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AEROSPACE INFORMATION REPORT

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Superseding AIR825/2

(R) Effects of Acute Altitude Exposure in Humans: Requirements for Physiological Protection

RATIONALE

AIR825/2 has been updated to reflect the latest developments in the consequences of altitude exposure in aviation: mild hypoxia and exposure to altitude after rapid decompression, taking in account the flight altitude and the decompression rate.

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1. SCOPE

The intent of this SAE Aerospace Information Report (AIR) is to describe the effects of the environmental changes on human physiology and the protection required to avoid negative consequences resulting from altitude exposure. A brief presentation of basic terms and considerations required to discuss the topic of human physiology at altitude is followed by an overview of the cardiovascular and respiratory systems. Issues specifically related to human exposure to altitude are discussed. Hypoxia, hyperventilation, barotrauma, and decompression sickness (DCS) are each addressed. One goal of this AIR is to demonstrate the necessity of oxygen use for prevention of physical and psychological problems, or loss of consciousness in an aircraft. This should provide a clear understanding as to why the use of supplemental oxygen is required for flight crew and healthy passengers at altitude greater than 10000 feet (3048 m).

1.1 Purpose

The purpose of this AIR is to describe the physiological needs for protection of humans against altitude in aviation.

2. REFERENCES

2.1 Applicable Documents

The following publications form a part of this document to the extent specified herein. The latest issue of SAE publications shall apply. The applicable issue of other publications shall be the issue in effect on the date of the purchase order. In the event of conflict between the text of this document and references cited herein, the text of this document takes precedence. Nothing in this document, however, supersedes applicable laws and regulations unless a specific exemption has been obtained.

2.1.1 SAE Publications

Available from SAE International, 400 Commonwealth Drive, Warrendale, PA 15096-0001, Tel: 877-606-7323 (inside USA and Canada) or +1 724-776-4970 (outside USA), www.sae.org.

AIR1069 Crew Oxygen Requirements Up to a Maximum Altitude of 45,000 Ft.

AIR6829 Oxygen Considerations for High Elevation Airport Operations (HEAO)

AS861 Minimum General Standards for Oxygen Systems

AS8010 Aviator's Breathing Oxygen Purity Standard

2.1.2 FAA Publications

Available from Federal Aviation Administration, 800 Independence Avenue, SW, Washington, DC 20591, Tel: 866-835-5322, www.faa.gov.

14 CFR Part 23 Airworthiness Standards: Normal, Utility, Acrobatic, and Commuters Category Airplanes

14 CFR Part 25 Airworthiness Standards: Transport Category Airplanes

TSO-C99 Flight deck (sedentary) Crewmember Protective Breathing Equipment

TSO C116a Crewmember Portable Protective Breathing Equipment

2.1.3 EASA Publications

Available from European Union Aviation Safety Agency, Konrad-Adenauer-Ufer 3, D-50668 Cologne, Germany, Tel: +49 221 8999 000, www.easa.europa.eu.

CS-23 Certification Specifications and Acceptable Means of Compliance for Normal, Utility, Acrobatic, and Commuters Category Airplanes

CS-25 Certification Specifications and Acceptable Means of Compliance for Large Aeroplanes

ETSO-C99 Protective Breathing Equipment

ETSO-C116a Crewmember Portable Protective Breathing Equipment

Commission regulation (EU) n° 965/2012: Technical requirements and administrative procedures related to air operations

2.1.4 NATO Publications

Copies of these documents are available online at https://www.sto.nato.int/publications/.

STANAG 3198AMD Physiological Requirements For Aircraft Oxygen Equipment And Pressure Suits.

2.2 Other Publications

2.2.1 General Publications

Gillies, J.A. (1965). “A textbook of aviation physiology.” *Pergamon Press*.

Gradwell, D. and Rianford, D. (2016). “Ernsting’s aviation and space medicine 5E.” *CRC Press*. ISBN 9781444179941.

U.S. Air Force. (1950). “German aviation medicine World War II.” *Surgeon General, U.S. Air Force*.

FAA. (n.d.). “Introduction to aviation physiology.” Available at https://www.faa.gov/pilots/training/airman_education/media/IntroAviationPhys.pdf.

2.2.2 Books or Papers

[1] Bert, P. (1979). “La Pression Barométrique (The Barometric Pressure).” Redited by *CNRS Paris*, Masson.

[2] Bert, P. (1943). “Barometric pressure,” translated by Hitchcock, M.A. and Hitchcock, F.A., *College Book Company*.

[3] Ernsting, J. (1986). “Mild hypoxia and the use of oxygen in flight.” *Aviat Space Environ Med, 55*(5):407-410.

[4] Gaume, J.G. (1970). “Factors Influencing the Time of Safe Unconsciousness (TSU) for Commercial Jet Passengers Following Cabin Decompression.” *Aerosp Med, 41*(4):382-385.

[5] Luft, U. (1965). “Aviation Physiology: The Effects of Altitude. Handbook of Physiology - Respiration.” Fenn, W.O. and Rahn, H., Eds., *Am. Physiol. Soc.*, Washington, DC: t.2, 44:1099-1145.

[6] Luft, U.C. and Bancroft, R.W. (1956). “Transthoracic Pressure in Man During Rapid Decompression.” *J Aviat Med, 27*:208-220.

[7] Marotte, H., Touré, C., Clère, J.M., and Vieillefond, H. (1990). “Rapid Decompression of a Transport Aircraft Cabin: Protection Against Hypoxia.” *Aviat Space Environ Med, 61*(1):21-27.

[8] Marotte, H., Touré, C., Florence, G., Lejeune, D., and Kerguélen, M. (1990). “Transport Aircraft Crew and Decompression Hazards: Study of a Positive Pressure Schedule.” *Aviat Space Environ Med, 61*(8):690-694.

[9] Otis, A.B. (1965). “Quantitative Relationships in Steady-State Gas Exchange. Handbook of Physiology - Respiration.” Fenn, W.O. and Rahn, H., Eds., *Am. Physiol. Soc.*, Washington, DC: t. 1, 27:681-698.

[10] Ruff, S. and Strughold, H. (1939). “Grundriβ der Luftfahrtmedizin.” Barth, J.A., Verlag - Leipzig.

[11] Violette, F. (1954). “Les Effets Physiologiques des Décompressions Explosives et Leurs Mécanismes.” *Rev. Méd. Aéro.*, 223-271.

[12] Wenzel, J., Luks, N., Plath, G., Wittkowski, M. (2009, May 8-12). “Rapid decompression from 8 to 45 kft in 20 seconds.” SAE-A10 Spring meeting, Denver, CO.

[13] Wenzel, J., Luks, N., Plath, G., Wittkowski, M., Bloch, N., Deutscher, W. (2011). “Rapid decompression from 8 to 45 kft in 20 seconds initially breathing air: experimental results from 10 human trials.” *Aviation, Space, and Environmental Medicine, 87*(3):249-250, abstract #127.

2.3 Definitions

BAROTRAUMA: Injury caused by pressure: most commonly referencing injury to the walls of the Eustachian tube and the ear drum due to the difference between atmospheric and intratympanic pressures, and

DECOMPRESSION SICKNESS (DCS): Related to an excess of nitrogen in the body tissues.

HYPERVENTILATION: An excessive rate of ventilation with ultimate consequences on acido-basic equilibrium,

HYPOXIA: An insufficient supply of oxygen to the tissues,

3. GENERAL

3.1 Units

In any serious discussion of altitude exposure, quantitative descriptions are required. The variables of primary interest being altitude, pressure, and temperature, a variety of units are commonly used with each of these parameters. Table 1 presents conversion factors for some of the most frequently used units for each of these parameters. In addition, the term Flight Level (FL) is commonly used in aeronautical practice. It is a valid reference for discussions of physiological limitations since it provides a common reference for the effects of pressure changes on the human body. It is, however, a valid means to determine absolute altitude with reference to sea level. Flight level is defined as the conventional altitude, expressed in feet, divided by 100 (for instance: FL 300 is 30000 feet, with pressure altitude equal to 301.5 hPa or 226.1 mm Hg).

The units of volume, in the metric system is dm^3 and the units of flow in the metric system is dm^3/s^{-1}. But the usual designation is the liter or liter per unit of time. In this text, we’ll use the liter (L), the liter per minute (L/min), and the liter per second (L/s).

Table 1 - Common conversion factors

Pressure to	**mm Hg**	**In H_2O**	**hPa**	**psi**
From	**Multiply by**			
mm Hg	1	0.5353	1.3333	0.0193
in H_2O	1.8683	1	2.4909	0.0361
hPa	0.7501	0.4014	1	0.0145
psi	51.7149	27.7000	68.9476	1

Length to	**Feet**	**Meters**
From	**Multiply by**	
Feet	1	0.3048
Meters	3.281	1
Volume: 1 l = 1 dm^3		

Temperature		
Fahrenheit (°F)	Celsius (°C)	Kelvin (K)
°F = 9/5 °C + 32	°C = 5/9 (°F - 32)	K = °C + 273.15

3.2 Mean Values and Safe Values

In many instances, data will be presented in this paper to demonstrate the concepts and issues being discussed. Confusion could result regarding the interpretation of these numbers if the differences between mean and safe values are not recognized. The mean of a set of data is the average value of all the observations. Usually, a measure of variability, such as standard deviation, among observations is also calculated. These measures are most meaningful when the observations are distributed in a manner that is referred to statistically as normal. For example, if one were to measure the height of 1000 individuals, the distribution of heights between the tallest and shortest individual would be expected to resemble that presented in Figure 1. Most biological data can be described or summarized in this manner. The sections of this AIR which address the fundamental functioning of human body will often utilize means and standard deviations to describe responses.

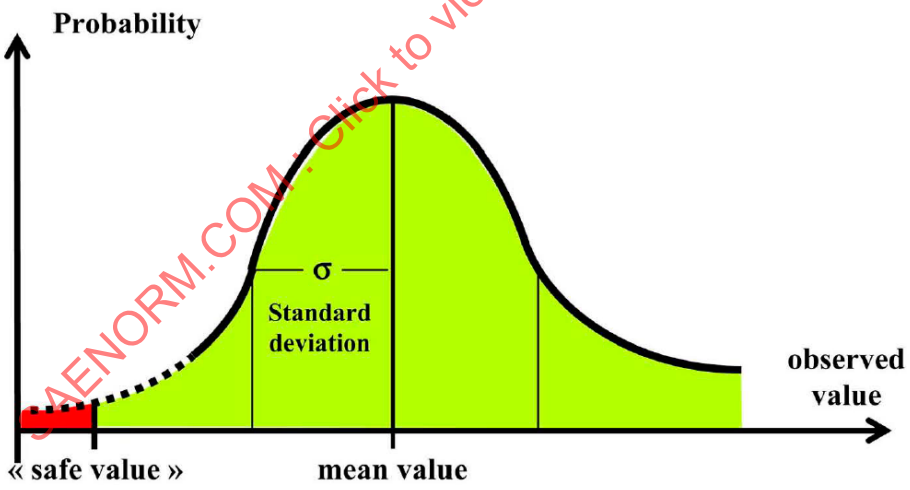

Figure 1 - Gauss curve, mean and safe values

NOTE: This curve describes the probability to observe a given value. The number of subjects, which are above this value, is in relation with the area under the curve. “Safe value” must leave ideally no subjects under the curve at the left of this value.

When the topic of discussion is the adaptative and performance limits of humans at altitude, a different perspective and analysis must be utilized. The object is flight safety. Therefore, statistically unusual responses must be considered. The part of this AIR which discusses data directly related with aviation safety will use safe data. Regulatory bodies often utilized safe data as the basis for their requirements. Although safe data can be described in statistical terms, mean data and safe data are not necessarily the same across the human population as a whole. For instance, subpopulations such as military people versus older passengers on commercial airline flights may be characteristically different for a given response.

4. ATMOSPHERE

As a biological species, humans are predominantly aerobic (i.e., dependent upon oxygen for metabolic energy production), requiring relatively specific environmental conditions for survival and comfort. Due to intelligence, humans have developed technologies that allow them to live in an extreme range of environments. Included within this range are the atmospheric changes associated with ascent to altitude in aircraft. If not controlled and regulated, the environmental changes associated with flying will result in physical and mental performance deficits that represent a serious threat to survival.

4.1 Atmospheric Pressure

Barometric pressure (P_B) decreases with altitude in an exponential relationship (see Figure 2). Just a few key values are required to show how fast the atmospheric pressure decreases as the altitude increases:

- Standard value at sea level (PB0): 1013 hPa (760 mm Hg)
- $P_{B0}/2$, i.e., 506 hPa (380 mm Hg) at about 5500 m (18050 feet)
- $P_{B0}/4$, i.e., 253 hPa (190 mm Hg) at about 10300 m (33800 feet)
- $P_{B0}/10$, i.e., 101.3 hPa (76 mm Hg) at about 16150 m (53000 feet)
- $P_{B0}/100$, i.e., 10 hPa (7.6 mm Hg) at about 30500 m (100000 feet)

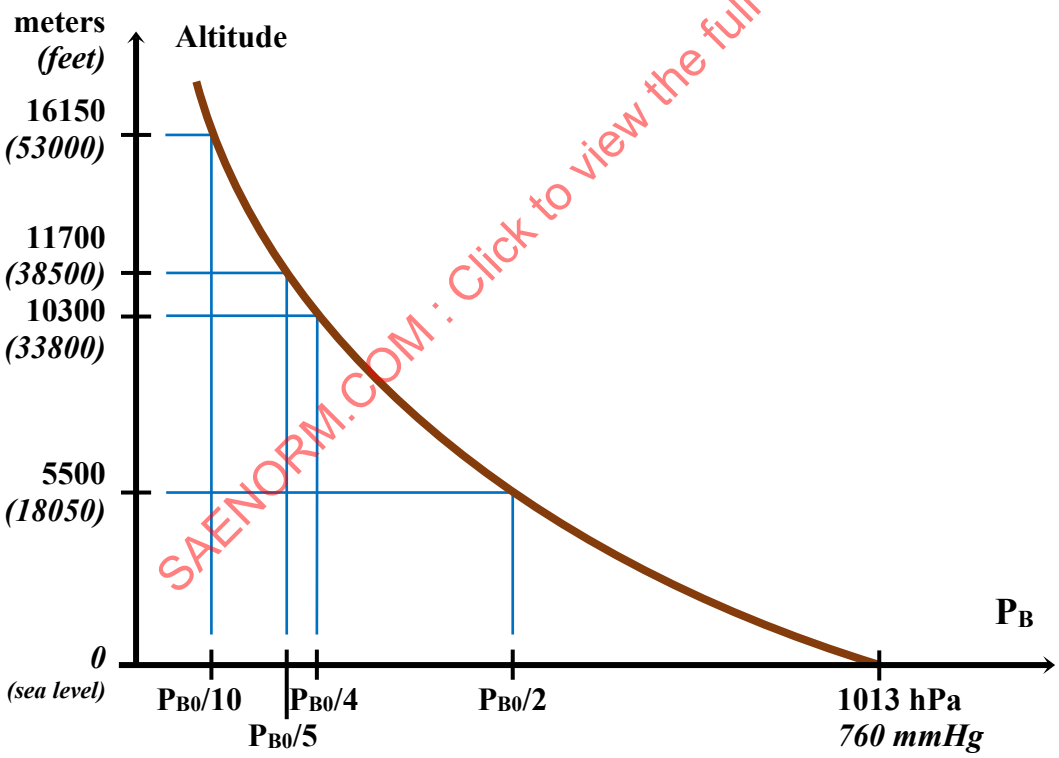

Figure 2 - Graph indicating atmospheric pressure (P_B) in relation to altitude

4.2 Temperature

The temperature of the atmosphere decreases as altitude increases, up to about 37000 feet (11300 m) where temperature reaches a mean value of approximately -56.5 °C (-69.7 °F, 216.7 K). Above this altitude, for all flight levels used in aeronautics, the temperature remains constant (Figure 3).

4.3 The Composition of the Atmosphere

For the majority of gases, the composition of the atmosphere is constant at all the flight levels operated in aeronautics. The primary exceptions are water vapor and carbon dioxide. Table 2 lists the components normally found in the lower layers of the atmosphere. Ozone is situated mainly between 50000 feet and 150000 feet (15 km and 50 km), with a maximum at 100000 feet (30 km).

For human respiration, the most important element in the atmosphere is oxygen. On a percentage basis, oxygen makes up 20.95% of the gas in the air. This percentage is important in that it is used in calculating the partial pressure of oxygen (P O_2). It is the P O_2 that provides the force to drive oxygen into the body. Since the percentage of oxygen in the air remains constant, the P O_2 varies in direct proportion with the atmospheric pressure. The gas pressures, partial or total, are most important when considering physiological parameters. The reason is the gas partial pressure will determine the extent to which the gas will be found in the body. Areas of the body containing gases can be divided into three categories:

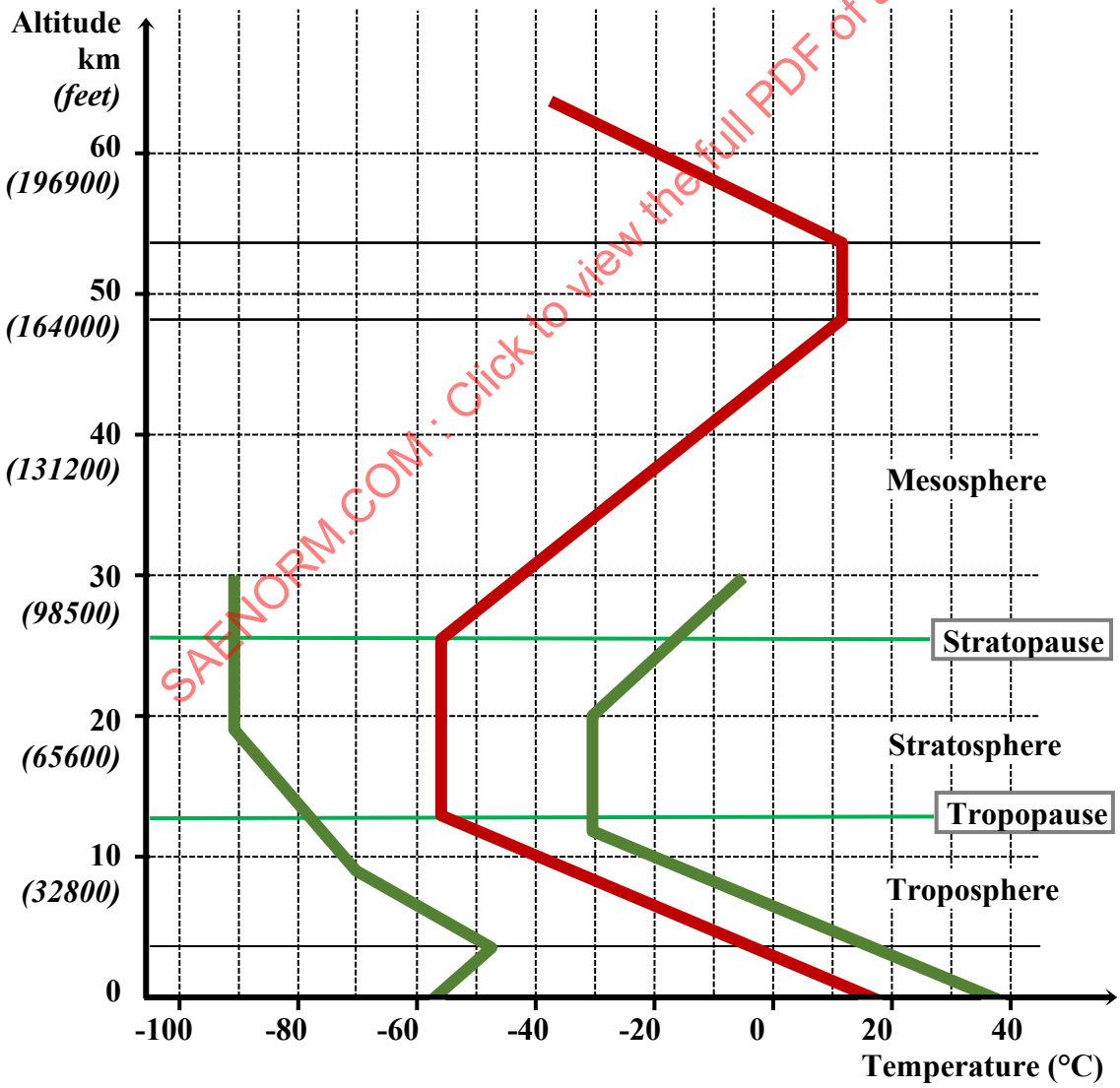

Figure 3 - Graph representing air temperature in relation to altitude

NOTE: Average temperature (central line) and extreme temperatures for a probability of 1 to 10 years, observed below latitude 45 °N between 0 feet and 200000 feet (0 m and 60000 m).

Table 2 - Composition of ambient air

Gas	Symbol	Fraction	Remarks
Nitrogen	N_2	0.78101	Constant
Oxygen	O_2	0.20946	Constant
Water	H_2O	$2 \cdot 10^{-2}$	Very variable
Argon	Ar	$9.17 \cdot 10^{-3}$	Constant
Carbon dioxide	CO_2	$3.30 \cdot 10^{-4}$	Variable
Neon	Ne	$1.82 \cdot 10^{-5}$	Constant
Helium	He	$5.24 \cdot 10^{-5}$	Constant
Methane	CH_4	$1.50 \cdot 10^{-6}$	Constant
Krypton	Kr	$1.14 \cdot 10^{-6}$	Constant
Ozone	O_3	$5 \cdot 10^{-7}$	Very variable

a. Gases in closed or semi-closed cavities. There are the gases contained in the digestive tract, the ear and sinus cavities, lungs, etc. They are believed to be the major cause of the pathological signs of barotrauma through the physical relationship defined by Boyle-Mariotte's Law (Pressure x Volume = Constant).

b. Gases dissolved in body fluids. These are the gases dissolved in the blood plasma, and other body fluids. The amount of gas dissolved in a fluid obeys Henry's Law which states that the quantity of gas dissolved in a liquid is proportional to the partial pressure of the gas in contact with the liquid. Movement of these gases out of body fluids are considered to result in the symptoms associated with the aviator's version of decompression sickness.[1]

c. Gases in chemical combination. The primary gases in respiration are oxygen and carbon dioxide. Blood transports a greater quantity of these gases than can be explained by simple adherence to Henry's Law. The explanation is the interactions of the gases with the red blood cells. Oxygen is chemically bound to hemoglobin. In normal conditions, 100 cm^3 of arterial blood contains 0.23 cm^3 (at 37 °C) of dissolved oxygen (by Henry's Law), and about 20 cm^3 of chemically combined oxygen. Carbon dioxide is a metabolic waste product which must be eliminated from the body. Carbon dioxide content is based on a complex set of equilibria, which involve dissolution of CO_2 molecules, followed by a subsequent combination with water. The result is a change in pH.

5. CARDIOVASCULAR AND RESPIRATORY ANATOMY AND PHYSIOLOGY

5.1 Blood Circulation

Blood is circulated through the body by the heart and vessels. The total volume of blood contained in these organs is approximately 4.5 dm^3 in normal adults.

5.1.1 Anatomy

The heart is a muscular pump divided into four chambers: left atrium, right atrium, left ventricle, and right ventricle. The right and left atria receive blood returning from the body and lungs, respectively, and work to assist in delivering the blood to the ventricles. The right ventricle pumps blood into and through the lungs (the pulmonary circulation). The left ventricle pumps blood into and through all other tissues (the systemic circulation). Separating atria and ventricles are muscular structures referred to as the interarticular and interventricular walls, respectively. The right heart and left heart are synchronized in rhythm, physiologically pumping an equal volume of blood through the pulmonary and systemic circulations. Blood circulations are organized in the following way (see Figure 4).

[1] Decompression sickness is a multifaceted condition brought about by a rapid change in atmospheric pressure. It is discussed in detail in Section 9.

Pressure resulting from the contraction of the left ventricle forces blood into an artery called the aorta. In the circulation, arteries carry oxygenated blood traveling away from the heart toward sites of cellular exchanges. Blood transporting the maximum amount of oxygen physically possible is said to be 100% saturated.[2] The aorta is the largest artery in the body and quickly gives rise to branches that ultimately supply blood to the entire body. The coronary arteries supply blood to the cardiac muscle and the carotid arteries supply the head. At the arch of aorta, the ascending aorta turns into the descending aorta, carrying blood to the trunk and lower limbs. As the large arteries travel throughout the body, they branch, giving rise to numerous small diameter vessels. The last small branches of the arterial system are referred to as arterioles. The role of the arteriole is to control the volume of blood flow into the capillaries. Capillaries are the smallest blood vessels in the body and are the site where gas exchanges between blood and cells take place.

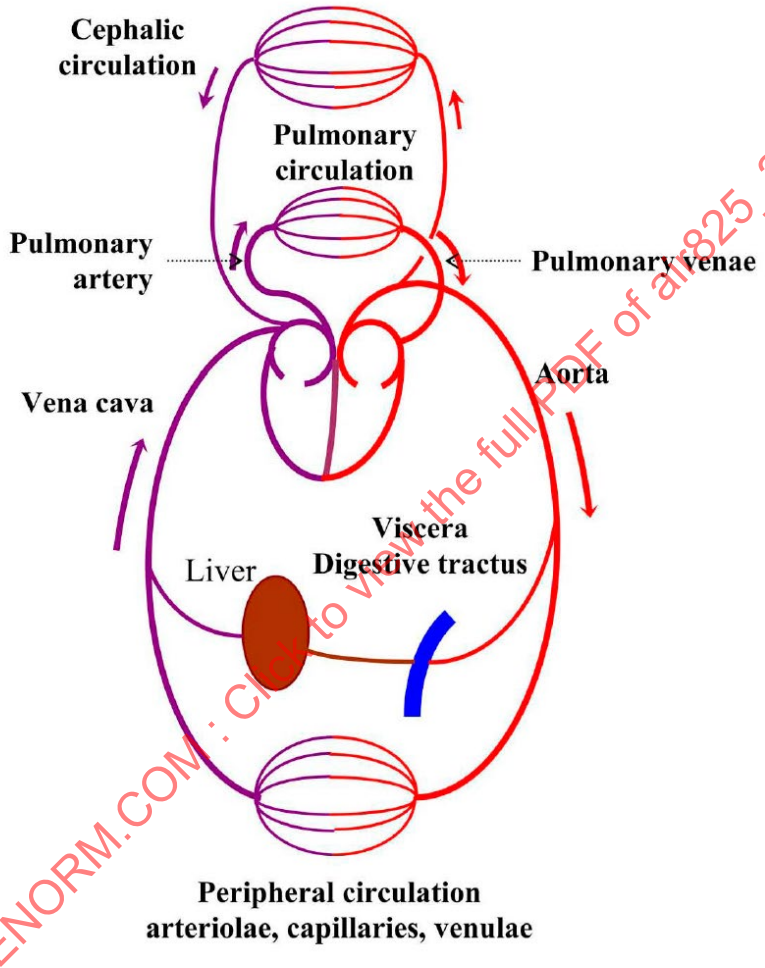

Figure 4 - General layout of the circulatory system

After exiting the capillaries, blood is collected by a network of blood vessels known as venules, which join together to form the veins that carry the oxygen depleted blood back to the heart and lungs. The two major veins in the body are the inferior vena cava which collects blood from the lower part of the body and the superior vena cava which collects blood from the head and upper limbs. The two venae cavae return blood to the right atrium. From the right atrium, the blood enters the right ventricle which pumps it through the pulmonary circulation. Anatomically similar to the peripheral circulation, the blood vessels branch repeatedly to ultimately form capillaries which then combine into veinules and veins on the return journey to the left atrium. From the left atrium, blood passes to the left ventricle for distribution through the body.

[2] Blood oxygen saturation (SaO_2) is often used as an index of hypoxia.

However, a major difference between the pulmonary and systemic circulation should be noted. The oxygenation state of the blood in the arteries or veins of the pulmonary circuit is reversed relative to the state in systemic circulation. The primary role of the pulmonary circulation is to provide for gas exchanges. Therefore, the pulmonary arteries carry deoxygenated blood away from the heart to the areas of gas exchanges within the lung (i.e., that returning to the right heart from the body), whereas the pulmonary veins carry oxygenated blood returning from areas of gas exchanges in the lung (i.e., that returning to the left heart from the areas of gas exchanges in the lung). Therefore, the oxygenation state of the blood being carried in pulmonary arteries and veins is opposite that of the systemic circulation.

5.1.2 Physiology

The basic function of the cardiovascular system is to provide a mechanism by which oxygen and nutriments can be delivered to the cells, and the waste can be eliminated from the cells. The cardiovascular system performs this role by the heart muscle contracting and creating pressure which forces the blood through the vascular system. The total blood volume is approximately 4.5 dm^3. Normal functioning of an individual depends upon this blood delivering an adequate supply of oxygen to the tissues, particularly the brain.

The rhythmic contraction of the cardiac muscle provides the pressure which constantly forces the blood through the vascular system. The relatively brief period of contraction, systole, is followed by a period of relaxation, diastole. Systolic and diastolic blood pressure readings reflect the pressure within the cardiovascular system during these periods of contraction and relaxation. The atria contract prior to the ventricles and work to complete ventricular filling. Valves which direct the blood and prevent backflow separate the atria from the ventricles and both sides of the heart, and also separate each ventricle from its corresponding artery. In discussing the anatomy of the heart, the cardiovascular system was divided into pulmonary and systemic circuits. Functionally, differences in the arterial and venous segments of the two circuits must be considered.

At rest, the amount of blood pumped by the heart, the cardiac output, is approximately 5 dm^3/min^{-1}. Cardiac output increases in response to increased demand for oxygen. For example, during a bout of maximum physical exercise, cardiac output can increase to 30 dm^3/min^{-1}. Contributing to this increase in output is an increase in heart rate from resting levels of 60 $beats/min^{-1}$, to rates approaching, and sometimes exceeding, 200 $beats/min^{-1}$. Other changes occurring under demanding conditions include more intense contraction of the heart muscle and the muscular walls of the vasculature.

In terms of the vasculature, the arteries make up a high-pressure system. Pressure in the aorta averages 100 mm Hg. Pressure drops as blood flows through the systemic circulation falling to approximately 0 mm Hg by the time it returns to the right atrium The venous side is a low-pressure system. The veins are very distensible and contain approximately 65% of the total blood volume. Under certain conditions, such as the exertion of increased G-force, the veins can actually distend and act as a reservoir for blood thus limiting the volume of blood returning to the heart. Obviously, the cardiovascular system must be able to adapt to such situational changes if normal functioning is to be maintained.

5.2 Ventilation

The tissue of the lung provides the interface for gas exchanges to take place. Ventilation is the process of moving volumes of gases in and of the lung.

5.2.1 Anatomy

The ventilatory system is made up of a network of branching tubes, an area dedicated to gas exchanges (see Figure 5), and a mechanical system to move air in and out of the lung. The transport tubes, i.e., bronchi, arise from the trachea and divide repeatedly as they descend into the lungs. The bronchial tree ends in what is referred to as the terminal bronchioles. Since no gas exchanges take place in these conducting bronchioles, they make up what is known as the anatomical dead space. The primary region of gas exchanges is the alveolar ducts which are completely lined with alveoli, the microscopic gas sacs of the lung. This alveolated area of the lung is known as the respiratory zone. The walls of the alveoli are heavily vascularized by the pulmonary capillaries. Gas exchanges take place across the extremely thin (1 µm, 10^{-6} m) alveolocapillary membrane, which represents a surface area of approximately 60 to 90 m^2.

Air is drawn into and forced out of the lung by pressure changes induced by changes in volume of the lung. Inspiration is an active event during which contraction of the diaphragm and intercostal muscles increases lung volume. The pressure becomes slightly negative with respect to the environment and air is drawn into the lung. Expiration is normally passive, the ventilatory musculature relaxes, the volume of the chest decreases, and the resulting increase in pressure forces air out of the lung. Typically, the movement of air results from pressure changes under 4 hPa (3 mm Hg) total.

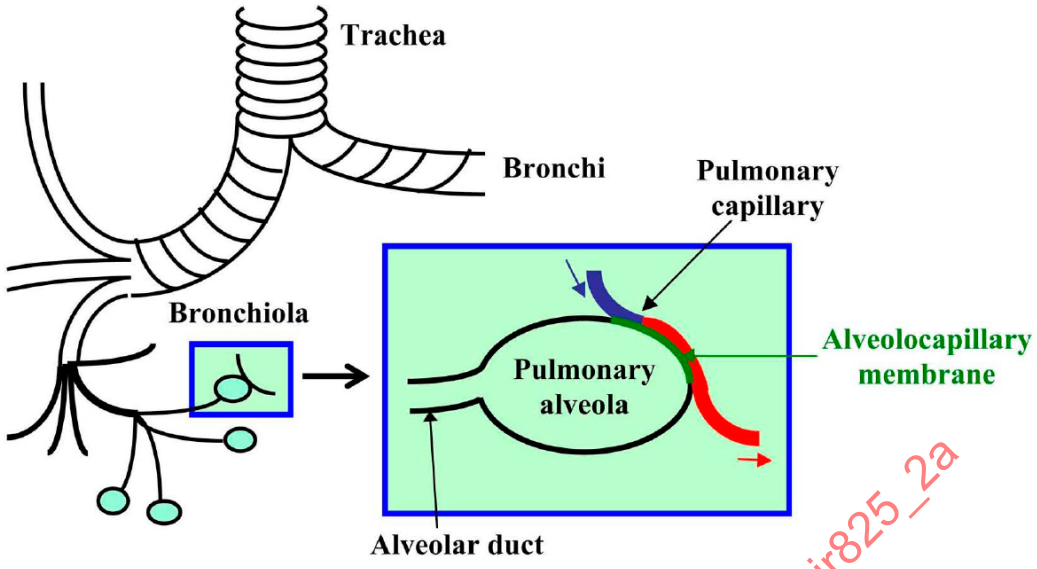

Figure 5 - Diagram of the ventilatory system

5.2.2 Mechanical Aspects of Pulmonary Ventilation

At rest, normal ventilation involves the movement of 0.5 to 0.7 L of gas, the tidal volume, in and out of the lung. Normally, 12 to 16 breaths are taken each minute. Therefore, the normal resting ventilation falls in the range of 6 to 8 L/min. There is an amount of gas left in the lung at the end of a normal expiration, the functional residual capacity. A maximal expiration still leaves some gas within the lung, the residual volume. The total lung capacity, which is the total amount of gas the lung can expand to contain, is the sum of the residual volume and the vital capacity. Ventilatory flows can be considered in terms of both mean and instantaneous flows, both of which are of importance to aviation. The mean flow, often named “minute volume,” is the volume of gas ventilated per unit time. It is usually given in L/min and is linked to the quantity of breathing gas that needs to be loaded on an aircraft to meet functional requirements. The instantaneous flow is linked to the mechanical qualities of the breathing system and is usually given in L/s.

Similar to cardiovascular system, the ventilatory system meets an increase in the demand for oxygen by increasing the ventilatory volume and associated flows. Again taking intense exercise as an example, the ventilatory volume can rise to a level of 100 L/min or more. This is brought about by an increase in both depth and rate of ventilation. The amount of work and energy cost associated with this increase is relatively low. However, there are circumstances in the aviation environment in which the effort associated with ventilation becomes significant. Anti-G straining maneuvers (AGSM) result in much higher instantaneous flow rates for ventilation. Instantaneous flow is normally less than 1 L/s but can increase to 3 to 6 L/s during AGSM. Similarly, positive pressure breathing significantly modifies the mechanical respiratory work and increases the work associated with a given respiratory volume. Therefore, it is very important that the resistance of a breathing system used aboard aircraft has minimal resistance levels associated with its flow rate characteristics.

5.2.3 Pulmonary Gaseous Exchanges

Understanding pulmonary gas exchanges, particularly under unusual environmental conditions, can be confusing. The most important item to be remembered when discussing gas exchanges is the partial pressure of the gas involved. Gases are exchanged in the lung by diffusion: they move from an area of higher partial pressure to an area of lower partial pressure. For example, the total atmospheric pressure at sea level is 1013 hPa (760 mm Hg). All the gases present in the atmosphere contribute to this total pressure based on their individual concentrations (Dalton's Law). Oxygen represents approximately 20.95% of the atmospheric gas, therefore the partial pressure of oxygen at sea level is 212 hPa (159 mm Hg). At an altitude where the total atmospheric pressure is only 666 hPa (500 mm Hg), corresponding to an altitude of 11000 feet (3350 m), the oxygen partial pressure falls to 140 hPa (105 mm Hg). Note that the concentration of oxygen remains at 20.95%. To reestablish a pressure gradient equivalent to that at sea level, the concentration of oxygen would have to be increased to 31.8% (P_B at 500 mm Hg * 31.8% O_2 = P_B at 760 mm Hg * 20.95% O_2).

Pulmonary gas exchanges can be described in terms of pressure and composition of the inhaled gases, the lung gases, and the blood gases. Since all of these are dynamic variables that change in response to external and internal conditions, the relationships among them are quite complex. The partial pressure of nitrogen (P N_2) and oxygen (P O_2) in the external environment vary with total atmospheric pressure. The partial pressure of water vapor (P H_2O) in atmospheric air is highly variable, whereas the partial pressure of carbon dioxide (P CO_2) is zero for all practical purposes. The internal environment consists of the lung gases and blood gases. Pressures and concentration of gases in the lung vary with location. Although they are correlated, conditions at the level of the trachea are different from conditions at the level of the alveoli. Pressures at the level of the alveoli are most important since that is the area where gas exchanges take place. In steady-state conditions, the mathematical equation for estimating the partial pressure of oxygen at the level of the alveoli ($P_A O_2$) is [9]:

$$P_A O_2 = F_I O_2 (P_B - P_A H_2O) - P_A CO_2 (F_I O_2 + [1 - F_I O_2/R]) \quad \text{(Eq. 1)}$$

where:

$F_I O_2$ = fraction of oxygen in the inhaled gas

P_B = atmospheric pressure

$P_A H_2O$ = partial pressure in water vapor in the alveolar gas

$P_A CO_2$ = partial pressure in carbon dioxide in the alveolar gas

R = respiratory exchange ratio (ratio between the quantity of carbon dioxide expelled and the quantity of oxygen consumed by minute); the normal value of R is 0.83 - note that in this context, R is not the symbol for ideal gas constant

The $P_A H_2O$ within the lung remains constant at 63 hPa (47 mm Hg). This value is based upon the gas in the lung being saturated with water vapor at a body temperature of 37 °C (BTPS conditions). Carbon dioxide levels in the human body are closely monitored and controlled. Normal $P_A CO_2$ levels have an average value of 53 hPa (40 mm Hg). This is also the value normally associated with the partial pressure of carbon dioxide in arterial blood ($P_a CO_2$). However, both $P_A CO_2$ and $P_a CO_2$ can fluctuate as a result of hyperventilation and/or metabolic disturbances. Physiological control systems exist to return such deviations to normal resting levels. Most importantly, $P_A O_2$ and $P_a O_2$ are roughly 138 hPa (100 mm Hg) in a resting individual at sea level. The venous blood that is returned to the lung after circulating through the body has a P O_2 ($P_v O_2$) of approximately 53 hPa (40 mm Hg). Internally these values are dictated by two external factors: the atmospheric pressure P_B and the fraction of oxygen in the inhaled gases ($F_I O_2$). Restructing Equation 1 with normal values for $P_A H_2O$ and $P_A CO_2$, the relationship for calculating $F_I O_2$ as a function of altitude (or barometric pressure P_B) is:

$$F_I O_2 = \frac{P_A O_2 + 63.5}{P_B - 52.5} \quad \text{(Pressures in hPa)} \quad \text{(Eq. 2A)}$$

$$F_I O_2 = \frac{P_A O_2 + 48}{P_B - 39} \quad \text{(Pressures in mm Hg)} \quad \text{(Eq. 2B)}$$

$P_A O_2$ is chosen as a function of the required level of protection against hypoxia: usually 80 hPa (60 mm Hg) in emergency conditions (for a crew).

5.2.4 How Oxygen is Carried by the Blood

Oxygen is dissolved in the blood and carried by the molecular compound hemoglobin (Hb). Hemoglobin allows the transport of approximately 20 mL of oxygen per 100 mL of blood. In comparison, only 0.23 mL of O_2 will be physically dissolved in the blood under conditions found at sea level. The quantity of oxygen carried by the blood is commonly given as the oxygen saturation level of arterial blood ($S_a O_2$). The $S_a O_2$ is the ratio of the quantity of O_2 actually carried by hemoglobin to maximum hemoglobin transport capacity. This measurement is given as a percentage (%). The methodology of this measure is based on the color change in the red-infrared spectrum of the hemoglobin. It's possible to measure differentially total hemoglobin versus oxygenated hemoglobin only. The result is expressed in percentage. This measure is possible in vivo through the skin, without invasive procedure.

Due to the chemistry of the oxygen/hemoglobin interaction, the oxygen/hemoglobin dissociation curve takes on a sigmoidal shape (see Figure 6). The shape of this curve offers a physiological advantage in that even if the P O_2 of the alveolar gas falls somewhat, loading and transport of O_2 by hemoglobin is not significantly affected. Other factors do influence the interaction between oxygen and hemoglobin, including carbon dioxide levels (P CO_2) and acidity (pH). Increases in temperature and/or P CO_2 along with decreases in pH shift the O_2/Hb dissociation curve to the right which favors the unloading of O_2 for a given concentration gradient.

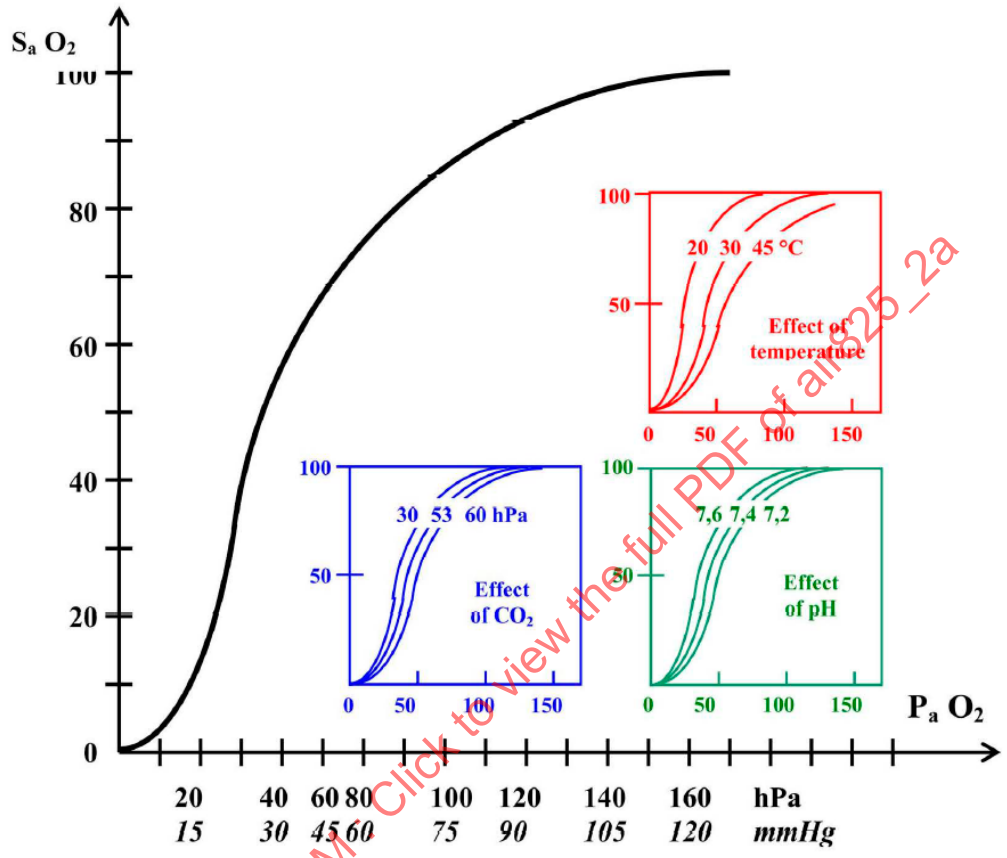

Figure 6 - Graph of hemoglobin dissociation: relationship between the oxygen saturation level of arterial blood (S_a O_2) and the partial pressure in oxygen (P_a O_2)

NOTE: This relationship depends on blood temperature (in °C), blood pH, and carbon dioxide pressure (in hPa). These values are drawn on the hypothesis of “safe value” (see Figure 1).

5.2.5 How Carbon Dioxide is Carried by the Blood and its Relationship with pH

Carbon dioxide is dissolved in the blood and forms carbonic acid (H_2CO_3) with water. H_2CO_3 dissociates into hydrogen ion (H^+) and bicarbonate ion (HCO_3^-). Bicarbonate ions combine with blood proteins and hemoglobin. These chemical reactions reverse when the blood passes through the lung. The resulting CO_2 diffuses into the alveoli. The pH of the blood fluctuates according to the relationship between carbonic acid and bicarbonate ions following Equation 3, as follows:

$$pH = pK + \text{Log}\frac{[HCO_3^-]}{\alpha \cdot P\ CO_2} \qquad \text{(Eq. 3)}$$

where:

pK = equilibrium point of the system protein-bicarbonate

HCO_3^- = HCO_3^- concentration in the blood

P CO_2 = partial pressure of CO_2 in the blood

α = constant (depending on the units)

In normal conditions, pH is near to 7.42 in arterial blood and 7.35 in venous blood.

5.3 Relationship Between Oxygen Consumption and Workload

Except for brief sport activities, such as sprinting 100 m or other explosive type event, aerobic metabolism provides the vast majority of energy for muscular work. The relationship between physical workload and oxygen consumption is normally proportional over the range of most sub-maximal workloads and is reported in terms of standard temperature and pressure, dry (STPD). This relationship can be important in defining the capabilities required of protective devices such as Portable Protective Breathing Equipment (PPBE).

At rest, oxygen consumption is about 0.25 to 0.30 L/min (STPD). Maximal efforts are generally characterized by oxygen consumption values in the range of 2.8 to 3.3 L/min. These values are typical of an individual who has not been specifically trained for aerobic endeavors. To consistently compare individuals of various sizes, oxygen consumption is often divided by body weight and expressed in terms of $cm^3O_2/min^{-1}/kg^{-1}$. The use of this set of units results in maximal oxygen consumption in the range of 42 to 45 $cm^3O_2/min^{-1}/kg^{-1}$ for a standard 70 to 75 kg male. However, when the body weight exceeds 75 kg, the maximal oxygen consumption does not exceed significantly 3.3 L/min (i.e., the oxygen consumption is a function of the body weight up to 75 kg and remains approximately constant when the body weight exceeds this value).

Aeronautical equipment is usually designed for individuals whose body masses fall in the interval between 48 kg and 99 kg (107 to 220 pounds). It is important to recognize that the oxygen supply is not intended to support physical endeavors that approach maximal exercise. In fact, a significant reduction in the time of protection during efforts exceeding 50% of maximum oxygen consumption can be anticipated.

6. HYPOXIA

The human body works as an aerobic, thermal machine. At altitude, the partial pressure of oxygen decreases. The most sensitive organ to the lack of oxygen (known as hypoxia) is undoubtedly the nervous system. The body instigates compensation mechanisms (automatic mechanisms of the vegetative functions), but these are limited [5]. What are the consequences of hypoxia which has exceeded the compensatory ability of the body?

6.1 Background

The physiological problems known to exist as a result of reduced O_2 levels, or hypoxia, have been recognized for centuries. One early report, published in 1590, of a pathological state linked to altitude comes from the Spanish Jesuit Father José de Agosta, who, when accompanying his country's armies to conquer the Andean high plateau, attributed the *soroché* (mountain sickness) to the air rarefaction. In 1787, de Saussure put these symptoms down to the lack of air. A century later (1878), Paul Bert published a monumental work on the subject, entitled La Pression Barométrique [1] (The Barometric Pressure [2]), in which he described and analyzed all the types of problems caused by variations in atmospheric pressure. Despite this work and subsequent warnings, early aviators (balloonists) Sivel, Crocé-Spinelli, and Tissandier took off with a goal of high-altitude flight in the "Le Zénith" balloon on April 15th, 1875. Due to the fact that they did not use their oxygen reserves appropriately, all three men suffered hypoxic syncope[3]. Only Tissandier survived. They had reached an altitude of 8600 m. Today, hypoxia remains a threat to the aviator flying at altitude.

[3] A temporary suspension of consciousness due to generalized reduction in oxygen flow in the brain.

6.2 Main Mechanisms and Classifications

6.2.1 Classification of the Different Types of Hypoxia

The following classification of the different types of hypoxia highlights certain associations which are particularly dangerous for aviators.

a. Ambient hypoxia is due to the decrease in oxygen partial pressure in inspired gases: this is what usually happens at altitude (hypobaric, then alveolar, hypoxia). It's the same when, at low altitude, the subject breathes a gas with less than 21% of oxygen.

b. Circulatory or stagnant hypoxia occurs when the blood contains a normal amount of oxygen, but oxygen delivery to tissues is impaired due to decreased blood flow. In military or acrobatic aviation, $+G_z$ accelerations can result in this type of hypoxia. In civilian applications, some passengers can have ischemic lesions, with potential ischemic hypoxia.

c. Anemic hypoxia is due to a reduction in the quantity functional hemoglobin in the blood (anemia) of oxygen carried by the blood. Several mechanisms can elicit this type of hypoxia in aeronautics, such as fumes or toxic vapors present in the cabin, like carbon monoxide. This considerably increases the impact of altitude hypoxia in the body.

d. Histotoxic hypoxia occurs when the cellular chains carrying oxygen are blocked; this can be caused by cyanide, for example. Several types of intoxication can occur in aeronautics, such as fumes or toxic vapors present in the cabin. This considerably increases altitude hypoxia.

6.2.2 Time-Based Classification of Types of Hypoxia

a. Hyperacute or fulminant hypoxia occurs in just a few seconds. Examples include the loss of cabin pressurization or oxygen supply systems at altitude in the 40000 feet (~12000 m) range. It results in sudden syncope with no warning symptoms.

b. Acute hypoxia occurs in a few minutes, due to slow exposure (over a few minutes) at medium altitude of 18000 feet (~5500 m). It is characterized by physiological and/or psychomotor reactions, sometimes followed by syncope. Acute hypoxia can cause a broad range of psychological problems in varying degrees of severity.

c. Prolonged hypoxia occurs when the hypoxic situation lasts several hours at altitudes between 8000 feet and 11500 feet (2500 m and 3500 m, respectively). Its primary symptom is fatigue.

d. Chronic hypoxia occurs when the hypoxic situation lasts several days, months, years, or even throughout an individual's entire lifetime. This is observed in mountainous regions.

6.3 Effects of Acute Altitude Hypoxia on the Major Vegetative Functions

In the following discussion, physiological changes associated with hypoxia are presented. Only the impact on organ systems and functions which are commonly considered most relevant to the aviator are reviewed, and the summary is in no way to be taken as comprehensive.

6.3.1 Effects of Hypoxia on Ventilation

Acute altitude hypoxia causes an increase in ventilation (hyperventilation) which is roughly proportional to altitude. The overall effects of hypoxia on ventilation are dictated by the severity and duration of hypoxic exposure.

6.3.2 Effects on Blood Circulation

Exposure to hypoxia has a tendency to trigger physiological reactions which improve oxygen delivery to tissues. For example, hypoxia causes the coronary vessels to dilate. Since it is easy to measure and record, the heart rate responses to hypoxia have been extensively studied. Acute hypoxia in humans significantly increases heart rate and cardiac output, particularly if the body has to perform muscular work.

6.4 Effects of Hypoxia on the Relation Functions

The relation functions are those which enable the individual to relate to his external environment. This covers all aspects of the nervous system function, including motor control, cognitive, sensory, and psychological functions. These very important relation functions are particularly sensitive to hypoxia. Hypoxic exposure can have a rapid effect on the cerebral cortex resulting in profound, insidious, and dangerous mental disturbances. Due to the importance of these functions to performance, descriptions of the interaction of hypoxia with the neural cells and the resulting impact on motor, sensory, and cognitive function follow.

6.4.1 Effects of Hypoxia on the Physiology of the Neurons

Basically, the special functions of the nervous system result from the ability of the cells to maintain concentration gradients across their membranes. These concentration gradients allow neuronal cells to transmit information through electrical impulses created by controlled movements of ions back and forth across the cell membrane. In order to control movement and location of ions, energy must be expended. In neuronal cells, the vast majority of energy is derived from the utilization of carbohydrates as a fuel source which requires high levels of O_2. Therefore, proper functioning of the neuronal cells is very sensitive to low levels of O_2 (hypoxia) in the blood. This sensitivity of the neurons to hypoxia forms the basis for the motor, sensory, and cognitive deficits commonly observed during altitude exposure.

6.4.2 Changes in the Motor Function

Motor neurons initiate the contraction process for voluntary muscle. The molecular processes responsible for muscular contractions are also dependent upon control of molecular concentration gradients. Therefore, hypoxia has the potential to influence all levels of motor functionality. The degradation in motor function resulting from hypoxia is positively correlated with increasing altitude, performance of complex tasks deteriorating more quickly than performance of simple tasks for a given altitude. Usually, the first sign indicating a loss of motor control is muscular tremor. These have been documented at altitudes of 4500 m (15000 feet). If the hypoxic stimulus is not removed, a total lack of motor coordination may follow and eventually develop into actual paralysis if the hypoxia remains severe. The risk of such a progression taking place is particularly dangerous because the initial symptoms of hypoxia often go unnoticed. The individual may not realize that their capabilities have deteriorated until they reach the point where they are incapable of action.

One suffering from hypoxia often has the will to perform a motor task but has neither the motor control nor strength to complete the job. Compounding the problem is the fact that the individual may not even realize that a particular ability is impaired. A motor task that has often been used in demonstrating or testing hypoxia tolerance is handwriting. Upon exposure to hypoxia, a person can become completely unaware of the deformation of their writing, or the type of silly things being written down. An example of this type of change is presented in Figure 7. It must be emphasized that the test subject did not realize that their writing was becoming illegible.

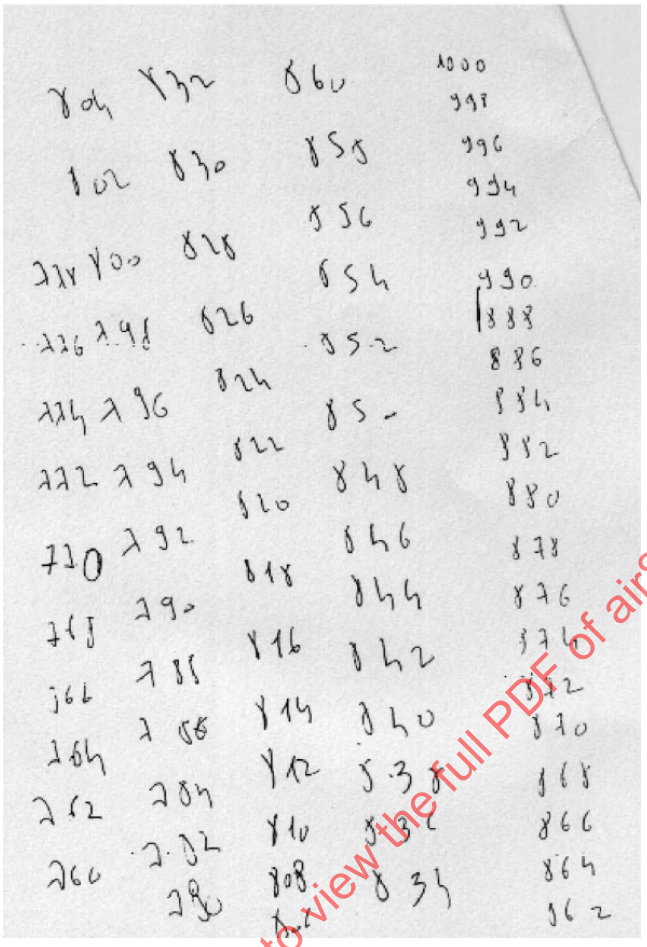

Figure 7 - Specimen writing test during acute exposure to altitude at 6000 m (20000 feet) without oxygen

NOTE: The climb was 25 m/s^{-1} (5000 ft/min). The goal of the test was to write numbers from 1000, by decreasing from 2 by 2. The subject started the test immediately before arrival at 6000 m (20000 feet). The total duration of test is 2 to 3 minutes. All the numbers on this page were written by the same subject, for the same test.

Observe that (a) surprisingly, the subject wrote from left to right; he began writing on the right side of the page, and then continued to write from right to left, (b) the subject did not realize that he was making major errors in the writing assignment (990 → 888), and (c) the handwriting was deeply altered. After restoring the oxygen supply to the subject, writing ability was recovered in about 5 to 10 seconds, but the subject is generally tired. (From a document of the French Aerospace Medical Laboratory, Flight Test Center.)

6.4.3 Sensory Organs

The senses of hearing and vision are affected by hypoxia in completely different ways.

a. Hearing is not considered to be significantly affected by hypoxia. This is of particular importance to the aviator because hearing is often the only communication link with an individual suffering from severe hypoxia. On numerous occasions, the ability to hear, recognize, and respond to simple orders has enabled crews suffering from hypoxia to perform essential emergency procedures and land aircraft that otherwise might have been lost. It is for this reason that pilots, and other personnel who may come into voice contact with aviators, must be taught to recognize and respond to situations in which hypoxia may be exerting an effect on one of their colleagues.

b. In contrast to hearing, vision is severely affected by hypoxia. Visual impairment can start as low as 1500 m (5000 feet). Problems include the inability for the lens of the eye to properly accommodate (focus) and a reduction in the field of vision. Color vision and night vision are also diminished. The only aspect of vision that is not highly sensitive to hypoxia is central vision acuity.

6.4.4 Effect of Acute Hypoxia on Behavioral Functions

Depending on its severity, hypoxia results in either behavioral abnormalities or unconsciousness. Behavior abnormalities are often caused by acute hypoxia. Many different types of afflictions are known to occur. Basically, this variety of behavioral problems covers anything that cannot be considered as normal under a given set of conditions. However, the following responses are characteristic of exposure to hypoxic conditions.

a. After the exposure, the individual can remember having judgmental or behavioral difficulties.

b. The onset and severity of behavioral alterations varies within individuals and among exposures. In other words, the response to a given hypoxic stimulus within an individual is relatively inconsistent.

c. The behavioral manifestations of hypoxia cover all types of abnormalities ranging from pseudo-maniac excitation to depressive syndromes.

d. A state like inebriation is characteristic symptom of hypoxia, although not always present. The subject is excessively jovial, a symptom which is not usually remembered by the subject him/herself.

e. Another symptom typically observed because of hypoxia is a depressive syndrome which includes a total abolition of the individual's will. Often the afflicted individuals analyze the situation correctly, yet draw no conclusions, even concerning matters of their own survival. A common result is no response to the situation, or a declaration that "nothing can be done" (this is a recurrent theme in cases of hypoxia).

f. On occasion, hypoxia has induced an utterly incoherent reaction, where the individual performs a totally illogical act in response to existing circumstances. An example of this type of behavior is the pilot who announces a (real) oxygen failure, then depressurizes the aircraft.

Loss of consciousness is most often observed in cases of hyperacute hypoxia. Following such a rapid onset, an individual loses consciousness in only a few seconds. Seldom does the individual feel anything before losing consciousness. Upon recovery some do not remember anything about the experience, often denying it ever happened. Others retain a dreamlike recollection associated with the loss of consciousness, but this is their only memory. Loss of consciousness is often accompanied by involuntary and/or uncoordinated movements of one or both of the upper limbs. If re-oxygenation procedures are initiated immediately, consciousness is regained within about 20 seconds. In summary, the effect of hypoxia on the relation functions presents a serious risk of incapacitating aircraft pilots, crews, and passengers. It is important for all individuals working in the aviation field to be informed about both acute and chronic hypoxia so that problems associated with altitude exposures can be minimized if not eliminated.

6.5 Tolerance to Altitude Hypoxia

Studying human tolerance to altitude hypoxia involves defining and quantifying the altitude limits which correspond to acceptable physical or behavioral performance in relation to given objectives. Tolerance depends on the altitude reached, the time spent and the speed of the ascent. Tolerance to altitude hypoxia must be defined for each separate type of hypoxia. For acute hypoxia, the limiting factor is the altitude reached. For hyperacute hypoxia, the limiting factor is the time spent before psycho motor incapacitation. For prolonged hypoxia, the limiting factor is the time spent before the onset of excessive fatigue.

Before giving any precise figures, it should be noted that these data are extremely variable. It can vary from one subject to another and, for the same subject, it can vary from one day to the next. This variability is very important. It has to be described in terms of probability (Gaussian curve) and, in control terms, it must be taken into account by using values which are reliable enough to cover the entire population ("safe values").

6.5.1 Tolerance in Relation to the Altitude Reached

Tolerance to hypoxia is related to the oxygen saturation of the arterial blood. The relationship between altitude and atmospheric pressure is not a simple one, and neither is the one between oxygen partial pressure and blood oxygen saturation. However, the relationships are relatively consistent, and the four following zones can be defined (see Figure 8) [10].

a. The unaffected zone is situated between 0 feet and 5000 feet (1500 m). The molecular characteristics of the hemoglobin molecule are such that no physiological deficiency results from hypoxia. However, other consequences of hypobarism may occur, such as barotraumas. The term unaffected zone only refers to the symptoms of hypoxia.

b. The total compensation zone is situated between 5000 feet and 11500 feet (1500 m and 3500 m, respectively). The body compensates for hypoxia by certain cardiorespiratory reactions. This compensation is almost total, except for some functions: night vision, learning ability, and fatigue if the exposure to altitude is prolonged. Night vision starts to deteriorate from 5000 feet (1500 m) and learning ability is reduced starting at 8000 to 10000 feet (2500 to 3050 m, respectively). The term total compensation zone is therefore not entirely correct.

c. The insufficient compensation zone is situated between 11500 feet and 18000 to 20000 feet (3500 m and 5500 to 6000 m). This zone is characterized by the risk of acute hypoxia and its corresponding symptoms deterioration of judgement, concentration difficulties, deterioration of memory, dysphoria (excitation or depression), personality disorders, and occasionally headaches and vertigo, risk of drowsiness, and, sometimes, the risk of loss of consciousness. This risk appears after a certain time of exposure, highly variable depending on the subject, generally a few minutes, if not corrected by appropriate supplemental oxygen administration.

d. The critical zone is situated above 18000 to 20000 feet (5500 to 6000 m). It is characterized by the risk of hypoxic syncope, whose suddenness increases with altitude. If the hypoxic situation is not corrected, syncope leads to death. The exact figures vary between individuals.

e. The reaction threshold is 1500 m (5000 feet), the trouble threshold is 3500 m (11500 feet) and the critical threshold is 5500 to 6000 m (18000 to 20000 feet).

The following limits for the effects of hypoxic should always be considered:

a. 5000 feet (1500 m) for normal flight with no problems, so without the use of additional oxygen; night vision can start to deteriorate above this altitude.

b. 8000 feet (2500 m) for the threshold of prolonged hypoxia without excessive fatigue or mild hypoxia, recognized by the civil aviation authorities as the statutory value for pressurization of passenger carrying aircraft (14 CFR and CS-25.841).

c. 12000 feet (3600 m) for the threshold for oxygen utilization in all conditions.

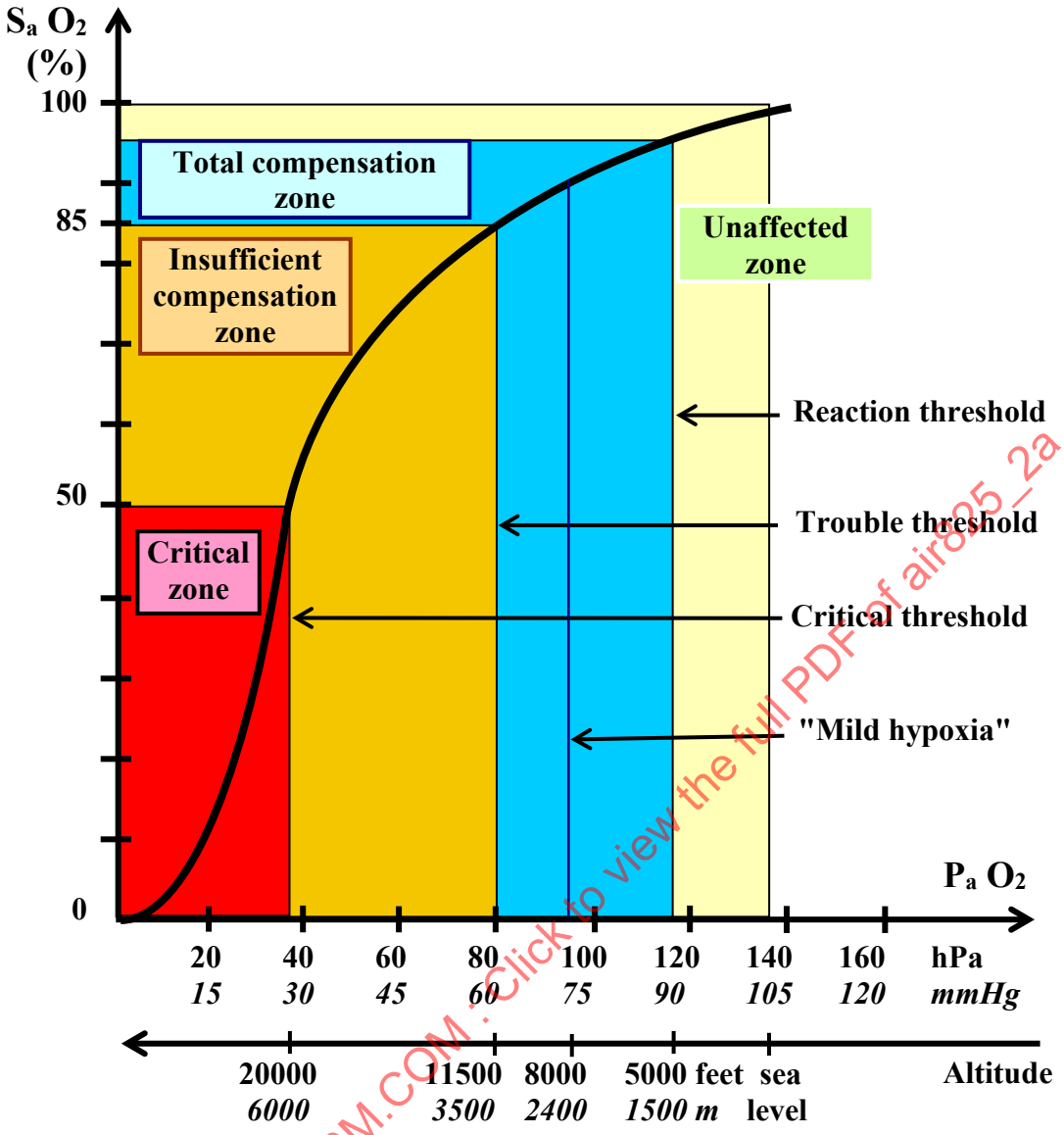

Figure 8 - Hypoxia tolerance zones in relation to altitude

NOTE: This figure is based on Figure 6 and includes the altitude hypoxia tolerance zones.

6.5.2 Development of Hypoxia Over Time

When hypoxia occurs within a brief period of time, following rapid decompression or oxygen supply failure for instance, the sequence of symptoms depends on the time spent at this altitude where: (1) unaffected phase, during which the individual is apparently normal, (2) total compensation phase, during which physiological (cardiorespiratory) reactions compensate for the reduced partial pressure of oxygen, (3) the insufficient compensation phase, during which the symptoms of hypoxia become apparent, (4) critical phase, during which loss of consciousness occurs. After the loss of consciousness, death or brain irremediable damages can occur after 3 minutes.

6.5.3 Tolerance to Hyperacute Hypoxia and Time of Useful Consciousness

Hyperacute hypoxia occurs when the critical threshold has been exceeded and exposure to hypoxia is rapid. The total and insufficient compensation phases are hardly visible, and in practice, the subject quickly moves from the unaffected phase to a state of severe incapacitation, just before the loss of consciousness. This is the notion of time of useful consciousness. Although the meaning and interpretation of time of useful consciousness has been debated, it remains a very useful index in operational terms. It is difficult to set values for the time of useful consciousness which are precise, realistic, and applicable in all circumstances. The time of useful consciousness varies depending on the altitude exposure conditions. Two factors are particularly important: the fraction of oxygen inhaled by the subject just before decompression and his level of metabolic activity when decompression occurs. Table 3 indicates the values of the time of useful consciousness for various scenarios of exposure to hypoxia (following explosive decompression or oxygen supply failure) with respect to the level of muscular activity of the subject.

In 1970, the notion of time of safe unconsciousness has been introduced with respect to passengers on commercial aircraft [4]. This is the period during which the severely hypoxic/anoxic nervous system can recuperate with no permanent lesions being evident. This time is estimated to be less than three minutes, which explains why certain airworthiness standards for air transport airplanes, based on the hypothesis that not all the passengers will put on their oxygen masks in the event of cabin decompression, specify that the aircraft must be designed such that occupants could not be exposed to cabin altitudes exceeding 25000 feet for more than 2 minutes (14 CFR part 25.841.(a).(2).(i)).

Table 3 - Time of useful consciousness

Altitude (feet)	Altitude (meters)	Subjects Inhaling Oxygen		Subjects Inhaling Air	
		At Rest	At Moderate Activity	At Rest	At Moderate Activity
22967	7000	10 minutes	5 minutes	5 minutes	2.5 minutes
26248	8000	5 minutes	2.5 minutes	2 minutes	1 minute
27889	8500	2.5 minutes	1 minute	1 minute	0.5 minute
29529	9000	1.5 minute	45 seconds	45 seconds	20 seconds
34451	10000	45 seconds	30 seconds	30 seconds	15 seconds
39372	12000	30 seconds	20 seconds	20 seconds	15 seconds
44294	13500	15 seconds	12 seconds	15 seconds	12 seconds
54137	16500	15 seconds	12 seconds	15 seconds	12 seconds
65620	20000	12 seconds	12 seconds	12 seconds	12 seconds

6.5.4 Tolerance to Mild Hypoxia

Historical descriptions of the effects of hypoxia have focused on severe forms of hypoxia, at high altitude. However, there are also non-severe but real forms of hypoxia, often grouped under the term mild hypoxia. In the aeronautical field, the exposure time remains limited to a few minutes or a few hours. These forms of "moderate" hypoxia affect flight at such an altitude that the relevance of the use of oxygen is not perceived by all, especially in general aviation, including in the practice of parachute jumping. To give a numerical indication, mild hypoxia is an exposure up to the altitude of 15000 feet (4572 m), as described by Ernsting [3]. The definition of an acceptable value of mild hypoxia is at the basis of the compromise studies for the pressurization of passenger transport aircraft.

Many studies have been and are dedicated to these forms of hypoxia. Almost all of them refer to disorders of the nervous system attributable to hypoxia. Several neurological functions are degraded: learning capacity, reaction time, decision-making, certain types of memory. Interindividual variability is important, and many factors come into play, including the heterogeneous response of neurons from different parts of the central nervous system. Neurocognitive disorders can be observed at a distance from exposure to altitude, up to 24 hours after exposure. However, the disorders observed up to 12000 feet are minimal. Particular attention is paid to vision, especially night vision which can be degraded from 4000 to 5000 feet (1220 to 1525 m). Fatigue, finally, can be observed following flights at an altitude between 10000 feet and 15000 feet, with the observation of accidents at the end of the flight, clearly attributable to fatigue following exposure to hypoxia.

6.6 Physiological Principles of Protection Against Altitude Hypoxia

6.6.1 General Case

Algebraic rearrangement of the Equation 1, with $P_A H_2O$ = 63 hPa (47 mm Hg) and $P_A CO_2$ = 53 hPa (40 mm Hg), results in relationships described by Equations 2A and 2B. Readily evident from this simplification are two approaches which can be utilized to prevent hypoxia at altitude. Either $F_I O_2$ has to be increased through inhalation of oxygen-enriched air, or P_B has to be maintained at levels near ground level. In aviation, P_B is usually maintained through aircraft pressurization. In circumstances where the aircraft is unpressurized or insufficiently pressurized for physiological needs (fighter aircrafts), or the pressurization system fails, the $F_I O_2$ required to maintain specific $P_A O_2$ can be calculated. For example, solving for an $F_I O_2$ in terms or desired $P_A O_2$ and P_B at 4500 m (15000 feet), 80 hPa and 575 hPa (60 mm Hg and 433 mm Hg) results in a $F_I O_2$ of 27%. This relationship is plotted in Figure 9 and is very important in that it highlights the necessity for pressure breathing above altitude at 39000 feet (11900 m), since the percentage of oxygen in breathing gas cannot exceed 100%.

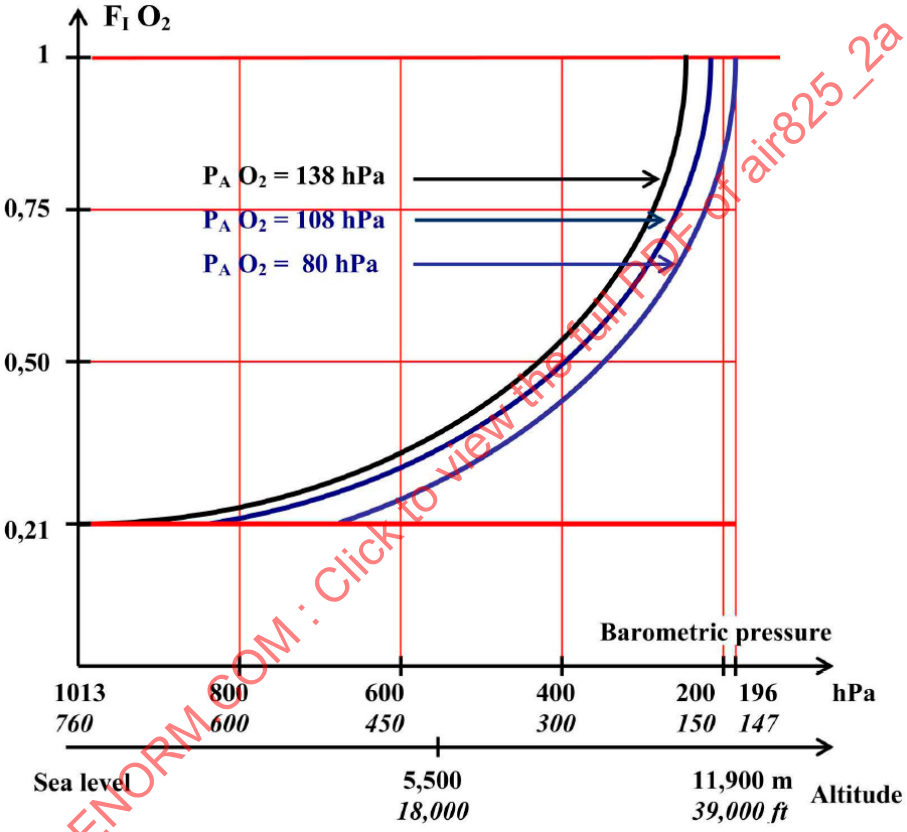

Figure 9 - Graph indicating the minimum values of $F_I O_2$ in relation to altitude and the value of $P_A O_2$ selected

where:

$P_A O_2$ = 138 hPa = 103 mm Hg: $P_A O_2$ equivalent to sea level

$P_A O_2$ = 108 hPa = 80 mm Hg: $P_A O_2$ equivalent to an altitude of 5000 feet

$P_A O_2$ = 80 hPa = 60 mm Hg: $P_A O_2$ equivalent to an altitude of 11500 feet

To maintain adequate $P_A O_2$ at altitudes above 39000 feet (11900 m), pressure breathing must be performed. Positive pressure breathing (PPB) provides additional pressure which augments the atmospheric pressure to deliver oxygen to a desired alveolar pressure. PPB involves creating, via the oxygen system, a permanent positive pressure in the gas passages and the lungs. The approach of increasing breathing pressure has a number of potential problems, which include:

a. Poor airtightness of the mask on the face which results in oxygen leaks.

b. Head and neck trouble resulting from distension of the tympanic membrane (i.e., eardrum), the floor of the mouth, and circulatory problems.

c. Ventilatory problems related to the mechanics of breathing. During normal ventilation, the differences in pressure in the lung are very low (1 to 2 mm Hg). Under PPB, the inspiratory and expiratory pressures are increased and reversed in time, i.e., inspiration is performed passively by a forced injection of breathing gas into the lung whereas expiration is active, effort being exerted against the positive pressure.

d. Circulatory problems resulting from the increased resistance for the blood returning to the heart and being pumped through the pulmonary circulation.

An untrained subject usually finds it difficult to tolerate PPB over 20 to 25 hPa (15 to 19 mm Hg). It is therefore essential to calculate the maximum altitude which can be tolerated with this level of PPB. For example, it was demonstrated that, if a decompression occurred at an altitude of 45000 feet (13700 m) and the aircraft was capable of rapidly descending to an altitude of 39000 feet (11900 m), an augmentation of P_B by a PPB value of 24 hPa (18 mm Hg) would be sufficient to prevent hypoxic effects if the duration of the 45000 feet exposure did not extend beyond 1 minute [8]. If the time spent at 45000 feet is extended, the resulting $P_A O_2$, about 56 hPa (42 mm Hg), would not be expected to be sufficient to prevent the effects of hypoxia.

6.6.2 Case of Rapid Decompression at High Altitude

The shortness of the time of useful consciousness following decompression at high altitude can be explained as follows (see Figure 10). Depicted in this curve is a hypothetical decompression from a cabin altitude of 8000 feet (2450 m, P_B = 753 hPa = 565 mm Hg) to an altitude of 39000 feet (11900 m, P_B = 197 hPa = 148 mm Hg). At 8000 feet, the alveolar partial pressure in oxygen is about 95 hPa (70 mm Hg). As a result of decompression, the alveolar oxygen partial pressures decrease in proportion to the changes in P_B. The $P_A O_2$ of the lung is reduced to approximately 25 hPa (18 mm Hg) under these circumstances. This decrease is compounded by the fact that the partial pressure in water vapor in the lung remains at the normal value of 63 hPa = 47 mm Hg. Due to increases in ventilation stimulated by physiologic control mechanisms, $P_A CO_2$ drops somewhat, reaching an intermediate value of around 30 hPa (22 mm Hg). Once decompression occurs, blood of very low oxygen saturation (S_aO_2 < 20%) leaves the lung. It reaches the brain in about 4 seconds. Neurons use up the available oxygen supply within a few seconds and start to malfunction. If normal oxygen supply is not restored within 3 to 4 minutes, neuronal cell death becomes imminent. This cascade of events explains the shortness of the time of useful consciousness after decompression at high altitude.

This has two effects at operational level:

a. The speed with which hypoxic syncope occurs means that in transport aircraft, flight crew oxygen masks which can be put on in under five seconds must be used. In the event of cabin decompression, the supplemental oxygen supply system must be immediately available.

b. Significant and sudden variations in $P_A O_2$ following rapid decompression can justify the administration, prior to decompression, of an oxygen-enriched gaseous mixture, which is calculated as a function of flight altitude and time expected for complete depressurization.

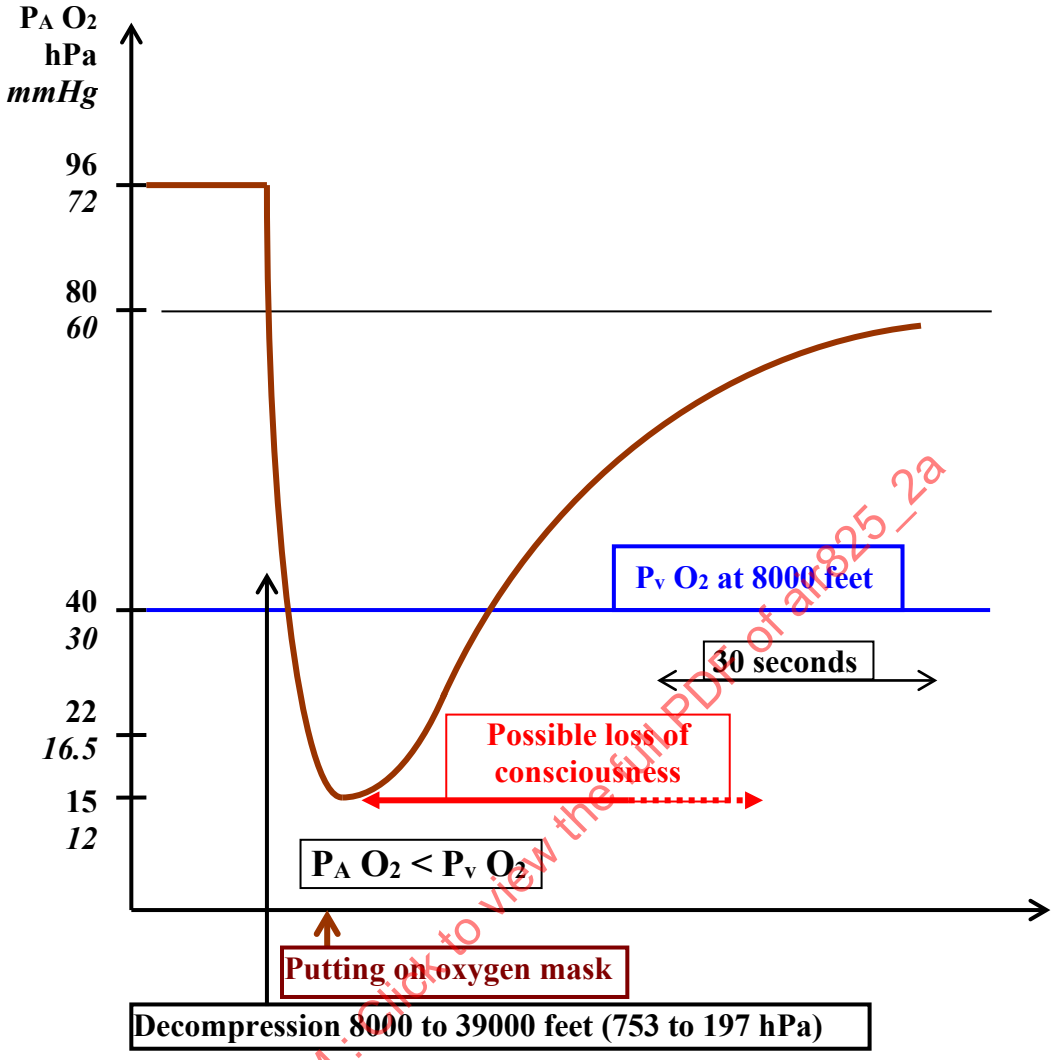

Figure 10 - Change in $P_A O_2$ during rapid decompression; the oxygen mask is put on in less than 5 seconds

NOTE: P_B decreases from 753 to 197 hPa (565 to 148 mm Hg). $P_A H_2O$ remains constant (63 hPa [47 mm Hg] at 37 °C), $P_A CO_2$ decreases with the same ratio as P_B, then rapidly increases to 30 hPa (22 mm Hg). At the initial part of the decompression, $P_A N_2$ and $P_A O_2$ decreases (only $P_A O_2$ is plotted on this figure). After putting on the oxygen mask, N_2 begins to disappear from alveolar gas and $P_A O_2$ increases to 80 to 90 hPa (60 to 67.5 mm Hg). About 50% of N_2 is eliminated in 15 to 20 seconds; 90% of N_2 is eliminated in 1 minute [7].

6.6.3 The Case of Decompression of a Large Aircraft at High Altitude

The presentation of the effects of rapid depressurization as shown in Figure 10 is valid on small aircraft. On some large aircraft, a breach in the cabin that would be responsible for complete depressurization in 3 seconds is not compatible with maintaining the integrity of the aircraft structure in flight. For these larger airliners, complete depressurization would more likely occur in 15 to 20 seconds. For this example, a hypothesis is made that the crew, warned of the depressurization, puts on its oxygen mask within 5 seconds. In this case, oxygen enrichment, in the form of an increase in $F_I O_2$, occurs at the same time as the pressure decreases in the cabin [12], [13]. Figure 11 shows a theoretical example of such a scenario.

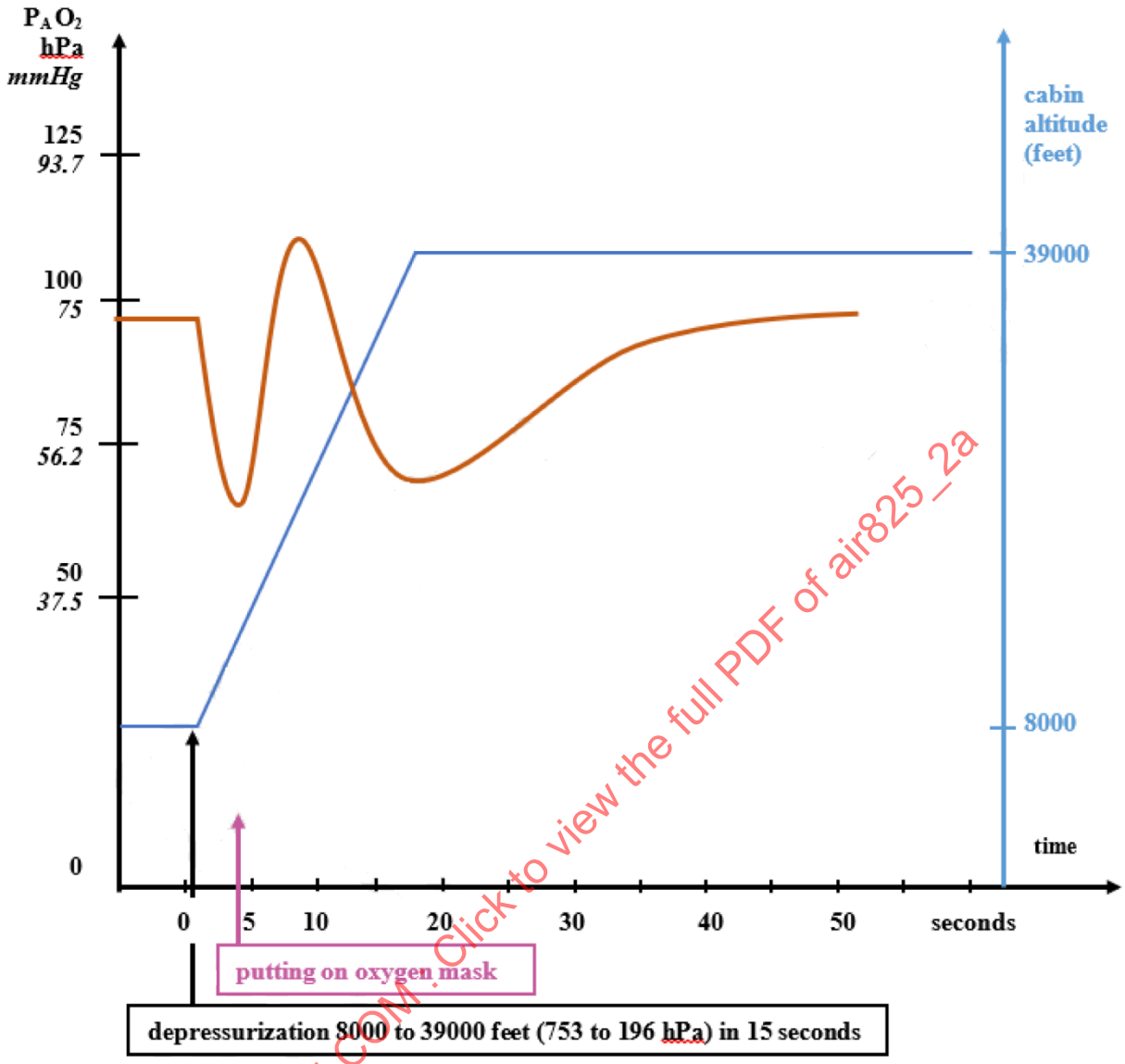

Figure 11 - Change in $P_A O_2$ during depressurization in 15 seconds

NOTE: The oxygen mask is put on in less than 5 seconds. The oxygen enrichment begins before the maximum cabin altitude be reached, there are some phases for describing the evolution of $P_A O_2$: an initial decrease, and, after putting on the oxygen mask, an increase in $P_A O_2$; then the altitude in still increasing and $P_A O_2$ decreases. Finally, nitrogen is eliminated the $P_A O_2$ is joining its definitive value (at altitude constant). In this scenario, with a limited rate of cabin pressure decrease, with immediate (<5 seconds) putting on the oxygen mask, the hazardous phase of hyperacute hypoxia can be strongly limited. The exact calculation depends on flight altitude, rate of decompression and time before putting on the oxygen mask (in “100% O_2”).

6.7 Rules of Protection Against Hypoxia

The following sentence is not a joke: a hypoxic man becomes stupid. Many aircraft crashes are due to the fact that, when a crew-member is becoming hypoxic, he or she is unable to judge the situation and respond appropriately. One of the frequent forms of behavioral troubles due to hypoxia is a personal inability to react, even when remaining conscious.

In the general sense, two approaches must be taken to minimize the risk of hypoxia in aviation. They are appropriate education of participants and responsible regulations for aircraft. Pilots and all other participants (i.e., navigators, air traffic controllers, flight attendants, etc.) directly involved in operation of the aircraft must be instructed of the risk and symptoms of hypoxia. Such training should include an experience in altitude chamber: “The most beautiful speech has never been better than a simple personal experience.” Clear rules and regulations must be established to avoid any hesitation or indecision about the steps to take to prevent hypoxia. The importance of clear rules cannot be overemphasized.

Several regulatory bodies have regulations addressing issues of hypoxia. For example, ICAO (International Civil Aviation Organization) regulations, FAR (Federal Aviation Regulations), EASA (European Aviation Safety Agency), and other regulatory bodies have guidelines addressing prevention of hypoxia in aviation. It is not the purpose of this text to describe or analyze these various regulations. However, it should be noted that many individuals find it difficult to remember a large number of complex regulations. Due to disastrous consequences that can result from hypoxia in aviation, basic data and associated safety limits must be embedded in the minds of flight crew. Recently, EASA dedicated new rules for oxygen use, applicable for all types of aircrafts and all types of flights [Commission regulation (EU) n° 965/2012].

From the preceding discussion, the necessity for appropriate oxygen levels should be clear. If oxygen is provided to the body at sufficient pressure, hypoxia, and all of the negative effects it can induce can be prevented.

7. HYPERVENTILATION

Hyperventilation is defined as an increased rate of breathing. When the rate of breathing is increased at rest, more CO_2 than normal is eliminated from the body unless CO_2 production has increased. Faster elimination of CO_2 lowers the level of CO_2 remaining in the body. A decrease in CO_2 levels of the body is called hypocapnia. Hypocapnia occurs when the partial pressure of carbon dioxide in arterial blood falls below its normal value (53 hPa = 40 mm Hg). The symptoms of hypocapnia are observed during hyperventilation. They can lead to a pre-syncopal state or actual syncope. A significant number of problems during flight are known to have occurred, at least to a degree, due to hyperventilation resulting from hypocapnia. The symptoms of hyperventilation can be similar to, and are more often confused with, those of hypoxia.

7.1 Physiological Mechanism of Hypocapnia

There is a risk of hypocapnia any time pulmonary excretion of carbon dioxide consistently exceeds metabolic production. Normally, the body responds to the increase in carbon dioxide by hyperventilating. This is what happens during muscular exercise. In this case, the role of hyperventilation is to increase carbon dioxide elimination by the lung. Any situation likely to cause hyperventilation which is not linked to the increase in the metabolic production of carbon dioxide causes a decrease in its partial pressure in the lung and blood, i.e., hypocapnia. Several situations encountered during flight can cause hyperventilation and hypocapnia.

Hypocapnia causes several problems. Vasomotor problems can lead to syncope. The impact of hypocapnia on the nervous problems gives rise to the condition's characteristic symptoms: abnormal and uncomfortable sensations in the extremities and lips, muscular spasms in the extremities of the limbs and in the face, with involuntary fasciculation, muscular attitudes, or movements. At worst, hypocapnia can result in a tetanic seizure complete with all its symptoms At the height of the seizure, the effect on psychomotor performance can entail complete or partial loss of consciousness.

7.2 Clinical Symptoms of Hypocapnia

In clinical terms, the subject suffers certain characteristic symptoms:

a. Increasing feeling of malaise: unlike hypoxic syncope, which is often accompanied by indifference or even euphoria, the symptoms of hypocapnia frequently include a feeling of intense panic, which in the worst cases can even lead to a feeling of impending death.

b. Sudation (i.e., sweating) of the face and extremities, which can be very abundant.

c. Tingling and numbness of the extremities, becoming more intense and affecting the whole limb.

d. At the start of the seizure, muscular cramp or involuntary contractions of small muscle groups, which are difficult for the subject to locate.

e. At worst, muscular cramp, which can lead to extremely painful, widespread contracture, despite diminishing consciousness.

7.3 Causes of Hyperventilation During Flight

There are several causes of hyperventilation during flight, other than the metabolic production of carbon dioxide:

a. Motion sickness, which can affect even experienced crew members in certain flight situations. Hyperventilation is one of the initial symptoms of motion sickness. It can increase ventilation three- to five-fold, which is considerable. In this case, hypocapnia is constant.

b. Anxiety or even fear, which any pilot may feel one day when involved in an accident, unexpected situation, or some kind of mistake.

c. A case of genuine hypoxia also causes hyperventilation. Elements of hypocapnia can often be observed in the symptoms described by pilots who have suffered hypoxia.

d. The final possible cause of hyperventilation is linked to the oxygen masks used by the crew. By its very design, an oxygen mask becomes more resistant as the inspiratory demand increases. Due to the system itself, a high inspiratory demand can result in an unusual resistance to inspiration. When this high inspiratory demand (rate and volume) is detected by the user, it can be alarming. Increased work of breathing is always uncomfortable and is sometimes (wrongly) interpreted as indicating a failure in the oxygen system. In an attempt to compensate, the crew member increases the rate and depth of their breathing. The resulting hyperventilatory response is triggered and self-sustained. The scenario emphasizes the importance of the supplemental oxygen delivery system. Good oxygen systems (characteristically such systems have very flat pressure/flow curves) seldom result in a hyperventilatory response by the user.

7.4 Procedure to Follow During Flight

It is important to know how to relieve a bout of hyperventilation with hypocapnia during flight. The cause of the problem must be correctly identified by eliminating other causes of discomfort which can occur during flight. However, it should be remembered that on single-seater aircraft, any pilot who suffers from hyperventilation with hypocapnia is effectively both physician and patient, which is far from being the ideal situation.

7.4.1 Recognizing the Problem

The feeling of discomfort increases progressively. Even when developing rapidly, the discomfort is still progressive. Unlike the hypoxic condition, the discomfort resulting from hyperventilation is very distressing. This is rarely the case during hypoxia which, even in its depressive forms, generally leaves the subject either calm and resigned or making a lot of mistakes, including inaction. Sudation of the head and extremities is a characteristic warning sign of hyperventilation. Tingling, prickling, and slight cramp in the extremities are easily recognizable initial symptoms. It is important to be aware of other possible causes of discomfort during flight in order to eliminate them. These include hypoxia and poisoning by toxic fumes inside the cockpit.

7.4.2 Discomfort Relief During Flight

During flight, the discomfort can only be relieved by the action of the pilot. As for any treatment, the assumed cause must be treated without neglecting any other possible cause (hypoxia or intoxication). If there is a sustained cycle of hyperventilation/inspiratory resistance, a good solution would be to rapidly descend to a cabin altitude below 10000 feet and undo the oxygen mask. However, this procedure is inadvisable since it is only correct if the diagnostic is certain. Therefore, it seems better to apply the following procedure:

a. Check the oxygen system.

b. Check the cabin altimeter.

c. Check that the oxygen mask fits properly.

d. Set the regulator to pure oxygen (100% O_2). This precaution avoids any risk of hypoxia and intoxication due to the cabin atmosphere. Intoxications during flight due to pollution of the oxygen system currently seem to be extremely rare. Breathing pure oxygen for relatively short durations is not toxic, and the old saying about getting high on oxygen just describes the symptoms of hyperventilation. In the event of hyperventilation-type malaises, oxygen as such is neither useful nor dangerous, but using it in all cases can compensate for mistaken diagnosis.

e. Apply the specific procedure which, during flight, has to be strict control of breathing, either by using short period of apnea (i.e., cessation of breathing) or by controlling the breathing rate using the aircraft chronometer. Two techniques are possible: (a) stop breathing for 20 seconds at a time or (b) regulate breathing to one inspiration every 6 to 7 seconds.

f. Remember the “blinker” operation principle: the blinker is a device directly sensing the oxygen flow. It is installed on the oxygen regulator panel or oxygen box. It only turns white or yellow if oxygen is flowing. The pilot must trust this indicator.

7.5 Specific Case: The Hyperventilation of a Subject Using PPBE (Portable Protective Breathing Equipment)

Hyperventilation while using portable protective breathing equipment (PPBE) results in an additional set of physiological problems. In PPBE, exhaled gases are recirculated. Chemical cartridges (containing alkaline absorber materials such as soda lime or lithium hydroxide) remove most of the metabolic CO_2 produced. However, CO_2 levels inside the hood still rise. This hypercapnic gas represents a powerful physiological stimulus to ventilation which can result in hyperventilation and its associated problems.

Additionally, there can be an increase in the resistance of breathing if the exhaled gases have to be driven through the CO_2 scrubbing system. The effects of hypercapnia and the resistance represent the primary causes of respiratory discomfort while wearing PPBE. These problems are often expressed as a sensation of dyspnea in the wearer. Therefore, for the most effective use of PPBE, potential users must be properly trained in the use of PBE devices and their performance characteristics.

7.6 Conclusion

Hyperventilation can cause several serious malaises which are often difficult to attribute to specific causes when the situation is subsequently examined. It is difficult to determine its real importance, but it seems certain that these malaises occur relatively often, with varying degrees of severity. These malaises occur totally unexpectedly, even in trained personnel. It is probably a reason which can explain (either wholly or partly) the physiological failure of a pilot.

8. GAS DILATION AND COMPRESSION

Gases are contained inside the closed or semi-closed cavities of the body. Applying Boyle-Mariotte's Law (P.V = constant) to these gases means that variations in volume and/or distension of the walls of these cavities will take place upon ascent to altitude. Distension of these cavities can be a very painful experience. This is what is known as barotraumas. Barotraumas affect all the body's closed or semi-closed cavities which contain gas. These include the ears, sinuses, and teeth, the digestive tract, and the lungs.