Aerospace Medicine: Part 2

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Citation

Abstract

AEROSPACE PULMONARY DISORDERS INTRODUCTION

There are many significant pulmonary problems in aerospace medicine. Occasionally, pulmonary problems in aviators are detected by abnormal pulmonary function tests (PFT's). Aeromedically, abnormal PFT's alone are not disqualifying but the aviator must be thoroughly evaluated. (7)

Some common conditions other than lung diseases, such as upper respiratory tract infections or chronic diseases also can increase the risk for serious complications in aerospace hypobaric setting. In addition, other hazards encountered in natural environments, such as high or low temperature, toxins, and pollutants, may amplify health risks from pressure changes. (6)

Environmental factors that may contribute to adverse outcomes in flight crew members include hypobaric hypoxemia, acceleration forces (+Gz) and anti-G maneuvers, breathing pure oxygen by mask, dry cabin air, ozone, smoke in the cockpit, and rapid decompression. (26) Other environmental factors associated with aerospace employment raise questions about possible causes of chronic occupational illnesses. These factors include disruption of circadian rhythms, exposure to noise and vibrations, cosmic radiation, and magnetic fields. (27,28)

An aging population combined with the increasing mobility of people with acute and chronic illnesses could make an increase in the frequency of in-flight medical events aboard commercial aircraft likely. In an interesting study, the demographic, medical, and flight characteristics of 1,115 passengers who were routinely referred for preflight medical screening by a major domestic air carrier were evaluated. The largest category consisted of 892 patients who requested in-flight O_2 therapy for cardiopulmonary conditions, of which COPD and cardiac disorders were the most frequent

indications; 561 of these patients were 60 years or older. The authors concluded that preflight medical screening and counseling can be important and helpful in assuring safe and comfortable air travel in this patient population. (29)

Medical complaints reported aboard a sample airline from July 1,1999 through June 30, 2000. There was an incidence of 22.6 medical complaints per million passengers and 0.1 deaths per million passengers. Variations in incidence of medical complaints cited in previous studies demonstrate the need for an industry-wide standardized reporting method of in-flight medical events. (30)

In an other study on 1197 reported in-flight medical events, the most frequent events were syncope or loss of consciousness; suspected myocardial infarction, angina, or chest pain; asthma; anxiety; otic barotrauma; and gastroenteritis. (31)

Since respiratory problems are estimated to make up about 11% of inflight emergencies it is reasonable to assume that the burden of risk surrounding the flight itself and later disruption of the journey is significant. (32)

People with abnormal lungs may be vulnerable to the relatively minor pressure changes by enlargement of a preexisting pneumothorax or rupture of an emphysematous bulla or other spaces containing air. People with respiratory disease who use long-term O_2 treatment will need to continue using O_2 during a flight. People with borderline hypoxemia at SL may also need supplementary O_2 to avoid becoming compromised at altitude. (33)

This section addresses selected acute illnesses that may occur in aerospace settings that related to respiratory system, including sudden decompression, altitude decompression sickness, venous gas embolism, pulmonary thromboembolism, pulmonary overinflation injury (systemic arterial gas embolism, pneumothorax), loss of consciousness

caused by gravity forces (g-LOC) and air-travel hypoxemia. Illnesses related to space travel and cardiopulmonary adaptations to chronic weightlessness are beyond the scope of this article.

1) SUDDEN DECOMPRESSION

It can occur following a breach in the fuselage of a pressurized aircraft, failure of cabin air compression systems, or an unrestrained ascent. Acute hypoxemia rapidly ensues because of reduced PaO_2. This occurrence limits subjects to a period of a few minutes to a few seconds, the "time of useful consciousness," to remedy the situation before hypoxic syncope ensues. Practical remedies consist of breathing O_2 from a contained source and rapid descent to lower altitude and higher PB. Commercial aircraft keep a temporary supply of O_2 for this contingency that typically cannot be accessed for one individual. (34)

The most effective method for providing physiological protection from reduced PB is aircraft pressurization. Aircraft pressurization is accomplished by increasing the PB above ambient pressure within the crew and passenger compartments, thus, in effect, reducing the cabin altitude to safe levels. During decompression the effects on the body are primarily due to rate of pressure loss from the aircrew compartment and to the pressure differential to which the body is exposed. (35)

In slow decompression, hypoxia symptoms may be the first alarm to a crewmember. While, in rapid decompression physiological injury occur, due to the expansion of gas in the lungs and gastrointestinal tract. There are also physiological consequences that can result after the decompression, such as acute hypoxia and decompression sickness. (35)

MECHANICAL EXPANSION OF GASES IN ACUTE DECOMPRESSION (35)

a) Gastrointestinal tract; One of the potential dangers during a rapid decompression is the expansion of gases within body cavities. The abdominal distress during rapid decompression is usually no more severe than that which might occur during slower decompression. The diaphragm is displaced upward by the expansion of trapped gas in the stomach, which can retard respiratory movements. Distention of these abdominal organs may also stimulate the abdominal branches of the vagus nerve, resulting in cardiovascular depression, and if severe enough, cause a reduction in blood pressure, unconsciousness, and shock.

b) The lungs; Because of the relatively large volume of air normally contained in the lungs, the delicate nature of the pulmonary tissue, and the intricate system of alveolar airways for ventilation, it is recognized that the lungs are potentially the most vulnerable part of the body during a rapid decompression. Whenever a rapid decompression is faster than the inherent capability of the lungs to decompress, a transient positive pressure will temporarily build up in the lungs.

If the escape of air from the lungs is blocked or seriously impeded during a sudden drop in the cabin pressure, it is possible for a dangerously high pressure to build up and to over distend the lungs and thorax. No serious injuries have resulted from rapid decompressions with open airways, even while wearing an oxygen mask, but disastrous, or fatal, consequences can result if the pulmonary passages are blocked, such as forceful breath-holding with the lungs full of air.

Under this condition, when none of the air in the lungs can escape during decompression, the lungs and thorax becomes over-expanded by the excessively high intrapulmonic pressure, causing actual tearing and rupture of the lung tissues and capillaries.

The trapped air is forced through the lungs into the thoracic cage, and air can be injected directly into the general circulation by way of the ruptured blood vessels, with massive air bubbles moving throughout the body and lodging in vital organs such as the heart and brain.

2) ALTITUDE DECOMPRESSION SICKNESS (DCS)

It can occur with ascent during flights, albeit much less commonly than after ascents from diving. The risk for altitude DCS increases with the degree of hypobaric exposure, duration of exposure, and exercise, and decreases with the duration of pre-exposure O_2 treatment. (36) The threshold for DCS has been described variously as 18,000 ft (5487 m) to 25,000 ft (7620 m) above SL. Exposure to 35,000 ft without preoxygenation (breathing 100% O_2 prior to decompression) can result in severe DCS. Exercise while decompressed increases the incidence and severity of symptoms. The subjects were monitored for venous gas emboli (VGE) with an echo-imaging system and observed for signs and symptoms of DCS. (37)

Incidence of decompression sickness Webb et al. (38) reported that DCS developed in 55% of subjects without prior depletion of O_2 at 22,500 ft. Exposures involving

strenuous and mild exercise resulted in higher incidence (p< 0.05) and earlier onset of symptoms (p< 0.05) of DCS than exposure at rest. Incidence at 30 min of exposure was 8% at rest and 23% while exercising. The results showed that current guidelines for 35,000-ft exposures keep DCS risk below 10% at rest. Exercise, even at mild levels, greatly increases the incidence and rate of onset of DCS. (37)

Data of another study indicated that repeated simulated altitude exposures to 25,000 ft with 100% O_2 breathing significantly reduce DCS and venous gas emboli incidence compared with a single continuous altitude exposure. (39)

PATHOPHYSIOLOGY OF PULMONARY BAROTRAUMA

The most serious problems resulting from a change in ambient pressure are pulmonary barotrauma with air embolism and DCS. Decompression sickness is a multiorgan system disorder that results from micro- or macroscopic N_2 bubble formation in the tissues from super-saturation of N_2 or helium absorbed under pressure. (40)

Serious DCS results from the entry of microbubbles into the systemic veins that cause injury by rupturing cell walls, accumulating in veins or arteries and impairing perfusion, activating coagulation or inflammation pathways, and other mechanisms. Large numbers of bubbles trapped in the lung cause an acute respiratory syndrome known as 'chokes'. If the lung filter is overwhelmed, or microbubbles pass into the systemic arteries through an atrial septal defect, they may open the blood-brain barrier, affecting brain and spinal cord function. Untreated DCS, demyelination with relative preservation of axons may occur, the pathological hallmarks of multiple sclerosis. The bubbles of respired gas that enter the systemic circulation often occlude cerebral arteries and may cause infarction. Joint pain--the 'bends'—is associated with gas in particular connective tissue. Gas bubble disease requires urgent compression in a hyperbaric chamber and the use of high partial pressures of O_2. Upper respiratory infections and allergic rhinitis are considered to increase the risk of barotrauma in the changing pressure environment. (41)

CLINICAL MANIFESTATIONS OF DECOMPRESSION SICKNESS

Susceptibility to altitude DCS is influenced by an individual's age. A study containing data on 1299 subject flight exposures conducted from 1983-94. Subjects were from 18-45 yr of age. Exposure altitudes ranged from 11,500 ft (3505 m) to 30,000 ft (9144 m). The results show a

significant three-fold increase in susceptibility between the age group 18-21 and the group > 42 yr of age. Therefore, there is a trend toward increased DCS susceptibility with increasing age, with a particularly strong trend for individuals over 42 yr of age. (42)

In most update study, about the effect of gender on DCS, no differences in altitude DCS incidence were observed between the sexes under the test conditions, although men developed VGE more often than women. Women using hormonal contraception showed significantly greater susceptibility to DCS than those not using hormonal contraception during the latter two weeks of the menstrual cycle. Persons of either sex with higher body mass index and lower physical fitness developed DCS more frequently. (43)

The symptoms of DCS occur from 10 minutes to 12 hours after surfacing in 90% of cases. Patients with type I DCS have mild manifestations, involving joint pain, (particularly in the knees), muscles, skin (pruritis), and lymph nodes. Patients with type II DCS have pulmonary, cardiac, or central nervous system involvement. Patients with pulmonary DCS may have prominent respiratory symptoms attributable to macroscopic nitrogen bubbles accumulating in the pulmonary artery ("the chokes"). Symptoms may include cough, dyspnea, chest pain or hemoptysis, and severe hypoxemia. (44)

Altitude DCS typically occurs during a flight because decompression occurs as the flight begins and symptoms typically improve with descent, but delayed symptoms occasionally occur. Treatment with O_2 and return to SL succeeds in most patients. Some patients require hyperbaric oxygen (HBO) for treatment for DCS. (44)

There was a study that reported the initial manifestations of DCS that occurred during a series of prospective hypobaric chamber studies. They found that of the 447 cases, 83.2% had musculoskeletal involvement, 2.7% had chokes, 2.2% skin manifestations, 10.8% paresthesia, and 0.5% frank neurological features. Their conclusion was that, the most common presenting feature was musculoskeletal, with knee pain predominating (occurring in 70% of these cases). A very low incidence of neurological features was seen in the database, which was in contrast to data from many other sources. Reasons for this difference may include the use of preoxygenation and the policy of prompt recompression upon symptom development. (44)

Type II altitude-related DCS, due to its wide spectrum of

symptoms, is often difficult to diagnose. A total of 133 cases of Type II altitude DCS were reviewed. The most common manifestation was joint pain (43.6%), associated with headache (42.1%), visual disturbances (30.1%), and limb paresthesias (27.8%). The next most common symptoms were mental confusion (24.8%), limb numbness (16.5%), and extreme fatigue 10.5%). Spinal cord involvement, chokes, and unconsciousness were rare (6.9%, 6%, and 1.5%, respectively). (45)

'Chokes' which are characterized by the triad of substernal pain, cough, and dyspnea, are considered to be associated with severe accumulation of gas bubbles in the pulmonary capillaries and may rapidly develop into a life-threatening medical emergency. Pulmonary altitude DCS with chokes (VGE that diagnosed with echo-imaging ultrasound) is confirmed to be a rare condition. When diagnosed early, recompression to ground level pressure and/or HBO treatment was 100% successful in resolving the symptoms. (46)

A retrospective study on reactions from 1986-94 in the hypobaric chamber-training unit at King Hussein Medical Center in Jordan was done; 39 cases were reported among 705 trainers in a 12-person rectangular hypobaric chamber. The most common reactions were found to be ear block (65%) and sinus block (25%). No any moderate or severe reaction. The authors concluded that, all reactions were minor, which reflects the efficacy of safety measures taken prior to and during training.(47)

TREATMENT FOR ALTITUDE DECOMPRESSION SICKNESS

Doppler-detectable microbubbles (DMB) are frequently used to evaluate altitude decompression stress due to its ability to detect microbubbles during decompression (Doppler test). The Doppler test was of greater utility in excluding DCS than confirming its presence, and was useful in making therapeutic decisions on DCS when confronted with non-specific symptoms at altitude. (48)

Treatment of DCS includes immediate administration of 100% O_2 and positioning the patient in the left decubitus and mild Trendelenburg positions. Patients with type II DCS should receive HBO as soon as possible. (49)

The mainstay of treatment of gas bubble disease is therapeutic recompression while the patient is breathing O_2. The patient should be recompressed as soon as possible; however, patients should be considered for recompression even after several days' delay. Treatments should be repeated if possible until symptoms have either resolved or stabilized. Appropriate hydration is essential. The use of HBO is generally safe, relatively nontoxic, and is possible even in neonates. Pharmacologic agents (e.g., anticoagulants, lidocaine, anti-platelet agents, corticosteroids, inhibitors of calcium flux) may be useful adjuncts to recompression therapy but they require further study. Portable hyperbaric chamber is recommended for the treatment of severe cases of AMS, as well as for risky descent to lower altitudes. (49)

Altitude decompression sickness has been treated with HBO therapy since 1941. Treatment has essentially followed the diving DCS paradigm. expanding space operations and higher flying, more remotely placed military aircraft have stimulated a re-examination of this paradigm. A new treatment table, which is USAF Treatment Table 8 (TT8) is applied. It consists of 100% O_2 delivered at 2 ATA for four 30-min periods with intervening 10-min air breaks (a total O_2 dose of 2 h). Treatment was successful in 91%. Its success suggests a new protocol for the treatment of altitude DCS. (50)

VENOUS GAS EMBOLISM

Venous gas embolism (VGE) occurs when gas enters the systemic venous system due to the presence of sub-atmospheric pressure in these vessels. The gas is transported to the lungs through the pulmonary arteries, causing interference with gas exchange, cardiac arrhythmias, pulmonary hypertension, right ventricular strain, and eventually cardiac failure. (51)

PATHOPHYSIOLOGY OF VGE

In insidious venous aeroembolism a series of gas bubbles resembling a string of pearls enters the venous system. Rapid entry or large volumes of gas put a strain on the right ventricle because of the migration of the emboli to the pulmonary circulation. The pulmonary arterial pressure increases, and the increased resistance to right ventricular outflow cause diminished pulmonary venous return. Because of the diminished pulmonary venous return, there is decreased left ventricular preload, resulting in diminished cardiac output and, ultimately, systemic cardiovascular collapse. The alteration in the resistance of the lung vessels and the mismatch between ventilation and perfusion cause intrapulmonary right-to-left shunting and increased alveolar dead space, leading to arterial hypoxia and hypercapnia. (16)

DIAGNOSIS

The so-called millwheel murmur, a splashing auscultatory sound due to the presence of gas in the cardiac chambers and great vessels, is often present and can be auscultated by a precordial stethoscope. A decrease in the end-tidal CO_2 levels, as determined by capnometry, suggests a change in the relation between ventilation and perfusion due to the obstruction of the pulmonary arteries. Doppler ultrasonography is a sensitive and practical means of detecting intracardiac air. More sensitive and definitive method for detecting intracardiac gas is transesophageal echocardiography. (51,52)

TREATMENT

Patients with VGE are placed in the flat supine position. In certain cases, therapy with catecholamines is required, and, if necessary, aggressive cardiopulmonary resuscitation is performed. Adequate oxygenation is often possible only with an increase in O_2 concentration of the inspired gas (up to 100 %O_2). Supplemental O_2 also reduces the size of the gas embolus by increasing the gradient for the egress of N_2 from the bubble. Rapid resuscitation with volume expansion is recommended to elevate venous pressure, thus preventing the continued entry of gas into the venous circulation. Some authors recommend attempting to evacuate air from the right ventricle with the use of a central venous catheter. Hyperbaric oxygen therapy is not a first-line treatment but may be a useful adjunct in severe cases. It should certainly be considered if there is evidence of neurologic changes. (51,16)

PULMONARY THROMBOEMBOLISM

Venous thromboembolism (VTE) (venous thrombosis and pulmonary embolism) has long been recognized as a potential complication of long-distance travel, particularly by air or by road. (53)

CAUSES

The predominant pathogenetic mechanism is no doubt a prolonged cramped position, leading in turn to blood flow stasis in the legs, which is one of the elements in Virchow's classic triad of the causes of thrombosis. (54) Additional environmental factors during air travel may possibly include dehydration and reduced atmospheric pressure with consequent desaturation. Other important risk factors, which apply in other circumstances and may be associated with VTE with aerospace medicine, are revealed as follows: (53)

Main risk factors for venous thromboembolism

- Older age (> 60 yr)
- Obesity
- Previous venous disease
- Immobility
- Cardiac failure
- Chronic medical illness
- Cancer
- Thrombophilia
- Recent major surgery or trauma (particularly involving the lower limbs)
- Contraceptive pill & hormone replacement therapy

INCIDENCE

Studies have shown an incidence of between 4.5% and 10% in DVT detected using ultrasound scanning in passengers flying for longer than 8 hours. (55) In a review of 182 cases of pulmonary embolism, 8 were reported to have been associated with long-distance travel. Another way of assessing the risk of travel has been to study patients admitted to hospital with VTE. Thus, among 207 such patients, 33 were found to have undertaken long-distance flights in the previous 31 days. (56)

A recent study has established that in high-risk subjects after long (>10 hours) flights the incidence of deep venous thrombosis (DVT) is between 4% and 6%. Below-knee, Scholl, Flight Socks, producing 14-17 mm Hg of pressure at the ankle, were used. The occurrence of DVT was evaluated with high-resolution ultrasound scanning. In total, 3.35% (6 subjects) had a thrombotic event. The difference (p<0.002) is significant. All thrombotic events were observed in passengers sitting in non-aisle seats. Also, Scholl Flight Socks are very effective in controlling edema and is effective significantly in reducing the incidence of DVT and thrombotic events in low-medium-risk subjects, in long-haul flights. (7-11 hours). (57) Moreover, Scurr et al. (55) demonstrated a reduction from 10% to 0% between two randomly allocated groups-one wearing Class-1 compressive stockings and the other not. Also, Belcarro et al. (58) reported a reduction from 4.5% to 0.24% in a similar type of study. The incidence in "high risk" passengers was 2.8% and 0% in the "low risk" group.

PROPHYLAXIS

There seems to be strong enough evidence to advocate the use of compressive elastic stockings in passengers with a recognized risk or in those particularly concerned. However, their use is unlikely to be widespread due to inconvenience. (53,55,57,58)

Prophylactic measures for all travelers include frequent active dorsiflexion of the foot while seated, occasional walks about the cabin, and physical activity at flight stops. (53)

MAINTENANCE OF FLUID INTAKE AND RESTRICTION OF ALCOHOL. (53)

Travelers with a mild to moderately increased risk may take low-dose aspirin (e.g. 160 mg) 2 hours before take-off and again 24 hours later, since this has been reported to reduce the risk of VTE by about one third. (59)

Travelers at moderate to high risk should consider requesting an aisle seat, the use of compressive stockings and be given low molecular weight heparin (e.g. dalteparin 5000 U or enoxaparin 40 mg) subcutaneously 2 hours before take-off. (54)

THE USE OF GRADED COMPRESSION STOCKINGS WITH EITHER ASPIRIN OR HEPARIN. (54)

In most update study, Campbell and Rayner (60) recommended the use of support tights or long socks with mobility and hydration in low risk passengers. In the passenger with a moderate to high risk of VTE, the recommendations are for compression stockings and aspirin or anticoagulation. While, guidelines for high risk individuals, pre-flight aspirin and use of compression stockings for every individual on hormone replacement therapy and the oral contraceptive pill. Unlike a postoperative patient, the traveler is mobile before and after flight as well as potentially during it. They added that, this information should be dispersed by airlines and public health agencies.

PULMONARY OVERINFLATION INJURY (POI)

Without exhalation, transpulmonary pressure within the lung increases as the ambient pressure declines upon ascent. Alveolar distention occurs, with the potential for tissue disruption. Factors that increase the likelihood of POI include ascending too rapidly, and having underlying lung disease such as bronchospasm, COPD, or a focal anatomic abnormality that traps air. (61)

Reviews of POI typically include findings of pneumomediastinum; systemic arterial gas embolism; pneumothorax; subcutaneous emphysema, and even pneumopericardium; pneumoperitoneum; and pneumoretroperitoneum. Such injuries can occur in normal subjects even with completely safe flying practices. As would be predicted by Boyle's law, the volume of a pneumothorax that occurs at depth will increase as ambient pressure declines during ascent. (61)

A proposed sequence for the pathogenesis of POI, that overinflation disrupts the continuity of the pulmonary epithelium. Alveolar rupture, particularly at the border of the alveolar base and vascular sheath, can occur at transpulmonary pressures of 80 mm Hg or less. (35)

Air enters the interstitial space and dissects centrally to the hilum along the bronchovascular compartment. If the mediastinal pleura remains intact, pneumomediastinum occurs without pneumothorax. From that point, subcutaneous emphysema or other manifestation can occur. Pneumothorax occurs if a defect develops in the mediastinal pleura, permitting air to enter the pleural space. Dissection of air to the periphery of the lung to cause pneumothorax also has been proposed. Direct entry of air into the pleural cavity through discontinuity in the visceral pleura can occur following piercing or blunt trauma to the lung. (62)

SYSTEMIC ARTERIAL GAS EMBOLISM (SAGE)

It can occur during air travel but is rare and usually is associated with underlying lung disease. Initial event consists of inflation of alveoli with tissue disruption or leakage of air into the interstitial compartment of the lung. Systemic arterial gas embolism (SAGE) is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation. Gas may enter the arteries as a result of over-expansion of the lung by decompression barotrauma. Although obstruction is possible in any artery, obstruction of either the coronary arteries or the nutritive arteries of the brain (cerebral arterial gas embolism) is especially serious and may be fatal. (16)

PATHOPHYSIOLOGY

Systemic arterial gas embolism (SAGE) may result from a variety of mechanisms. These include passage of venous air bubbles through an asymptomatic intracardiac shunt or through pulmonary overinflation injury with passage of air from the pulmonary interstitial space into pulmonary capillaries or veins. SAGE may occur after the accumulation

of bubbles in the pulmonary artery raises right heart pressure enough to create right-to-left shunt through a previously closed patent foramen oval (PFO). Other mechanisms such as passage of air bubbles directly through the pulmonary capillary bed or through a pulmonary arteriovenous malformation could contribute to the pathogenesis of SAGE. (63)

The entry of gas into the aorta causes the distribution of gas bubbles into nearly all organs. Small emboli in the vessels of the skeletal muscles or viscera are well tolerated, but embolization to the cerebral or coronary circulation may result in severe morbidity or death. Embolization into the coronary arteries induces electrocardiographic changes typical of ischemia and infarction; dysrhythmias, and cardiac arrest are all possible, depending on the amount of gas embolized. Cerebral arterial gas embolization typically involves the migration of gas to small arteries. The emboli cause pathologic changes by two mechanisms: a reduction in perfusion distal to the obstruction and an inflammatory response to the bubble. (64)

SYMPTOMS

The symptoms of SAGE develop suddenly. The clinical presentation, however, is determined by the absolute quantity of gas and the areas that are affected. Thus, there may be minor motor weakness and headache or moderate confusion; complete disorientation, hemiparesis, convulsions, loss of consciousness, and coma may occur. Asymmetry of the pupils, hemianopia, and impairment of the respiratory and circulatory centers (manifested as bradypnea, Cheyne–Stokes breathing, cardiac arrhythmias, and circulatory failure) are other well-known complications. (16)

DIAGNOSIS

In aerospace flier who has an injury such as DCS or SAGE, thorough evaluation is appropriate. Evaluation with pulmonary function tests, high-resolution CT scanning of the lung parenchyma, and bubble contrast echocardiography will detect the cause of many injuries. (6)

However, pathologic changes are sometimes very subtle and not well visualized on CT, and the diagnosis of cerebral arterial gas embolism must be considered early. Magnetic resonance imaging of the cerebrum can sometimes show an increased volume of water concentrated in the injured tissue. Another finding that is nonspecific but that has been described in a number of cases is hemoconcentration with an increase in the hematocrit, possibly as a direct consequence of the extravascular shift of fluid into the injured tissues. (65)

TREATMENT

The primary goal of treatment is the protection and maintenance of vital functions. If necessary, cardiopulmonary resuscitation should be performed. Oxygen should also be administered, at as high a concentration as possible. Hyperbaric oxygen is the primary therapy in established acute condition. (64)

Administration of O_2 is important not only to treat hypoxia and hypoxemia but also to eliminate the gas in the bubbles by establishing a diffusion gradient that favors the egress of gas from the bubbles. It is currently recommended that patients with SAGE be placed in the flat supine position. Adjunctive therapy in the form of lidocaine, antiepileptic agents and physical therapy may be indicated. (64)

b) Pneumothoraces may occur during air travel but do so relatively rarely considering the large number of people who travel by air each year. Expansion of a pre-existing subclinical pneumothorax during air travel also could occur and lead to symptoms during flight. Under such circumstances, a pneumothorax could expand by up to 34.5% of its initial volume in going from sea level to 8000 ft (2438 m). (62)

The safety of air travel for patients sustaining a recent traumatic pneumothorax has long been a subject of debate. The Aerospace Medicine Association has suggested that patients should be able to fly 2 to 3 weeks after radiographic resolution of their pneumothorax. To validate these recommendations, a prospective study was performed. Twelve consecutive patients with recent traumatic pneumothorax expressing a desire to travel by commercial airline were evaluated. Ten patients waited at least 14 days after radiographic resolution of their pneumothorax before air travel (mean, 17.5+/-4.9 days), and all were asymptomatic in-flight. One of two patients who flew earlier than 14 days developed respiratory distress in-flight, with symptoms suggestive of a recurrent pneumothorax. The authors conclude that commercial air travel appears to be safe 14 days following radiographic resolution of a traumatic pneumothorax. (66)

GRAVITATIONAL-FORCE LOSS OF CONSCIOUSNESS (G-LOC)

It is an interesting condition that afflicts pilots of high-

performance aircraft, typically military fighter jets, in situations that require tactical maneuvering for evasion. Transient reduction of cerebral perfusion leading to loss of consciousness can ensure. Gravitational forces occur with turning at high speeds and with rapid ascent or descent. Counter measures for g-LOC include positive-pressure breathing maneuvers that may have other consequences. The study of this condition typically includes noninvasive monitoring of cerebral O_2 saturation and systemic O_2 saturation during centrifuge experiments or other simulation. (67)

AIR-TRAVEL HYPOXEMIA

Travel by commercial aircraft is convenient, safe and increasingly being sought by patients with chronic pulmonary conditions. The major health concern with flying involves exposure to hypoxic stress, and how best to identify those at risk of having negative consequences. Commercial airplanes cruise between 6,700 and 13,400 meters above SL. At this altitude, hypoxia is incompatible with life. In modern aircraft, cabin pressurization varies to produce cabin altitude between 1,433 and 2,440 meters. Such exposures present no significant risk to normal subjects but can cause severe hypoxemia in patients with lung disease such as COPD even if home O_2 delivery is not required at SL. (68) This still represents a significant hypobaric environment, the PO_2 of inspired air is reduced, and therefore, the PaO_2 in the arterial blood of those with normal lungs will drop to 55 to 85 mmHg. In healthy individuals, the response to this hypoxic stress is hyperventilation and increase in cardiac output via tachycardia. The ability of the patient with cardiopulmonary disease to compensate would be variable. (69)

Traveling by means of a commercial aircraft is equivalent to visiting any location with an altitude of 6000-8500 ft for the duration of the flight. Studies in adults have confirmed the expected 6-8% decrease in baseline saturations. This is generally well tolerated because the patient is not only adapted to hypoxemia but is also at rest. As expected, cyanotic patients who tolerate living at moderate altitudes also tolerate commercial air travel, whereas patients who require O_2 supplementation at moderate altitudes should continue to receive O_2 during travel to lower altitudes. Patients at risk because of air travel are those who have previously demonstrated an intolerance to brief decreases in oxygenation. (70)

Published guidelines advocated maintenance of PaO_2 greater than 50 mm Hg during flight if the predicted in-flight PaO_2 is <50, then supplemental O_2 is prescribed. This recommendation seems appropriate for older patients with COPD with spirometrically severe but otherwise uncomplicated disease taking long flights. Maintenance of PaO_2 during flight equal to PaO_2 at ground level may be desirable for patients with COPD complicated by right heart failure or a need for home O_2 delivery, and for patients with multiple defects in organ function, such as lung disease, coronary artery disease, cerebrovascular disease, anemia, and other illnesses. (71)

Measurements of inflight cabin altitude on 204 regularly scheduled commercial aircraft flights were carried out on 16 different types of aircraft, operated by 28 airlines. Cabin altitudes ranged from SL to 8,915 feet (2717 m). Inspired PO_2 falls from 159 mm Hg at SL to 127 mm Hg at 6,200 feet and further declines to 113 mm Hg at 9,000 ft. New generation aircraft fly at higher altitudes than older aircraft and are associated with greater altitude exposures to passengers (p = 0.002). (35)

The recent statement, from the Canadian Thoracic Society regarding patients and air travel, underscored the importance of ABG's determination close to the travel date, as the best predictor of in-flight PaO_2. Those individuals with PaO_2>70 are unlikely to be at risk for significant in-flight hypoxic stress. Those with PaO_2 between 60 and 70 are in a gray zone where the in-flight PaO_2 is difficult to predict, and may depend on the ability to hyperventilate to compensate. They recommended high altitude stress test (HAST) for selected patients. They, like others, recommended O_2 at 2 liters/min for those gray zone patients requiring in-flight O_2, and an increase of 1 to 3 liters/min for those who had already been on home O_2 therapy prior to the flight. (72)

Patients with COPD often undertake airline travel without prior medical supervision. In one study, 38% of patients with severe COPD by spirometry took at least one airline trip within a 2-year period. A minority of patients (27.3%) consulted a physician before the trip, but 18.2% of patients reported clinical signs and symptoms during or after air travel. Patients who do not require home O_2 may not be aware of their potential risk for hypoxemia during air travel. Physicians can educate patients to seek evaluation for air-travel hypoxemia. (73)

Considerably fewer studies related to air-travel hypoxemia have been performed on patients with lung diseases other than COPD, such as cystic fibrosis (CF), interstitial lung disease, and sleep apnea. Patients with CF develop

hypoxemia during altitude chamber exposures but, because of younger age and less severe obstruction in some cases, some authors maintain that patients with CF may be more tolerant to moderate hypoxemia than older patients with severe COPD. (74)

The American Medical Association (AMA) takes action to improve airport and airline accommodations for passengers requiring medical O_2. The AMA Council on Scientific Affairs determined that commercial air travel exposes passengers to altitude-related hypoxia and gas expansion, which may cause significant symptoms and medical complications to some passengers during flight. Medical guidelines are available to help physicians to evaluate passengers who are at increased risk of inflight hypoxemia. Supplemental O_2 may be needed for some passengers to maintain adequate tissue O_2 and prevent hypoxemia. For safety and security reasons, federal regulations prohibit travelers from using their own portable O_2 system onboard commercial aircraft. Oxygen-dependent passengers must make additional arrangements for the use of supplemental O_2 in airports. Uniform standards are needed to specify procedures and equipment for the use of medical O_2 in airports and aboard commercial aircraft. (75)

The availability and cost of O_2 on commercial aircraft varies. Patients with heart and lung disease should contact their doctors early and arrange specialized assessment if necessary. Oxygen can be supplied via a Hudson mask (patients using venturi masks or nasal cannulae can bring these with them) at flows up to 4 l/min. (8)

Figure 1

Table (III): Clinical manifestations of some aerospace medical problems and selected causes

Manifestations	Causes
Chest pain	Coronary thrombosis Systemic arterial gas embolism Pneumomediastinum Pneumothorax
Dyspnea	Bronchospasm Pneumothorax Hypercapnia Anxiety
Hemoptysis	Pulmonary artery gas embolism Pulmonary edema Middle ear squeeze* Sinus squeeze*
Paralysis	Spinal decompression sickness Cranial artery gas embolism
Seizures	Central nervous system O_2 toxicity High pressure neurologic syndrome Systemic arterial gas embolism (SAGE) Decompression sickness
Loss of consciousness	Systemic arterial gas embolism Hypoxia Hypercapnia Nitrogen narcosis Hyperoxia Vasovagal syncope
Paresthesias	Cutaneous decompression sickness Spinal decompression sickness

*Conditions that may mimic hemoptysis.

References

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