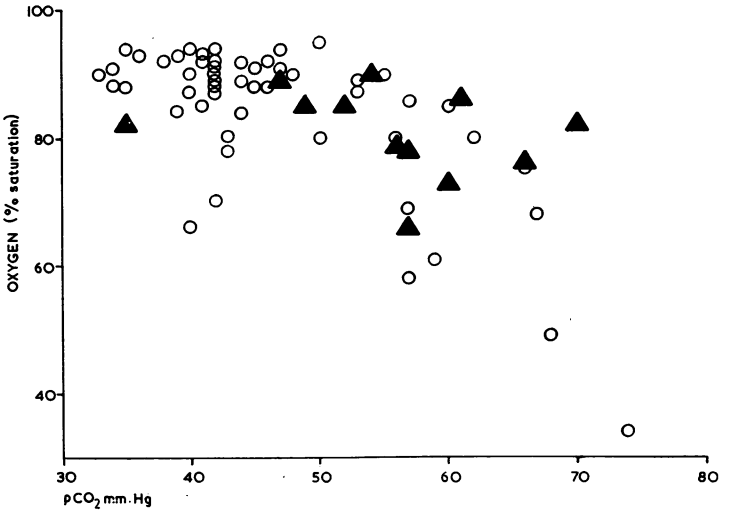

FIG. 1.—Arterial blood gases of patients with chronic pulmonary disease who had not suffered from heart failure. O Patients with no history of heart failure. ▲ Patients who subsequently developed heart failure.

Eleven patients were observed before they developed heart failure and again during their first attack (Table I). With the onset of heart failure the oxygen saturation of the arterial blood invariably fell, but the changes in the carbon dioxide tension were less consistent; only three of the 11 patients showed a rise of more than 5 mm. Hg.

Most patients with pulmonary heart failure are more anoxic and hypercapnic than patients with similar pulmonary disease who have not suffered from congestive cardiac failure (Fig. 2). Eighty-five per cent. of the patients with no history of pulmonary heart failure had arterial blood which was more than 70% saturated with oxygen and in three quarters the carbon dioxide tension was less than 55 mm. Hg. On the other hand, two-thirds of the 36 patients with untreated pulmonary heart failure had arterial blood which was less than 70% saturated with oxygen and in 89% the carbon dioxide tension was 55 mm. Hg or more.

TABLE I

ARTERIAL BLOOD GASES BEFORE AND DURING FIRST ATTACK OF PULMONARY HEART FAILURE

Patient No.	Date	pCO_2 (mm. Hg) A	pCO_2 (mm. Hg) B	O_2 % Saturation A	O_2 % Saturation B
1	2.3.54 (Sept. '54)	35		82	
	10.11.54		17*		—
2	29.3.55 (5.4.55)	47		89	
	11.4.55		47		88
3	16.10.51 (17.1.53)	—		82	
	26.1.53		57		77
4	13.11.50 (11.2.52)	—		82	
	5.4.52		62		62
5	20.9.54 (Feb. '55)	49		—	
	16.2.55		69		77
6	31.3.52 (14.2.55)	51		90	
	14.2.55		65		63
7	23.2.55 (Jan. '56)	56		79	
	27.1.56		53		57
8	1.2.55 (11.2.55)	60		73	
	16.2.55		65		41
9	6.3.56 (Jan. '57)	61		86	
	18.1.57		55		70
10	6.6.56 (Jan. '57)	66		76	
	14.1.57		66		70
11	1.2.55 (Jan. '56)	57		78	
	9.2.56		64		65

A = Before onset of congestive failure.
B = During first attack of congestive failure.
Dates in brackets are those on which oedema first developed.
* At the time this sample was taken, there was gross hyperventilation due to pulmonary artery thrombosis.

The difference between the arterial blood gases in the two groups of patients is sufficiently consistent to provide a basis for differential diagnosis in heart failure. Respiratory acidosis is peculiar to patients with pulmonary heart failure and is not found in other forms of cardiac failure (Campbell and Poulton, 1920; Fraser, 1927; Barach and Richards, 1931; Platts, 1953; Platts and Whitaker, 1954). Although the diagnosis of pulmonary heart failure is not usually difficult, cyanosed patients are occasionally encountered in whom it is difficult to determine, on clinical grounds, whether they are suffering from pulmonary heart failure or congenital heart disease. Two such patients were seen in this series.

The first patient was a man who presented with congestive cardiac failure, central cyanosis, and a loud parasternal systolic murmur. The right ventricle was enlarged and he had signs of bronchitis and emphysema. It was doubtful whether he was suffering from pulmonary heart failure or a right-to-left shunt of blood through a patent ventricular septum. However, the carbon dioxide tension in the arterial blood was 61 mm. Hg. The conclusion that he was suffering from cor pulmonale was subsequently confirmed by post-mortem examination.

The second patient was a woman aged 55 years with a long history of productive cough and breathlessness. She had congestive cardiac failure with central cyanosis and right-sided cardiac hypertrophy. As she was not hypertensive and had no cardiac murmurs, she was first

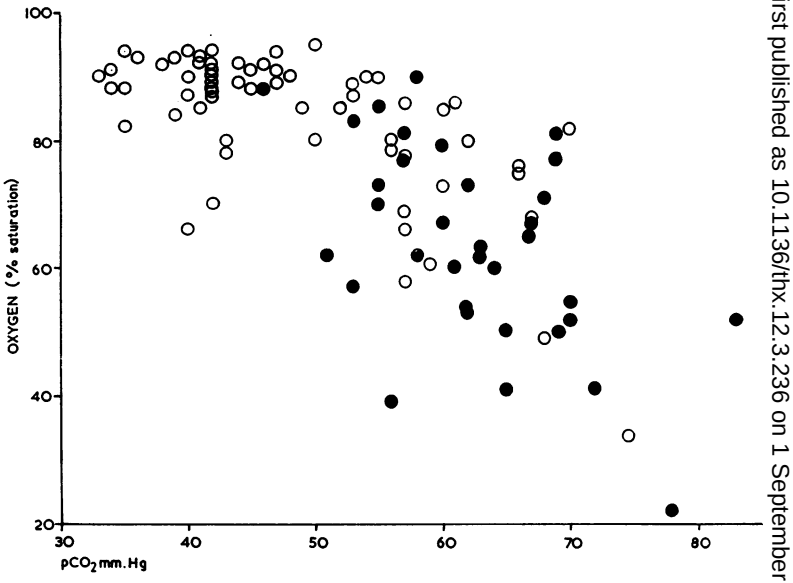

FIG. 2.—Arterial blood gases of patients with chronic pulmonary disease. O Patients with no history of pulmonary heart failure. ● Patients with untreated pulmonary heart failure.

considered to be suffering from pulmonary heart failure. However, the carbon dioxide content of the blood was only 45.4 vol. %, a value which is incompatible with a high carbon dioxide tension unless a gross metabolic acidosis coexists. At necropsy she was found to have a widely patent ductus arteriosus.

Clinical observation shows that certain patients with pulmonary heart failure will lose their venous congestion and oedema when treated with antibiotics and bronchodilator drugs alone, but more severely affected patients remain oedematous in spite of such treatment and may die unless they also receive mercurial diuretics. Again, some patients, after recovery from heart failure, may be kept free from oedema simply by moderate restriction of their salt intake. Others rapidly become oedematous again unless they receive diuretics regularly.

We have attempted to correlate the blood gas findings with the patients' need for diuretic treatment (Fig. 3). Thirty-six patients were examined at the onset of their first attack of pulmonary heart failure before any diuretics had been given. Thirteen of these patients did not require diuretics. Eight of them had arterial blood with a carbon dioxide tension under 60 mm. Hg at the beginning of the attack of failure. Of the 23 patients who received prolonged treatment with mercurial diuretics, only four had a carbon dioxide tension below 60 mm. Hg. Thus at the onset of an attack of pulmonary heart failure, those patients who did not require diuretics had less disturbance of the arterial blood gases than those who subsequently needed intensive and prolonged treatment with these drugs.

The severity of anoxia and hypercapnia closely paralleled the mortality in our different groups of patients (Table II). The values for the oxygen saturation of blood given in Table II are those of the same specimens for which the figures for carbon dioxide

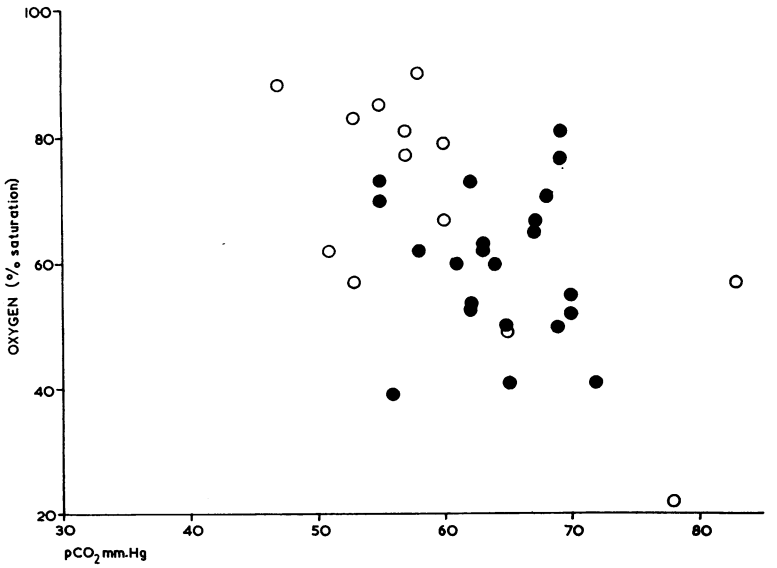

FIG. 3.—Arterial blood gases of patients with pulmonary heart failure. O Patients who recovered without treatment with diuretics. ● Patients who received prolonged treatment with diuretics.

TABLE II
MORTALITY OF PATIENTS WITH CHRONIC PULMONARY DISEASE

	30–44 mm. Hg	45–54 mm. Hg	55–64 mm. Hg	More than 64 mm. Hg
Highest tension of carbon dioxide recorded in each patient:				
Total number of patients ..	38	24	31	20
Average period of observation (months)	21·2	19·8	17·3	17·7
Percentage of patients dead..	11	13	42	55
Average interval between blood analysis and death (months)	5·5	18·7	6·2	15·2
	100–80%	79–60%	59–40%	Less than 40%
Oxygen saturation of the same specimen of blood for which carbon dioxide tensions are given:				
Total number of patients ..	65	28	13	3
Average period of observation (months)	22·2	15·9	14·5	20
Percentage of patients dead..	14	39	69	33
Average intervals between blood analysis and death (months)	12·2	5·8	13·3	1

tension are quoted in the rest of this paper. They are therefore not necessarily the lowest oxygen saturation recorded in any particular patient and the blood was not always taken at the time when the patient appeared most cyanosed. A particularly high mortality was encountered in patients whose arterial blood was at any time found to be less than 50% saturated with oxygen. This occurred in 20 patients, all of whom had suffered from pulmonary heart failure. Eighteen of these died, eight within one month of the estimation. However, some survived for as long as two years though the oxygen saturation of the arterial blood exceeded 50% for most of this time. These were patients from whom a specimen of blood had been taken during an acute, severe pulmonary infection.

DISCUSSION

West and others (1951) did not find a close correlation between the severity of emphysema as judged by the lung capacity, the volume of residual air, or the efficiency of intrapulmonary mixing of gas, and the development of pulmonary heart failure. Bates, Knott, and Christie (1956) found that the only measurement of pulmonary function which was considerably more abnormal in patients with emphysema who died within the ensuing six months than in the less severely affected patients was the carbon monoxide diffusing capacity in the lungs. No series of such measurements in emphysematous patients with pulmonary heart failure of varying severity has been published.

While observing many patients with chronic bronchitis and emphysema, we have learnt to recognize the serious import of a high or rising carbon dioxide tension in the arterial blood. We have found this a more useful sign of deterioration than other tests of pulmonary function. We have correctly predicted the onset of cardiac failure in several of our patients on the basis of a persistently high carbon dioxide tension in the arterial blood. Others are under close supervision because this complication appears imminent. In this way we hope to prevent the development of heart failure by treatment of pulmonary infection or to detect and treat heart failure at an early stage.

There is sometimes doubt whether a patient will remain free from oedema once diuretic treatment has been stopped, and a number of patients may receive unnecessary injections of mersalyl because of this. We have ceased giving diuretics to some patients in whom the carbon dioxide tension of the arterial blood had fallen below 50 mm. Hg, and found that they remained free from oedema for many months. In those patients with very high carbon dioxide tensions, on the other hand, heart failure often recurred in spite of further treatment with diuretics.

An abnormally high carbon dioxide tension in the arterial blood indicates that pulmonary ventilation is impaired. It is not known why patients with this abnormality are liable to develop heart failure. However, experiments on animals and man have shown that anoxia or hypercapnia raises the pulmonary artery pressure (Binet and Bourlière, 1941; von Euler and Liljestrand, 1946; Motley, Cournand, Werko, Himmelstein, and Dresdale, 1947; Westcott, Fowler, Scott, Hauenstein, and McGuire, 1951). A linear relation has also been demonstrated between the carbon dioxide tension or the arterial oxygen desaturation and the pulmonary artery pressure in patients with pulmonary heart failure (Borden, Wilson, Ebert, and Wells, 1950; Harvey, Ferrer, Richards, and Cournand, 1951; Yu, Lovejoy, Joos, Nye, and McCann, 1953; Whitaker, 1954). Since it seems probable that pulmonary hypertension is an important cause of the right ventricular hypertrophy which characterizes pulmonary heart failure, the abnormalities in the arterial blood gases may be a direct precipitating factor in pulmonary heart failure. The retention of salt and water which must occur before peripheral oedema becomes apparent takes several days. If the patient should die or recover from the pulmonary infection in a shorter time, the signs of cardiac failure may not develop in spite of a temporary alteration of the arterial blood

gases to levels characteristic of pulmonary heart failure.

The development of heart failure causes an increase in anoxia, presumably because pulmonary oedema hinders the diffusion of oxygen through the alveolar membrane. Since carbon dioxide is much more soluble than oxygen, its passage through the alveolar membrane is not greatly impeded by pulmonary oedema and therefore the carbon dioxide tension in the arterial blood is usually not greatly affected by the onset of pulmonary heart failure. The increased anoxia caused by pulmonary oedema is a potential cause of a further increase in pulmonary blood pressure and aggravation of heart failure. Thus a vicious circle may develop and it is well known that untreated patients with pulmonary heart failure usually deteriorate rapidly. Relief of anoxia and carbon dioxide retention, either by treatment of the pulmonary infection or by abolition of pulmonary oedema with diuretics, usually reverses this process.

Pulmonary heart failure occasionally develops in patients with no carbon dioxide retention. Patient 1 (Table I) was the only instance encountered in this series. He was suffering from pulmonary fibrosis due to sarcoidosis and had the physical signs of severe pulmonary hypertension. Similar patients have been described elsewhere (Austrian, McClement, Renzetti, Donald, Riley, and Cournand, 1951; Stone, Schwartz, and Green, 1956). Their pulmonary hypertension is believed to result from the obliteration of the pulmonary vasculature by fibrosis.

SUMMARY

The carbon dioxide tension and oxygen saturation of the arterial blood from a series of patients with chronic pulmonary disease have been measured.

In 85% of the patients who had not suffered from pulmonary heart failure the arterial blood was more than 70% saturated with oxygen. The blood of 76% of these patients also had a carbon dioxide tension under 55 mm. Hg.

Two-thirds of patients with untreated pulmonary heart failure had an arterial oxygen saturation of less than 70% and the great majority of these patients had an arterial carbon dioxide tension of more than 54 mm. Hg.

Fifty per cent. of patients who had a carbon dioxide tension persistently over 54 mm. Hg and had not suffered from pulmonary heart failure developed this condition during the following year.

Patients whose arterial blood was more than 80% saturated with oxygen or had a carbon dioxide tension of less than 55 mm. Hg at the onset of an attack of pulmonary heart failure usually recovered without treatment with mercurial diuretics.

Forty-four per cent. of patients whose arterial blood was less than 50% saturated with oxygen died within one month.

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