Air Transportation of Patients with Acute Respiratory Failure: Theory

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Adult respiratory distress syndrome (ARDS) that results from severe trauma often occurs in remote places, making it necessary to transport the patients to tertiary medical facilities by air. Since these severely hypoxic patients are exposed to additional risk of reduced inspired oxygen tension due to decreased barometric pressure, the feasibility of transportation of these patients was investigated by computer analysis. Mathematical models of pulmonary gas exchange in patients with ARDS were developed to calculate arterial and mixed venous blood tensions while breathing room air and oxygen at sea level, 8,000 ft, and 40,000 ft. Under each condition the following parameters were varied: alveolar ventilation ($\dot{V}A$), cardiac output ($\dot{Q}$), metabolic rate ($\dot{V}O_2$), hematocrit (Hcrit), and membrane diffusing capacity for oxygen (D_mO_2). Most of the gas exchange problems at altitude could be overcome by breathing oxygen as long as cardiac output and hematocrit were adequate. Hypoxemia in ARDS patients will not be greatly affected by the reduced inspired oxygen tensions at altitude in much the same way that hypoxemia in ARDS is poorly responsive to increased inspired oxygen tensions at sea level.

SEVERE TRAUMA on a large scale, whether it is due to earthquakes, industrial accidents, or armed conflict, often occurs in remote areas of the world. Patients that develop adult respiratory distress syndrome as a result of these problems can be difficult to manage in local medical facilities where intensive care units are often lacking, or insufficient to deal with large numbers of patients. Thus, there is a need for these patients to be transported rapidly to medical centers where appropriate treatment can be instituted. Transportation by air is the most efficient approach to this problem, but it raises the question of whether the reduced inspired oxygen tension that occurs with altitude might cause an overwhelming burden on these patients who are already severely hypoxic. Over long distances, aircraft commonly cruise at an altitude of 40,000 ft with the cabin maintained at a barometric pressure of 564 mm Hg which is approximately 75% of the barometric pressure at sea level. In the event of sudden decompression, barometric pressure could fall through 141 mm Hg, which is less than 20% of sea level barometric pressure.

To investigate the feasibility of transporting these severely hypoxic patients by air, we developed a computer model of pulmonary gas exchange in these patients to assess the effects of reduced barometric pressure. This approach has the advantage of being able to test changes in a large number of different parameters to assess their effect on pulmonary gas exchange. With this information it would be possible to identify the gas exchange parameters that need particular attention in this type of endeavor.

MATERIALS AND METHODS

Pulmonary gas exchange in patients with adult respiratory distress syndrome (ARDS) was simulated with a computer model. Patients were assumed to be ventilated artificially with a constant minute ventilation. Other assumptions of the model included: continuous alveolar ventilation rather than tidal breathing, continuous —rather than pulsatile—pulmonary blood flow, and a constant metabolic rate. Dead space and regulatory mechanisms, such as hypoxia pulmonary vasconstriction

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or the effects of hypoxia and hypercapnia on cardiac output, were not taken into account.

The pulmonary gas exchange abnormality of ARDS was based on the data of Dantzker *et al.* (1) and was simulated with a 10-compartment lung model. The compartments were ventilated and perfused in parallel, therefore, collateral ventilation did not occur. One compartment was designated as shunt while the other compartments were assigned ventilation-perfusion ratios from 0.005–100.0, equally spaced on a logarithmic scale. The model was assigned a shunt of 48%. Apart from the shunt, the dispersion of ventilation-perfusion ratios in ARDS is mild (1). Therefore, we generated a narrow degree of ventilation-perfusion inequality with a small log standard deviation of 0.25 using techniques described in detail elsewhere (8). Because of the narrow dispersion, only 2 of these 10 compartments receive appreciable blood flow (greater than 10^{-6} L · min^{-1}). Therefore, the trivial amount of blood flow to the other compartments was reassigned to these two compartments in order to reduce computation time.

For the purpose of comparison, we simulated gas exchange in a computer model of a normal subject being ventilated mechanically. A single homogeneous lung compartment was used for this study, and the minor degree of alveolar ventilation-perfusion mismatch that occurs in normal subjects was ignored because its effect of gas exchange is small (6).

Input Data

Under all conditions body temperature was held at 37°C, respiratory exchange ratio was maintained at 1.0, P_{50} was set at 26.8 mm Hg at pH 7.4, nitrogen exchange and base excess were assumed to be zero. Baseline values that were assigned to other variables were as follows: hemoglobin concentration of 15 gm %, hematocrit of 0.45, oxygen uptake of 300 ml · min^{-1} STPD, carbon dioxide output of 300 ml · min^{-1} STPD, and cardiac output of 6.0 L · min^{-1}. Both the normal and ARDS models were ventilated to an arterial P_{CO_2} of 40 mm Hg, which required an alveolar ventilation of 6.4728 L · min^{-1} BTPS for the normal and 8.729 L · min^{-1} BTPS for the ARDS model.

Computer Program

The computer program was written in FORTRAN and executed on a VAX computer. For most calculations we assumed that there is complete equilibration between alveolar and end-capillary blood gases (i.e. no diffusion defect). Since diffusion limitation has been demonstrated even in normal subjects at altitude, this factor was incorporated into some of the calculations of gas exchange at extreme altitude. To take diffusion into account, it was necessary to use a Bohr integration procedure that has been described previously (5). For this procedure, a constant ratio (0.533) of membrane diffusing capacity for oxygen (D_mO_2) to capillary blood volume (V_c) was assumed. The fraction of D_mO_2 assigned to each compartment, except shunt, was directly dependent on compartmental blood flow ($\dot{Q}$). This relation is likely to occur if increases of pulmonary blood flow result in recruitment of capillaries. This simplifying assumption enabled compartmental V_c to be estimated from compartmental D_mO_2, and the transit time in each lung compartment to be estimated from its V_c and $\dot{Q}$. For this procedure, the membrane diffusing capacity for carbon dioxide was assumed to be 20 times the corresponding value for oxygen. The multiple chemical reactions involved in CO_2 transfer were described by a single reaction rate with a half-time of 0.15 s. The oxygen reaction rate was calculated from the O_2 saturation according to the data of Staub *et al.* (4). An important assumption used here is that these reaction rates measured *in vitro* can be used to simulate *in vivo* reaction rates.

The computer program used iterative techniques (Newton-Raphson) to estimate the arterial and end-capillary blood gas tensions within each lung compartment, given barometric pressure, inspired gas tensions, and mixed venous blood gas composition. Arterial blood gas composition was estimated from the flow-weighted average of the end-capillary blood gas contents of each compartment. Oxygen uptake ($\dot{V}O_2$) and carbon dioxide output, mixed venous and arterial blood gas composition. The mixed venous blood gas composition was then altered by another iterative process to adjust values of $\dot{V}O_2$ and $\dot{V}CO_2$ to preassigned values.

Sensitivity Analysis

A sensitivity analysis was performed to determine the relative importance of the gas exchange variables on the system. Five factors that affect pulmonary gas exchange were selected for examination because of their clinical importance: alveolar ventilation, cardiac output, hematocrit, metabolic rate, and membrane diffusing capacity for oxygen. Each parameter was varied over a wide range to determine at what level pulmonary gas exchange was compromised to a level that life was unlikely to be sustained. We chose arbitrarily these limits to be a decreased mixed venous oxygen tension to less than 10 mm Hg or an increased P_aCO_2 of greater than 82 mm Hg (which was associated with an arterial pH of less than 7.1).

RESULTS

Control Data

Table I compares the published data on gas exchange variables in patients with ARDS (1) to the values obtained in the computer model at sea level while breathing oxygen. The values obtained with the computer model were all within 1 S.D. of those reported in ARDS patients (1), which indicates the close simulation that the computer model provides of pulmonary gas exchange in ARDS.

Table II provides the values of arterial and mixed venous blood oxygen tensions for both the normal and the ARDS computer simulations for five conditions: breathing air at sea level and 8,000 ft, and breathing oxygen at sea level, 8,000 ft, and 40,000 ft. Barometric pressures of 564 and 141 mm Hg were assumed for conditions of 8,000 and 40,000 ft, respectively. With increasing altitude, the differences between the normal and ARDS lungs diminish due to the fact that the falling

TABLE I. COMPARISON OF GAS EXCHANGE VARIABLES.

Gas Exchange Variable*	ARDS Patients†	ARDS Model‡
F_{IO_2}	0.83 ± 0.16	0.99
$\dot{V}_A$ ($L \cdot min^{-1}$)	9.3 ± 1.9	8.7
$\dot{Q}$ ($L \cdot min^{-1}$)	6.7 ± 2.6	6.0
Shunt (%)	48 ± 15	48
P_{aO_2} (mm Hg)	66 ± 19	52
$P_{\bar{v}O_2}$	36 ± 7	35

*F_{IO_2} is inspired gas fraction; $\dot{V}A$ is alveolar ventilation; $\dot{Q}$ is cardiac output; shunt is the percent of cardiac output which is shunted from the pulmonary to systemic circulation; P_{aO_2} and $P_{\bar{v}O_2}$ are arterial and mixed venous blood oxygen tensions, respectively.

†Indicates the ±1 S.D. of the gas exchange variable in a group of patients with adult respiratory distress syndrome published by Dantzker *et al.* (1).

‡Indicates values of gas exchange variables in the computer model.

TABLE II. COMPARISON OF BLOOD OXYGEN TENSIONS AT DIFFERENT ALTITUDES.

Blood Oxygen Tension		Air		Oxygen		
		Sea Level	8,000 ft	Sea Level	8,000 ft	40,000 ft
P_{aO_2}*	N†	109	68	666	472	53
	ARDS‡	42	38	52	49	37
$P_{\bar{v}O_2}$	N	43	40	53	51	35
	ARDS	29	27	35	33	26

*P_{aO_2} and $P_{\bar{v}O_2}$ are arterial and mixed venous blood oxygen tensions respectively, expressed in mm Hg.

†N refers to computer model of the normal lung.

‡ARDS refers to computer model of patients with adult respiratory distress syndrome.

inspired oxygen tension rather than shunt becomes the dominant cause of hypoxemia.

Sensitivity Analysis

Alveolar ventilation, cardiac output, hematocrit, metabolic rate, and membrane diffusing capacity for oxygen were varied systematically to determine the values that result in a limitation of pulmonary gas exchange. Not surprisingly, the major limit for gas exchange due to the reduction of alveolar ventilation ($\dot{V}A$) (Fig. 1) was a rising arterial carbon dioxide tension and falling arterial pH. With inhalation of O_2, oxygenation was a limiting factor only at 40,000 ft for both the ARDS lung and the normal lung.

In contrast, reduction of the cardiac output was poorly tolerated in the ARDS lung compared with the normal lung (Fig. 2). The intolerance to the reduced cardiac output, however, was affected only to a small extent by inspired oxygen fraction or altitude. A similar pattern of results was obtained by varying hematocrit (Fig. 3). These results confirm the concept that oxygen delivery to the tissues (the product of cardiac output and

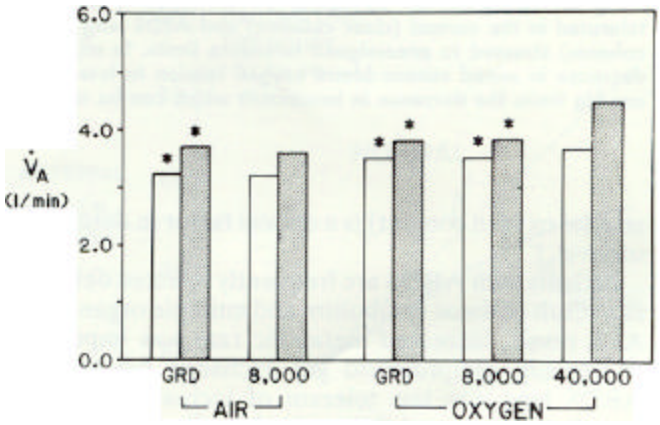

Fig. 1. Histogram of the lowest levels of alveolar ventilation that can be tolerated in the normal (clear columns) and ARDS lung (shaded columns) stressed to preassigned limits. The asterisk (*) indicates that the limiting factor is a decrease of arterial pH to 7.1 and an increase of arterial blood carbon dioxide tension to 82 mm Hg. Data without an asterisk indicate that a decrease in mixed venous blood oxygen tension to less than 10 mm Hg limits the decrease in ventilation which can be tolerated.

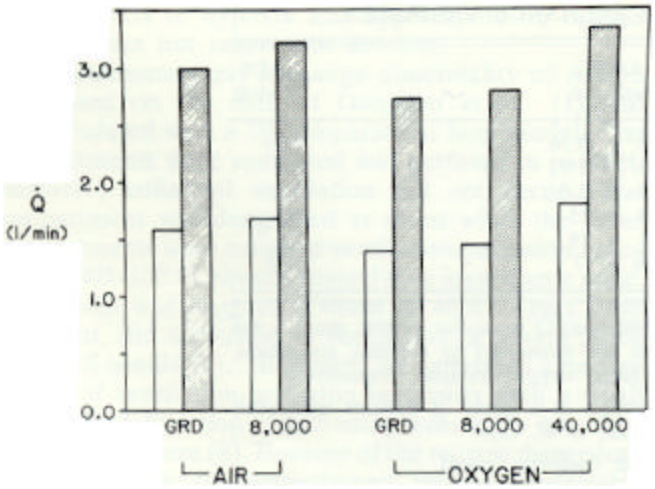

Fig. 2. Histogram of the levels of cardiac output that can be tolerated in the normal (clear columns) and ARDS lung (shaded columns) stressed to preassigned limits. In all cases a decrease in mixed venous blood oxygen tension to less than 10 mm Hg limits the decrease in cardiac output which can be tolerated.

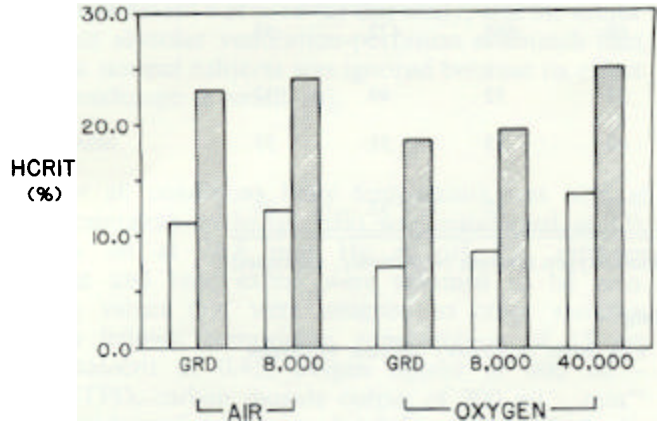

Fig. 3. Histogram of the levels of hematocrit that can be tolerated in the normal (clear columns) and ARDS lung (shaded columns) stressed to preassigned tolerance limits. In all cases a decrease in mixed venous blood oxygen tension to less than 10 mm Hg limits the decrease in hematocrit which can be tolerated.

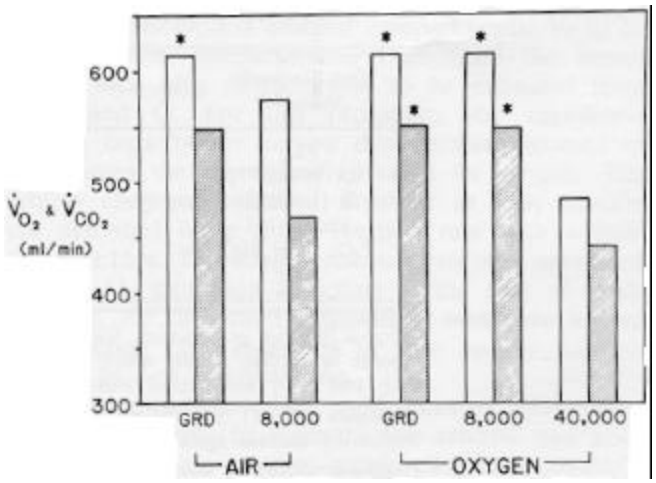

Fig. 4. Histogram of the levels of oxygen uptake and carbon dioxide output that can be tolerated in the normal (clear columns) and ARDS lung (shaded columns) stressed to preassigned limit. Both oxygen uptake and carbon dioxide output are equal in magnitude and are displayed on the ordinate. The asterisk (*) indicates that the limiting factor is a decrease of arterial pH to 7.1 and an increase of arterial blood carbon dioxide tension to 82 mm Hg. Data without an asterisk indicates that a decrease in mixed venous blood oxygen tension to less than 10 mm Hg limits the increase in oxygen uptake and carbon dioxide output which can be tolerated.

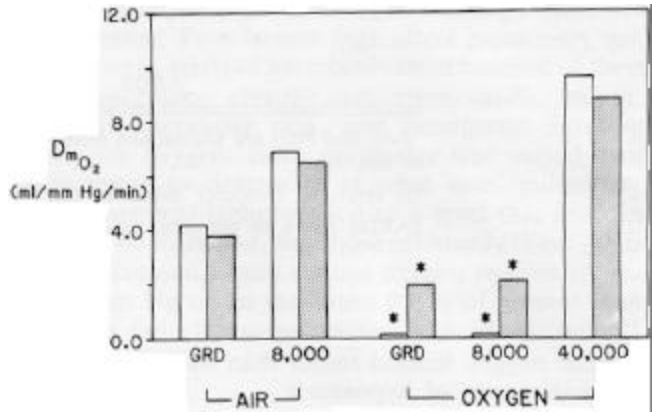

Fig. 5. Histogram of the levels of membrane diffusing capacity for oxygen that can be tolerated in the normal (clear columns) and ARDS lung (shaded columns) stressed to preassigned limits. The asterisk (*) indicates that the limiting factor is a decrease of arterial pH to 7.1 and an increase of arterial blood carbon dioxide tension to 82 mm Hg. Data without an asterisk indicates that a decrease in mixed venous blood oxygen tension to less than 10 mm Hg limits the decrease in membrane diffusing capacity which can be tolerated.

arterial oxygen content) is a critical factor in determining survival.

Patients with ARDS are frequently infected or stressed as a result of tissue catabolism and multiple organ failure. As a result, increased metabolic rate was imposed on an already compromised gas exchange function. The ARDS lung was less tolerant of increasing metabolic rate than the normal lung at both sea level and altitude when air was inspired (Fig. 4). On the other hand, with inspiration of pure oxygen this difference at all three altitudes was considerably reduced, but not abolished.

A diffusion defect not only affects oxygen transfer, but also can impact CO_2 transfer (5). Impairment of carbon dioxide transfer limited gas exchange at sea level and 8,000 ft when oxygen was inspired (Fig. 5). For all other conditions, oxygen transfer was the limiting facto and similar reductions of D_mO_2 were tolerated in both the normal and ARDS lung. Table III indicates the maximum change in any of the five tested parameters expressed as a percentage of the baseline value, which can be tolerated under the different conditions o altitude and inspired O_2 fraction. Two columns are provided for D_mO_2. The next to last column on the right indicates the maximum percent reduction in D_mO_2

TABLE III. LIMITING VALUES OF PHYSIOLOGIC PARAMETERS[a] AS PERCENT OF BASELINE.

Condition	$\dot{V}A$[b]	$\dot{Q}$	Hcrit	$\dot{V}O_2$ & $\dot{V}CO_2$	DmO_2[c]	DmO_2[d]
Breathing Air						
Sea level						
N	49*	27	25	205*	11	11
ARDS	42*	50	51	183	10	18
8,000 ft						
N	49	28	28	191	17	17
ARDS	41	53	54	156	17	32
Breathing 100% O_2						
Sea level						
N	54*	23	16	205*	<2*	<2*
ARDS	44*	45	41	183*	5*	9*
8,000 ft						
N	54*	23	18	205*	<2*	<2*
ARDS	44*	46	44	183*	5*	10*
40,000 ft						
N	56	30	30	161	24	24
ARDS	51	55	56	147	22	42

[a]$\dot{V}A$ is alveolar ventilation; $\dot{Q}$ is cardiac output; Hcrit is hematocrit; $\dot{V}O_2$ and $\dot{V}CO_2$ are oxygen uptake and carbon dioxide output; DmO_2 is the membrane diffusing capacity for oxygen.

[b]The baseline values of alveolar ventilation in the normal and ARDS lungs were chosen to maintain an arterial PCO_2 of 40 mm Hg and are 6.4728 and 8.729 $L \cdot min^{-1}$ BTPS, respectively. Baseline values of $\dot{Q}$, Hcrit, and $\dot{V}O_2$ and $\dot{V}CO_2$ are the same for both normal and ARDS Lungs.

[c]Baseline DmO_2 in these calculations is assumed to be equal in both circumstances.

[d]The absolute value of the baseline DmO_2 in the ARDS lung is assumed to be 52% of the value in the normal lung, analogous to the presence of a 48% shunt in the ARDS lung.

*Values followed by asterisks indicate that these limiting bounds are the result of a decrease in arterial pH to 7.1 and an increase in arterial pH to 7.1 and an increase in arterial PCO_2 to 82 mm Hg. In all other cases limitation is reached due to mixed venous oxygen tension falling below 10 mm Hg.

tolerated when both the normal and ARDS lungs have an identical baseline DmO_2. This comparison does not take into account that DmO_2 may be reduced in ARDS. It seems likely that a pathological process which causes a reduction in functioning alveoli may produce a similar reduction in DmO_2. Therefore, the importance of the diffusing capacity was also assessed with the assumption that DmO_2 of the ARDS lung was reduced in direct proportion to the degree of shunt. The last column on the right of Table III displays the percent reduction in DmO_2 which is tolerated when the baseline DmO_2 in the ARDS lung is reduced to 52% of the normal value.

Table III indicates that, compared with the normal lung, the two variables that are most sensitive to gas exchange limitation in ARDS are cardiac output and hematocrit. Supplemental oxygen only partially diminished the adverse effects of altitude.

Supplemental Oxygen

The ARDS lung was able to achieve sufficient pulmonary gas exchange at 40,000 ft as long as supplemental oxygen was provided. The inspired oxygen fraction was reduced progressively at this altitude to determine the minimum oxygen fraction required to prevent limitation of oxygen transfer. Fig. 6 and 7 indicate the arterial and mixed venous oxygen tensions due to alteration of the inspired oxygen fraction. These results indicate that at least 65% oxygen is required to avoid life threatening limitation of oxygen exchange

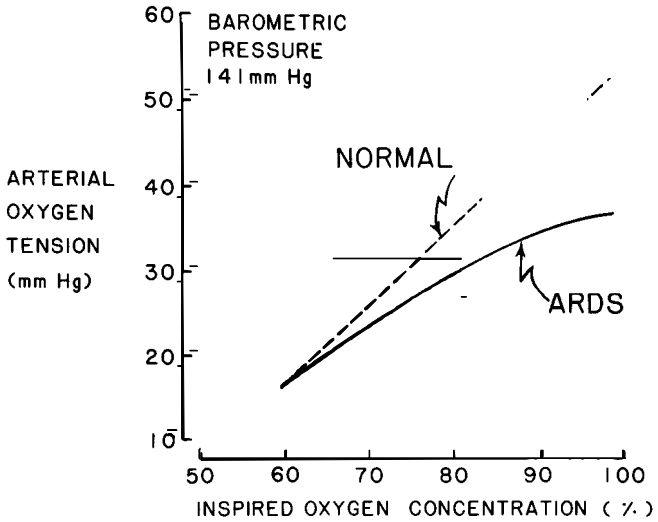

Fig. 6. Effects of changing inspired oxygen fraction (abscissa) on arterial blood oxygen tension at a barometric pressure of 141 mm Hg (40,000 feet altitude) for the normal lung (dashed lines) and the ARDS lung (solid lines).

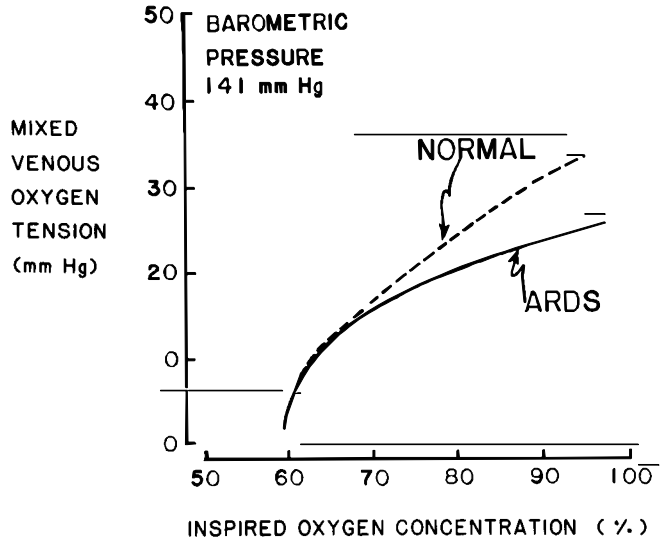

Fig. 7. Effects of changing inspired oxygen fraction (abscissa) on mixed venous blood oxygen tension at a barometric pressure of 141 mm Hg (40,000 feet altitude) for the normal lung (dashed lines) and the ARDS lung (solid lines).

for both the normal and ARDS lung. In addition, the smaller increases of the arterial and mixed venous blood gas tensions accompanying increases in the inspired oxygen concentration indicate that the hypoxemia of ARDS is more resistant to supplemental oxygen than the hypoxemia of the normal lung.

DISCUSSION

An interesting feature of the model computations was the demonstration that the arterial oxygen tension was relatively more resistant to change during hypobaric hypoxia in the ARDS lung than in the normal lung. As seen in Table II, ascent to 8,000 ft altitude resulted in a 41 mm Hg drop in P_aO_2 from 109 to 68 mm Hg in the normal lung. In comparison, the P_aO_2 in the ARDS model decreased only 4 mm Hg to 38 mm Hg under the same circumstances. When oxygen was inspired, this difference between normal and ARDS lungs was even more prominent with alterations in altitude which resulted in changes of several hundred mm Hg in the normal lung. In contrast, the total range of P_{O_2} in the ARDS model is only 15 mm Hg. These differences stem largely from the non-linear characteristics of the oxygen dissociation curve. In these calculations, gas exchange in the normal lung for the most part takes place on the upper portion of the dissociation curve. The slope of this portion of the dissociation curve is much less steep than the portion which is utilized in exchange in the ARDS lung.

Sensitivity Analysis

Fig. 1 illustrates that the ARDS and normal lungs have similar lower limits of alveolar ventilation necessary for survival by the criteria chosen. However, since the absolute quantity of ventilation required for maintenance of eucapnia is greater in the ARDS lung (8.729 vs. 6.4728 $L \cdot min^{-1}$), the relative reduction in ventilation which could be tolerated is, in fact, greater in the ARDS lung than the normal lung (Table III).

Hypoxemia was the factor that limits reductions in alveolar ventilation while breathing air except in the normal lung at sea level. In that circumstance a 42 mm Hg elevation of alveolar P_{CO_2}, the maximum change under the chosen criteria, was accompanied by a 42 mm Hg decrease of alveolar P_{O_2}. This decrease in alveolar P_{O_2} is insufficient to produce hypoxemia severe enough to reach the preassigned limits of $P_{\bar{v}O_2}$. Respiratory acidosis is the factor that limited reductions in alveolar ventilation while breathing oxygen except at 40,000 ft. Barometric pressure at this altitude was only 141 mm Hg so that inspired oxygen, even while breathing pure oxygen, was only 94 mm Hg. The difference between these two pressures was due to water vapor (47 mm Hg) which has a marked effect on inspired O_2 at this altitude. Under these circumstances, reductions in alveolar P_{O_2} caused by decreases in ventilation produced severe hypoxemia before the concommitant increases in P_{CO_2} reached an intolerable level.

The data illustrating limitations imposed by reductions in cardiac output and hematocrit (Fig. 2 and 3) are remarkably similar. These results confirm the concept that oxygen delivery to the tissues (the product of cardiac output and arterial oxygen content) is a critical factor in determining survival. Reduction in either parameter was poorly tolerated in the ARDS lung. In contrast, in the normal lung either blood flow or hematocrit could be reduced to approximately one half the lower limiting value seen in the ARDS lung. Interestingly, tolerated reductions in both models were little influenced by either altitude or inspired oxygen concentration.

Increases in metabolic requirements were less well tolerated in the ARDS model than the normal lung, but the differences were relatively small in most cases. As indicated in Fig. 4, the maximum possible increase in metabolic rate which can be tolerated, under the criteria chosen, was an approximately twofold increase over the baseline value. Ventilation was held constant. Therefore, a twofold increase in CO_2 production resulted in an approximate doubling of alveolar, and hence, arterial P_{CO_2}. A doubling of the baseline arterial P_{CO_2} of 40 mm Hg reached the preset limit of 82 mm Hg since distribution of both ventilation and blood flow remained invariant in this analysis. When tolerable increments in metabolic rate were substantially less than twice the baseline value, reduction in oxygen delivery with a fall in $P_{\bar{v}O_2}$ to less than 10 mm Hg usually set the tolerable limit.

The effect of diffusion limitation on gas exchange in ARDS is unknown. D_{mO_2} has been estimated to be 40 ml $\cdot min^{-1} mm\ Hg^{-1}$ or greater in normal subjects (2) but it is unknown in patients with ARDS. Substantial reductions in D_{mO_2} can be tolerated, especially when supplemental oxygen is administered (Fig. 5). The reduction in D_{mO_2} is overcome by increasing the gradient of oxygen tension across the alveolar-capillary membrane. Alveolar P_{O_2} is incremented with supplemental O_2, and the resulting increased partial pressure gradient augments O_2 transfer into the capillary blood. Because of the uncertainty of the effect of ARDS on the membrane diffusing capacity,

the comparisons were made with two baseline values of D_mO_2 in the ARDS model. A normal baseline value of D_mO_2 was assumed initially, a baseline value assumed for a second set of calculation was reduced in proportion to the right to left shunt in the ARDS model. The results differed quantitatively to a moderate degree but there were no qualitative differences (Table III).

Impaired CO_2 exchange with resulting hypercapnia can occur under conditions of extreme reduction of D_mO_2 while breathing oxygen. When air (21% O_2) is inspired, progressive reduction of the membrane diffusing capacity would result in increasing hypoxemia as oxygen transfer is reduced. However, inhalation of pure oxygen increases the driving force for O_2 diffusion into the blood almost fivefold. Under these circumstances, oxygen transfer is sufficient to maintain life, and progressive reduction of the membrane diffusing capacity begins to affect CO_2 transfer from blood to alveolus. Oxygen transfer can be enhanced by increasing the O_2 fraction in the inspired air, which results in an increased driving force for O_2 diffusion. Because air is free of O_2, the driving force for CO_2 exchange cannot be increased by alteration of the inspired gas.

Survival is theoretically possible in ARDS even with decompression to 40,000 ft altitude. It is essential that a high inspired oxygen concentration (at least 65%) be maintained in this circumstance (Fig. 6 and 7). The low barometric pressure (141 mm Hg) sets a limit of 94 mm Hg as the maximum possible alveolar oxygen tension. A patient who is mechanically ventilated at this altitude is at a disadvantage compared to the patient breathing spontaneously, even if the ventilator does not fail due to decompression. With fixed ventilation, alveolar P_{CO_2} will remain at 40 mm Hg, reducing alveolar oxygen tension from the inspired value of 94 mm Hg (breathing 100% O_2) to 54 mm Hg. The patient who is breathing spontaneously will increase ventilation as a result of the hypoxic stimulus, thus lowering alveolar P_{CO_2} and increasing alveolar P_{O_2}. The P_{O_2} ventilatory response to altitude exposure is thought to be a major compensatory mechanism in this circumstance (7).

The data in Fig. 6 and 7 were somewhat surprising in that there was relatively little difference in arterial and mixed venous P_{O_2} in both the normal and ARDS lungs. However, this finding is consistent with the observation made four decades previously that right-to-left shunt has little effect on the $(A\text{-}a)O_2$ when alveolar P_{O_2} is reduced to a low level (3).

It should be emphasized that this study only addressed the problem of gas exchange during air transportation of ARDS patients. Although the ARDS lung tolerated 40,000 ft when ventilated with enriched oxygen mixtures, it is likely that sudden decompression at this altitude would result in severe hypothermia, barotrauma, and other serious complications which could cause death. Nevertheless, the results of this theoretical study indicate that air transportation of ARDS patients is feasible and that these patients could possibly survive sudden decompression.

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