Ventilation/Perfusion Changes During Mechanical Ventilation*

Jerome H. Modell, M.D., F.C.C.P.

Changes in arterial blood gases occurring during mechanical ventilation of a patient with extensive pulmonary disease have been presented. The \( P_{A}O_2 \) and \( A-aDO_2 \) values observed in this patient demonstrate that various areas of \( V_A/Q \) imbalance exist simultaneously in the diseased lung. By altering the tidal volume and the pressure transmitted to the pulmonary capillary bed, the proportion of various types of \( V_A/Q \) match or mismatch can be changed almost instantaneously. These findings suggest that single determinations of arterial oxygen tension cannot be used to predict what will happen from moment to moment nor at changing patterns of ventilation. Considerable thought and effort should be given to the feasibility of continuous monitoring and recording of arterial oxygen tension.

**INTRODUCTION**

During the past decade considerable progress has been made in the treatment of patients requiring prolonged mechanical ventilation. While innovations in equipment have contributed, improved therapy has resulted primarily from study of both normal and abnormal lungs. Studies of distribution of ventilation and its relationship to perfusion (\( V_A/Q \)) have shown that the normal lung is not a homogenous unit, but rather, many combinations of \( V_A/Q \) can be present simultaneously.\(^1\)\(^-\)\(^3\) A case report is presented which suggests, not only that the diseased lung contains many combinations of ventilation-perfusion relationships, but that the predominant \( V_A/Q \) ratio can be changed by the pattern of ventilation supplied.

**CASE REPORT**

A 31-year-old Negro man, with a history of idiopathic grand mal epilepsy, convulsed while fishing and fell into a salt water bay. When it became apparent to his companions that he was unconscious, they retrieved him and gave him mouth-to-mouth ventilation. On arrival at the hospital he was breathing spontaneously, but was extremely dyspneic, disoriented and agitated. On physical examination diffuse rales and rhonchi were heard throughout the chest. There was flaring of the nostrils, grunting respiration, and pink frothy fluid was present in his airway. Vital signs were: blood pressure 180/70, heart rate 108 per minute and irregular, respiratory rate 60 per minute. Nasotracheal intubation was performed and the patient's ventilation was assisted with a pressure limited ventilator delivering 100 percent oxygen (\( F_{i}O_2 = 1.0 \)).

Venous blood was drawn for determination of hemoglobin, hematocrit and serum electrolyte concentrations: these were Hgb 13.6 gm per 100 ml, Hct 43 vol percent, Cl 117 mEq/L, Na 160 mEq/L, K 4.0 mEq/L. An intravenous infusion of 5 percent dextrose in water was started and the patient was treated with increments of sodium bicarbonate, totaling 495 mEq, to correct his metabolic acidosis. Sodium cephalothin and steroids were administered intravenously. He was transferred to the Intensive Care Unit (ICU) where central venous pressure, intra-arterial pressure and ECG were monitored. A sinus tachycardia with occasional premature ventricular contractions was noted. An x-ray film of the chest showed diffuse hazy infiltrates in both lung fields (Fig 1).

The patient was curarized and ventilation was controlled with a volume limited ventilator at a tidal volume of 1,000 ml, rate of 15 per minute and \( F_{i}O_2 \) of 1.0. Periodic deep

*Alveolar-arterial oxygen tension difference.
Ten hours postaspiration his PaO₂ had increased to 80 and 120 mm Hg. The FIO₂ was decreased to maintain the PaO₂ between 80 and 120 mm Hg. The patient's sensorium improved and he could carry out a conversation by writing on a memo pad. Specimens taken through the endotracheal tube and urinary catheter for culture showed no growth. Intravenous antibiotics were: oxacillin and nystatin were started. His temperature decreased within an hour and by the following morning it was maintained at less than 103°F (39.4°C) without external cooling. Daily cultures continued to show Pseudomonas aeruginosa. Sodium colistimethate, sodium oxacillin and nystatin were started. His temperature decreased within an hour and by the following morning it was maintained at less than 103°F (39.4°C) without external cooling. Daily cultures continued to show Pseudomonas aeruginosa in spite of antibiotic therapy. The patient’s compliance progressively decreased, but his arterial oxygenation could be maintained within acceptable limits by mechanical ventilation with 60 percent inspired oxygen, until the tenth hospital day when he became extremely apprehensive and expressed both the fear and desire to die. The FIO₂ was increased to 1.0 and blood gases obtained: pH 7.43, PaO₂ 35 mm Hg, PaCO₂ 49 mm Hg. Chest x-ray taken at this time showed almost complete obliteration of both lung fields (Fig 2). Ventilation was attempted by hand with a nonrebreathing valve and reservoir bag without improvement. One hour later he had a grand mal seizure following which circulatory arrest occurred. Resuscitation attempts were started immediately by physicians in attendance. After one hour, resuscitation measures were discontinued and the patient was pronounced dead.

Abnormal gross autopsy findings were limited to the lungs

**All values reported for FIO₂ were measured at the inspiratory port of the endotracheal tube with a paramagnetic oxygen analyzer.**

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**FIGURE 1. X-ray film of the chest one hour after sea water aspiration.**

Blood gas values showed him to be severely hypoxic: PaO₂ 35 mm Hg, A-aDO₂ 640 mm Hg, pH 7.44, PaCO₂ 41 mm Hg. Mechanical ventilation was reinstituted and inspired oxygen tension adjusted to maintain an acceptable PaO₂.

One hundred four hours postaspiration, 82 percent inspired oxygen concentration was required to maintain a PaO₂ of 68 mm Hg (pH 7.38, PaCO₂ 48 mm Hg). The tidal volume was increased from 1,000 ml to 1,200 ml and the rate decreased from 20 to 16 per minute; FIO₂ increased minimally from 0.82 to 0.86. This produced a marked increase in arterial oxygen tension, PaO₂ 205 mm Hg, pH 7.43, PaCO₂ 41 mm Hg. Subsequently, ventilation was either controlled or assisted at a tidal volume of 1,200 ml, with an FIO₂ of 0.80 able to maintain a normal PaO₂.

On the fifth day of hospitalization the patient's rectal temperature which had been between 100 and 101°F (37.8 and 38.3°C), spiked acutely to 105°F (40.6°C). He was wrapped in an external cooling blanket and was given curare to stop shivering. The tracheal aspirates obtained on the third hospital day were reported at this time to be positive for Pseudomonas aeruginosa. Sodium colistimethate, sodium oxacillin and nystatin were started. His temperature decreased within an hour and by the following morning it was maintained at less than 103°F (39.4°C) without external cooling. Daily cultures continued to show Pseudomonas aeruginosa in spite of antibiotic therapy. The patient’s compliance progressively decreased, but his arterial oxygenation could be maintained within acceptable limits by mechanical ventilation with 60 percent inspired oxygen, until the tenth hospital day when he became extremely apprehensive and expressed both the fear and desire to die. The FIO₂ was increased to 1.0 and blood gases obtained: pH 7.43, PaO₂ 35 mm Hg, PaCO₂ 49 mm Hg. Chest x-ray taken at this time showed almost complete obliteration of both lung fields (Fig 2). Ventilation was attempted by hand with a nonrebreathing valve and reservoir bag without improvement. One hour later he had a grand mal seizure following which circulatory arrest occurred. Resuscitation attempts were started immediately by physicians in attendance. After one hour, resuscitation measures were discontinued and the patient was pronounced dead.

Abnormal gross autopsy findings were limited to the lungs

**FIGURE 2. X-ray film of the chest 10 days following sea water aspiration.**
and the brain. The brain showed evidence of bilateral golden yellow cystic degeneration consistent with old contusion injury of the anterior half of both mediofrontal gyri. There was also marked cortical cystic degeneration consistent with old contused areas over the inferior surface of the right temporal lobe. The right lung weighed 1,120 gm and the left 840 gm. There was approximately 1,000 ml of cloudy sanguineous fluid and yellow fibrinous adhesions of the parietal and visceral pleurae in the left hemithorax. The right lung showed evidence of widely diffuse nodular red hepatization while the left lung showed diffuse nodular red and gray hepatization. A small area of bullous emphysema was noted in the upper lobe of the right lung. Scattered areas of 1 to 2 cm of cloudy pinkish-red abscess cavities were present in the parenchyma of the left lower lobe. The superior segment of the lobe showed an area of infarction due to thromboemboli measuring approximately three inches in diameter. The remainder of the pulmonary vasculature appeared patent. The lower trachea and both bronchi down to the segmental branches were congested and contained cloudy pinkish-red purulent fluid. The terminal segmental branches were grossly occupied by cloudy, yellowish purulent material. A representative microscopic section from the right lung is shown in Figure 3.

The larynx and upper tracheal mucus were normal in appearance. Representative blocks of tissue were taken from each lung for determination of pulmonary surfactant activity after extraction by the mincing method. The surface tension activity of the extract was measured on a modified Wilhelmy balance as the surface area was alternately compressed to 20 sq cm and expanded to 100 sq cm at a rate of one cycle every 90 seconds. The values for surface tension at minimum and maximum surface area were 21/46 dynes per cm for the left lung, and 20/48 dynes per cm for the right lung.

**DISCUSSION**

This patient's death was attributed to secondary pulmonary infection complicating treatment of a near-drowning episode. The factors contributing to the hypoxia seen after near-drowning and its treatment, as well as the significance of infection with *Pseudomonas aeruginosa*, have recently been reviewed, and will not be discussed here further.

Frequent monitoring of arterial blood gases in this patient (72 determinations in all) demonstrated considerable fluctuation of arterial oxygen tension. The frequency of changes in PaO₂ and their relation to changes in tidal volume suggest that a variety of lung elements with various combinations of ventilation and perfusion coexisted in this patient's lungs. The type of relationship between alveolus and capillary, or ventilation and perfusion, which predominated at any given time was apparently influenced by the pattern of ventilation. For purposes of simplification, these Vₑ/Qₑ relationships will be discussed in terms of the three types of alveoli (ventilation) and four types of capillary (perfusion) diagrammed in Figure 4. By combining the various patterns of ventilation with the types of perfusion diagrammed, 12 combinations for Vₑ/Qₑ are possible. (A fourth alveolus; ie, one that is ventilated, but has a diffusion deficit or block, is also possible. Since this can mimic alveolus b or c, depending upon the extent of the block, it will not be considered separately).

On admission to the hospital, this patient's arterial oxygen tension was only 27 mm Hg. Since 100 percent oxygen breathing tends to obscure the contribution to intrapulmonary shunting by hypventilated alveoli (type c), we can assume that his lung contained significant numbers of nonventilated, but perfused alveoli (type b1, b3, or b4). Whether these alveoli were nonventilated because of physical collapse or whether they were fluid-filled, the effect on gas exchange would be similar. Experimental evidence in animals suggest that fluid-filled, but perfused alveoli are the etiology of the hypoxia seen immediately after sea water aspiration.
During the following 36 hours his arterial P02 gradually increased permitting a reduction in the inspired oxygen concentration necessary for oxygenation of arterial blood. This could occur if either the nonventilated alveoli had decreased in quantity or perfusion to these areas had diminished (eg, combinations of b2 or b3 predominating over b1 or b4). This shift in perfusion away from nonventilated areas has been described previously.\textsuperscript{10}

At one stage, increasing the tidal volume from 1,000 ml to 1,200 ml with a constant F\textsubscript{IO2} increased the arterial P02. This suggests that "type b" alveoli were expanded as the tidal volume was increased, thus taking an active part in ventilation as "type a" or "c" alveoli rather than contributing to the true or absolute intrapulmonary shunt.\textsuperscript{11}

On another occasion the arterial oxygen tension was observed to increase dramatically when the tidal volume was reduced from 1,500 ml to 1,100 ml. Decreasing the tidal volume also reduced the peak inspiratory pressure from approximately 45 cm of water to 35 cm of water. Let us assume that large areas of this patient's lung contained nonventilated alveoli while other areas were normally ventilated. The pressure generated in ventilating the patient at 1,500 ml may have reduced the blood flow to the ventilated alveoli producing primarily an "a3" V\textsubscript{A}/Q relationship. The high pressure would not be transmitted to capillaries perfusing nonventilated alveoli as readily, especially if they were grouped in blocks as in lobar pneumonia. The remaining portion of the cardiac output would seek the path of least resistance and perfuse the nonventilated alveoli producing a "b4" type of V\textsubscript{A}/Q. The net result was an increase in intrapulmonary shunt. When the intrathoracic pressure was dropped in association with the decrease in tidal volume, there would be less resistance to flow, permitting greater perfusion of ventilated alveoli resulting in more "a1" V\textsubscript{A}/Q segments (V\textsubscript{A}/Q = 1). Proportionately, the number of "b4" alveoli would decrease by a reduction of blood flow to the nonventilated alveoli ("b1, 2 or 3"). The net result is a better matched V\textsubscript{A}/Q for the lung as a whole and an increase in arterial oxygen tension.

Obviously, changes in cardiac output can also change the degree of intrapulmonary shunting. As mean intrathoracic pressure decreased we would expect the cardiac output to increase. If the arterial oxygen tension remained constant this would increase the shunt. On the other hand, increasing the cardiac output also results in a higher oxygen content in mixed venous blood (C\textsubscript{VO2}). Under these circumstances, if V\textsubscript{A}/Q remained constant in all areas of the lung, with an increased C\textsubscript{VO2} the shunt blood would have a higher P02 than at the lower cardiac output. This would result in a higher arterial oxygen tension after mixing of shunted and non-shunted blood. The net result could be a decrease in calculated shunt.\textsuperscript{12} Cardiac output was not measured in this patient. His blood pressure and pulse rate were comparable at both ventilatory patterns, however, making it unlikely that a profound change in cardiac output occurred.

The "absolute shunt" through the lungs (type bl, 3, or 4 alveoli) can be estimated by determining the A-aDO\textsubscript{2} after 100 percent oxygen breathing.\textsuperscript{13} This method has proved to be reliable in patients with normal lungs. When our patient was changed from the mechanical ventilator delivering a 1,000 ml tidal volume at an F\textsubscript{O2} of 0.60 to spontaneous ventilation at a tidal volume of 450–500 ml and F\textsubscript{O2} of 1.0, his arterial oxygen tension dropped from 80 to 35 mm Hg. Thus, a higher absolute shunt or true intrapulmonary shunt was calculated at an F\textsubscript{O2} of 1.0 than with the F\textsubscript{O2} of 0.60. This can be explained by the presence of extremely unstable alveoli that collapsed at the lower tidal volume. Surfactant determinations performed on this patient's lungs at autopsy showed abnormally elevated minimum surface tension values on compression. This indicates an inability to maintain stable alveoli was present and rapid collapse was possible.\textsuperscript{4} It is also possible the shunt measurement was influenced by changes in cardiac output occurring during the conversion from controlled to spontaneous ventilation. In either case, these findings emphasize the importance of keeping all parameters constant when quantitating A-aDO\textsubscript{2} in the diseased lung.

Changes in shunt with changes in ventilatory pattern have been reported not to occur in patients with normal lungs.\textsuperscript{14} An increase in shunt with increasing airway pressure during mechanical ventilation in an animal model with occlusion of the airway to one lung has been described.\textsuperscript{15} This model...
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is similar in principle to what was observed in this patient. It seems likely, therefore, that the effect of changing patterns of mechanical ventilation on A-aD02 and shunting is different in diseased lung than in the normal lung.

Conclusions

This patient illustrated that the treatment of acute respiratory insufficiency can be associated with unpredictable changes in the distribution of pulmonary blood flow. By altering the tidal volume and the pressure transmitted to the pulmonary capillary bed, the proportion of various types of Va/Q match or mismatch can be changed almost instantaneously. This suggests that single determinations of arterial oxygen tension, while of some value in determining what is going on at that instant, cannot be used to predict what will happen from moment to moment, nor at changing patterns of ventilation. It would be helpful in the clinical care of these patients if continuous long-term monitoring of PaO2 were possible. This would allow the physician to change the patterns of ventilation frequently depending on the status of the patient's lungs. The ventilatory pattern selected would be that producing the optimal Va/Q.

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Reprint requests: Dr. Modell, University of Miami School of Medicine, PO Box 875, Biscayne Annex, Miami 33143

GREATNESS THROUGH CONJONT DEDICATION

Four doctors of superior talents and unique personalities made the medical side of Johns Hopkins University great. They became “The Four Physicians” who have been immortalized in the painting by Sir John Singer Sargent, known the world over and now reposing in the great hall of the Welch Memorial Library Building at Hopkins. Welch, Osler, Halsted and Kelly became “The Four Saints” really, and the story of the early Hopkins is chiefly their story—collectively and indivisibly. All four were present at the birth of the Hopkins (1889, capacity 220 beds) and a new era in American medical education dawned. Actually, Welch was a bit beforehand. Pathologist, physician, surgeon, gynecologist, the Four took the new infant to themselves and literally swept it to maturity and first rank without so much as a gesture to the usual childhood years. Working as a team, but individuals just the same, they plotted new courses and followed through with a judgment and a verve and an enthusiasm that made them preeminent in American medicine almost overnight. They had spirit, they had humanity, they had knowledge and deep understanding of illness, disease and people.


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