Air Travel and Oxygen Therapy in Cardiopulmonary Patients*

Henry Gong, Jr., M.D., F.C.C.P.†

(Chest 1992; 101:1104-13)

BP = barometric pressure; CHO = carbohydrate; FAA = Federal Aviation Administration; HAST = hypoxia-altitude simulation test; PIo_2 = inspired partial pressure of oxygen; R = respiratory quotient; RQ = respiratory exchange ratio; SaO_2 = arterial oxygen saturation

Air travel today is relatively convenient, expeditious, affordable, and safe.[1,2] The more than 400 million individuals who fly annually aboard commercial carriers in the United States usually adapt well to exposures to moderate altitude. The flying public also includes patients with cardiopulmonary disorders who choose commercial air travel for business, pleasure, or medical reasons. The number of flying patients is probably increasing annually. Their capability for air travel has been enhanced by several factors. For example, reliable clinical assessment of oxygen (O_2) needs during rest, sleep, exercise, and altitude exposure is readily available, lending confidence to O_2 prescribing and usage. Lightweight portable O_2 units and efficient delivery systems (oxygen-conserving devices and tracheal catheters[3]) can markedly improve the portability of O_2, decrease O_2 requirements, and increase mobility. Pulmonary rehabilitation programs promote quality-of-life issues and functional independence, including long-distance travel by oxygen-dependent patients.[4,5] Air carriers generally recognize and accept the concept that in-flight O_2 therapy is manageable and safe, although many deficiencies and issues remain in obtaining and using supplemental O_2.

This review focuses on the acute responses of cardiopulmonary patients to altitude hypoxia and the methods for providing adequate oxygenation during commercial flights, which temporarily induce a hypobaric, hypoxic stress in an isolated environment. Although the database is limited, this review will also summarize clinical information, practical guidelines, and procedures applicable to patients requiring supplemental O_2 during air travel.

*From the Pulmonary and Critical Care Medicine Division, Department of Medicine, UCLA Medical Center, Los Angeles.
†Professor of Medicine.
Reprint requests: Dr. Gong, Pulmonary and Critical Care Medicine, UCLA Medical Center, Los Angeles 90024-1690

Environmental Oxygen at Altitude

Although the proportion of atmospheric O_2 remains approximately 21 percent of the total barometric pressure (BP) with increasing altitude, the partial pressure of oxygen (Po_2) falls considerably. For example, atmospheric Po_2 is approximately 159 mm Hg at sea level and 118 mm Hg at 8,000 ft (2,438 m). The corresponding inspired Po_2 (PIo_2) as designated by $PIo_2 = 0.21 \times (BP - 47)$, is 149 mm Hg at sea level and decreases approximately 4 mm Hg per 1,000 ft of elevation, resulting in 108 mm Hg at 8,000 ft. Thus, atmospheric Po_2 and PIo_2 can be estimated from the BP at different altitudes.[6,7]

Commercial aircraft cruise between 22,000 ft (6,706 m) and 44,000 ft (13,411 m) above sea level to improve operating efficiency.[8] The intolerable and lethal hypobaric effects at these high elevations are ameliorated by partial environmental modification of the aircraft cabin, *ie*, pressurization to a safer and more comfortable lower altitude.[1,6,8] Compressors draw in external air and force it into the cabin. The outflow of cabin air is regulated via outlet valves to achieve a pressure differential of approximately 8.6 pounds per square inch (psi) between the cabin and outside environment during flight.[1] The pressurization is added to the ambient BP at a given altitude, resulting in a cabin altitude of 5,000 ft (1,529 m) to 8,000 ft during most cruising altitudes. Sea-level cabin BP and Po_2 can be maintained during flights at ≤22,500 ft (6,858 m) in most pressurized aircraft[1] but is neither cost-effective nor necessary at higher altitudes from the perspectives of structural design, operating efficiency, and, in most cases, the well-being of the passengers. In fact, this ideal pressurization can significantly weaken the structural integrity of aircraft which undergo frequent pressurization-depressurization cycles over time. The Federal Aviation Administration (FAA) requires aircraft to maintain an 8,000-ft cabin altitude at the highest operating altitude.[9] However, the regulations[10] for actual flight operations are more complicated and

flexible, allowing for temporarily higher flight and cabin altitudes such as during turbulence or adverse weather. Cabin altitudes greater than 10,000 ft are effectively prohibited since all flight crew and passengers must then use supplemental O_2.

A wide range of cabin altitudes can occur during individual flights and among different types of aircraft, rendering the prediction of cabin altitude to be inexact. The differential pressurization of commercial jets varies according to the type of aircraft and outside altitude.[6,11,12] Some aircraft may be unable to operate at designated peak differential pressurization, resulting in higher cabin altitudes. Aircraft of the same or different model may have identical pressurization capabilities but may be otherwise designed to cruise at different flight altitudes, such as seen with new-generation aircraft that fly several thousands of feet higher than older models.[12] The flight altitude is also influenced by changing weather, turbulence, or air traffic. In one study,[12] the median cabin altitude was 6,214 ft (1,894 m) with a range of 0 (sea level) to 8,915 ft (2,717 m) during cruising altitudes between 10,000 and 60,000 ft (18,288 m). The difference in cabin altitudes between domestic and intercontinental flights was not statistically significant. However, new-generation aircraft had significantly higher mean cabin altitude (7,004 ft or 2,135 m) than older aircraft (5,820 ft or 1,774 m), indicating that modern aircraft generally fly higher than their forerunners and expose passengers to greater hypoxia.

Responses to Altitude Hypoxia

The acute physiologic responses to high altitude have been reviewed elsewhere,[7,8,11,13-17] although the effects in individuals with preexisting cardiopulmonary conditions are less understood. Briefly, acute hypoxia in normal individuals initiates reflex responses that reduce the P_{O_2} gradient between the atmosphere and body tissues and prevent a large fall in PaO_2.[1,7,8,14,16] Hypoxia-induced stimulation of peripheral chemoreceptors (carotid bodies) varies in threshold and magnitude of response from individual to individual. Hyperventilation is the primary physiologic response to acute hypoxia and maximizes alveolar P_{O_2} (P_AO_2) and PaO_2, assuming that O_2 consumption is stable. Minute ventilation increases, primarily as a result of increased tidal volume rather than tachypnea. Concomitant respiratory alkalosis may blunt the hypoxic response. The alveolar-to-arterial P_{O_2} gradient ([A-a]P_{O_2}) narrows because of the steepness of the oxyhemoglobin dissociation curve at low P_{O_2}. Thus, most healthy individuals initially demonstrate a resting PaO_2 of 50 to 60 mm Hg and arterial oxygen saturation (SaO_2) of 80 to 90 percent at 8,000 to 10,000 ft.[17]

Blood flow and O_2 delivery to the heart and brain (organs with high O_2 requirements) are normally maintained during acute hypoxia.[14] Clinically benign and reversible hypoxia-induced pulmonary vasoconstriction increases pulmonary arterial pressure and pulmonary vascular resistance in proportion to the degree of hypoxia.[18] Cardiac output characteristically increases initially with hypoxia in a dose-dependent fashion,[19] primarily due to tachycardia. The cardiac response slowly decreases over time despite continued hypoxia for unclear reasons.[14] Hypoxia overcomes the cerebral vasoconstrictor effect of hyperventilation and dilates the cerebral vessels to maintain O_2 delivery within the brain. Subtle neuropsychologic deficits may be initially detected between 5,000 and 8,000 ft as altered perception, impaired judgment and vision, inefficiency of learning, and increased fatigability or drowsiness.[7,8,20] More severe symptoms of headache, nausea, listlessness, insomnia, altered personality and breathing pattern, seizures, and coma occur with severe hypoxia, suggesting a relationship with acute mountain sickness.[21]

The above compensatory responses to acute hypoxia may also occur to varying degrees in patients with cardiopulmonary disorders (Fig 1). However, the decrease in altitude oxygenation in patients with preexisting pulmonary disorders will depend largely on the individual's mechanism(s) for hypoxemia, the rate of ascent and the duration at the final altitude (which determine the pace of physiologic compensations), and the final (cabin) altitude achieved (which determines the maximum ambient P_{O_2} and PI_{O_2}).[16,22] The mechanism(s) of hypoxemia may be hypoventilation, ventilation-perfusion mismatching, shunting, diffusion impairment, low mixed venous P_{O_2}, or combinations of

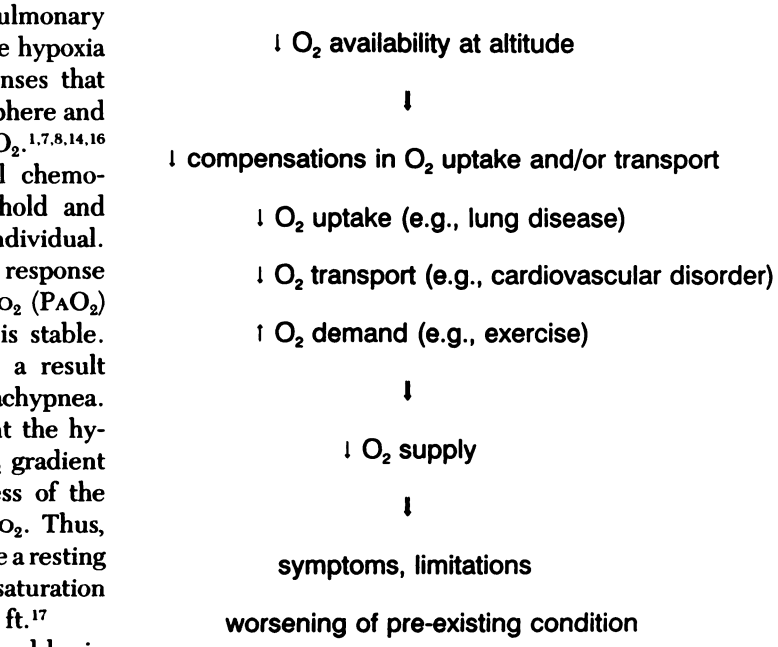

Figure 1. Pathophysiology of responses to acute altitude exposure in patients with preexisting cardiopulmonary conditions.

these abnormalities, resulting in an unpredictable net effect on gas exchange at altitude. Acute hypoxia may also increase airway resistance in patients with chronic obstructive pulmonary disease (COPD),[23] although neither this finding nor an enhancement of nonspecific bronchial hyperresponsiveness has been confirmed.[24,25] The arterial partial pressure of carbon dioxide ($PaCO_2$) changes minimally in patients with COPD exposed acutely to 8,000 ft, indicating less hyperventilation than expected with acute hypoxia.[22,26-28] Low PaO_2 at ground level may lie on the steep portion of the oxyhemoglobin dissociation curve and the SaO_2 will decrease rapidly at higher elevations.[17,22,26] Exercise at altitude increases O_2 demand and hypoxemia because of limited compensatory cardiopulmonary mechanisms.[29-32] Preexisting cardiac dysfunction and/or pulmonary hypertension in patients with pulmonary disease may significantly worsen during acute hypoxia,[33] preventing an increase in cardiac output necessary for adequate systemic O_2 transport. New or worsening cardiac arrhythmias, right-axis deviation, and right ventricular strain may occur without symptoms.[22] On the other hand, patients with either compensated congestive heart failure or chronic anemia alone can generally maintain adequate arterial oxygenation and tolerate the stress of altitude better than patients with respiratory disease[13,30] because of their intact capability to increase O_2 uptake with hyperventilation.

Other potential promoters of hypoxemia during flight may be clinically relevant. Personal or passive smoking increases the blood concentration of carboxyhemoglobin (COHb) and, thus, decreases the oxygen-carrying capacity of circulating hemoglobin.[8,22] Anemia, sedation, sleep, and drying of respiratory secretions (due to low cabin humidity) may increase hypoventilation, ventilation-perfusion mismatching, and/or work of breathing, resulting in further hypoxemia at altitude.[17,34] Immobilization during long flights predisposes individuals with preexisting venous disease to pulmonary embolism.[35] Expansion of trapped gases in the lung (within poorly ventilated bullae or blebs)[36] or abdomen may compress functioning lung. The decreased gas density at 8,000 ft does not significantly reduce turbulent flow in the airways to the extent of increasing maximum expiratory flow rates in healthy individuals and COPD patients.[37] Any beneficial effects may not be clinically significant until elevations $\geq$10,000 ft are reached[7,38] and are probably negated by the reduced PIo_2 and factors that increase lung distensibility, gas trapping, and maldistribution of ventilation during rapid ascent.[15]

Dietary intake may influence requirements for O_2 uptake by the lung and, possibly, arterial oxygenation.[39,40] Ethanol metabolism results in a low respiratory quotient (R) and respiratory exchange ratio (RQ) of 0.67 and decreases PAO_2 and PaO_2 during normoxia and hypoxia.[41,42] On the other hand, carbohydrate (CHO) metabolism results in a high RQ (1.00), and a high CHO diet (*eg*, >70 percent of total calories) can stimulate ventilation, resulting in higher PAO_2 and PaO_2 during hypoxia at 16,000 ft (3,350 m).[43] Thus, CHO feeding has been recommended for mountain climbers[43] and patients with pulmonary disease.[41-43] However, excessive dietary CHO concomitantly increases lipogenesis, which leads to an increase in CO_2 production, $PaCO_2$, and ventilatory requirements.[44-47] Whereas normal individuals increase ventilation, some patients with limited ventilatory reserve, such as with chronic airways obstruction or neuromuscular weakness, may not effectively increase ventilation. Hypercapnia may result or worsen, leading to greater hy-

Table 1—*Selected Surveys of In-Flight Medical Emergencies*

	Reference				
	61 (Speizer et al)	60 (Cummins and Schubach)	59 (Skjenna et al)	50 (Cottrell et al)	49 (Hordinsky and George)
Locus	Los Angeles	Seattle-Tacoma	Air Canada	United Air Lines	FAA
Period	10/1/85-3/31/86	9/1/86-8/31/87	1/1/88-12/31/88	7/1/86-6/31/87	8/1/86-7/31/87
No. of passengers ($\times 10^3$)	8,735	14,400	13,553	55,000	ns*
In-flight incidents, No. (%)	260 (0.003)	190 (0.001)	464 (0.003)	218 (0.0004)†	1,016†
Categories of incidents, No. (%)					
Cardiovascular	34 (13)	21 (20)	235 (50.6)	34 (15.6)	177 (17.4)
Respiratory	20 (7.5)	15 (7.9)	48 (10.3)	22 (10.1)	94 (9.3)
Neurologic	49 (18.8)	23 (12.1)	33 (7.1)	72 (33)	256 (25.2)
Gastrointestinal	69 (26.5)	28 (14.7)	59 (12.7)	ns*	109 (10.7)
Trauma	13 (5)	26 (13.7)	31 (6.7)	ns*	12 (1.2)
Other	76 (29.2)	60 (31.6)	58 (12.5)	90 (41.3)	368 (36.2)
Incidents/100,000 passengers	2.9	1.3	3.4	0.4	. . .
In-flight deaths, No.	7	0	2	3	9
Medical kit uses, No.	—	—	167	362	1,016

*ns = not stated.

†With medical kit use only.

poxemia, respiratory acidosis, and possibly acute ventilatory failure.[44] Even some increase in ventilation may result in greater work of breathing and dyspnea. Thus, it is premature to advocate high CHO intake at altitude in patients with advanced lung disease and who are prone to hypercapnia.

In-Flight Emergencies

Incidence data for health problems or emergencies and their causes during and after flight are incomplete and are likely underestimations due to the lack of standardized and medically accurate reporting.[34,48,49] A central registry that accumulates data about the diagnoses, disposition, and outcome of flying patients does not exist. Air carriers are not required to report in-flight medical events unless the emergency medical kit[49,50] is used, the flight is diverted, or a death of a passenger (or crew member) occurs. Anecdotal reports[51-53] and retrospective[2,54-59] and prospective[49,50,60,61] surveys have consistently indicated that cardiopulmonary and neurologic disorders are the most frequent causes of major in-flight morbidity and mortality (Table 1). The data suggest that the incidence of in-flight exacerbations of pulmonary disorders is very low. However, the number of at-risk passengers with preexisting pulmonary problems and of preventable in-flight events and the role of O_2 therapy during these events are unknown. A small number of studies[35,50,55,56,61] have evaluated the postflight medical status of ill passengers. The incidence of some disorders (*eg*, pulmonary embolism[35]) may be considerably underestimated since passengers may not manifest symptoms until after arrival. Thus, flight-related medical illnesses probably occur more frequently than reported or believed, although true emergencies are rare during flight. Similarly, in-flight deaths occur very infrequently, and respiratory-related deaths are rare.

Preflight Evaluation for Oxygen Therapy

The clinical significance of altitude hypoxemia and its role in in-flight morbidity and mortality remain unclear and speculative. Beighton[55] estimated that <50 percent of patients admitted to a hospital after deplaning had conditions exacerbated by altitude hypoxia. Results from studies in small numbers of pulmonary patients exposed to altitude hypoxia aboard aircraft,[26,62] in hypobaric chambers,[27-30,33,38] following rapid ground transport to a mountainous location,[31] and in controlled hypoxic breathing studies[22,26,32] indicate that stable patients with severe lung disease (primarily COPD) are relatively asymptomatic at rest and can maintain good short-term tolerance at 5,000 to 10,000 ft, despite PaO_2 of 30 to 40 mm Hg. These studies primarily evaluated stable, normocapnic patients at rest and without coexisting ischemic cerebrovascular and/or cardiac conditions. Thus, the advising physician must still rely largely on clinical evaluation and judgment and individualize recommendations (including O_2 therapy) for each patient who wishes to fly.[34]

The most specific and effective treatment of significant altitude hypoxemia is supplemental O_2.[22,27,32,63,64] The goal of O_2 therapy at altitude is to maintain adequate tissue oxygenation and to prevent hypoxemic complications. Although no specific criteria exist for recommending supplemental O_2 during flight, we either prescribe O_2 for patients who may develop a PaO_2 of less than 50 mm Hg at any airborne altitude or advise flying aboard an air ambulance or avoiding flying and using surface transportation.[34] (Similarly, the primary indication for O_2 therapy at ground level is a resting PaO_2 consistently ≤55 mm Hg on room air.[65]) Sea-level PaO_2 values of ≥68 mm Hg and ≥72 mm Hg in most normocapnic COPD patients result in a PaO_2 ≥55 mm Hg at 5,000 ft and ≥50 mm Hg at 8,000 ft, respectively.[22] Cottrell[12] estimated that a preflight PaO_2 of 70 mm Hg would be required to maintain an adequate SaO_2 at 6,214 ft. A preflight PaO_2 <70 mm Hg would require supplemental O_2. Pulmonary patients not already receiving supplemental O_2 should have pulmonary function and arterial blood gases measured to estimate altitude hypoxemia and tolerance. Patients already receiving continuous or intermittent O_2 therapy at ground level will require in-flight supplementation at higher fractional inspired oxygen (FIO_2), although the risks of hypercapnia and respiratory acidosis may be increased in some patients.[63]

Traditional contraindications for air travel in pulmonary patients have included a vital capacity <50 percent of predicted, maximum voluntary ventilation <40 L/min, respiratory acidosis, and a PaO_2 <50 mm Hg.[1,17,29,30,34,66-69] However, most of these abnormalities do not adequately predict acute altitude intolerance or the ability to correct it with supplemental O_2.[22,26-32] Abnormal diffusing capacity for carbon monoxide[22] and exercise studies[26] do not adequately predict altitude PaO_2. The ground-level PaO_2 is superior to spirometric values, lung volumes, diffusing capacity, SaO_2, (A-a)PO_2, and (a/A)PO_2 in predicting altitude PaO_2.[22,26-28,70] Equations and nomograms[22,27,28,62] are available for predicting acute PaO_2 and assessing the need for O_2 therapy at moderate altitudes (5,000 to 10,000 ft). The predictive accuracy of altitude PaO_2 improves if ground-level PaO_2 is measured within hours prior to flight,[26] although this practice is usually impractical. Nevertheless, it is still not possible to predict precisely the altitude PaO_2 of a given patient since the ability to compensate for a reduction in PIO_2 varies among individuals (even in healthy persons), depending on the ability of the patient to increase ventilation and the nature of the hypoxemia.[16] The

variability of cabin altitudes is another factor.[6,12] Furthermore, simply predicting altitude PaO_2 does not provide relevant information about individual cardiovascular, neuropsychologic, and other symptomatic responses at rest or during mild exertion or the effectiveness of supplemental O_2 at altitude.[22,48]

An altitude stress test is perhaps the most reliable and objective method of clinically assessing a patient's overall responses to a predetermined altitude. Supervised monitoring with noninvasive oximetry[71,72] or arterial blood gases[26,62] during actual flight is the most direct method. Alternatively, rapid surface ascent to a moderate altitude[31] may be considered. Finally, flight simulation in an altitude (hypobaric) chamber is an attractive test.[27-30,33,64] However, these methods are neither clinically practical nor cost-effective for most physicians. Thus, a preflight hypoxia-altitude simulation test (HAST)[22,26] can be readily performed in the pulmonary function laboratory to evaluate acute altitude-equivalent responses in gas exchange, symptoms, and cardiovascular function. The basic premises of the HAST are that altitude hypoxia is the primary stress or threat to patients with cardiopulmonary disorders[22,26] and that the FIO_2 at altitude can be replicated according to known pressure-altitude relationships.[7] This normobaric procedure uses a hypoxic breathing mixture (15 percent O_2) to simulate an anticipated altitude (usually 8,000 ft as the typical "maximum" cabin altitude) and monitoring with an oximeter or arterial catheter and electrocardiogram. The HAST-measured PaO_2 correlates well with actual altitude PaO_2.[26,28,70] The HAST can also involve exercise (*eg*, walking on a treadmill at the slowest speed)[32] and supplemental oxygenation.[22,32] These data can help the physician set altitude limits and determine appropriate O_2 therapy for individual patients. A HAST may be particularly relevant in patients with preexisting hypercapnia or concomitant neurologic and/or cardiovascular disorders.

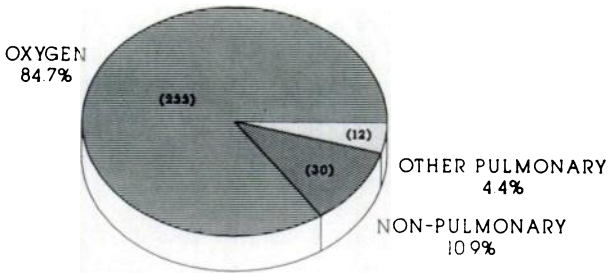

FIGURE 2. Distribution of 275 medical screenings by a commercial airline. The numbers in parentheses represent the number of screenings per category.

MEDICAL CLEARANCE PROCEDURES

Commercial airlines based in the United States are not legally obligated to accept all patients or to manage specific medical needs during flight.[73] Patients with special needs or services must satisfy the individual airline's policy regarding medical clearance. Airlines typically require written medical clearance from the patient's physician >48 h prior to the scheduled flight. The physician's letter must contain information regarding the fitness, stability, and safety of the patient for commercial air travel, as well as the patient's diagnosis, specific travel conditions and requirements, and authorization for the airline to transport the patient at 8,000 ft. Generally, medical clearance is granted by the airline as long as the patient's condition is not contagious or a discomfort, interference, or hazard to the patient, other passengers, and the flight crew. Requests for special services (including O_2) must not be extraordinary or significantly different from those routinely extended to other passengers (unless the passenger is accompanied by a knowledgeable care-

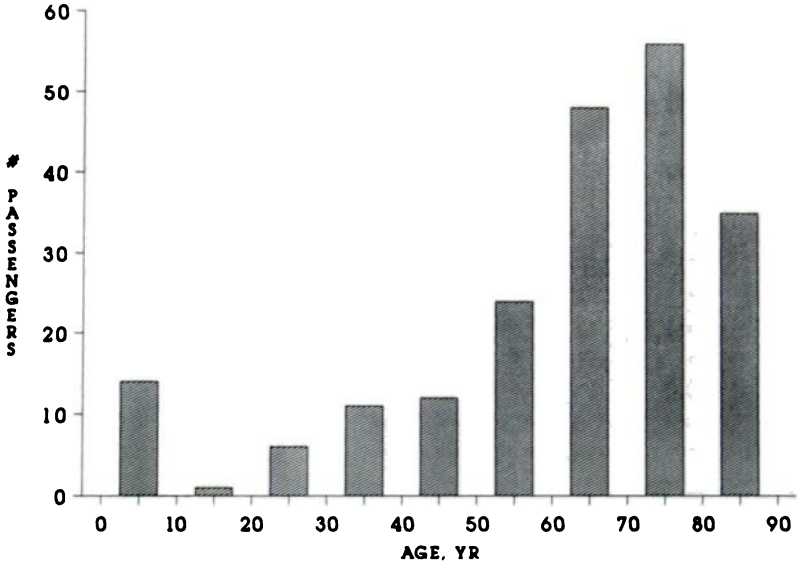

FIGURE 3. Distribution of ages of 215 passengers who were screened for in-flight oxygen.

giver). Air carriers will reject passengers known to be too unstable for flight or likely to interfere with the safety or punctuality of the flight which might result in diversion or unscheduled landing.

Use of nonemergent in-flight O_2 therapy is not infrequent. The precise number of passengers using supplemental O_2 aboard commercial aircraft is not known, but United, Trans World, and American Airlines carry 8 to 12 passengers with O_2 each week.[63] Thus, several thousand passengers fly with O_2 each year in the United States. One major American-based airline (Delta) referred 275 preflight screenings to a medical advisory service (Air Ambulance Inc, San Carlos, Calif) during January through March 1991. During this 90-day period, 233 (84.7 percent) evaluations involved requests for in-flight O_2 use (Fig 2). This group consisted of 130 female and 103 male subjects with a mean age (± 1 SD) of 58.9 ± 24.0 years (median, 67 years; range, 1 month to 94 years); 139 (59.6 percent) passengers were 60 years of age or older (Fig 3). The most frequent diagnostic categories (confirmed by patient, patient's family, and/or physician or nurse) were COPD (45 percent of passengers) and cardiac disorders (31 percent); 19 patients had combined COPD and cardiac disorders (Table 2). Five patients were flying to have an evaluation for heart and/or lung transplantation. Two hundred eleven (90.6 percent) patients were cleared for flight with an O_2 flow rate of 3 ± 1.2 L/min, usually via nasal cannulae. Five passengers had a tracheostomy and two used transtracheal catheters. Fifty-two patients (22.3 percent) had been discharged from an acute care hospital within two weeks of the scheduled flight. One hundred thirty-six (58.4 percent) passengers flew within the previous two years, and 59 (43.4 percent) in this group had flown with O_2. Sixteen passengers with COPD or other cardiopulmonary disorders had flown previously without O_2 and recalled in-flight dyspnea. The mean total duration for 228 flights was 54 ± 115 min (median, 210 min) and the total layover time in 111 itineraries was 98 ± 126 min (median, 60 min) with an average of one aircraft change during 97 flights, according to the passengers' booked flight schedules. Thus, these data indicate that in-flight supplemental O_2 is a recognized

Table 2—*Characteristics of 233 Passengers Evaluated for Commercial Air Flight and Oxygen Therapy**

	No. (%)	
Sex		
Male	103 (44.2)	
Female	130 (55.8)	
Diagnostic categories		
COPD	104 (44.6)	
Asthma	15 (6.4)	
Interstitial lung disease	14 (6.0)	
Cystic fibrosis	3 (1.3)	
Neuromuscular disorder	7 (3.0)	
Pneumonia (recent)	11 (4.7)	
Pulmonary embolism	3 (1.3)	
Pulmonary hypertension	6 (2.6)	
Lung cancer	18 (7.7)	
Other malignancy	9 (3.8)	
Cardiac disorder	72 (30.9)	
Seizure disorder	6 (2.6)	
Patients using medications	189 (81.1)†	
Bronchodilators	97 (51.3)	
Oral corticosteroids	56 (29.6)	
Cardiac medications	85 (44.9)	
Anticoagulants	12 (6.3)	
Antibiotics	21 (11.1)	
Antiseizure medications	7 (3.7)	
Other medications	59 (31.2)	
Preflight oxygen therapy	157 (67.4); 92 continuously 65 intermittently	
Discharged from hospital (<2 wk)	52 (22.3)	
Prior commercial flight (<2 yr)	136 (58.4)	
With supplemental oxygen	59 (43.4)	
		Mean + SD (median; range)
Scheduled flight segments, No.	375 (231 flights)	2 ± 0.8 (1; 1-4)
Plane changes, No.	109 (97 flights)	1 ± 0.4 (1; 1-2)
Total flight duration, min	54,519 (228 flights)	239 ± 115 min (210; 42-675)
Total layover time, min	10,843 (111 flights)	98 ± 126 (60; 10-210)

*COPD = chronic obstructive pulmonary disease.

†Total percent of subcategories exceeds 100 percent because of concurrent medication use.

airline service that is primarily used by an elderly population with chronic cardiopulmonary disorders and who are frequent fliers. A significant proportion of patients were recovering from a recent cardiorespiratory exacerbation. The mean duration of exposure to altitude hypoxia (in pressurized aircraft cabins) was <60 min although most flights lasted >3 h.

In-Flight Oxygen Therapy

Numerous practical, medicolegal, and policy issues regarding in-flight O_2 therapy must be recognized and resolved by patients and health providers.[34,48,74] Although many foreign-based air carriers permit passenger-supplied O_2, carriers in the United States cannot allow the in-flight use of personal O_2 systems, according to FAA regulations.[75] Any transported O_2 equipment belonging to a passenger must be emptied (to a pressure <40 psi) and shipped as luggage. The air carriers lack uniform or standardized information and guidelines about air travel with O_2.[34,48,74,76-78] Each carrier establishes its own policy regarding the processing of O_2 requests and administering O_2 aboard its aircraft. Although most major carriers supply requested O_2, some airlines impose rigid restrictions or categorically refuse to provide in-flight O_2, except in cases of emergencies (the onboard emergency units provide >20 min of O_2). Regional and local air carriers with commuter flights usually do not supply nonemergent O_2. This diversity of policies and lack of standardized rules and procedures governing supplemental O_2 during air travel make frustrations frequent and generalizations difficult. As a result, the potential flier should plan far ahead and obtain advice and assistance from knowledgeable or experienced physicians, nurses, respiratory therapists, oxygen vendors, rehabilitation programs, and airline special services. Other oxygen users who have previously traveled by commercial aircraft may be the most useful resource.[74] Experiences of passengers and caregivers[34,74,76-78] have generated many of the following recommendations and principles that are applicable to most patients who are medically cleared for air travel with O_2.

Oxygen Prescription

Air carriers in the United States require a physician's prescription stating the duration of in-flight O_2 use (intermittent or continuous) and flow rate at 8,000 ft. The carrier's medical department can recommend an appropriate FIO_2. A reasonable method of ensuring adequate oxygenation during flight (assuming no significant change in $PaCO_2$) is to maintain the FIO_2 or PIO_2 at a value that produces adequate oxygenation at ground level. This can be estimated by results from a HAST with supplemental O_2 or empiric calculation,[27,79,80] such as the following: $FIO_2 \times BP$ (ground level) = $FIO_2 \times BP$ (altitude). Approximately 30 percent FIO_2 is adequate at 8,000 ft for patients with readily reversible ventilation-perfusion mismatching and who are not using continuous O_2 on the ground.[80] The supply of supplemental O_2 must be matched for the flow rate, duration of O_2 usage and the flight, and some margin for preflight and arrival times and unexpected in-flight delays, *eg*, an additional 30- to 60-min supply of O_2. The patient should carry several copies of the O_2 prescription during the entire itinerary.

Oxygen Vendor

The airlines do not provide O_2 for ground (terminal) use. Planning a trip with a major O_2 provider is advantageous since the vendor may be able to arrange for O_2 and other respiratory services at layovers and at the final destination. A company representative may be able to meet the patient at the airport gate with a portable O_2 unit and to set up a stationary tank at the patient's lodging. Higher O_2 flow rates may be required if the elevation at the destination is higher than that at the patient's departure. The vendor can measure SaO_2 with an oximeter and determine an adequate flow rate in the new environment.

Personal Oxygen System

Passengers do not need to bring along their O_2 equipment (as checked in luggage) if comparable or identical substitute equipment can be arranged at the destination. A personal O_2 unit, when brought for later use, must be protected in a securely packed box or hard suitcase. The vendor at the final destination should check the patient's portable O_2 unit for proper functioning and assure its compatibility with the stationary O_2 reservoir. Unfortunately, universal or standardized fill adapters are not available for portable units from different manufacturers.

Airline Arrangements

The patient should always understand the carrier's policy regarding in-flight O_2 use and directly confirm that O_2 will be available during the entire flight itinerary. Airlines charge a basic service fee for providing in-flight O_2, ranging from $40 to $150, based on either the number of flight segments (*ie*, per landing or plane charge) or O_2 cylinders.[74] The patient should arrive at the airport early because the O_2 service charge is usually paid at the ticket counter and the transaction may take 20 min to process.[74] Passengers with O_2 should be able to board the plane early. The patient's insurance company or Medicare may reimburse the expense,[74] although reimbursement is variable even with submission of flight receipts. On flights of >6 h duration, smoking is frequently permitted in certain sections, although passive exposure to environmental tobacco smoke remains problematic

throughout the cabin.[81-83] Oxygen can only be used >10 ft from smoking sections. Seating near a lavatory is advisable. The flight attendants can provide only limited attention to any individual passenger and cannot administer medications or provide continuous medical assistance. Attendants can turn on and change the O_2 units but they are not trained in O_2 therapy or trouble-shooting malfunctioning systems.

Nonstop flights are recommended. Although itineraries can be arranged with ground-level O_2 therapy at layovers, nonstop flights (with adequate O_2 supply) avoid additional time and procedures at layovers and the purchasing and setting up of new O_2 units on the next aircraft. A direct flight (layovers on the same plane) is the next recommendation.

Onboard Oxygen Equipment

The commercial airlines are not regulated by industry standards or other regulations that standardize O_2 sources and delivery devices. Airlines provide either a face mask or nasal cannulae but usually not both.[63,74] Masks are provided most frequently and vary from airline to airline but are essentially rebreathing masks that deliver a high FIO_2.[63] Such masks are effective and safe for normocapnic patients but may be hazardous for hypercapnic patients. Significantly greater hypoxemia and hypercapnia occur when the mask is removed at altitude than were present prior to donning the mask,[63] thus predisposing the patient to severe tissue hypoxia, possible cardiac arrhythmias, and even sudden death. Controlled O_2 therapy via a Venturi mask is safer in hypercapnic patients since the FIO_2 at a given setting is accurately controlled, unaffected by hypobaric changes to 10,000 ft, and minimizes respiratory acidosis.[63] However, masks inhibit speaking and eating. Nasal cannulae are more comfortable than masks but may have limitations in providing an adequate FIO_2 during changes in ventilatory pattern (*eg*, while talking) and exercise.[84] Whereas both nasal cannulae (at 4 L/min) and Venturi masks (at 24 percent and 28 percent O_2) can effectively raise PaO_2 in COPD patients at 8,000 ft, only nasal cannulae significantly restore PaO_2 to ground-level values.[64] Nasal cannulae should always be carried aboard as a backup in case of malfunction of the provided delivery device. Patients with a tracheostomy can receive in-flight O_2 therapy via a tracheostomy collar. Similarly, oxygen-conserving cannulae and transtracheal or intratracheal catheters can be effectively used at altitude and reduce total O_2 requirements.[3] A crucial consideration is an adapter that connects the patient's delivery device with the aircraft's O_2 source since airlines use different types of O_2 units with variably sized outflow ports and a universal adapter is not available. Passengers can bring aboard and use different adapters or connectors and tape to accomplish this task effectively,[74] although airlines discourage this practice.

Oxygen in the form of compressed gas is most frequently available and contained in either large cylinders holding >3,000 L or small cylinders holding 300 L.[74] The large cylinders have an adjustable flowmeter with rates ranging from 2 to 8 L/min and are secured under the passenger's seat or the adjacent seat, which may need to be purchased. The small cylinders usually deliver only two predetermined flows: 2 L/min (lasting about 150 min) and 4 L/min (lasting about 75 min). Most onboard O_2 units have fixed flow rates. In units with adjustable flow rates, patients should carefully titrate the O_2 flow rate depending on their symptoms or when exercising or sleeping during the flight (although airlines discourage this practice). An adequate length of O_2 tubing is necessary for brief walks or visits to the lavatory. Regardless of the type of O_2 source, the user's leg or arm space may be restricted. Isometric exercises are encouraged to decrease venous stasis.

Other Methods to Improve Altitude Oxygenation

Alternative methods may maintain or improve PaO_2 at altitude. The patient must take adequate amounts of all prescription medications (*eg*, inhaled and oral bronchodilators or corticosteroids) on board and not leave them at home or in the checked-in luggage. Oxygen-dependent patients should avoid sedating medications, overeating (especially CHO), carbonated beverages, and ethanol-containing beverages during flights. Patients with a traheostomy or indwelling tracheal catheter or with abundant respiratory secretions must continue with ample oral hydration to couteract the dry air in pressurized cabins. Obviously, patients should avoid inhalation of respiratory irritants, including personal or passive tobacco smoke. Pursed lips breathing may temporarily increase SaO_2.[85] Almitrine bismesylate, a peripheral chemoreceptor agonist that increases PaO_2 during wakefulness and sleep in COPD patients at ground level,[86,87] may be beneficial at altitude. Unfortunately, almitrine is not commercially available in the United States, although some American patients with COPD have obtained almitrine from other countries. The efficacy of acetazolamide or medroxyprogesterone to improve oxygenation at altitude in patients with pulmonary disease is not known. Emergency O_2 units are aboard every commercial aircraft and are available if the passenger's purchased O_2 supply is depleted or malfunctioning and the patient has respiratory distress. If necessary, most domestic flights can land an unstable patient at an airport within 30 to 40 min. However, the decision to divert cannot be made lightly by the pilot because of the inconvenience and financial impact of a disrupted flight schedule. The crew will request the

voluntary advice and assistance of a passenger-physician (acting as a "good samaritan")[57,88] and inquire about the medical necessity of an unscheduled or priority landing. Alternatively, the pilot may cruise at a lower altitude (<22,500 ft) to restore a sea-level cabin pressure and possibly alleviate altitude-related (hypobaric) problems.

ACKNOWLEDGMENT: The author is grateful to Julia Lee Mark, R.N., C.C.R.N., and Michael N. Cowan, M.D. (Air Ambulance Inc) for access to their records.

References

1 AMA Commission on Emergency Medical Services. Medical aspects of transportation aboard commercial aircraft. JAMA 1982; 247:1007-11

2 Cummins RO, Chapman PJC, Chamberlain DA, Schubach JA, Litwin PE. In-flight deaths during commercial air travel: how big is the problem? JAMA 1988; 259:1983-88

3 Tiep BL, Lewis MI. Oxygen conservation and oxygen-conserving devices in chronic lung disease. Chest 1987; 92:263-72

4 Santoro K. A vacation cruise for COPD patients. Respir Ther 1985; 15:31-5

5 Burns MR. Cruising with COPD. Am J Nurs 1987; 87:479-82

6 Aldrete JA, Aldrete LE. Oxygen concentrations in noncommercial aircraft flights. South Med J 1983; 76:12-4

7 Luft UC. Aviation physiology: the effects of altitude. In: Fenn WO, Rahn H. Handbook of physiology; section 3: respiration. Washington, DC: American Physiological Society, 1965; 2: 1099-1145

8 McFarland RA. Human factors in relationship to the development of pressurized cabins. Aerospace Med 1971; 12:1303-18

9 Code of Federal Regulations, Title 14 CFR. Part 25.841. Washington, DC: US Government Printing Office, 1986

10 Code of Federal Regulations, Title 14 CFR. Part 121.327-.331. Washington, DC: US Government Printing Office, 1986

11 Liebman J, Lucas R, Moss A, Cotton E, Rosenthal A, Ruttenberg H. Airline travel for children with chronic pulmonary disease. Pediatrics 1976; 57:408-10

12 Cottrell JJ. Altitude exposures during aircraft flight: Flying higher. Chest 1988; 92:81-4

13 Luft UC. Physiologic alterations of altitude in aviation. In: Gordon BL, Careleton RA, Faber LP. Clinical cardiopulmonary physiology. 3rd ed. New York: Grune & Stratton, 1969:631-48

14 Lenfant C, Sullivan K. Adaptation to high altitude. N Engl J Med 1971; 284:1298-1309

15 Coates G, Gray G, Mansell A, Nahmias C, Powles A, Sutton J, et al. Changes in lung volume, lung density, and distribution of ventilation during hypobaric decompression. J Appl Physiol 1979; 46:752-55

16 California Thoracic Society. Travel at high altitude: information for physicians. Oakland, Calif: American Lung Association of California, 1989

17 Gong H Jr. Advising pulmonary patients about commercial air travel. J Respir Dis 1990; 11:484-99

18 Cudowicz L. Mean pulmonary artery pressure and alveolar oxygen tension in man at different altitudes. Respiration 1970; 27:417-30

19 Phillips BA, McConnell JW, Smith MD, Moffett C. The effects of hypoxemia on cardiac output: a dose-response curve. Chest 1988; 93:471-75

20 Kelman GR, Crow TJ. Impairment of mental performance at a simulated altitude of 8,000 feet. Aerospace Med 1969; 40:981-82

21 Houston C. Altitude illness and pulmonary edema. In: Simmons DH, ed. Current pulmonology. Chicago: Year Book Medical Publishers Inc, 1986:227-39

22 Gong H Jr, Tashkin DP, Lee EY, Simmons MS. Hypoxia-altitude simulation test: evaluation of patients with chronic airway obstruction. Am Rev Respir Dis 1984; 130:980-86

23 Astin TW, Penman RWB. Airway obstruction due to hypoxemia in patients with chronic lung disease. Am Rev Respir Dis 1967; 95:567-75

24 Parsons PE, Grunstein MM, Fernandez E. The effects of acute hypoxia and hypercapnia on pulmonary mechanics in normal subjects and patients with chronic pulmonary disease. Chest 1989; 96:96-101

25 Alberts WM, Colice GC, Hammond MD, Goldman AL. Effects of mild hypoxemia on bronchial responsiveness. Ann Allergy 1990; 65:189-93

26 Schwartz JS, Bencowitz HZ, Moser KM. Air travel hypoxemia with chronic obstructive pulmonary disease. Ann Intern Med 1984; 100:473-77

27 Mathur PN, Dowdeswell IRG, Stonehill RB. A method to assess and prescribe oxygen therapy during commercial air travel [abstract]. Am Rev Respir Dis 1984; 129(part 2):A51

28 Dillard TA, Berg BW, Rajagopal KR, Dooley JW, Mehm WJ. Hypoxemia during air travel in patients with chronic obstructive pulmonary disease. Ann Intern Med 1989; 111:362-67

29 Shillito FH, Tomashefski JF, Ashe WF. The exposure of ambulatory patients to moderate altitudes. Aerospace Med 1963; 34: 850-57

30 Tomashefski JF, Shillito FH, Billings CE, Ashe WF. Effects of moderate altitude on patients with pulmonary and cardiac impairment. Calif Med 1964; 101:358-62

31 Graham WGB, Houston CS. Short-term adaptation to moderate altitude: patients with chronic obstructive pulmonary disease. JAMA 1978; 240:1491-94

32 Bjorkman BA, Selecky PA. High-altitude simulation at rest and exercise to determine oxygen therapy needs in hypoxemic patients during airplane travel: a community hospital experience [abstract]. Chest 1988; 94(suppl):31S

33 Matthys H, Volz H, Ernst H, Konietzko N, Kleeberg HR. Kardiopulmonale belastung von flugpassagieren mit obstruktiven ventilationsstorungen. Schweiz Med Wschr 1974; 104: 1786-89

34 Gong H Jr. Air travel and patients with chronic obstructive pulmonary disease. Ann Intern Med 1984; 100:595-97

35 Cruickshank JM, Gorlin R, Jennett B. Air travel and thrombotic episodes: the economy class syndrome. Lancet 1988; 2:497-98

36 Parker GW, Stonehill RB. Further considerations of the roentgenologic evaluation of flying personnel at simulated altitude. Aerospace Med 1961; 32:501-04

37 Dillard TA, Berg BW, Rajagopal KR, Dooley JW, Mehm WA. Effect of altitude exposure on pulmonary function in chronic airflow obstruction (CAO) [abstract]. Am Rev Respir Dis 1989; 139(pt 2):A296

38 Finkelstein S, Tomashefski JF, Shillito FH. Pulmonary mechanics at altitude in normal and obstructive lung disease patients. Aerospace Med 1965; 36:880-84

39 Askanazi J, Weissman C, Rosenbaum SH, Hyman AI, Milic-Emili J, Kinney JM. Nutrition and the respiratory system. Crit Care Med 1982; 10:163-72

40 Dillard TA, Berg BW, Rajagopal KR, Mehm WJ. The respiratory exchange ratio and hypoxemia at altitude in COPD patients [abstract]. Chest 1990; 98:104S

41 Hansen JE, Claybaugh JR. Ethanol-induced lowering of arterial oxyhemoglobin saturation during hypoxia. Aerospace Med 1975; 46:1123-27

42 Hansen JE. Diet and flight hypoxemia. Ann Intern Med 1989; 111:859-60

43 Hansen JE, Hartley H, Hogan RP III. Arterial oxygen increase by high-carbohydrate diet at altitude. J Appl Physiol 1972; 33:

441-45
44 Covelli HD, Black JW, Olsen MS, Beckman JF. Respiratory failure precipitated by high carbohydrate loads. Ann Intern Med 1981; 95:579-81
45 Angelillo VA, Bedi S, Durfee D, Dahl J, Patterson AJ, O'Donohue WJ. Effects of low and high carbohydrate feedings in ambulatory patients with chronic obstructive pulmonary disease. Ann Intern Med 1985; 103:883-85
46 Berger R. Oxygen, food, and air travel. Ann Intern Med 1990; 112:236
47 Dillard TA. Dietary carbohydrate, alveolar gas, and chronic obstructive pulmonary disease. Ann Intern Med 1990; 112: 309-10
48 Gong H Jr. Advising patients with pulmonary diseases on air travel [editorial]. Ann Intern Med 1989; 111:349-51
49 Hordinsky JR, George MH. Response capability during civil air carrier inflight medical emergencies. Aviat Space Environ Med 1989; 60:1211-14
50 Cottrell JJ, Callaghan JT, Kohn GM, Hensler EC, Rogers RM. In-flight medical emergencies: one year of experience with the enhanced medical kit. JAMA 1989; 262:1653-56
51 Callanan JG, et al. Medical hazards of air travel. BMJ 1977; 1:1473-74, 2:44, 515, 637-38 and 1981; 282:320
52 Wakeford R. Death in the clouds. BMJ 1986; 293:1642-43
53 Cockerell CJ. Basic medical equipment on commercial airlines. N Engl J Med 1986; 314:1052
54 Iglesias R, Cortes MDCG, Almanza C. Facing air passengers medical problems while on board. Aerospace Med 1974; 45: 204-06
55 Beighton PH. Medical hazards of air travel. Practitioner 1967; 198:668-72
56 Richards PR. The effects of air travel on passengers with cardiovascular and respiratory diseases. Practitioner 1973; 210: 232-41
57 Hays MB. Physicians and airline medical emergencies. Aviat Space Environ Med 1977; 48:468-70
58 Davies GR, Degotardi PR. Inflight medical facilities. Aviat Space Environ Med 1982; 53:694-700
59 Skjenna OW, Evans JF, Moore M-S, Thibeault C, Tucker AG. Helping patients travel by air. Canad Med Assoc J 1991; 144: 287-93
60 Cummins RO, Schubach JA. Frequency and types of medical emergencies among commercial air travelers. JAMA 1989; 261: 1295-99
61 Speizer C, Rennie CJ III, Breton H. Prevalence of in-flight medical emergencies on commercial airlines. Ann Emerg Med 1989; 18:26-9
62 Henry JN, Krenis LJ, Cutting RT. Hypoxemia during aeromedical evacuation. Surg Gynecol Obstet 1973; 136:49-53
63 Stonehill RB, Peoples AG. The accuracy of venturi masks at altitude. Aviat Space Environ Med 1982; 53:818-21
64 Berg BW, Dillard TA, Mehm WJ, Dooley JW, Rajagopol KR. Oxygen supplementation during altitude exposure in COPD [abstract]. Chest 1988; 94(suppl):32S
65 Fulmer JD, Snider GL. American College of Chest Physicians (ACCP)-National Heart, Lung, and Blood Institute (NHLBI) conference on oxygen therapy. Chest 1984; 86:234-47
66 Committee on Medical Criteria of Aerospace Medical Association. Medical criteria for passenger flying: scheduled commercial flights. Arch Environ Health 1961; 2:124-38
67 Stonehill RB. Considerations for the ambulatory cardiopulmonary patient as concerned with commercial supersonic transportation. Dis Chest 1967; 52:606-10
68 Parsons CJ, Boechko WP. Aeromedical transport: its hidden problems. Can Med Assoc J 1982; 126:237-43
69 Mills FJ, Harding RM. Fitness to travel by air, I: physiological considerations. BMJ 1983; 286:1340-41
70 Apte NM, Karnad DR. Altitude hypoxemia and the arterial-to-alveolar oxygen ratio. Ann Intern Med 1990; 112:547-48
71 Cissik JH, Yockey CC, Byrd RB. Evaluation of the Hewlett Packard ear oximeter for use during routine air transport of patients. Aviat Space Environ Med 1981; 52:312-14
72 Mehm WJ, Dillard TA, Berg BW, Dooley JW, Rajagopal KR. Accuracy of oxyhemoglobin saturation monitors during simulated altitude exposure of men with chronic obstructive pulmonary disease. Aviat Space Environ Med 1991; 62:418-21
73 Code of Federal Regulations, Title 14 CFR. Part 382-Nondiscrimination on the basis of handicap in air travel. Washington, DC: US Government Printing Office, 1991:572-86
74 Livingstone G. Airline travel with oxygen. In: Travel for the oxygen dependent adventurer, ALA/ATS International Conference, May 14, 1991
75 Code of Federal Regulations, Title 14 CFR. Part 121.574. Oxygen for medical use by passengers. Washington, DC: US Government Printing Office, 1980:371-72
76 Poundstone W. Air travel and supplemental oxygen: friendly skies for respiratory patients? Respir Ther 1983; 13:79-82
77 Gong H Jr. Guidelines for travel with oxygen in advanced COPD. Respir Times 1988; 3:10-11
78 Maguire J, Randazzo J, Lundstedt D, Bobeck J. Requirements for traveling with oxygen. Am Assoc Respir Care (AARC) Times 1988; 12:31-5
79 Lachenmyer J. Physiological aspects of transport. Int Anesth Clin 1987; 25:15-41
80 Saltzman AR, Grant BJB, Aquilina AT, Ackerman NB Jr, Land P, Coulter V, et al. Ventilatory criteria for aeromedical evacuation. Aviat Space Environ Med 1987; 58:958-63
81 Committee on Airliner Cabin Air Quality. The airliner cabin environment: air quality and safety. Washington, DC: National Academy Press, 1986
82 Mattson ME, Boyd G, Byar D, Brown C, Callahan JF, Corle D, et al. Passive smoking on commercial airline flights. JAMA 1989; 261:867-72
83 Crawford WA, Holcomb LC. Environmental tobacco smoke (ETS) in airliners: a health hazard evaluation. Aviat Space Environ Med 1991; 26:580-86
84 Dixon JP. Arterial oxygen saturation at altitude using a nasal cannula. Aviat Space Environ Med 1982; 53:1207-10
85 Tiep BL, Burns M, Kao D, Madison R, Hererra J. Pursed lips breathing training using ear oximetry. Chest 1986; 90:218-21
86 Bell RC, Mullins RC, West LG, Bachand RT, Johanson WG. The effect of almitrine bismesylate on hypoxemia in chronic obstructive pulmonary disease. Ann Intern Med 1986; 105: 342-46
87 Gothe B, Cherniack NS, Bachand RT Jr, Szalkowski MB, Bianco KA. Long-term effects of almitrine bismesylate on oxygenation during wakefulness and sleep in chronic obstructive pulmonary disease. Am J Med 1988; 84:436-44
88 Zimmerly JG. More on in-flight emergencies. Md Med J 1987; 36:423-24