

Aerospace Medicine: Part 1

A Darwish

Citation

A Darwish. *Aerospace Medicine: Part 1*. The Internet Journal of Pulmonary Medicine. 2002 Volume 3 Number 2.

Abstract

HISTORY OF AEROSPACE MEDICINE

As the human body was introduced to flight and its unfamiliar, hostile atmospheric environment, the medical profession had to become familiar with its effects in order to develop preventive measures. In this way, the field of aerospace medicine had its origin as aviation medicine.

Aerospace medicine has had rich history. It has evolved into a legitimate specialty through a long process of historical observation and technical achievement. However, most public literature on the topic has only covered information written through the late 1960s.

After World War II the technologic advances were applied to the airline industry. Increases in aircrew led to increases in civilian aviation medicine. In the 1960's advances were made to meet the challenge of manned atmospheric and space flight. Therefore, aviation medicine evolved to aerospace medicine.

WHAT IS AEROSPACE MEDICINE?

Aerospace medicine is a branch of preventive medicine that deals with the clinical and preventive medical requirements of man in atmospheric and space flight. It concerned with the physiological and psychological effects of living and working in an environment beyond the atmospheric and gravitational forces of the earth.

The 21st century technological advances are creating new demands and new opportunities in aerospace medicine to address the health needs of military and civil aircrew, astronauts, ground support personal, and airline passengers. Two areas of aerospace medicine are of particular general interest: the medical care of passengers aboard commercial aircraft and the application of telemedicine, which used in the space program since 1960s, to patient care on Earth.

FLIGHT SURGEON

“Flight Surgeon” (FS) is a vocation commonly known in the

USA and in Europe where there are many pilots. The FS is a doctor specialized in the health care for pilots and astronauts and in the aerospace medical research. His primary role is to ensure the health and safety of the pilots and air travelers. His work is mainly composed of selecting pilots, periodic medical examinations, health monitoring of the pilots before and during atmospheric and space flight. (3)

Pilots are examined annually to determine whether they may be participated in space flight. The examination includes physical examinations performed by the FS, clinical laboratory tests (analyses of blood, urine and feces), electrocardiography, pulmonary function tests, audio and visual examinations, and dental examinations. (4)

There are many problems with the abnormal environment encountered in aviation and space. The flight surgeon is just one of many highly trained individuals working to minimize the effects of these adverse effects so that man can continue to have mastery over the air and space. (3)

The scope of this review article is limited to piloting aircraft, passenger air travel, and mountain climbing. It considers the physiological response to aerospace environment and the manner in which common lung diseases may reduce tolerance to exposures. Also, it reviews aerospace pulmonary illnesses, altitude related disorders and fitness for flight duty. Moreover, potential complications for air travelers with respiratory disorders are summarized. Also, it includes guidelines and recommendations for preflight assessment for air travelers for patients with common pulmonary disorders. Lastly, telemedicine in respiratory diseases, which is the most recent revolution in aerospace medicine, has been highlighted.

THE ATMOSPHERE (AEROSPACE ENVIRONMENT)

COMPOSITION

The atmosphere surrounding the earth is a vast mixture of

gases and trace quantities of liquids and solids held earthward by gravity. The gases that make up the greatest percentage of the atmosphere are nitrogen and oxygen. The percentage or ratio of oxygen to nitrogen is constant up to an altitude of approximately 60 miles (96 kilometers) where layering begins. Water vapour varies with time, location and meteorological conditions as well as with altitude, since the presence of water vapour is governed by temperature. The primary particles, which make up cosmic radiations, are photons, neutrons, alpha particles and heavy nuclei.

The atmosphere has been classically considered as being divided into several concentric “shells” around the Earth, each with its own characteristics. These shells are Troposphere, Stratosphere, Ionosphere and Exosphere. The innermost shell is the troposphere, which extends from ground level to 9144 m (30,000 ft) at the poles and 18,288 m (60,000 ft) at the Equator. Conventional aircraft operates in this region. It is characterized by a relatively constant decline in temperature with increasing altitude at a rate of 1.98°C/305 m (1000 ft) ascent. Air is compressed by gravity. Therefore, atmospheric pressure is greatest at sea level and declines logarithmically with ascent.

The troposphere has constant composition containing 21% oxygen, 78% nitrogen, and 1% other gases (including argon and carbon dioxide, the latter being present at a concentration of 0.03%). It is the fall in the partial pressure of oxygen as total pressure declines on ascent that can give rise to hypobaric hypoxia, not a change in its percentage in air. Boyle's law predicts that, as pressure falls on ascent, there will be an inversely proportional increase in gas volumes. This affects body parts where gases are trapped, including the middle and inner ear, sinuses, and intestines. The same effect occurs in the lungs. The volume of a gas is also related to temperature, but the temperature of gases trapped in the body stays constant at 37°C.

To understand how the flight influences physiology and occasionally pathology, it is useful to consider the physiological properties of the atmosphere environment and changes that occur on ascent to altitude.

PHYSIOLOGICAL FEATURES OF THE ATMOSPHERE

The aerospace environment, it is the human's relation to that environment which is of primary concern. Therefore, it is useful to consider physiological responses at various levels of the atmosphere, and to divide the atmosphere into three physiological zones as follows:- (4)

1. **Physiological zone.** The region of the atmosphere to which humans are adapted physiologically extends from sea level (SL) to 10,000 feet. The oxygen (O₂) level within this zone is sufficient to keep a normal, healthy person physiologically fit without the aid of special protective equipment. The changes in pressure encountered with rapid ascents or descents within this zone can produce ear or sinus difficulties.
2. **Physiologically deficient zone.** This zone extends from 10,000 ft to about 50,000 ft (FL 500). Because of reduced atmospheric pressure, this is the zone in which O₂ deficiency becomes an ever-increasing problem. Supplemental O₂ is required when flying above 10,000 ft. Trapped gas in the intestinal tract, lung and evolved gas problems occur within this zone. In addition, protection must be provided against decreasing temperature.
3. **Space-equivalent zone.** From a physiological viewpoint this zone begins when 50,000 ft is reached since supplemental O₂ (100%), even when supplied under pressure, no longer protects one from the problem of hypoxia. The means of protecting a person above 50,000 ft are such that they will also offer protection in true space (i.e., pressure suits, sealed cabins). The only additional physiological problems occurring within this zone, which extends from 50,000 ft to 120 miles, are possible radiation effects and the boiling of body fluids in an unprotected individual. Boiling of body fluids will occur when the total barometric pressure (PB) is less than the vapor pressure of water at 37°C (47 mmHg), which is reached at 63,500 ft (Armstrong's Line).

Aerospace hypobaric exposures, including operation of jet aircraft and airplane travel, often involve rapid changes in total PB, partial pressures of gases, and acceleration forces that entail risks for barotrauma, decompression sickness, and other manifestations. Terrestrial hypobaric exposures, such as mountain climbing, usually involve a slower rate of change in conditions; permitting acclimatization to lower partial pressure of O₂, but often involve longer exposure, and variable temperature. (6)

HYPOBARIC ENVIRONMENTS: ALTITUDE

The barometric pressure of ambient air declines in a

nonlinear manner as altitude above SL increases. Table (I) shows the international standard atmosphere for elevations above SL and PB. These values represent average conditions. Local weather conditions can cause significant deviations from the standard atmosphere. Although the fraction of inspired O₂ (FiO₂) remains constant, at 20.9%, as altitude increases, the partial pressure of O₂ in ambient air declines progressively in proportion to declining barometric pressure. (6)

When one is breathing pure O₂ at 33,700 ft, the partial pressure of O₂ in the alveoli is the same as the pressure at SL when breathing air. Above 34,000 ft, the partial pressure of O₂ in the lungs begins to fall below the pressure at SL, even though 100 % O₂ is breathed. At altitudes greater than 40,000 ft, the partial pressure of O₂ decreases rapidly and falls below the limit that maintains the body in a physiologically safe condition. (6)

It is generally recognized that the most serious danger for the aircrew member is the decreased partial pressure of oxygen encountered at low barometric pressures. Without the proper use of oxygen equipment and cabin pressurization, hypoxia can quickly lead to incapacitation or death, depending on the altitude. (5)

Combination exposures, such as air travel followed by mountain climbing or skiing, frequently occur. Patients may seek medical supervision to accelerate acclimatization by pharmacologic means or to prepare for treatment of altitude sickness if it develops. The mountain climbing or air travel carries increased risk for decompression sickness. (6)

Figure 1

Table (I): Pressure equivalents at various altitudes above sea level

ft	m	mm Hg	kPa	ATA	PSIA	PN ₂	PO ₂	Vol
SL	SL	760	101	1.00	14.7	593	159	1000
3	0.9	684	91	0.90	13.2	534	143	1111
6	1.9	608	81	0.80	11.8	475	127	1250
10	3.1	532	71	0.70	10.3	415	111	1429
13	4.1	456	61	0.60	8.8	356	96	1667
18	5.6	380	51	0.50	7.4	297	80	2000
23	7.2	304	41	0.40	5.9	237	64	2500
30	9.4	228	30	0.30	4.4	178	48	3333
38	11.9	152	20	0.20	2.9	119	32	5032
53	16.6	76	10	0.10	1.5	59	16	10,000

ft = altitude in thousands of feet above sea level, m = altitude in thousands of meters above sea level, SL = sea level, kPa =kilopascal, ATA = pressure in atmospheres absolute pressure, PSIA = pressure in pounds per square inch absolute pressure, PN₂ =nitrogen partial pressure in mm Hg, PO₂ = oxygen partial pressure in mm Hg, Vol = relative volume of gas in milliliters. Assumes dry gas.

Cabin pressurization in modern aircraft ensures that the effective altitude to which occupants are exposed is much

lower than that at which the aircraft is flying. Commercial aircraft are not pressurized to sea level but to a relatively modest intermediate cabin altitude. This allows the aircraft to fly at much higher altitudes, which is fuel efficient for jet engines and more comfortable since it avoids much turbulence. Aircraft cabin altitude can thus approach 2438 m (8000 ft) while the aircraft is flying at 11,582 m (38,000 ft). Therefore, a pressure differential exists across the cabin wall, commonly of up to 9 pounds per square inch (psi). International aviation regulations stipulate that, at a plane's maximum cruising altitude, the cabin pressure should not exceed 2438 m (8000 ft). This may be exceeded in emergencies. (5)

In the event of failure of the cabin pressurization system at high altitude, all occupants would require supplemental oxygen to prevent an unacceptable degree of hypoxemia. Commercial aircraft are thus equipped with an emergency oxygen system for passengers, demonstrated before each flight in accordance with civil aviation regulations. However, some passengers with impaired respiratory function may be unusually susceptible to the effects of ascent even to normal cabin altitudes. (6)

PHYSIOLOGICAL RESPONSES TO AEROSPACE ENVIRONMENT

Although the vast majority of humans live in lowland areas of the Earth at SL or slightly higher elevations, increasing numbers of people work or engage in leisure activities at moderate-to-high altitude. The extremes of barometric pressure entail physiologic effects and hazards even for normal subjects. These effects typically result from rapid changes in total pressure or from changes in partial pressures of gases. (7)

OXYGEN AVAILABILITY AND ALTITUDE

Moderate altitude is defined as 5,000-10,000 ft above SL. As the altitude increases, the barometric pressure (PB) decreases. This fall in the PB affects the available PO₂. The O₂% remains stable at about 21%. At sea level, the partial pressure of O₂ available in the environment is equal to 0.21 times the PB (760 mm Hg), or 159 mm Hg. After saturation with water and expired CO₂, the partial pressure of alveolar O₂ (PAO₂) is 103 mm Hg, as calculated by the following equation: PAO₂ = FiO₂ (PB - PH₂O) - PaCO₂ [FiO₂ + (1 - FiO₂/R)]

(PB is the ambient barometric pressure, PH₂O is the pressure exerted by water vapor at body temperature, FiO₂ is the

fraction of inspired oxygen, $PaCO_2$ is the alveolar carbon dioxide pressure, and R is the respiratory exchange quotient.)

Although the $O_2\%$ in inspired air is constant at different altitudes, the fall in atmospheric pressure at higher altitude decreases the partial pressure of inspired oxygen and hence the driving pressure for gas exchange in the lungs. An ocean of air is present up to the ends of troposphere layer. The weight of air above us is responsible for the atmospheric pressure, (100 kPa at SL). This atmospheric pressure is the sum of the partial pressures of the constituent gases, oxygen and nitrogen, and also the partial pressure of water vapour (6.3 kPa at 37°C). (8)

The decrease in PB with increasing altitude results in a fall in the PaO_2 . Atmospheric pressure and inspired oxygen pressure fall roughly linearly with altitude to be 50% of the SL value at 5500 m and only 30% of the SL value at 8900 m (the height of the summit of Everest). For example, the PaO_2 decreases from 103 mm Hg at SL to 81 mm Hg in Denver, Colo (5,280 ft, 1,610 m), and 48 mm Hg at the top of Pikes Peak (14,110 ft, 4,300 m). (9)

A fall in inspired O_2 pressure reduces the driving pressure for gas exchange in the lungs and in turn produces a cascade of effects right down to the level of the mitochondria, the final destination of the O_2 . (8)

The lungs are a delicate interface between the atmosphere and our bodies across which O_2 diffuses from the air we breathe to the blood. In healthy lungs at SL where there is a surfeit of O_2 , this process occurs easily, whereas, in lungs with disease it becomes a task, which may not be fully successful and hypoxemia may ensue or worsen. At high altitude where the PB and thus the supply of O_2 is lower, the job of getting O_2 to the blood, even in the healthy lung is more difficult, and in the diseased lung it may be impossible. (10)

PHYSIOLOGICAL EFFECTS OF EXPOSURE TO ALTITUDE ON DIFFERENT ORGANS

1) LUNG

HYPOXIC VENTILATORY RESPONSE

At sea level CO_2 is the main stimulus to ventilation. At altitude hypoxia does increase ventilation (hyperventilation), but usually only when the inspired oxygen pressure is reduced to about 13.3 kPa (3000 m altitude). At this inspired oxygen pressure the PAO_2 is about 8 kPa, and with further

increases in hypoxia ventilation rises. This hypoxic ventilatory response is mediated by the carotid body, and response varies widely among subjects. However, the ability to tolerate altitude does not seem to relate to the presence of a brisk hypoxic ventilatory response. Some climbers with poor hypoxic ventilatory response do particularly well—for example, Peter Habeler, who in 1978 became (with Reinhold Messner) the first to climb Everest without oxygen. (8)

Breathing air at 2438 m (8000 ft) and 1524 m (5000 ft) is equivalent to breathing 15.1% and 17.1% O_2 at SL. In healthy subjects exposed to these conditions, their PaO_2 will be influenced by their age and minute ventilation but the PaO_2 fall to 53–64 mmHg (SaO_2 85–91%). However, healthy passengers do not generally have symptoms. (6)

Hypoxia in aviation is a syndrome that is usually acute and results from inadequate oxygenation of tissues secondary to a decreased partial pressure of O_2 in the inspired air. It is evident that all forms of hypoxia may become problems in flight. However, hypoxic hypoxia is the most common type reported in flying due to reduced atmospheric pressure that causes a reduced PAO_2 . (6)

STAGES OF HYPOXIA (8)

Hypoxia of high altitude may be divided into four stages related to the altitudes, and the O_2 saturations of the blood. This is illustrated in table (II):

Figure 2

Table (II): Stages of hypoxia

Stage	Altitude in Feet		Arterial O_2 saturation (%)
	Breathing air	Breathing 100% O_2	
1- Indifferent	0 - 10,000	34,000 - 39,000	95 - 90
2- Compensatory	10,000 - 15,000	39,000 - 42,500	90 - 80
3- Disturbance	15,000 - 20,000	42,500 - 44,800	80 - 70
4- Critical	20,000 - 23,000	44,800 - 45,500	70 - 60

In the Critical stage, there is loss of consciousness. It may be the result of circulatory failure (“fainter”) or a central nervous system failure (“non-fainter,” unconsciousness with maintenance of blood pressure). The former is more common with prolonged hypoxia, the latter with acute hypoxia. With either type there may be convulsions and eventual failure of the respiratory center.

CLINICAL PICTURE OF HYPOXIA

Hypoxia during flying most often results from depletion of compressed air supplies and other types of equipment failure. The manifestations of hypoxia begin when dry inspired PO₂ declines below 0.14 ATA (equivalent to 14% oxygen at SL). Tachycardia, increased systemic blood pressure, tachypnea, and cyanosis typically occur. Other manifestations include inability to concentrate on task, loss of coordination, loss of color vision, drowsiness, generalized weakness, incapacitation (0.11 ATA O₂), loss of consciousness (< 0.1 ATA O₂), and death. (6)

FACTORS INFLUENCING HYPOXIA (8)

The appearance of the signs and the severity of the symptoms of acute hypoxic hypoxia depend on the following variables:-

- a. Altitude level.
- b. Rate of ascent.
- c. Duration at altitude.
- d. Ambient temperature.
- e. Physical activity.
- f. Individual factors:-
 - Inherent tolerance.
 - Physical fitness.
 - Emotional state.
 - Acclimatization.

PROPHYLAXIS AND TREATMENT OF HYPOXIC HYPOXIA (11)

The treatment for hypoxia consists in giving 100% O₂ by inhalation. The aviator recognizing hypoxia must immediately switch to 100 % O₂ and emergency. If respiration has ceased, artificial respiration along with simultaneous use of 100% O₂ is indicated. If peripheral circulatory failure persists, the type must be determined and treated accordingly.

Recovery from hypoxia is rapid when sufficient O₂ is supplied. An individual on the threshold of unconsciousness may regain full faculties within 15 seconds after receiving an abundance of oxygen.

TIME OF USEFUL CONSCIOUSNESS

Time of useful consciousness (TUC) is the period of time from the interruption of the O₂ supply or exposure to an oxygen-poor environment, to the time when useful function is lost. The individual is no longer capable of taking proper

corrective and protective action. It is not the time to total unconsciousness. A rapid decompression can reduce the TUC by up to 50% caused by the forced exhalation of the lungs during decompression and the extremely rapid rate of ascent. (6)

CLINICAL MANIFESTATIONS OF HYPERVENTILATION (12)

a) Neuromuscular. The increased sensitivity and irritability of neuromuscular tissue, due to an elevation in blood pH, gives rise to a superficial tingling of the extremities (this tingling is not limited to the extremities, but is usually encountered there). The tingling usually precedes muscular spasm (i.e., a fixation of the hand wherein the fingers are drawn back toward the wrist). In severe cases facial muscles will be tetanically contracted, and the face will give an appearance of being pulled downward. The most dire and dramatic reaction is the “stiffening” of the entire body due to generalized muscular tetany. It is believed that a reduction in the partial pressure of alveolar CO₂ to 24-30 mmHg is the critical level for the onset of these symptoms.

b) Psychomotor. Deterioration of muscular control and coordinated activity is invariably seen during severe hyperventilation. Performance deterioration is encountered whenever the partial pressure of alveolar CO₂ is reduced below 25 mmHg. As the value falls below this level, performance deterioration becomes more marked.

TREATMENT OF HYPERVENTILATION (12)

Voluntary reduction in the rate or depth or both of respiration of the individual affected is the most effective method of treatment, when applicable. It is conceivable, however, that an extremely apprehensive person would not respond to directions to slow respiration.

It should be noted that the symptoms of hypoxia and hyperventilation are virtually indistinguishable. The individual must treat for both simultaneously. If either occurs, a decrease in the respiratory rate and breathing 100% O₂, will correct the condition. In the presence of hypoxia, if other disturbances coexist, or in more severe cases, it is imperative to return to ground level before more serious developments occur.

PULMONARY CIRCULATION AT HIGH ALTITUDE

In the systemic circulation hypoxia acts as a vasodilator, but in the pulmonary circulation it is a vasoconstrictor. The purpose of hypoxic pulmonary vasoconstriction is unclear. It

may help match ventilation and perfusion within the lung, but in hypoxia of altitude the reflex leads to pulmonary hypertension and is associated with high altitude pulmonary oedema (HAPE). (8)

An estimated 20% of the general population responds to a hypoxic stimulus with a marked increase in the pulmonary vascular resistance. These individuals are referred to as hyper-reactors. Clinically significant increases in the right ventricular pressure can be seen in such individuals who have an elevated pulmonary vascular resistance secondary to the hypoxic environment of a higher altitude. This can be exacerbated by acute pulmonary disease, exercise, upper airway obstruction, or congenital heart defects associated with an increase in pulmonary blood flow or restriction to pulmonary venous return. (13)

An increase in pulmonary vascular resistance is seen in normal subjects during hypoxic breathing at SL, in acclimatized lowlanders and in high-altitude natives. Hypoxic pulmonary hypertension in all these circumstances is most generally moderate, except in high-altitude natives at exercise. Pulmonary hypertension may become severe during HAPE, during infantile or adult forms of subacute mountain sickness, and during chronic mountain sickness. Subacute and chronic mountain sickness may be associated with a right heart failure that would be the human counterpart of brisquet disease described in cattle. (13)

Susceptible subjects will present with HAPE with a slight increase in pulmonary vascular resistance at rest and at exercise, and often with an enhanced pulmonary vascular reactivity to hypoxia compared to normal person. Noninvasive echo-Doppler studies of the pulmonary circulation at sea level are of little predictive value of tolerance to altitudes on an individual basis. (13)

GASEOUS DIFFUSION

At SL gaseous diffusion is probably limited by ventilation/perfusion matching in the lung. At high altitude, however, the alveolar-arterial difference for O₂ is higher than would be predicted from the measured ventilation/perfusion inequality. This is because the decreased driving pressure for O₂ from alveolar gas into arterial blood is insufficient to fully oxygenate the blood as it passes through the pulmonary capillaries. This is more evident on exercise as cardiac output increases and blood spends less time at the gas-exchanging surface (diffusion limitation). (9)

2) HEART

The heart works remarkably well at altitude. Initially there is an increase in cardiac output in relation to physical work but later this settles to SL values. At all times there is increased heart rate and decreased stroke volume for a given level of work, though the maximum obtainable heart rate falls as higher altitudes are reached. (8)

3) BRAIN

Hypoxia has progressive effects on the functioning of the central nervous system. Accidents that occur at extreme altitude on Everest and other mountains may be due to poor judgment as a consequence of hypoxic depression of cerebral function. More worrying is that these effects on cerebral function may be permanent. The American Medical Research Expedition to Everest studied its climbers a year after return to SL and found some enduring abnormalities of cognitive function and ability to perform fast repetitive movements, although most functions tested had returned to pre-expedition values. (8)

4) BLOOD

Initially on traveling to altitude, hemoglobin concentrations rise through a fall in the plasma volume due to dehydration. Later, hypoxia stimulates production of erythropoietin by the juxtaglomerular apparatus of the kidney so hemoglobin production increases and hemoglobin concentrations may rise to 200 g/l. The increased viscosity of the blood coupled with increased coagulability increases the risk of stroke and venous thromboembolism. Some authors advocate regular venesection in high altitude climbers; others recommend prophylactic aspirin. Neither has been shown scientifically to reduce the incidence of venous or arterial thrombosis. (14)

5) EFFECT ON PREGNANCY AND FETUS

The chronic hypoxia associated with moderate altitudes can affect the fetus. Birth weights are smaller, uterine blood flow is decreased, placental morphology may be different, and the incidence of prematurity and pregnancy-induced hypertension is increased at higher altitudes. Maternal smoking at high altitudes can have an additive effect. Travel of pregnant women from a low to a high altitude and vice versa has the potential to initiate premature labor caused by the changing PB on the amniotic sac. No difference in hematocrit levels has been reported in neonates born at higher altitudes compared with those born at SL. (15)

PULMONARY FUNCTION TESTS AT ALTITUDE

Lung function during altitude exposure has importance in

part because changes in lung function may worsen the severity of hypoxemia beyond that anticipated for ambient PB. Decline in lung functions also reduces ventilatory reserves available for exercise and may predispose to altitude illness. The subsequent paragraphs briefly review some studies that illustrate the changes in pulmonary function tests encountered in various altitude exposure scenarios. It should be kept in mind that, compared with altitude chamber studies, terrestrial field studies may have greater potential for factors such as acclimatization, weight loss, dehydration, sleep deprivation, thermal stress, exposure to pollutants, and other factors that confound results. (6)

Earlier studies described time-dependent changes in pulmonary function at altitude that may reflect adaptation in some cases. Gray and colleagues (16) previously reported a decrease in vital capacity (VC) in normal subjects at 5 hours that diminished by 12 hours of exposure to 14,000 ft of altitude. They also reported an increase in residual volume (RV) by helium dilution but no change in total lung capacity (TLC). Using body plethysmography daily for 6 days in normal subjects at an altitude of 3457 m (11,342 ft), Gautier et al. (17) also reported a time-dependent change in static lung volumes, including a reduction in VC. These investigators reported decreased elastic recoil at altitude, especially at low lung volumes.

In a study of an aerospace scenario, Dillard and co-workers (18) measured spirometric variables using pneumotachography and lung volume subdivisions by helium dilution in nine healthy subjects and 18 patients with chronic obstructive pulmonary disease (COPD) at SL and within 1 hour of exposure equivalent to 8000 ft (2438 m) above SL in a hypobaric chamber. Average forced vital capacity (FVC) declined by 0.123 L ($P < 0.05$). The magnitude of decline in FVC correlated with increasing RV ($P < 0.05$). Total lung capacity did not change significantly. Changes in spirometric variables; increased peak expiratory flow (PEF), reduced FEF₂₅₋₇₅, increased RV; for patients and controls did not explain significant variability in the arterial blood gas (ABG's) variables PaO₂ and carbon dioxide PaCO₂, or pH at this altitude. Peak expiratory flow increased slightly in the normal group ($P < 0.05$).

In a terrestrial field study, Pollard and coworkers (19) collected spirometric data at SL and after arrival at Mount Everest base camp in Nepal (altitude 5300 m) using a pocket turbine spirometer during the British Mount Everest Medical Expedition. A total of 205 spirometric measurements were made on the 51 subjects during the first 6 days after arrival.

Further measurements were made before and after inhalation of O₂ or a β_2 agonist. They found no evidence of hypoxic bronchoconstriction. Forced vital capacity fell at altitude, and there was a greater drop in FVC for subjects with lower pulse oximetry saturations and lower end-tidal CO₂ at this altitude.

Mason and colleagues (20) reported further on data from the British Medical Expedition. Forty-six subjects were studied twice daily during an ascent from 2800 m to 5300 m during a period of between 10 and 16 days. Forced vital capacity fell with altitude, by 4% from SL values at 2800 m, and 8.6% at 5300 m. Forced expiratory volume in 1 second (FEV₁) did not change with increasing altitude. Peak expiratory flow (PEF) increased by 8.9% at 2800 m, and by 16% at 5300 m. The increase in PEF was less than would be predicted from the change in gas density. Those authors reported no significant correlation between changes in spirometry and pulse oximetry saturation.

Welsh et al. (21) reporting altitude chamber data from Operation Everest II, previously described a decline in FVC by 4% at 15,000 ft and 13.6% at 29,000 ft from initial values at SL. They also observed interstitial pulmonary edema on post exposure chest radiographs.

Levine et al. (22) previously found that hypobaric hypoxia increased pulmonary lymph flow, a measure of pulmonary interstitial fluid flux and lung water, at moderate altitude (2600 m) and at higher altitudes (4600 m and 6600 m) in sheep. They mentioned that reduced PB alone caused some increase in lung water, independent of hypoxia, possibly because of alteration of Starling forces. The presence of subclinical edema during altitude exposure also has been suggested in other human study. (23)

Respiratory function test were measured at SL and at Everest Base Camp (5300 m). Mean FVC fell by 5%. FVC was lowest in the mornings and did not improve significantly with acclimatization. The cause of the fall in FVC at 5300 m is unknown but may be attributed to changes in lung blood volume, interstitial lung oedema, or early airways closure. Lower PEF values were observed on morning readings and were associated with higher acute mountain sickness scores. There was no change in forced expiratory volume in one second (FEV₁) at altitude. (8)

The preceding studies illustrate typical pulmonary function changes. Forced expiratory volume in 1 second and TLC change little. Decline in FVC can be detected around 2500 m

but may not be detected with milder exposures. Forced vital capacity declines further at higher altitude. Peak expiratory flow increases modestly at altitude in normal subjects. Correlation of spirometric changes with gas exchange variables may not be consistent or significant. Increasing RV mirrors decreasing FVC.

These pulmonary function changes with altitude exposure involve interesting questions of pathophysiology. Plausible mechanisms for changes in pulmonary function include pulmonary interstitial edema, pulmonary vasoconstriction, redistribution of pulmonary blood volume, and regional changes in lung elastic recoil, among others. (16,17,20-23) Mechanisms for reduced FVC that have less credibility include distention of abdominal gas (18) and reduced peak

respiratory muscle strength. (24)

A variety of portable devices developed primarily for home use or in-hospital bedside monitoring of PEF in asthmatic patients have emerged in the medical market place. Application of portable spirometers and peak flow meters in the setting of variable barometric pressure raises considerable concern for accuracy. Jensen et al., (25) tested several devices mechanically in an altitude chamber and found that portable flow meters underestimate PEF as a function of increasing altitude and increasing target peak flow.

See Aerospace Medicine: Part 2 for continuation

References

Author Information

Amr Abd-El Monem Darwish

Assistant Professor of Pulmonary Medicine, Faculty of Medicine, Minuffya University