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Arterial blood gases during treatment of congestive heart failure

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ARTERIAL BLOOD GASES DURING TREATMENT OF CONGESTIVE HEART FAILURE

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The congestive syndromes of heart failure are classified as 1) acute pulmonary edema (acute left ventricular failure) and 2) chronic heart failure in Principles of Internal Medicine edited by Harrison, et al[11]. Acute left ventricular failure is characterized by paroxysmal dyspnea. It may be caused by an elevated filling load (most common), acute myocardial infarction, ectopic tachycardia or fulminant myocarditis--the first causing a high-output syndrome with cardiogenic shock occurring late; the remaining causes characterized by an abrupt decline in cardiac output. Diagnosis is made by the presence of pulmonary edema, its signs and symptoms, and the signs of cardiac disease.[11]

Harrison, et al[11], define chronic heart failure "as a syndrome induced by disease of the heart and characterized by long-standing engorgement in the pulmonary and/or systemic vascular beds." This type of heart failure usually involves both sides of the heart, ie, persistent elevation of pulmonary pressures due to left sided failure leads to right sided failure. Right sided failure alone is rare, and most disorders of the heart involve the left side of the heart first. Episodes of acute pulmonary edema are often found superimposed upon chronic heart failure.

Chronic heart failure can be further divided into latent heart failure, and overt heart failure. These are characterized as follows: 1) latent type--undue dyspnea and fatigue present only with effort; and 2) overt type--(failure at rest) usually follows the latent type after a variable time period with the signs of left sided failure present at rest. Without treatment right sided failure soon develops, along with its well recognized signs and symptoms.[11] An excellent review of the pathophysiology of pulmonary edema is available in the American Physiological Society's Handbook of Physiology[10], and has more recently been reviewed by Hultgren and Flamm[12]. Further discussion of congestive heart failure may be obtained from such textbooks as Friedberg[9], or the American Journal of Cardiology[3].

According to Comroe, et al[4], the function of ventilation, diffusion and blood flow is the maintenance of normal partial pressures of oxygen and carbon dioxide in alveolar gas and arterial blood. Measurement of arterial blood gases will determine how adequately ventilation, diffusion and blood flow have carried out their job. Early investigators[7,8,14] found normal arterial oxygen saturation and pH, with decreased carbon dioxide tension in patients with congestive heart failure. Pulmonary function studies have revealed decreased vital capacities in patients with cardiac failure[6,15,16]. In 1954, Vitale, Dumke, and Comroe[21], reported finding little

correlation between diffuseness of rales and arterial oxygen saturation in patients with congestive heart failure. In some of their patients with the most diffuse rales, the arterial oxygen saturation was found to be greater than 93 per cent.

Oxygen and blood combine in two ways: 1)physical solution and 2) chemical combination with hemoglobin. The amount of oxygen in the blood depends on the pressure of oxygen to which the blood is exposed. The amount of oxygen dissolved in physical solution is directly proportional to the partial pressure in the plasma, no matter how low or high the pressure. The amount of oxygen combined with hemoglobin depends on the partial pressure, but is not a linear relationship. This is shown by the oxygen-hemoglobin dissociation curve.[4]

An adequate pressure of oxygen is necessary for the loading of oxygen onto hemoglobin in the lungs and for the diffusion of oxygen from capillaries to the cells. In studying blood gases certain advantages can be obtained by measuring PO_2 (oxygen tension), as it is a more sensitive test due to the shape of the oxygen-hemoglobin dissociation curve. Normal values are found on page 145 of The Lung[4].

Carbon dioxide is produced by metabolism in the tissues and diffuses into the blood. Some reacts with water to form carbonic acid. However, most of it is carried in the blood as dissolved carbon dioxide. pH and pCO_2 (carbon dioxide tension)

can be measured by electrodes and are related by the Henderson-Hasselbach equation. Elimination of carbon dioxide is important in maintaining normal acid-base balance and therefore arterial blood pH should be measured whenever interpretation of pCO_2 is done.[4]

In 1965, Saunders[18], reported finding an increased alveolar-arterial oxygen gradient over normal in patients with congestive heart failure. He presented supporting evidence to show this to be due to shunting of blood. In [illegible] blood gas studies of 1966, Valentine, et al[20], reported finding a significant reduction in arterial oxygen tension and an increased alveolar-arterial oxygen gradient was observed in acute myocardial infarction. It took three to four weeks for these changes to revert to normal after the acute infarction. There has been no report in the literature of blood gases during treatment of congestive heart failure. This study is the first to follow arterial blood gases in patients with congestive heart failure as they are treated.

Methods

Patient Selection: All patients were studied while hospitalized in the University of Nebraska Hospital or the Douglas County Hospital between May and October, 1968. Patients were selected on the following criteria: signs and symptoms of congestive heart failure, absence of adequate previous treatment and

absence of an underlying disease process which is known to modify blood gases. All patients were examined by the author prior to admitting them to the study. All patients had x-ray evidence of cardiomegaly and pulmonary congestion. One patient was on digitalis prior to the study. One patient had chronic lung disease.

Treatment: All patients received digitalizing doses as calculated by the house staff. The use of diuretics is noted in the accompanying chart. Blood gases were drawn prior to the initiation of treatment, when one-half the digitalizing dose had been administered, at full digitalization and before being discharged from the hospital.

Blood Sampling: Blood was collected in the following manner:

1) Patency of radial and ulnar arteries was determined by Allen's Test[5], and respiratory rate was counted and recorded.

2) The skin surrounding the puncture site was cleansed with an antiseptic solution.

3) The skin and subcutaneous tissue at the puncture site was injected with 1% xylocaine.

4) Radial artery puncture was performed using anaerobic technique with a 21 gauge needle and heparinized syringe. The sample was placed in ice, and blood gases determined within 30 minutes of collection.

5)Pressure was held over the puncture site for 5 minutes.

Patient	1	2	3	4	5**	6	7
PaO_2 (mm Hg)							
Time 0	61	64	42	58	60	60	45
Time 1	64	60	56	60	--	--*	--*
Time 2	72	66	72	51	--	--	--
Time 3	93	72	84	72	84	--	--
$PaCO_2$ (mm Hg)							
Time 0	29	30	29	44	30	19	7
Time 1	29	28	33	44	--	--*	--*
Time 2	39	42	35	46	--	--	--
Time 3	35	40	44	47	32	--	--
pH							
Time 0	7.34	7.40	7.45	7.37	7.42	7.20	7.05
Time 1	7.34	7.37	7.44	7.40	----	----*	----*
Time 2	7.41	7.36	7.43	7.39	----	----	----
Time 3	7.40	7.38	7.42	7.40	7.40	----	----
Respiratory Rate							
Time 0	34	36	32	40	28	38	41
Time 1	32	30	29	25	--	--*	--*
Time 2	24	18	27	18	--	--	--
Time 3	20	18	27	22	26	--	--
Age	75	76	78	52	52	67	59
Etiology	ASHD	ASHD	ASHD	unk	hyper-tension	unk	unk

Figure 1: Blood gases during treatment of Congestive heart failure. PaO_2=arterial oxygen tension.; $PaCO_2$= arterial carbon dioxide tension.; *=patient expired; **=patient digitalized when admitted to the hospital.; Unk=unknown; ASHD=arteriosclerotic heart disease.; Time 0=prior to institution of therapy.; Time 1= one-half the digitalizing dose given.; Time 2=full digitalization.; Time 3= prior to being discharged from the hospital.; Patient 4 had FEV_1= 1.4 L. and Peak Flow=39% predicted after treatment. He was cardioverted for atrial fibrillation between times 0 and 1.

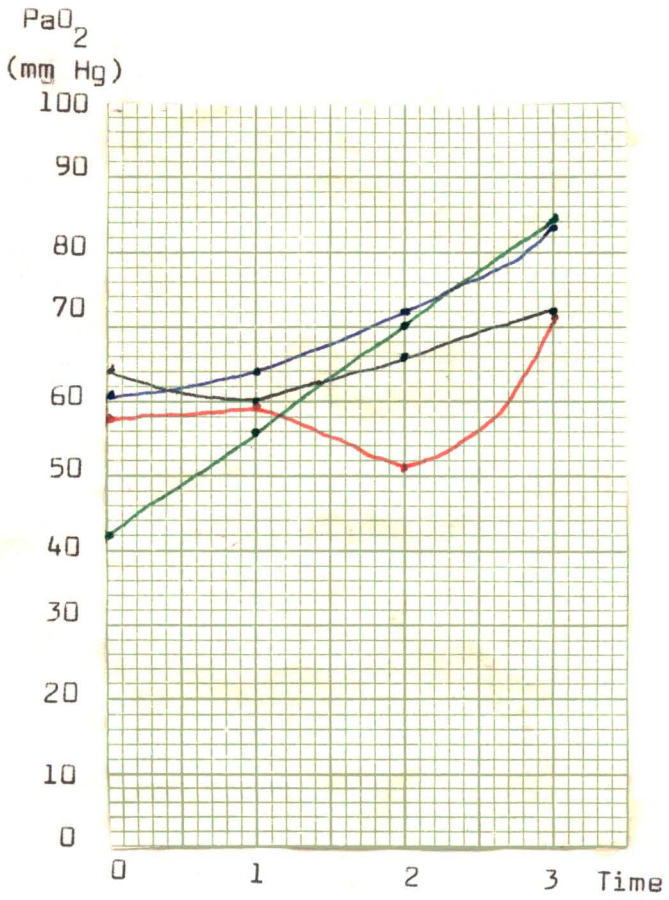

Figure 2: Arterial oxygen tensions in four patients as they are treated for congestive heart failure. Time 0, 1, 2, 3 as in figure 1. Patient 4 has chronic lung disease. Patients numbered as in figure 1.

Patient 1 ——
Patient 2 ——
Patient 3 ——
Patient 4 ——

Blood Gas Analysis: No patient received oxygen in the thirty minute period prior to collection of the blood sample. All determinations were made within thirty minutes of blood sampling. The electrode blood-gas apparatus of the Anesthesia department was used to make the determinations. Before each sample was run, the apparatus was standardized with gases of known composition and with a standard pH solution. All procedures were performed by the author. Excellent reviews of methods, apparatus and error are available[1,19].

Etiology: The etiology included in figure 1 is that given on the chart after the patient had been worked-up on the Medicine service.

Results

PaO_2: (See Figures 1 and 2) All patients had initial low values for PaO_2, including those who expired. Improvement was noted in three of the four patients in figure 2, at full digitalization. Improvement was also noted in all five surviving patients at the time of discharge from the hospital.

$PaCO_2$: All patients, except number 4 (who had lung disease), had low $PaCO_2$ prior to treatment. With treatment the CO_2 tension became more normal. The very low CO_2 tensions observed in the two patients who died are striking.

pH: Deviations from normal were not marked except for the markedly acidotic state of the two patients who died.

Respiration: An increased rate of respiration was noted in all patients. It changed to a more normal level as treatment was instituted.

Arterial Puncture: No complications occurred in the twenty arterial punctures performed during this study.

Discussion

Fraser, et al[8], reported low CO_2 tensions in patients with congestive heart failure. The results of this study are in agreement with this. Fraser[7] also reported that arterial oxygen saturation and pH were normal in the congestive syndromes. The results here show a hypoxemia in untreated congestive heart failure and point to the need for supplemental oxygen in the therapy of congestive heart failure. The difference in part is in the inherent properties of the oxygen-hemoglobin dissociation curve, which gives more accuracy to the measurement of arterial oxygen tensions.

The observed decrease in oxygen tension could be due to either shunting of blood or to diminished oxygenation in the lungs. Saunders and Contab[18] have reported evidence that this is due to shunting of blood in the lungs. Peabody and Wentworth[15] showed a decrease in the vital capacity of the lungs in congestive failure. By monitoring PaO_2 and determining diffusion rates in acute pulmonary edema induced in anesthetized dogs, it has been shown that the decreased oxygen tension that develops in these animals cannot be

explained by the decrease in vital capacity alone, but that it is due to a shunting effect, ie, perfusion of non-ventilated alveolar units[17]. All PaO_2 values improved with treatment in surviving patients, suggesting that blood gases may be used to assess effectiveness of therapy in heart failure.

The decreased $PaCO_2$ in these patients can only be obtained by hyperventilation. Only in the patient with lung disease was $PaCO_2$ at normal levels. The high initial respiratory rate which declined with therapy, and the associated rise in CO_2 with therapy confirms this. These patients seem to be in a state of metabolic acidosis compensated by a respiratory alkalosis prior to treatment.

The patients who died showed a marked acidosis and very low $PaCO_2$ levels. One should consider a blood gas status such as this before administering drugs (such as morphine) which might depress the respiration. An increase in CO_2 would lead to further acidosis in these patients.

The low $PaCO_2$ levels observed in these patients without pulmonary disease suggests that one can differentiate congestive heart failure from respiratory failure on this basis. High levels of CO_2 have been reported[2], however, in four patients with near terminal pulmonary edema. This was not observed in the near terminal patients of this study.

Summary

Arterial blood gases were followed in seven patients as they were treated for congestive heart failure. All patients had a decreased arterial oxygen tension prior to the institution of therapy. The arterial oxygen tension improved in all surviving patients with treatment. Arterial CO_2 tensions were less than normal in six of the seven patients prior to treatment. It returned to normal levels as the respiratory rate decreased with treatment. The two patients who expired had very low pH values and $PaCO_2$ values. This should be kept in mind when treating near terminal congestive heart failure. The low CO_2 tensions observed in these patients suggest that arterial blood gases may be useful in differentiating congestive heart failure and respiratory failure.

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