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Ventilatory Criteria for Aeromedical Evacuation

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Ventilatory requirements during simulated aeromedical transportation were investigated in normal dogs and animals with oleic acid-induced lung injury. Inspired oxygen fractions of 0.21 and 1.0 were used to ventilate the normal and injured dogs, respectively. Both groups were ventilated with a constant-volume piston ventilator. After a control period, animals were exposed to a simulated altitude of 8,000 ft (barometric pressure 564 mm Hg), followed by a second control period at ground level. Both groups of animals had no change in carbon dioxide production, arterial P_{CO_2} or ventilation during exposure to reduced barometric pressure. Systemic blood pressure, heart rate, cardiac output, and lung volume were all lower in oleic acid-injured animals than controls; the alveolar-arterial oxygen difference was larger in the oleic acid group. With altitude exposure, arterial and mixed venous oxygen tensions were decreased in both groups. Adequate gas exchange can be maintained during exposure to altitude even in animals with abnormal function provided that ventilation is constant and the inspired oxygen fraction is increased to compensate for the reduced barometric pressure.

PATIENTS REQUIRING mechanical ventilatory support occasionally must be transported to other medical facilities where more appropriate or sophisticated diagnostic or therapeutic help is available. If great distances are involved, this movement must be accomplished by air.

Despite past experience with large-scale transportation of injured personnel and an obvious need for scientific guidelines in this matter, there is a paucity of relevant studies in the medical literature. Some studies are anecdotal reports of one or more cases or descriptions of the process of air transport (1,4). Many other studies involving large numbers of patients were reported during or just after World War II. Therefore, they lack information pertinent to current medical therapy.

Kirby *et al.* (6) described the function of the Bird Mark VIII positive pressure ventilator at altitude. These authors studied normal dogs at altitude and concluded that this particular ventilator was adequate to maintain ventilation of a healthy animal, even though the delivery characteristics of the instrument varied substantially at altitude. They did not study animals with abnormal lungs. In a technically difficult study, Henry *et al.* (5) documented severe arterial hypoxemia in casualties being evacuated from the Republic of Viet Nam to Japan. These patients were all reasonably stable from a medical standpoint and none required mechanical ventilation. There are no studies which describe the physiological consequences of exposure to reduced barometric pressure in either humans or animal models that require mechanical ventilation to sustain life.

These considerations led us to examine the potential problems which may occur with exposure to reduced barometric pressure during air transportation. Our theoretical analysis (3) indicated that maintenance of ventilation and oxygen delivery was a critical factor in insuring survival in adult respiratory distress syndrome (ARDS) during air evacuation. Therefore, this investigation was designed to monitor these functions in normal and abnormal animals during exposure to altitude. The animal model of acute lung disease used in these experiments was induced by intravenous injection of oleic acid. This model results in a pathological and physiological picture quite similar to that accompanying ARDS in humans (7).

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METHODS

Ten male mongrel dogs were used for these experiments. The animals were premedicated with 4% thiamylal (6 mg·kg^{-1}) given by rapid intravenous bolus injection. Anesthesia was induced and maintained with alpha-chloralose and sodium tetraborate. The dose of alpha-chloralose/tetraborate solution was 120 mg·kg^{-1} of alpha-chloralose given intravenously followed by a continuous infusion of 42.8 mg·kg^{-1}·h^{-1} of alpha-chloralose. A constant-infusion pump was used to maintain accurate infusion rate. Muscle paralysis was obtained using repeated intravenous injections of pancuronium bromide. An initial dose of 0.1 mg·kg^{-1} was given and supplemental doses of 0.04 ml·kg^{-1} were administered as needed. No data were collected for at least 10 min after an injection of pancuronium bromide. Each dog was intubated with a cuffed oral endotracheal tube and ventilated with a Harvard constant-volume ventilator. Control dogs received 21% oxygen. Oleic acid animals were ventilated with 100% oxygen. Since the severity of the oleic acid-induced injury varied among animals, an F_{IO_2} of 1.0 was chosen to ensure an acceptable P_{aO_2} in all animals. Initial tidal volume was set at approximately 15 ml·kg^{-1} with a respiratory rate of 12–16 breaths per minute. Adjustments in ventilator rate were made prior to data collection to ensure adequate gas exchange as determined by blood gas analysis. No further adjustments were made in ventilator settings.

The dogs were instrumented with a jugular venous catheter for fluid and drug administration, and a femoral arterial catheter for blood pressure monitoring and collection of arterial blood samples. A heating blanket was used to maintain body temperature. A triple lumen Swan-Ganz thermodilution catheter was placed in the pulmonary artery via a femoral vein for pulmonary arterial and pulmonary capillary wedge pressure monitoring and collection of mixed venous blood samples. Body temperature was monitored by the thermistor at the tip of the Swan-Ganz catheter.

The oleic acid model of acute respiratory distress syndrome was induced in 5 of the 10 dogs by injection of oleic acid, 0.1 ml·kg^{-1}, infused into the right atrium via the Swan-Ganz catheter at 0.4 ml·min^{-1}. The dogs were allowed to breathe spontaneously from a reservoir containing 100% oxygen. Arterial blood gases were monitored and, if needed, the animal was ventilated intermittently with an Ambu bag to prevent severe respiratory acidosis. Approximately 90 min after the oleic acid infusion, the animals were placed on the constant volume ventilator with 2-cm H_2O positive end-expiratory pressure. When blood gases were stable, which was approximately 2 h after oleic acid infusion, data collection began.

Data were recorded with both a Gould 8-channel recorder and a Hewlett-Packard 8-channel FM tape recorder. The Gould recorder was used for on-line monitoring. All calculations were obtained from tape recorder data. The variables recorded were EKG, systemic and pulmonary artery pressures (Gould P-23 transducers), pulmonary artery wedge pressure, airway pressure at the proximal end of the endotrachial tube (Validyne MP-45 transducer), airflow obtained from a Fleish No. 3 pneumotachograph placed between the ventilator and the endotracheal tube, inspired volume by electrical integration of the inspiratory flow signal, and end tidal O_2 and CO_2 concentrations obtained from a Perkin-Elmer MGA 1100 mass spectrometer. The end tidal sample site was at the proximal end of the endotracheal tube. In addition, mixed expired O_2 and CO_2 were obtained from a mixing chamber attached to the expiratory port of the ventilator. Arterial and mixed venous blood samples were obtained and analyzed at 37°C using a Corning blood gas analyzer. Since all animal temperatures ranged from 36°–38°C, no blood gas corrections for these minor temperature variations were made. Cardiac output was determined using an Edwards cardiac output computer. Using 10 cc injections of saline at room temperature, 3–5 sequential cardiac output determinations were made. The cardiac output computer continually monitored the temperature of the injectate solution. Lung volume was estimated by nitrogen dilution using a closed-circuit rebreathing technique.

The same data collection protocol was employed for both control and oleic acid animals. After being placed on the constant volume ventilator, several preliminary arterial blood gases were obtained at 15–20 min intervals. When both arterial P_{O_2} and P_{CO_2} were stable, data collection was begun.

The initial data at ground level were collected on two occasions 20–30 min apart, and are designated as Ground 1 and 2 conditions. All parameters were measured each time except for lung volume, which was determined only with the second collection of each set. Following data collection, the hypobaric chamber in which the animal was situated was closed and decompressed to a simulated level of 8,000 ft (barometric pressure equal to 564 mm Hg). The rate of ascent and descent to and from 8,000 ft was 2,000–5,000 ft·min^{-1}. Once altitude was reached, all pressure transducers (airway, systemic, and pulmonary artery pressure) were recalibrated. After a 25–30 min stabilization period, two sets of altitude data (Altitude 1 and 2) were collected using the same protocol as at ground level. The hypobaric chamber was returned to ground level and, after 25–30 min of stabilization, another two sets of measurements (Ground 3 and 4) were obtained. Pressure transducers were recalibrated again prior to these measurements. At the conclusion of the experiments, animals were sacrificed by rapid intravenous injections of a saturated KCl solution.

The data recorded were digitized by a 12-bit analog-to-digital converter in a NorthStar Horizon computer and stored on a floppy disc. Data from the recorder were sampled over 20-s periods at the rate of 50 Hz. Mean values for heart rate, airway pressure, and pulmonary wedge pressure were obtained as well as systolic and diastolic systemic and pulmonary arterial pressures. Pulmonary vascular pressures were measured at end expiration. Ventilator rate and inspired volume also were determined. Mixed expired gas concentrations were used to calculate CO_2 production (V_{CO_2}). Inspired P_{O_2} and alveolar P_{O_2} were calculated, as was the alveolar-arterial P_{O_2} difference, $(A\text{-}a)_{O_2}$. The cardiac outputs determined at each data collection time were averaged, and lung volume was calculated from mixing syringe volume and nitrogen dilution data, and corrected for instrument dead space.

Data were analyzed with analysis of variance for a factorial experimental design (1,6). The two factors studied in these experiments were oleic acid and altitude. Each of the 15 variables listed in Table I was analyzed separately. Four aspects of the data were examined. First, the ground data between the oleic and control dogs were compared to deter-

TABLE I. SUMMARY OF RESULTS.

Parameter							
Tidal Vol (ccBTPS)	Control	413 ± 126	412 ± 125	411 ± 121	416 ± 116	417 ± 122	411 ± 122
	Oleic	413 ± 31	412 ± 32	410 ± 31	410 ± 35	411 ± 31	412 ± 36
Resp Rate (breaths/min)	Control	19 ± 3	19 ± 3	19 ± 3	19 ± 3	19 ± 3	19 ± 3
	Oleic	20 ± 4	20 ± 4	20 ± 4	20 ± 4	20 ± 4	20 ± 4
$\dot{V}_{CO_2}$STPD (cc/min)	[a]Control	79 ± 28	77 ± 24	74 ± 22	74 ± 18	73 ± 19	72 ± 16
	Oleic	68 ± 25	63 ± 21	64 ± 22	61 ± 17	62 ± 22	61 ± 25
P_{Airway} (mm Hg)	[b]Control	6 ± 1	7 ± 1	6 ± 1	6 ± 1	7 ± 1	7 ± 1
	Oleic	8 ± 2	9 ± 2	9 ± 2	9 ± 2	10 ± 2	10 ± 2
FRC (ccBTPS)	[c]Control		908 ± 322		670 ± 41		872 ± 288
	Oleic		319 ± 116		249 ± 74		252 ± 88
$P_{Art,Mean}$ (mm Hg)	[d]Control	128 ± 14	126 ± 13	122 ± 12	120 ± 18	119 ± 16	119 ± 14
	Oleic	108 ± 20	110 ± 17	108 ± 20	110 ± 19	109 ± 19	110 ± 20
$P_{Pulm,Mean}$ (mm Hg)	[e]Control	13 ± 3	14 ± 2	16 ± 2	13 ± 4	13 ± 2	13 ± 2
	Oleic	17 ± 5	19 ± 7	20 ± 4	21 ± 6	21 ± 6	21 ± 6
$P_{Wedge,Mean}$ (mm Hg)	Control	3 ± 3	3 ± 3	4 ± 2	3 ± 3	4 ± 3	4 ± 3
	Oleic	3 ± 2	4 ± 2	5 ± 1	5 ± 2	4 ± 2	5 ± 2
Heart Rate (beats/min)	[f]Control	110 ± 32	106 ± 34	102 ± 21	103 ± 25	101 ± 23	100 ± 25
	Oleic	71 ± 14	79 ± 12	80 ± 11	83 ± 16	88 ± 20	88 ± 21
$\dot{Q}$ (L/min)	[g]Control	2.8 ± .8	3.2 ± 1	3.3 ± .7	3.3 ± .9	3.0 ± .7	2.9 ± .7
	Oleic	2.2 ± .8	2.3 ± .7	2.1 ± .6	2.0 ± .4	1.8 ± .4	1.9 ± .3
pHa	[h]Control	7.42 ± .08	7.40 ± .09	7.43 ± .11	7.44 ± .10	7.44 ± .12	7.45 ± .10
	Oleic	7.34 ± .07	7.35 ± .07	7.36 ± .08	7.36 ± .07	7.36 ± .07	7.37 ± .08
P_{aCO_2} (mm Hg)	[i]Control	35 ± 7	36 ± 7	34 ± 8	33 ± 7	33 ± 9	31 ± 7
	Oleic	47 ± 6	46 ± 6	44 ± 8	43 ± 7	43 ± 8	42 ± 8
P_{aO_2} (mm Hg)	[j]Control	92 ± 6	92 ± 3	67 ± 11	82 ± 15	100 ± 6	103 ± 4
	Oleic	209 ± 138	172 ± 125	106 ± 67	100 ± 61	157 ± 131	159 ± 134
$P\bar{v}_{O_2}$ (mm Hg)	[k]Control	45 ± 3	36 ± 20	32 ± 18	33 ± 19	25 ± 23	34 ± 19
	Oleic	52 ± 13	50 ± 15	43 ± 13	41 ± 12	42 ± 12	42 ± 11
$(A\text{-}a)O_2$ (mm Hg)	[l]Control	15 ± 20	13 ± 18	−1 ± 19	4 ± 16	7 ± 21	6 ± 18
	Oleic	420 ± 147	465 ± 134	362 ± 75	368 ± 67	478 ± 130	485 ± 136

[a] $\dot{V}_{CO_2}$ = Control > Oleic, $p < 0.01$.
[b] P_{Airway} = Oleic > Control, $p < 0.01$.
[c] FRC = Control, Altitude n = 3; Oleic, Ground #1 n = 4.
No difference within Control or Oleic groups by ANOVA or *t* test for multiple samples.
Control > Oleic by *t* test, $p < 0.001$.
[d] $P_{Art,Mean}$ = Control > Oleic, $p < 0.01$.
[e] $P_{Pulm,Mean}$ = Oleic > Control, $p < 0.01$.
[f] Heart Rate = Control > Oleic, $p < 0.01$.
[g] $\dot{Q}$ = Cardiac Output: Control > Oleic, $p < 0.01$, $\dot{Q}$ falls with time: Ground 1,2 > Ground 3,4; $p < 0.05$ (due to Oleic values).
[h] pHa = Control > Oleic, $p < 0.01$.
[i] P_{aCO_2} = Oleic > Control, $p < 0.01$.
[j] P_{aO_2} = Oleic > Control, $p < 0.01$.
P_{aO_2} falls with altitude, both groups, $p < 0.05$.
Note: 2/5 Controls on 30% O_2 for Altitude 2.
[k] $P\bar{v}_{O_2}$ = Oleic > Control, $p < 0.01$.
$P\bar{v}_{O_2}$ falls with altitude, both groups, $p < 0.01$.
[l] $(A\text{-}a)O_2$ = Oleic > Control, $p < 0.01$.
Decrease with altitude, both groups, $p < 0.01$.
Oleic shows greater decrease with altitude than control, $p < 0.05$.

mine the effects of oleic acid on the variable. Second, data obtained at altitude were compared with data obtained at ground to determine the effects of altitude. Third, an interaction effect was evaluated to determine if the effects of altitude on oleic acid dogs differed from the effect of altitude on normal dogs. Finally, the pre-altitude data (Ground 1 and 2) were compared with the post-altitude data (Ground 3 and 4) to determine if there was a temporal effect on the baseline data due to either barotrauma or instability of the preparation. For the lung volume variable, analysis of variance could not be used because of incomplete data. Instead, we used Student's *t* tests and the Bonferroni correction for repeated samples. For all statistical techniques, significance was accepted at the 5% level.

RESULTS

A total of 12 mongrel dogs was required for 10 successful experiments. Two oleic acid animals died before measurements were completed. Dogs weighed 14.4–27.2 kg. There was no significant difference in the weight of the five dogs used as controls (20.6 kg) compared to the five oleic acid animals (19.1 kg).

Results are summarized in Table I. The respirator delivered a constant tidal volume at ground level and at altitude. Its performance was not affected by a simulated altitude of 8,000 ft. Ventilator frequency was also constant. Airway pressure was higher in the oleic acid animals compared to controls, probably related to a decreased lung compliance secondary to the oleic acid lung injury in this group. An increased resistance to airflow may also have been present, related to the decreased lung volume in the oleic acid animals. Functional residual capacity (FRC) was approximately three times larger in the controls than the oleic acid group ($p < 0.001$).

Arterial carbon dioxide tension, P_{aCO_2}, was higher in the oleic acid animals than controls, despite a similar minute ventilation in both groups. This information, combined

with the finding that CO_2 production (V_{CO_2}) was actually lower in the oleic acid animals, suggests that major alterations in gas exchange occurred in the oleic acid animals with lung injury. These data indicate that physiologic dead space and the dead space to tidal volume ratio V_D/V_T were both increased in the oleic acid animals. The elevated P_{aCO_2} noted in the oleic acid group produced a mild respiratory acidosis, as indicated by a decrease in arterial pH in these animals compared to controls.

Oxygen exchange was also grossly abnormal in the oleic acid-injured group. Despite being ventilated with 100% O_2, which resulted in an inspired P_{O_2} of approximately 700 mm Hg at ground level, these animals had a mean P_{aO_2} of 174 mm Hg for the four ground observations. There was a wide range of P_{aO_2} in this group, and pre-altitude (Ground 1 and 2) P_{aO_2} ranged from 62–344 mm Hg. At 8,000 ft, inspired P_{O_2} was 521 mm Hg and mean P_{aO_2} was 103 mm Hg. The mean alveolar-arterial oxygen difference $(A\text{-}a)O_2$ of the group was 462 mm Hg at ground level (Ground 1–4) and 365 mm Hg at altitude (Altitude 1 and 2). This large $(A\text{-}a)O_2$ on 100% O_2 is evidence for a large right-to-left intrapulmonary shunt, as would be expected in a model of lung injury simulating ARDS.

The control group had normal blood gas values under ground conditions. At 8,000 ft, P_{IO_2} decreased to 108 mm Hg from about 147 mm Hg under ground conditions. This 39-mm Hg decrease in inspired P_{O_2} resulted in an average decline in P_{aO_2} of 25 mm Hg, from 92 mm Hg (Ground Levels 1 and 2) to 67 mm Hg during the first altitude period. During the second altitude period, two control animals received 30% O_2 rather than room air. The higher inspired O_2 concentration resulted in a 35 mm Hg increase in inspired P_{O_2} and restored P_{aO_2} to ground level values in these two animals. The mean values (± S.E.) for P_{aO_2} during the ground level periods bracketing the altitude mean values are shown in Fig. 1 and 2 for the control group and the oleic acid animals, respectively.

Mixed venous P_{O_2} was lower in the control group than the oleic acid group. This is not unexpected since the oleic acid animals all received 100% O_2 and had much higher P_{aO_2}. There was a slight decrease in P_{vO_2} over time during these experiments.

Heart rate and cardiac output were significantly lower in the oleic acid group compared to controls. A decrease in cardiac output after oleic acid-induced pulmonary edema has been reported by others and is felt to be related to the increase in pulmonary vascular resistance seen in this model (8). There was a decrease in cardiac output over time due to a small, but steady, decrease in cardiac output in the oleic acid animals. This can be clearly seen in Fig. 3, which plots the mean cardiac output during ground and altitude periods. Fig. 4 shows similar data for normal animals; the relative stability of cardiac output in this group is apparent.

Mean systemic arterial blood pressure was lower in the oleic acid animals than the controls. This difference was probably due to the decreased cardiac output noted in the oleic acid dogs. Mean pulmonary artery pressure was higher in the oleic acid group, most likely as a result of their lung injury and hypoxemia. There was a tendency for the mean pulmonary artery pressure to increase over time, but this did not reach statistical significance. Pulmonary artery wedge pressure was similar in both oleic acid animals and controls.

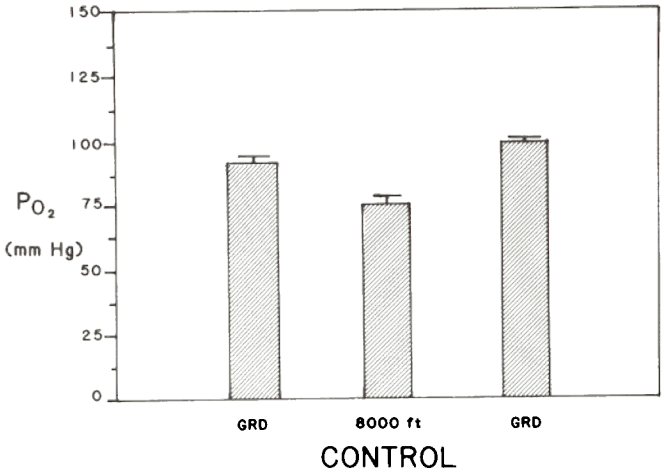

Fig. 1. Arterial P_{O_2} in normal dogs during the three experimental periods. Bars in the figure indicate S.E.M.

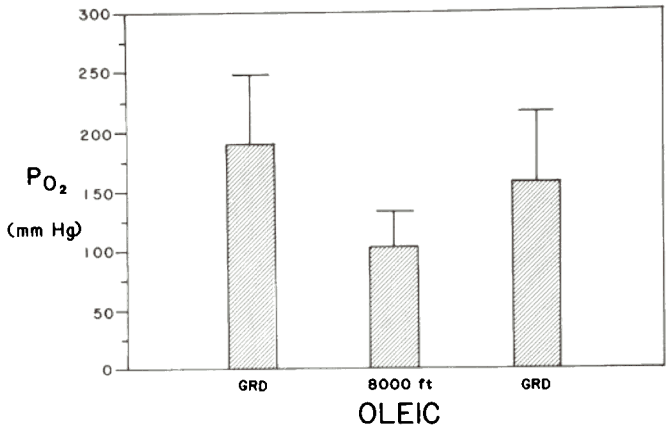

Fig. 2. Arterial P_{O_2} in dogs given intravenous oleic acid during the three experimental periods. Bars in the figure indicate S.E.M.

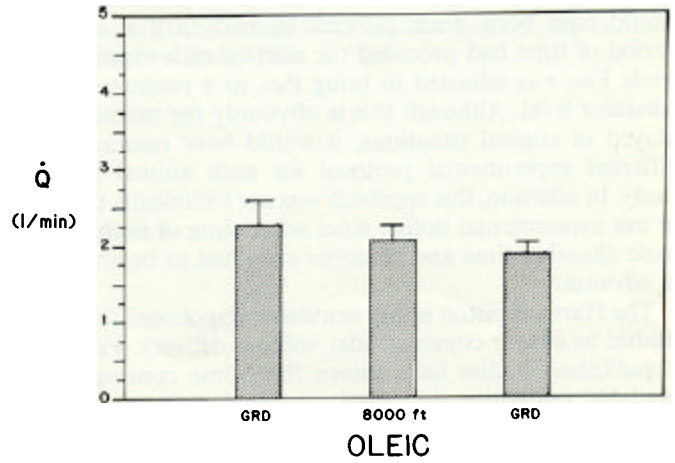

Fig. 3. Cardiac output in dogs given intravenous oleic acid during the three experimental periods. Bars in the figure indicate S.E.M.

DISCUSSION

This study was performed to determine the ventilatory criteria which would allow successful aeromedical evacua-

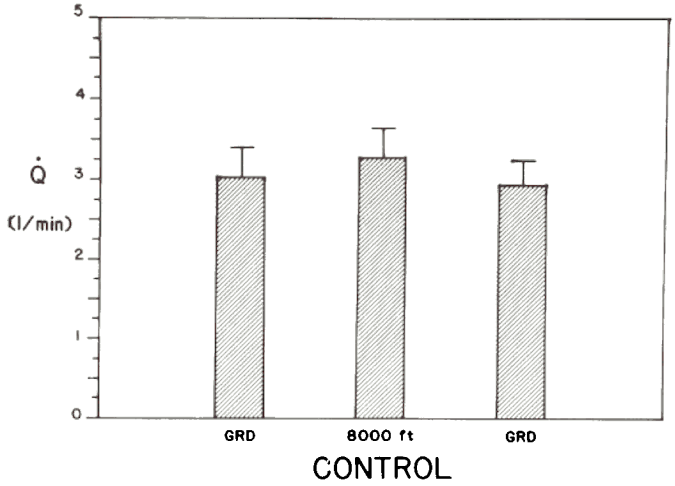

Fig. 4. Cardiac output in normal dogs during the three experimental periods. Bars in the figure indicate S.E.M.

tion of patients with respiratory failure requiring mechanical ventilation. This issue was approached first by evaluating cardiorespiratory and gas exchange information obtained from normal, anesthetized, apneic animals ventilated under ground level conditions and at a simulated altitude of 8,000 ft. This hypobaric condition is similar to that maintained with commercial and military aircraft flying at an altitude of approximately 40,000 ft. Acute lung injury was induced in a second group of animals by intravenous injection of oleic acid, which produces a lung injury similar to the adult respiratory distress syndrome. These animals were ventilated with 100% O_2, and the same hemodynamic and gas exchange measurements were made as in the control group.

The use of only 100% oxygen in the oleic acid animals was employed for several reasons. First, it ensured an adequate P_{aO_2} in all animals, even in those with the most severe lung injury. Second, it imposed a constant total experimental time on all oleic acid animals. Time-related changes would have been more difficult to analyze if a variable period of time had preceded the start of each experiment, while F_{IO_2} was adjusted to bring P_{aO_2} to a predetermined, adequate level. Although this is obviously the method employed in clinical situations, it would have resulted in a different experimental protocol for each animal in our study. In addition, this approach was not technically feasible in our experimental design since scheduling of both hypobaric chamber time and chamber crew had to be arranged in advance.

The Harvard piston pump ventilator was chosen for these studies to ensure constant tidal volume delivery. Previous unpublished studies have shown that some commercially available ventilators decreased volume output at altitude. By using the piston pump ventilator, this important variable was controlled. Our results indicated that the tidal volume delivered by the ventilator remained constant at both ground level and at a simulated altitude of 8,000 ft. Because of the constant ventilation and carbon dioxide production, P_{aCO_2} and pH remained unchanged both at ground and at altitude in the control group. The oleic acid animals showed evidence of impaired carbon dioxide exchange, which was associated with an increased physiological dead space. This occurred despite a significant decrease in CO_2 production compared to the control group. Nonetheless, even in the face of their lung injury, P_{aCO_2} and V_{CO_2} remained constant at ground level and at altitude. Therefore, as long as a ventilator delivers the same tidal volume at altitude as it does at ground level, no change in P_{aCO_2} or acid-base status will occur. If ventilation, as assessed by P_{aCO_2}, is adequate at ground level in ventilator patients with respiratory failure, no change in ventilation should occur during aeromedical evacuation if the ventilator employed maintains rate and volume delivery and the patient's physiologic status does not change.

The normal animals exhibited a drop in P_{aO_2} at 8,000 ft approximately equal to the decrease in P_{IO_2} which occurred from ground level to altitude. At ground level, P_B was approximately 747 mm Hg and P_{IO_2} was 147 mm Hg; at 8,000 ft, P_B was 564 mm Hg, and P_{IO_2} was 108 mm Hg. When the inspired O_2 concentration was increased to 30% at 8,000 ft, the P_{IO_2} was 155 mm Hg, slightly greater than that at ground level. Two control animals were evaluated at 8,000 ft while breathing 30% oxygen. This increment in F_{IO_2} restored P_{aO_2} in both animals to values observed at ground level. Most control animals had a slight increase in pulmonary artery pressures when taken to a simulated altitude of 8,000 ft. Pressures in the two animals given 30% oxygen decreased noticeably with administration of this mixture. These findings indicate that it is sufficient to administer only 30% oxygen during aeromedical evacuation to prevent alteration of physiological parameters in a patient with normal lungs who requires mechanical ventilation, e.g. respiratory failure caused by neuromuscular disease.

The five animals with oleic acid-induced lung injury had markedly elevated alveolar-arterial oxygen differences. However, since all were ventilated with 100% oxygen, none were severely hypoxemic at ground level. Several animals did become hypoxemic at altitude; arterial oxygen tension approached 40 mm Hg in several cases. From these data, it is apparent that if a patient requires 100% oxygen at ground level and has a P_{aO_2} less than 100 mm Hg, there is significant risk that severe hypoxemia may develop during air transportation. This could adversely affect the likelihood of successful aeromedical evacuation. We did not verify this conclusion experimentally in this study. Our previous theoretical investigation set limits on acceptable levels of ventilation, inspired oxyen, and oxygen delivery compatible with survival during air transport of patients with acute respiratory failure (3).

The best means of ensuring adequate oxygenation in patients being evacuated by air is to maintain the inspired oxygen tension (P_{IO_2}) at a value that produces adequate oxygenation at ground level. All that is required is to increase the inspired oxygen concentration at altitude to an extent which will counteract the decrease in barometric pressure. Table II serves as a guide to therapy for patients requiring supplemental oxygen during aeromedical evacuation. This table lists the F_{IO_2} necessary to maintain P_{IO_2} at the same value present at sea level with different oxygen mixtures. For example, P_{IO_2} can be maintained at 149 mm Hg at a simulated altitude of 8,000 ft if inspired oxygen is increased to just under 30%. If 50% oxygen were required to maintain adequate O_2 exchange at sea level, then 71% oxygen would be necessary at 8,000 ft. Note that if the F_{IO_2}

TABLE II. INSPIRED O_2 FRACTION REQUIRED TO MAINTAIN INSPIRED O_2 TENSION.

Barometric Pressure (mm Hg)	760	656	609	564
Equivalent Altitude (ft)	0	4000	6000	8000
P_{IO_2}	F_{IO_2}			
149	0.21	0.25	0.27	0.29
214	0.30	0.35	0.38	0.41
285	0.40	0.47	0.51	0.55
366	0.50	0.60	0.65	0.71
428	0.60	0.70	0.76	0.83
499	0.70	0.82	0.89	0.97
517	0.725	0.85	0.92	1.00
562	0.79	0.92	1.00	
570	0.80	0.94		
609	0.85	1.00		

exceeds 0.725 at sea level, the equivalent P_{IO_2} cannot be achieved at 8,000 ft. This suggests an upper limit in F_{IO_2} for patients being evaluated for possible aeromedical evacuation. If an F_{IO_2} of greater than 0.70 at sea level is needed to maintain a satisfactory P_{aO_2}, deterioration in P_{aO_2} will almost certainly occur at altitude, even if 100% O_2 is inspired. This could have an adverse effect on patient survival. The risk-benefit ratio of transporting such a patient would have to be carefully weighed.

An alternative choice would be to transport patients requiring very high oxygen concentrations at lower altitudes to maintain cabin pressure at a higher value. Table II lists the F_{IO_2} necessary to maintain P_{IO_2} at intermediate barometric pressures. As shown in the table, these intermediate altitudes permit transportation of patients with greater oxygen requirements.

It is difficult to predict the consequent fall in arterial oxygenation if patients had to be transported under conditions which would result in lowered P_{IO_2}. The resulting decrease in oxygenation would be a function of the mixed venous oxygen tension, the degree of ventilation-perfusion inequality, and the magnitude of right-to-left shunt. These factors could vary widely from one patient to another.

This approach to oxygen therapy depends upon the ability to measure arterial blood gases prior to evacuation. Under extremely adverse circumstances, this may not be possible. If so, the most expedient approach would be to administer 100% oxygen to all severely ill patients requiring mechanical ventilation. The risk of producing oxygen toxicity while breathing 100% oxygen for durations of less than 24 h is extremely small in normal individuals. However, little data are available to predict the effect of pure oxygen in a patient who already has an existing lung lesion. Nevertheless, it seems likely that this risk is less than the consequences of possible severe hypoxemia.

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