TECHNICAL SPECIFICATION

**ISO/TS 16976-3**

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# Respiratory protective devices — Human factors —

## Part 3: Physiological responses and limitations of oxygen and limitations of carbon dioxide in the breathing environment

*Appareils de protection respiratoire — Facteurs humains —*

*Partie 3: Réponses physiologiques et limitations en oxygène et en gaz carbonique dans l'environnement respiratoire*



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# Foreword

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ISO/TS 16976-3 was prepared by Technical Committee ISO/TC 94, *Personal safety — Protective clothing and equipment*, Subcommittee SC 15, *Respiratory protective devices*.

ISO/TS 16976 consists of the following parts, under the general title *Respiratory protective devices — Human factors*:

— *Part 1: Metabolic rates and respiratory flow rates*

— *Part 2: Anthropometrics*

— *Part 3: Physiological responses and limitations of oxygen and limitations of carbon dioxide in the breathing environment*



# Introduction

Due to the nature of their occupations, millions of workers worldwide are required to wear respiratory protective devices (RPD). RPD vary considerably, from filtering devices, supplied breathable gas devices, and underwater breathing apparatus (UBA), to escape respirators used in emergency situations (self-contained self-rescuer or SCSR). Many of these devices protect against airborne contaminants without supplying air or other breathing gas mixtures to the user. Therefore, the user might be protected from particulates or other airborne toxins but still be exposed to an ambient gas mixture that differs significantly from that which is normally found at sea level. RPD that supply breathing air to the user, such as an SCBA or UBA, can malfunction or not adequately remove carbon dioxide from the breathing space, thus exposing the user to an altered breathing gas environment. In special cases, RPD intentionally expose the wearer to breathing gas mixtures that significantly differ from the normal atmospheric gas mixture of approximately 79 % nitrogen and 21 % oxygen with additional trace gases. These special circumstances occur in aviation, commercial and military diving, and in clinical settings.

Breathing gas mixtures that differ from normal atmospheric can have significant effects on most physiological systems. Many of the physiological responses to exposure to high or low levels of either oxygen or carbon dioxide can have a profound effect on the ability to work safely, to escape from a dangerous situation, and to make clear judgements about the environmental dangers. In addition, alteration of the breathing gas environment can, if severe enough, be dangerous or even fatal. Therefore, monitoring and controlling the breathing gas, and limiting user exposure to variations in the concentration or partial pressure of oxygen and carbon dioxide, is crucial to the safety and health of the worker.

This Technical Specification discusses the gas composition of the Earth's atmosphere; the basic physiology of metabolism as the origin of carbon dioxide in the body, respiratory physiology and the transport of oxygen to the cells and tissues of the body; and the subsequent transport of carbon dioxide from the tissues to the lungs for removal from the body. Following the basic physiology of respiration, this Technical Specification addresses the physiological responses to altered breathing environments (hyperoxia, hypoxia) and to the effects of excess carbon dioxide in the blood (hypercarbia). Examples are given from the relevant biomedical literature.

Finally, it deals with the impact of altered partial pressures/concentrations of oxygen and carbon dioxide on respirator use. The content of this Technical Specification is intended to serve as the basis for advancing research and development of RPD with the aim of minimizing the changes in the breathing environment, thus minimizing the physiological impact of RPD use on the wearer. If this can be accomplished, the health and safety of all workers required by their occupation to wear RPD will be enhanced.

# Respiratory protective devices — Human factors —

# Part 3: Physiological responses and limitations of oxygen and limitations of carbon dioxide in the breathing environment



## 1 Scope

This Technical Specification gives:

— a description of the composition of the Earth's atmosphere;

— a description of the physiology of human respiration;

— a survey of the current biomedical literature on the effects of carbon dioxide and oxygen on human physiology;

— examples of environmental circumstances where the partial pressure of oxygen or carbon dioxide can vary from that found at sea level.

This Technical Specification identifies oxygen and carbon dioxide concentration limit values and the length of time within which they would not be expected to impose physiological distress. To adequately illustrate the effects on human physiology, this Technical Specification addresses both high altitude exposures where low partial pressures are encountered and underwater diving, which involves conditions with high partial pressures. The use of respirators and various work rates during which RPD can be worn are also included.

## 2 Terms and definitions, symbols and abbreviated terms

### 2.1 Terms and definitions

For the purposes of this document, the following terms and definitions apply.

**2.1.1**
**alveoli**
**s. alveolus**
terminal air sacs of the lungs in which respiratory gas exchange occurs between the alveolar air and the pulmonary capillary

NOTE The alveoli are the anatomical and functional unit of the lungs.

**2.1.2**
**ambient temperature pressure saturated**
**ATPS**
standard condition for the expression of ventilation parameters related to expired air

NOTE Actual ambient temperature and atmospheric pressure; saturated water pressure.

**2.1.3**
**body temperature pressure saturated**
**BTPS**
standard condition for the expression of ventilation parameters

NOTE Body temperature (37°C), atmospheric pressure 101,3 kPa (760 mmHg) and water vapour pressure (6,27 kPa) in saturated air.

**2.1.4**
**carbaminohaemoglobin**
**$HbCO_2$**
haemoglobin that has bound carbon dioxide at the tissue site for transport to the lungs

**2.1.5**
**dead space**
‹anatomical› conducting regions of the pulmonary airways that do not contain alveoli and, therefore, where no gas exchange occurs

NOTE These areas include the nose, mouth, trachea, large bronchia, and the lower branching airways. This volume is typically 150 ml in a male of average size.

**2.1.6**
**dead space**
‹physiological› sum of all anatomical dead space as well as under-perfused (reduced blood flow) alveoli which are not participating in gas exchange

NOTE The volume of the physiological dead space can vary with the degree of ventilation. Thus, the physiological dead space is the fraction of the tidal volume that does not participate in gas exchange in the lungs.

**2.1.7**
**dyspnoea**
sense of air hunger, difficult or laboured breathing, or a sense of breathlessness

**2.1.8**
**end-tidal carbon dioxide**
**ET $CO_2$**
volume fraction of carbon dioxide in the breath at the mouth at the end of exhalation

NOTE End-tidal carbon dioxide corresponds closely to alveolar carbon dioxide.

**2.1.9**
**haemoglobin**
**Hb**
specific molecules contained within all red blood cells that bind oxygen or carbon dioxide under normal physiological states and transport either oxygen or carbon dioxide to or from the tissues of the body

**2.1.10**
**hypercarbia**
**hypercapnia**
excess amount of carbon dioxide in the blood

**2.1.11**
**hyperoxia**
volume fraction or partial pressure of oxygen in the breathing environment greater than that which is found in the Earth's atmosphere at sea level, which contributes to an excess of oxygen in the body

NOTE This can occur when a person is under hyperbaric conditions (i.e. diving), subjected to breathing gas mixtures with an elevated oxygen fraction, or during certain medical procedures

**2.1.12**
**hypoxia**
volume fraction or partial pressure of oxygen in the breathing environment below that which is found in the Earth's atmosphere at sea level

NOTE Anaemic hypoxia is due to a reduction of the oxygen carrying capacity of the blood as a result of a decrease in the total haemoglobin or an alteration in the haemoglobin constituents.

**2.1.13**
**hypocapnia**
volume fraction or partial pressure of carbon dioxide in the breathing environment or in the body that is lower than that which is found in the Earth's atmosphere at sea level

NOTE This usually occurs under hyperventilation conditions (i.e. diving) or in medical settings that contribute to a reduction of carbon dioxide in the body

**2.1.14**
**inotropic**
affecting the force of muscle contraction

NOTE A negative inotropic effect reduces and a positive inotropic effect increases the force of muscular contraction (e.g. both skeletal and heart muscle).

**2.1.15**
**medulla oblongata, pons**
areas of the brain where the respiratory control centre is located

**2.1.16**
**oxyhaemoglobin**
**$HbO_2$**
haemoglobin that has bound oxygen from the lungs for transport to the body tissues

**2.1.17**
**partial pressure**
pressure exerted by each of the components of a gas mixture to form a total pressure

EXAMPLE Air is a mixture of oxygen, nitrogen, carbon dioxide, inert gases (argon, neon), and water vapour. The volume fraction of oxygen in air is about 20,9 %. At sea level, total atmospheric pressure is 101,3 kPa (760 mmHg). Water vapour pressure is 6,26 kPa (47 mmHg) (fully saturated in the lungs at a body temperature of approximately 37 °C). To find partial pressure of oxygen, subtract vapour pressure from total atmospheric pressure and then multiply the oxygen volume fraction by the dry atmospheric pressure. Thus, 101,3 – 6,3 = 95,1 kPa (760 mmHg – 47 mmHg = 713 mmHg); 0,21 × 95,1 kPa = 19,9 kPa (= 149 mmHg). If the ambient pressure increases (as in diving), the partial pressure of each component gas increases. Thus, at 2 atm absolute, the partial pressure of oxygen in dry gas is 101,3 × 2 = 202,6 kPa (760 mmHg × 2 = 1 520 mmHg); 0,21 × 202,6 = 42,6 kPa (0,21 × 1520 mmHg = 319 mmHg) oxygen.

NOTE 1 Partial pressure is dependent on the volume fraction of the component gas.

NOTE 2 The partial pressure of a gas can increase or decrease while its relative volume fraction remains the same. Partial pressure drives the diffusion of gas across cell membranes and is, therefore, more important than relative volume fraction of the gas.

**2.1.18**
**respiratory quotient**
**$R_Q$**
ratio of volume of carbon dioxide exhaled to the volume of oxygen consumed as follows

$$R_Q = V\mathrm{CO}_2 / V\mathrm{O}_2$$

where

$\dot{V}CO_2$ is the volume of carbon dioxide exhaled;

$\dot{V}O_2$ is the volume of oxygen consumed

NOTE $R_Q$ gives an estimate of the content of substrate utilization during steady-state respiration and metabolism. At rest, $R_Q$ = 0,82 reflecting a substrate utilization of a combination of carbohydrates and fats as the primary energy source.

**2.1.19**
**respiratory system**
tubular and cavernous organs (mouth, trachea, bronchi, lungs, alveoli, etc.) and structures which bring about pulmonary ventilation and gas exchange between ambient air and blood

**2.1.20**
**standard temperature pressure dry**
**STPD**
standard conditions for expression of oxygen consumption

NOTE Standard temperature (0 °C) and pressure (101,3 kPa, 760 mmHg), dry air (0 % relative humidity).

**2.1.21**
**ventilation (general)**
process of exchange of air between the lungs and the ambient environment

## 2.2 Symbols and abbreviated terms

| | |
|---|---|
| APR | air purifying respirator |
| BSA | body surface area, expressed in $m^2$ |
| PAPR | powered air purifying respirator |
| SAR | supplied air respirator |
| SCBA | self-contained breathing apparatus |
| UBA | underwater breathing apparatus |
| $PCO_2$ | partial pressure of carbon dioxide |
| $P_ACO_2$ | alveolar partial pressure of carbon dioxide |
| $P_aCO_2$ | arterial partial pressure of carbon dioxide |
| $P_vCO_2$ | venous partial pressure of carbon dioxide |
| $PO_2$ | partial pressure of oxygen |
| $P_AO_2$ | alveolar partial pressure of oxygen |
| $P_aO_2$ | arterial partial pressure of oxygen |
| $P_iO_2$ | partial pressure of inspired oxygen |
| $P_vO_2$ | venous partial pressure of oxygen |

$V_E$ minute ventilation (expired)

total volume expired from the lungs in 1 min, in l/min (BTPS)

$V_I$ minute ventilation (inspired)

total volume of air inspired into the lungs in 1 min, in l/min (BTPS)

$VO_2$ oxygen consumption

volume of oxygen consumed by the human tissues, in l/min, derived from the difference between the minute volume of inhaled oxygen and the minute volume of exhaled oxygen.

$VCO_2$ carbon dioxide elimination rate

volume of carbon dioxide produced per minute, derived from the product of minute ventilation and the difference between the fractional concentrations of exhaled and inhaled carbon dioxide

# 3 Oxygen and carbon dioxide in the breathing environment: physiological responses and limitations

## 3.1 General

The Earth's atmosphere is composed primarily of nitrogen and oxygen along with some trace gases. Atmospheric carbon dioxide occurs in very low concentrations (approximately 0,03 %). Humans require oxygen as a primary element in the production of energy during aerobic cellular metabolism. Low atmospheric oxygen concentrations or partial pressures (such as occur at high altitude) can limit production of metabolic energy, leading to a compromise in physiological function. On the other hand, low concentrations of carbon dioxide in the breathing atmosphere do not appear to have any physiological consequence. Carbon dioxide is produced as a by-product of cellular metabolism and it is this source of carbon dioxide, not the normal atmospheric concentration, which carries a physiological consequence. However, increased environmental levels of carbon dioxide, as in the breathing space of respirators or in confined areas, can also have a profound effect on the respiratory system.

High concentrations of either oxygen or carbon dioxide can have dramatic physiological consequences. Hyperoxia, especially under ambient pressures greater than one atmosphere (atm), such as occur in diving, can be toxic and even fatal to humans. High concentrations of carbon dioxide can also have a profound effect on respiration and metabolism. This overview will address several issues:

— Oxygen and carbon dioxide in normal human physiology;

— Effects of hypoxia and hyperoxia on physiology;

— Effects of hypercarbia on physiology;

— Relevance to respiratory protective devices.

## 3.2 Oxygen and carbon dioxide gas exchange in the human lung

Normal minute ventilation takes place as a result of neural activity in the respiratory centres in areas of the brainstem known as the medulla oblongata and the pons. The movement of air in and out of the lungs facilitates the gas exchange necessary for normal metabolic function.

Gas exchange does not occur in all regions of the pulmonary system. Anatomical dead space (regions where gas diffusion to the blood does not occur) comprises about 150 ml volume within the pulmonary system. However, the physiological dead space can add a much larger volume depending on activity level. Inhaled gas passes through the regions of dead space to the pulmonary alveoli. Gas exchange occurs in the alveoli, which are in contact with blood capillaries.

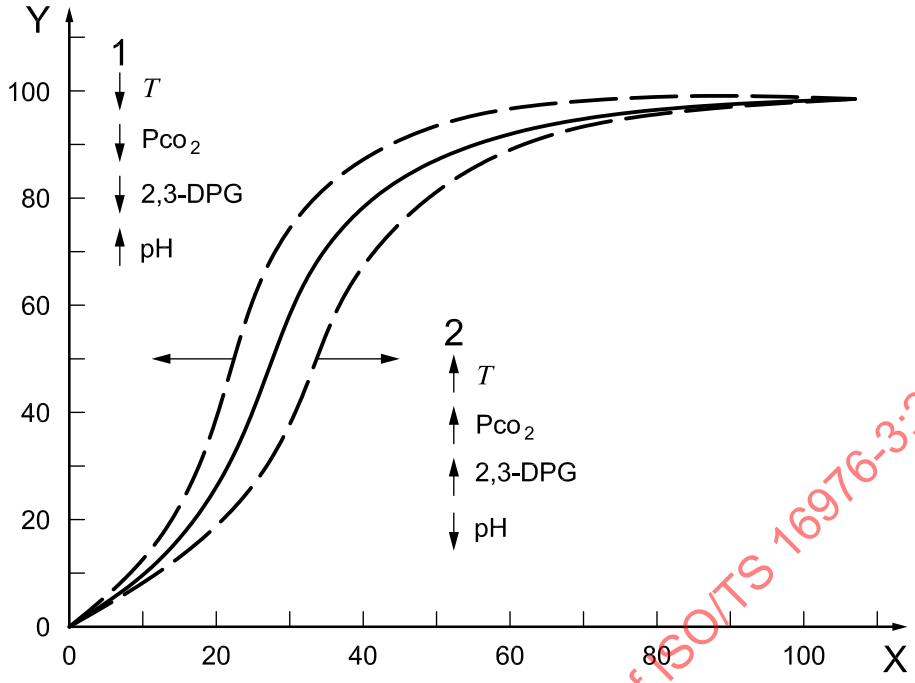
The exchange of oxygen into the blood stream and carbon dioxide out of the blood stream into the alveoli is driven by simple diffusion down a partial pressure gradient. The partial pressure of oxygen in the alveoli ($P_AO_2$) is approximately 13,3 kPa (100 mmHg) whereas the partial pressure of oxygen in the venous blood ($P_vO_{2)}$ is approximately 5,3 kPa (40 mmHg). Therefore, oxygen will move from the area of higher concentration of oxygen in the alveoli to the area of lower concentration of oxygen in the venous blood. Oxygen will also be transported into the red blood cells along a similar partial pressure gradient to be bound to haemoglobin. Conversely, the partial pressure of carbon dioxide in the venous blood ($P_vCO_2$) is roughly 6,1 kPa (46 mmHg) and is only 5,3 kPa (40 mmHg) in the alveoli. Therefore, carbon dioxide will move from the venous blood to the alveoli to be exhaled to the atmosphere.

After this gas exchange has taken place, arterial blood contains a $P_aO_2$ of 13,3 kPa (100 mmHg) and a $P_aCO_2$ of 5,3 kPa (40 mmHg). The arterial blood arriving at the cells will release oxygen and take up carbon dioxide based on a similar process of moving along a partial pressure gradient. After oxygen delivery to the cells has taken place, the red blood cells have a $PO_2$ of 5,3 kPa (40 mmHg) and a $PCO_2$ of 6,1 kPa (46 mmHg). Upon return to the lungs for another round of gas exchange, each gas again moves along its partial pressure gradient to repeat the process. Proper oxygen delivery to the cells and carbon dioxide removal from the body will occur as long as a match exists between ventilation of the lungs and blood perfusion driven by a healthy circulatory system.

### 3.3 Oxygen and carbon dioxide transport in the blood

Oxygen has a very low solubility in the blood. Therefore, oxygen is transported to the vital organs, working muscles, and brain by a special transport mechanism in the blood. When oxygen from the atmosphere diffuses from the alveoli to the circulation, about 25 % of the oxygen present in the alveoli is rapidly transported into the red blood cells and binds to haemoglobin to form oxyhaemoglobin. Oxyhaemoglobin in the red blood cells is carried through the arterial circulation to the capillaries where the oxygen diffuses from the red blood cells to the cells of the target tissues. The oxygen is then utilized in the aerobic metabolic processes in the cell mitochondria.

Several factors affect the affinity of oxygen for haemoglobin. For any given ambient $PO_2$, an increase in body temperature, blood lactic acid (↓ pH), increased $P_aCO_2$, or an increase in 2,3-diphosphoglycerate (DPG, a product of anaerobic metabolism in red blood cells) can decrease the affinity of oxygen for haemoglobin[4]). This phenomenon is known as the Bohr Shift, which makes oxygen delivery easier under acidotic conditions.




**Key**

| | |
|---|---|
| X | oxygen partial pressure (torr) |
| Y | haemoglobin saturation (%) |
| 1 | decreased P50 (P50 = one half saturation pressure)(increased affinity) |
| 2 | increased P50 (decreased affinity) |
| T | temperature |
| $PCO_2$ | partial pressure of carbon dioxide |
| 2,3-DPG | 2,3-diphosphoglycerate |
| pH | measure of the acidity or basicity of a solution |

NOTE 1 1 torr = 133 Pa.

NOTE 2 See Reference [4].

**Figure 1 — Shift of the oxyhaemoglobin dissociation curve by pH, carbon dioxide temperature, and 2,3-diphosphoglycerate (2,3-DPG)**

By contrast, carbon dioxide is about 20 to 25 times more soluble in blood than oxygen. Carbon dioxide produced as a by-product of metabolically active tissues diffuses from the cells of the tissue to the red blood cells in the circulation along a concentration gradient. Some of the carbon dioxide (approximately 5 to 10 %) is carried to the lungs in solution in the blood plasma. A portion of the carbon dioxide combines with water to form carbonic acid according to the equation:

$$CO_2 + H_2O \leftrightarrows H_2CO_3 \quad (1)$$

This reaction occurs slowly in the plasma and most of the carbon dioxide remains in solution in the plasma. However, a small amount of carbonic acid in the plasma dissociates to bicarbonate following the equation:

$$H_2CO_3 \leftrightarrows H^+ + HCO_3 \quad (2)$$

Whereas the reaction in Equation (2) occurs in very small amounts in the plasma, it occurs to a very large extent in red blood cells. Red blood cells contain the enzyme carbonic anhydrase (CA), which catalyzes the reversible reaction between carbon dioxide and $H_2O$ extremely rapidly (approximately $10^6$ reactions per second)[3] in the following manner:

$$CO_2 + H_2O \overset{CA}{\leftrightarrows} H_2CO_3 \leftrightarrows H^+ + HCO_3 \quad (3)$$

Approximately 70 % of the carbon dioxide is transported to the lungs in the form of bicarbonate. In addition, carbon dioxide combines with haemoglobin to form carbaminohaemoglobin. The affinity of haemoglobin for carbon dioxide increases as oxygen dissociates from haemoglobin during delivery of oxygen to the tissues (see also the Haldane effect[5]). Approximately 15 % of the carbon dioxide in the blood is transported to the lungs in the form of carbaminohaemoglobin.

### 3.4 Oxygen and carbon dioxide and the control of respiration

Human life is strongly dependent on an adequate supply of oxygen to support the metabolic processes that produce energy. As a result, the ability to sense changes in ambient $PO_2$ has evolved. In addition, although atmospheric carbon dioxide concentrations are almost negligible, carbon dioxide is produced as a product of metabolism and has a profound effect on the respiratory system. Thus, mechanisms for sensing $PCO_2$ in the blood have also evolved. Indeed, changes in $PCO_2$ are more powerful stimulators of respiration than changes in ambient $PO_2$. A detailed discussion of the physiological mechanisms involved in sensing changes in oxygen and carbon dioxide in the atmosphere or the blood is beyond the scope of this Technical Specification. However, a brief overview of the process is given below.

Chemical sensors (chemoreceptors) are present in both the central nervous system (medulla oblongata in the brain stem) and the peripheral nervous system integrated with the vascular system (i.e. carotid bodies in the carotid artery in the neck and chemoreceptors in the aorta) that are capable of sensing changes in $P_aO_2$, $P_aCO_2$ and pH in the arterial blood. When these areas sense changes in $P_aO_2$ and $P_aCO_2$, neural signals are integrated into a respiratory response that usually results in a normalization of the $P_aO_2$ and/or $P_aCO_2$. Under conditions of hypoxia, the decreased $P_aO_2$ is sensed primarily by peripheral chemoreceptors in the carotid bodies and the aortic bodies. The respiratory response is an increase in ventilation in order to increase the oxygen uptake to maintain metabolic energy production. However, if the carotid and aortic bodies are removed or damaged, a decrease in $P_aO_2$ can result in a decrease in ventilation because a reduction in brain $P_aO_2$ can act directly to depress respiratory cells in the brain. Low $P_aO_2$ also increases brain blood flow, thereby lowering $P_aCO_2$ and $[H^+]$ and decreasing ventilation. Figures 2 and 3 illustrate the basic relationships involved in the control of respiration.

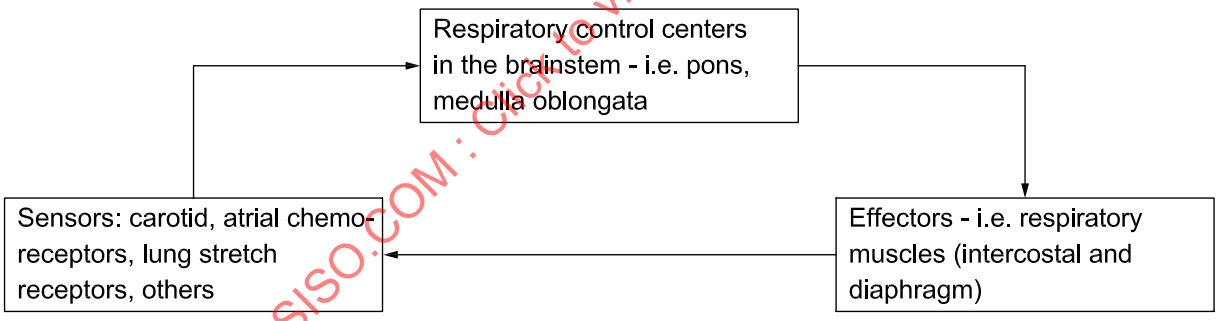
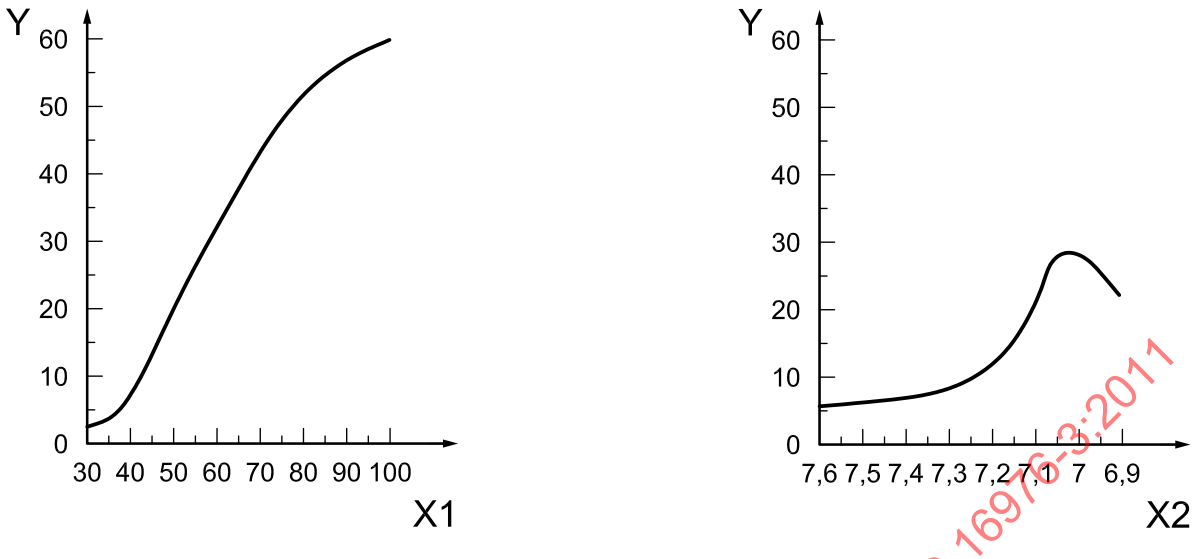



**Figure 2 — Basic relationships between sensor inputs, processing and outputs from the respiratory control mechanisms in the central nervous system, and the effectors (respiratory muscles) that actuate the respiratory process**




**a) carbon dioxide response**

**b) pH response**

**Key**

X1 arterial $PCO_2$ [mm Hg]

Y total ventilation, in l/min

**Key**

X2 arterial pH

Y total ventilation, in l/min

**Figure 3 — Ventilatory responses to changes in arterial carbon dioxide partial pressure and arterial pH for a person at rest**

Inhalation of supra-atmospheric concentrations of carbon dioxide also increases pulmonary ventilation. However, the increased $P_aCO_2$ stimulates ventilation largely in central chemoreceptors located in the medulla oblongata and pons area of the brainstem and, to a much lesser extent, in the peripheral carotid bodies. The increase in ventilation with increased $P_aCO_2$ is exaggerated in the presence of hypoxia. From a functional standpoint, ventilation is stimulated either in the presence of a decreased $P_aO_2$ (hypoxia) or an increased $P_aCO_2$ (hypercapnia). This results in a ventilatory response that ensures appropriate oxygenation of the blood and excretion of carbon dioxide as a product of metabolism.

## 3.5 Hyperoxia: physiological effects

One does not normally encounter an elevated oxygen level (hyperoxia) in the atmosphere. Hyperoxia is normally encountered in a hospital setting (e.g. when breathing 70 % oxygen) or during the use of special gas mixtures for underwater diving. Hyperoxia is defined as an excess of oxygen in the body due to exposure to an oxygen concentration above 20,9 % in the breathing environment or to a normoxic gas concentration under hyperbaric conditions. Breathing mild hyperoxic gas mixtures (i.e. with ambient $PO_2$ of approximately 1,3 kPa or 10 to 300 mmHg higher than normal) for a limited period of time (e.g. a working shift) is usually not harmful However, breathing hyperoxic gas mixtures under hyperbaric conditions above 1 atm can be harmful.

Oxygen toxicity can occur when the partial pressure of inspired oxygen reaches a level where neurological or pulmonary changes become pathological. At sea level, breathing a hyperoxic gas mixture over many hours can result in pulmonary changes through a direct effect of oxidative stress on alveolar cells. Under greater pressure than 1 atm (101,3 kPa or 760 mmHg), such as occurs during diving and caisson work, hyperoxic exposure can have effects on the nervous system as manifested by seizures. Seizures will not occur while breathing 100 % oxygen at sea level (1 atm). However, seizures are a potential risk while breathing 100 % oxygen at 2 atm or more (≥ 202,6 kPa or 1520 mmHg). Much of the research on hyperoxia has been performed on the professional underwater diving community (commercial and military). Professional divers are often required to breathe gas mixtures other than air during the dive. Greater than normal oxygen can be administered to effect “nitrogen washout”, thereby limiting the potential for decompression sickness and inert gas narcosis. However, breathing hyperoxic gas mixtures carries the risk of oxygen toxicity since the partial pressure of oxygen increases with depth. The potentially heavy exercise performed by commercial and military divers and an increase in hypercarbia can accelerate the effects of oxygen toxicity[11].

### 3.6 Hypoxia: physiological effects

Much of the physiological research on hypoxia has been performed during high altitude studies (mountain climbing or aviation). In extreme hypoxia, there is not enough oxygen to maintain basal metabolism and the person dies.

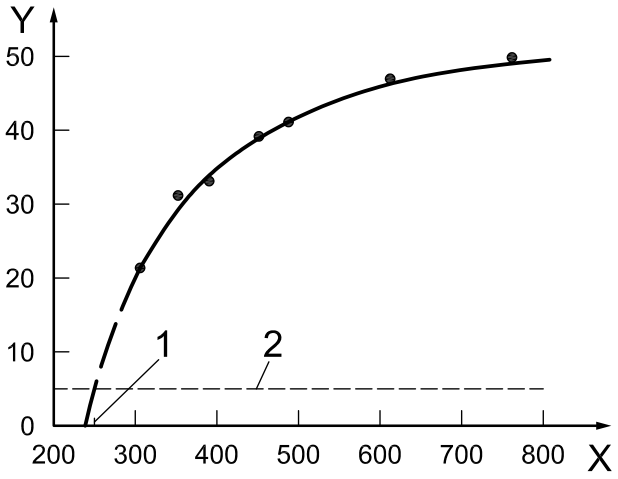
Before the first successful ascent to the summit of Mt. Everest without supplemental oxygen in 1978, research suggested that the ambient $PO_2$ at that altitude (8 848 m or 29 028 ft) was lower than that needed to sustain basal metabolic needs. Therefore, ascent to the summit could not be performed without supplemental oxygen[36]. Calculations indicated that the maximal oxygen uptake was equal to that required for basal metabolism. Therefore, there was not enough "extra" oxygen available to perform physical work. However, the air density (and therefore the atmospheric $PO_2$) at the summit of Mt. Everest varies seasonally. Refined calculations based on data collected indicated that, during the months of May to October, the atmospheric $PO_2$ was enough to perform the physical work required to reach the summit, whereas during the winter months the ambient $PO_2$ was not enough. The atmospheric $PO_2$ at the summit of Mt. Everest is 6,6 kPa (49,3 mmHg) and is approximately the same as breathing a gas mixture containing only 5 to 6 % (ambient $PO_2$ of 5,1 to 6,0 kPa or 38 to 45 mmHg) oxygen at sea level.

NOTE Atmospheric $PO_2$ at sea level is 21,2 kPa or 159 mmHg.

Exposure to hypoxia results in several significant physiological adjustments. The most noticeable change occurs with pulmonary ventilation. Acute hypoxia results in an increased ventilatory response[9] and if the hypoxia is sustained, the peripheral chemoreceptors become hypersensitized and the ventilatory response to hypoxia and hypercapnia increases. The increased ventilatory response serves to increase the oxygen content of the blood and eliminate the increased $PCO_2$ in the lungs[4] and is accompanied by a concomitant increase in cardiac output as a result of central nervous system stimulation[11]. At the summit of Mt. Everest, alveolar $PO_2$ is 4,7 kPa (35 mmHg) despite the low atmospheric $PO_2$ (see Figure 4). This level of alveolar $PO_2$ is maintained primarily by extreme hyperventilation, which results in a decrease in $PCO_2$ leading to respiratory alkalosis[35]. However, the ventilatory suppression normally associated with respiratory alkalosis is overcome by the hypoxic stimulation of ventilation. Whereas measurable adaptive changes in the cerebrovascular response to mild hypoxia occur over days to weeks[40], when the ambient $PO_2$ falls below a critical value, there is not enough oxygen being transported to the vital organs and the central nervous system to sustain life and health. With atmospheric oxygen concentrations below about 4 to 5 % (ambient $PO_2$ is 3,9 to 5,3 kPa or 30 to 40 mmHg), a loss of consciousness and death will ensue within minutes. The victim is often unaware of the progression to loss of consciousness[11].

Much of our knowledge of the consequences of quick and complete removal of oxygen comes from research on rapid decompression of pilot cabins and the re-supply of emergency gas. Of particular relevance is effective performance time (EPT), the duration of time that one is able to conduct useful flying duties, e.g. taking appropriate corrective action, in this situation. At 10 000 m where $P_iO_2$ is approximately 40 mmHg, i.e. lower than normal $P_vO_2$, EPT will be less than 1 min. This is relevant to users of RPD who become disconnected from their breathable gas supply[10].

NOTE EPT is also known as "time of useful consciousness".




**Key**

X barometric pressure (mmHg)

Y maximum oxygen uptake [(ml/min)/kg]

1 summit of Mt. Everest

2 basal oxygen uptake

NOTE 1 The maximal oxygen uptake at the summit was predicted to be the same as the basal oxygen uptake, indicating that no work would be possible. Also note that $\dot{V}O_{2\,max}$ near the summit is exquisitely sensitive to barometric pressure.

NOTE 2 See Reference [36].

**Figure 4 — Maximal oxygen uptake in acclimatized subjects plotted against barometric pressure using the data from the Silver Hut expedition**

Humans can adapt to chronic hypoxia. Some 40 million people live and work at altitudes between 3 048 m and 5 486 m (10 000 to 18 000 ft). There are some immediate adaptive responses to exposure to hypoxia, e.g. an increase in ventilation. Nevertheless, full adaptation to chronic hypoxia can take months, or even years. Native populations in the Peruvian Andes and the Himalayas reside at altitudes as high as 5 486 m (18 000 ft)[22]. The barometric pressure at this altitude is approximately 50,6 kPa (380 mmHg), about half of that measured at sea level. At this altitude, atmospheric $PO_2$ is roughly 10,5 kPa (79 mmHg). The physiological adaptations to this low atmospheric $PO_2$ from living at high altitude include increases in the number of pulmonary alveoli, increased blood concentration of haemoglobin and myoglobin (Mb) in the muscle, increased pulmonary ventilation, and a decreased ventilatory response to hypoxia[11]. In spite of an atmospheric $PO_2$ of 10,5 kPa (79 mmHg), and an arterial $PO_2$ of 5,1 kPa (38 mmHg), the blood haemoglobin is still 73 % saturated. Because the oxyhaemoglobin dissociation curve is sigmoidal, even a small decrease in atmospheric $PO_2$ at this altitude can result in a rapid oxygen desaturation of haemoglobin, down to about 50 %.

Although the vast majority of high altitude acclimatized individuals show little or no adverse effects under these circumstances, a small minority of those acclimatized individuals develop Monge's disease (chronic mountain sickness) over time, characterized by a poor ventilatory response to hypoxia, low $P_aO_2$ and high $P_aCO_2$, high hematocrit, pulmonary hypertension, right heart failure, dyspnoea, and lethargy[20]. Unacclimatized individuals rapidly exposed to high altitude would eventually be incapacitated from the potentially life-threatening health effects of hypoxia.

There is also evidence that hypoxia can affect the thermoregulatory response to cold stressors. Exposure to intermittent hypobaric hypoxia sufficient to cause acclimation resulted in a blunted thermoregulatory response to a standard cold air exposure test at sea level. Much of the response was through peripheral vasoconstriction that might have been driven by hypocapnia due to the increase in the ventilatory response to hypoxia[18].

In studies conducted by Angerer and Norwak[1], designed to determine if human subjects could tolerate short-term intermittent exposure to hypoxia, it was found that humans could tolerate daily occupational exposure to an atmosphere composed of 13 to 15 % (atmospheric $PO_2$ of 13,2 to 15,2 kPa or 99 to 114 mmHg) oxygen (balance nitrogen) for periods of approximately 8 h without significant physiological or health consequences. However, drops in $P_aO_2$ to values of less than 50 mmHg have been recorded at an oxygen concentration of 14,8 % (approximately equal to $PO_2$ at an altitude of 3 400 m). The results of this study have been used to support the suggestion that healthy workers could function in a hypoxic atmosphere designed for fire suppression with no significant ill-effects. However, the authors cautioned that workers with cardiovascular or pulmonary disease might not tolerate a hypoxic work environment. A summary of the effects of hypoxia and hyperoxia appear in Table 1 below.

**Table 1 — Potential effects and limitations on human tolerance imposed by exposure to decreasing concentrations of oxygen in the inspired air while at rest and at an extremely high workrate (see References [1] and [14])**

| | | At rest | | Extremely high workrate | |
|---|---|---|---|---|---|
| **Average level of oxygen in the air** (%) | **Average ambient $PO_2$ *(i.e. altitude) in the breathing space** (mmHg) | **Potential effects and/or limitations** | **Exposure limit** (time) | **Potential effects and/or limitations** | **Exposure limit** (time) |
| **100** | **760** | Mild respiratory depression, followed by stimulation, pulmonary injury | Many hours | Slight increase in exercise performance, pulmonary injury due to toxic effects of oxygen | hours |
| **100** | **1520** (2 atm) e.g. underwater diving | Seizure, loss of consciousness, depression of the cardiopulmonary system | ~30 min | Seizure, loss of consciousness, cardiopulmonary depression, death | <30 min |
| **70** | **532** (1 atm) | No restrictions within the exposure limit, well tolerated | days | No restrictions within the exposure limit, well tolerated | days |
| **20,9** | **158** (1 atm) e.g. normal atmospheric pressure | Normal – no symptoms in a healthy person | indefinite | Normal – no symptoms in a healthy person | indefinite |
| **20,9** | **79** (5 486 m) | Large increase in ventilation, severe limitations on activity | <30 min | Collapse, unconsciousness | <30 min |
| **20,9** | **49** (8 848 m) | Large increase in ventilation, severe limitations on activity | <30 min | Collapse / unconsciousness | <2 min, unable to perform work |
| **19,5** | **148** (1 atm) | Easily tolerated, no symptoms | indefinite | Easily tolerated, no symptoms | indefinite |
| **13 to 15** | **99 to 114** (1 atm) | Tolerated in a healthy individual, cardiopulmonary patients might show symptoms | hours | Decrease in exercise tolerance, cardiopulmonary patients might show symptoms | <5 min |

### 3.7 Hypercarbia: physiological effects

The physiological effect of increasing the $PCO_2$ in the breathing atmosphere has been studied extensively for decades. Hypercarbia actually serves a protective purpose due to its stimulatory effect on ventilation. As previously noted, alveolar and arterial $PO_2$ can be maintained by hyperventilation by direct stimulation of the chemoreceptors in the carotid bodies as well as stimulation of the respiratory centres in the brain and brainstem[4].

Endogenously produced carbon dioxide is known to induce pronounced vasodilation in heavy working muscles. Systemic hypercarbia is also a potent stimulus of peripheral vasculature. In cerebral blood vessels hypercarbia also induces vasodilation due to the critical importance of maintaining full oxygenation to the brain[38]. This effect of carbon dioxide seems, therefore, to increase both the oxygen uptake by stimulating ventilation, but also oxygen delivery through increased cerebral blood flow (probably due to vasodilation of cerebral blood vessels). In fact, when atmospheric carbon dioxide was chemically scrubbed while human subjects were exposed to simulated high altitude in a hypobaric chamber, both regional cerebral oxygen and peripheral oxygen levels decreased[16]. Indeed, breathing gas mixtures containing 3 % carbon dioxide and 35 % oxygen have been used at altitude to increase both pulmonary ventilation and cerebral oxygen delivery by increasing cerebral vasodilation and peripheral oxygen delivery to skeletal muscle, thereby increasing human performance[17].

In spite of the use of supplemental carbon dioxide in both clinical and high altitude settings, there are some drawbacks to breathing elevated concentrations of carbon dioxide. Stereoacuity and perception of coherent motion are reduced at atmospheric concentrations of only 2,5 % carbon dioxide[31], [37]. Breathing carbon dioxide concentrations ranging from 2,5 to 8 % (balance oxygen) has been shown to reduce retinal blood flow[21], and increase the rate of body core temperature heat loss during snow burial[14]. Nevertheless, there appear to be no pronounced disabling physiological effects or clinical symptoms associated with exposures of up to 5 % carbon dioxide[39]. Breathing gas mixtures containing > 6,5 % carbon dioxide decreased performance on reasoning tasks and subjectively increased both irritability and discomfort[27]. Subjects participating in a simulated emergency Space Shuttle egress while wearing the launch and entry suit experienced a build-up of more than 6 % carbon dioxide in the non-conformal helmet during a five minute walk on a treadmill at 5,6 km/h (3,5 mph). This carbon dioxide concentration limited the ability of most subjects to complete the simulated egress[6]. Breathing 10 % carbon dioxide can result in a resting $V_E$ of 75 l/min which is maximal for many healthy middle-aged individuals[28]. Thus, while breathing increased carbon dioxide concentrations might seem beneficial by increasing cerebral blood flow, which serves to protect brain oxygenation during exposure to high altitude, this strategy is tempered with the consideration that carbon dioxide can also significantly impede performance at sea level. Inhalation of carbon dioxide is known to induce anaesthesia in animals and inhalation of 30 % carbon dioxide can induce anaesthesia in humans. However, the administration of carbon dioxide to achieve anaesthesia in humans is complicated by the frequent incidence of seizures during the exposure[20]. Carbon dioxide can induce narcosis in patients with ventilatory failure, probably through changes in intracellular pH that alters the metabolic processes that underlie the narcosis[20].

Increased concentrations of carbon dioxide in the breathing space can result in physiological effects and responses that limit human performance.

As mentioned above, elevated concentrations of carbon dioxide or increased ambient $PCO_2$ affect pulmonary minute ventilation disproportionately to the level of exercise, thus increasing the metabolic cost of breathing as well as inducing a sense of dyspnoea that limits tolerance to exercise[6]. Increasing levels of carbon dioxide to 2,5 % can impair visual stereoacuity and motion detection[31],[37], while carbon dioxide levels of more than 6,5 % affect reasoning time[27]. In addition, higher concentrations of carbon dioxide in the breathing space can increase the displacement of oxygen from haemoglobin resulting in shifting the oxygen dissociation curve to the right and reducing the oxygen carrying capacity of the blood[20]. This could exacerbate the effects of concurrent hypoxia. A major concern is the potential of elevated levels of carbon dioxide to induce cardiac arrhythmias[20]. These arrhythmias are seldom serious but, combined with ischemic heart disease, could be life threatening. All of these effects will impact the ability of an individual to perform tasks while exposed to elevated levels of carbon dioxide in the breathing atmosphere.

Other effects of breathing high carbon dioxide are also significant. Hypercarbia can cause a decrease in cerebral cortex excitability, induce the release of enough catecholamines (adrenaline and noradrenline) from the sympathetic nervous system to cause cardiac arrhythmias, and reduce cardiac contractility. Hypercarbia also increases the pain threshold through its effects on the central nervous system. All of the above can affect the ability to think clearly, negatively impact the cardiovascular system, and reduce the ability to feel pain. Effects of carbon dioxide can also include dyspnoea, irritability, nausea, tunnel vision and reduced capacity for exercise.

A report by the National Institute for Occupational Safety and Health (NIOSH)[24] summarized 19 studies on the effects of carbon dioxide on human subjects. Both the physiological responses to acute and longer term exposure were described. For high work rates, (exercise on a treadmill at 7 km/h with a 10 % gradient), exposure to 5 % carbon dioxide during this level of activity represented the upper limit of tolerance. Exposure to 3 % carbon dioxide posed no strict limitation on activity. Note, however, that breathing concentrations consistently above 3 % reduced the time to exhaustion during high intensity running on a treadmill[8]. Absolute duration of exposure to carbon dioxide (while at rest) was also summarized. Human exposure to 10 % carbon dioxide could only be tolerated for a few minutes before loss of consciousness, while exposure to 7 % carbon dioxide was tolerable for less than 30 min and usually resulted in a carbon dioxide induced headache. Exposure to 5 % carbon dioxide was tolerable for up to 8 h and 3 % carbon dioxide could be tolerated for up to 15 h. Exposure to 1,5 % carbon dioxide could be tolerated essentially indefinitely (Table 2).

**Table 2 — The potential effects and limitations on human tolerance imposed by exposure to increasing concentrations of carbon dioxide in inspired air at rest and at an extremely high workrate[1].**

| | At rest | | Extremely high workrate | |
|---|---|---|---|---|
| **Average % $CO_2$** (in air) | **Potential effects and/or limitations** | **Exposure limit** (time) | **Potential effects and/or limitations** | **Exposure limit** (time) |
| **1,5** | No restrictions on activity | Indefinite exposure | Increase in ventilation | unknown |
| **2,5** | Increase in ventilation | unknown | Increase in ventilation | 2 h |
| **3,0** | Increase in ventilation, no restrictions within the exposure limit | 15 h | Increase in ventilation | 30 min |
| **5,0** | Increase in ventilation, no restrictions within the exposure limit | 8 h | Increase in ventilation, collapse/unconsciousness | 15 min |
| **7,0** | Increase in ventilation, severe limitations on activity | <30 min | Collapse/unconsciousness | n/a |
| **10,0** | Increase in heart rate, collapse/unconsciousness | <2,0 min | Collapse/unconsciousness | n/a |



The summarized NIOSH data in Table 2 clearly indicate that increasing the environmental (inhaled) concentration of carbon dioxide significantly attenuates the duration of exercise on a treadmill at a constant rate. This effect has been corroborated by a study conducted at NASA which clearly showed that the increase in carbon dioxide concentration in the breathing space of the non-conformal helmet of the Launch Entry Suit worn by US astronauts significantly reduced the ability to complete a simulated emergency egress (5 min treadmill walk at 5,6 km/h). The effects of 5 % to 6 % carbon dioxide in the breathing space of the non-conformal helmet resulted in only one third of the subjects wearing the Launch Entry Suit being able to complete the five minute walk. The effect was clearly dose-related and aggravated by increasing the antigravity suit pressure bladders situated in the lower extremities and abdominal region to more than 6,9 kPa (1,0 psi)[6].

The published work used to construct Table 2 suggests that the reduction of the duration of exercise with increasing concentrations of carbon dioxide in the breathing atmosphere could be predicted from the increase in metabolic acidosis, as evidenced by a decrease in blood pH, the increase in blood $PCO_2$, and the increase in blood potassium and phosphorus[7],[8],[23],[29],[19]. In any case, it is apparent that increased $PCO_2$ in the breathing space or hypercapnic physiologic state will result in a decrease in overall physical performance for all the reasons outlined in Table 2 and in this section.

The use of respirators while at rest or during exercise has a significant effect on the concentration of inhaled carbon dioxide. Assuming that the respirator is used in a normal atmosphere at sea level, it is likely that there will be incomplete elimination of carbon dioxide from the respirator breathing space. Thus, with each respiratory cycle, a small amount of carbon dioxide will remain in the breathing space. This small amount of carbon dioxide will be re-inhaled with the next breathing cycle. Over time, the concentration of carbon dioxide in the breathing space could continue to increase to levels that have a significant impact on the physiology of the wearer. In addition, wearing a filtering device inevitably increases the work of breathing because of the resistance to air flow across the filter media. Even with breathing gas supplied devices, the exhalation resistance increases the effort of breathing. The increased effort of breathing can have two possible results:

a) hypoventilation;

b) increased production of carbon dioxide due to the increased metabolism of intercostals and diaphragmatic muscle effort required to overcome breathing resistance.

Hypoventilation is one of the most important causes of hypercapnia. The effort of maintaining ventilation in the presence of breathing resistance can create dyspnoea, as well as a feeling of distress under some circumstances. These combined factors will therefore increase the amount of carbon dioxide the respirator wearer is exposed to. However, the effect of carbon dioxide on the physiology will be the same whether or not the person is wearing a respirator or breathing in an atmosphere that contains an elevated concentration or partial pressure of carbon dioxide (see 3.8).

Nevertheless, as in the case of hypoxia, human beings have a remarkable capacity to adapt to long term low level (1,2 % carbon dioxide) atmospheric exposure leading to hypercarbia[12]. Research into the experiences of submarine crews and astronauts exposed for days or months to mild hypercarbia in submarines or space stations has indicated that metabolic adjustments to hypercarbia occur within days[20].

As mentioned previously, one of the most visible physiological responses to hypercarbia is an increase in pulmonary ventilation. However, after several days of exposure to elevated carbon dioxide in the breathing atmosphere, ventilation returned to baseline. The mechanism of this adaptive response appears to be an attenuation in the central chemoreceptor response since the compensatory response to hypercarbia is too rapid to be the result of a blood acid-base buffering system that involves the renal resorption of bicarbonate[12]. However, long-term hypercarbia tends to reduce the resorption of bicarbonate in spite of the fact that arterial pH returns toward baseline[20]. Moreover, the increase in brachial blood flow in response to hypercapnia gradually decreases to baseline (this effect is not observed in the cerebral vasculature) indicating an adaptive process to hypercapnia[38].

Unexpectedly, humans can survive exposure to extreme hypercarbia as long as the patient is well oxygenated with a blood $PO_2$ of more than or equal to 7,3 kPa (55 mmHg). In clinical cases in which extreme hypercarbia in the absence of hypoxia was reported, all patients recovered completely with no evidence of short or long-term sequelae (e.g. no neurological deficit)[13][25]. The critical fact to bear in mind is the normal oxygenation of the patient during exposure to the episodes of extreme hypercarbia. Should the blood $PO_2$ have fallen below 7,3 kPa (55 mmHg) (hypoxia), the result would have been almost certainly a far worse clinical outcome.

NOTE Extreme hypercarbia (also known as supercarbia) is a blood $PCO_2$ of more than 19,9 kPa (150 mmHg). This is generally only observed in the patient population with certain types of respiratory disease, but illustrates the wide variation in sensitivity to carbon dioxide.

### 3.8 Relevance to the use of respiratory protective devices (RPD)

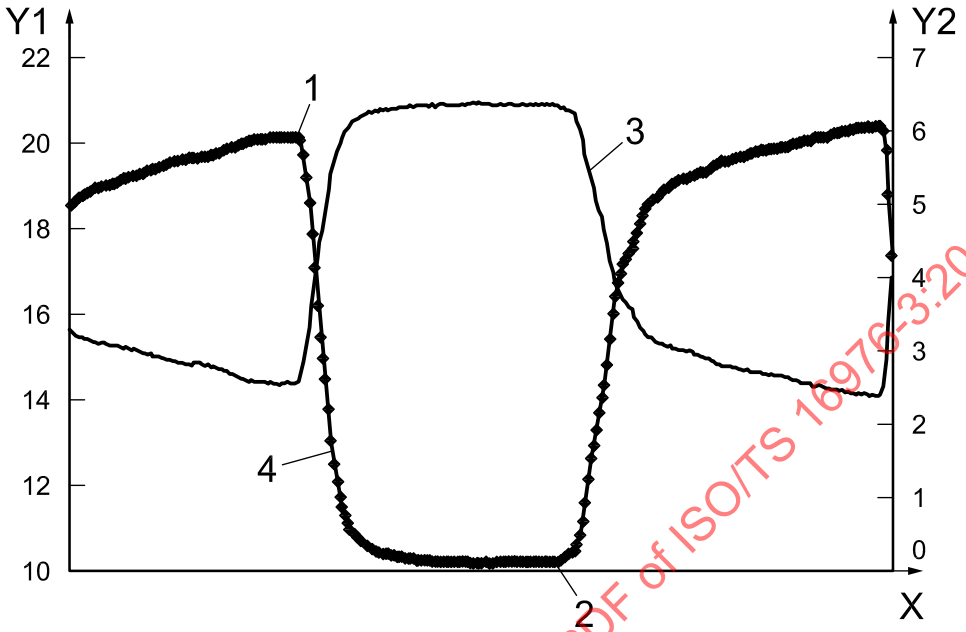
The use of RPD can lead to breathing gas mixtures that differ significantly from that found in the normal atmosphere. There is always the potential for the concentration of exhaled carbon dioxide in the breathing zone of the respirator to increase because of incomplete removal by the specific respirator. With incomplete carbon dioxide removal upon exhalation, an amount of carbon dioxide above atmospheric levels will be re-inhaled during the following inhalation. Exercise compounds the effects of respirator carbon dioxide accumulation due to increased metabolic carbon dioxide production. At rest, $\dot{V}CO_2$ is approximately 0,23 l/min. During moderate to heavy exercise, $\dot{V}CO_2$ can increase to 1,65 to 2,0 l/min while during maximal exercise $\dot{V}CO_2$ can exceed 4,0 l/min. Breathing an atmosphere containing 10 % carbon dioxide can stimulate $\dot{V}_E$ to approximately 75 l/min[28]. At this level of $\dot{V}_E$ (induced entirely by breathing carbon dioxide and not due to exercise), issues such as effort of breathing and respirator breathing resistance can become dependent upon the type of respirator that is being worn. Withdrawal of the carbon dioxide leads to a rapid reversal of these effects[11].

It has been shown that an increase in breathing resistance results in hypoventilation[2]. Hypoventilation is the primary cause of hypercarbia. The increased resistance to breathing increases the amount of muscular work required at any given workload which will, in turn, lead to increased metabolic carbon dioxide production, increased end-tidal carbon dioxide, heart rate and a reduced tolerance for work[26]. Paradoxically, the greatest percent change in the decreased workload tolerance occurred at the lighter workloads. Clearly, occupations that do not involve heavy workloads but require the use of RPD are also affected significantly by increased breathing resistances imposed by the RPD. Indeed, should the resistance to breathing involve an increase in breathing frequency concurrent with a decrease in tidal volume, the result will be hypercapnia[26]. It has been shown that an increase in inspiratory breathing resistance will result in a decrease in $\dot{V}_E$ when the breathing concentration of carbon dioxide is 3 %. As the metabolic carbon dioxide increases (i.e. increased $\dot{V}CO_2$), ventilation will be stimulated but might not increase in the presence of an increased breathing resistance imposed by the respirator. Thus, a negative cycle is initiated by the increased breathing resistance which induces hypoventilation, leading to increased hypercarbia. The resulting hypercarbia stimulates ventilation and increased re-breathing of carbon dioxide which further exacerbates the hypercarbia. Eventually, dyspnoea caused by hypercarbia might be so great as to be intolerable. Further research in this area is required to determine the degree to which this putative cycle influences respirator wearers in dangerous environments. In any case, Babb et al (1989)[2] demonstrated that an increased respirator breathing resistance in conjunction with an increased level of carbon dioxide (3 %) in the breathing space resulted in a decrease in physical performance.

As noted, a sufficient increase in carbon dioxide or a decrease in oxygen can stimulate the respiratory system and increase ventilation, thereby placing an increased demand on the respirator. A sufficient build up of carbon dioxide in the breathing space of the respirator can negatively impact the ability to perform tasks. Similarly, a decrease in the oxygen level in the breathing space of a respirator can result in a stimulation of ventilation and a loss of consciousness if the oxygen level is less than 5 % (ambient $PO_2$ of 3,9 to 5,3 kPa or 30 to 40 mmHg). For oxygen concentrations below about 15 % (at 1 atm), the ability to work is seriously diminished and can induce early symptoms such as chest pain or discomfort in persons with coronary artery disease. However, the duration of exposure is important because it usually influences the degree of clinical symptoms observed. High levels of oxygen (even up to 100 %) can be potentially toxic but is generally well tolerated at normal atmospheric pressures for several hours. Breathing increased $PO_2$ while exposed to greater than 2 atm also can result in oxygen toxicity and should be avoided (see Table 2).

It should be noted that the carbon dioxide limitations for respirators stated herein reflect maximal allowable average concentrations in the inspired air over time. During normal respiration, concentrations of carbon dioxide in the breathing zone at the end of exhalation can be as high as 8 %, particularly during exercise. However, for a respiratory device with a small dead space, at the start of the following inhalation, concentrations quickly decrease as the breathing zone is flooded with carbon dioxide-deficient and oxygen-enriched air. As such, carbon dioxide levels decrease to near atmospheric conditions in the early stages of inhalation and remain at such levels until inhalation ends and another exhalation begins (Figure 5). As long as this occurs, carbon dioxide concentration averaged for the complete inhalation phase of a breath is unlikely to exceed maximal allowable concentrations. The example presented in Figure 5 illustrates that with an end exhalation carbon dioxide concentration in the breathing zone of an APR of roughly 6 %, the average

concentration for all data of the inspired air was approximately 0,7 %. Therefore, it is important to understand that average inspired carbon dioxide concentrations are not the same as single maximal values normally found at the end of an exhaled breath.




**Key**

X time
Y1 oxygen concentration (%)
Y2 carbon dioxide concentration (%)
1 end exhalation
2 end inhalation
3 line representing oxygen measurements
4 line representing carbon dioxide measurements

NOTE The end of exhalation and end of inhalation markings are approximate for the breath.

**Figure 5 — Typical breath recording of oxygen and carbon dioxide concentrations in the breathing zone of a filtering RPD during exhalation, followed by inhalation while exercising at sea level**

From Figure 5, it is apparent that, with a breath recording cycle (exhalation followed by inhalation followed by exhalation), the concentration of carbon dioxide in the breathing space of the RPD decreases to nearly zero because of the influx of fresh air during the inspiratory phase of breathing (with a concurrent increase in oxygen). However, for this pattern to continue, it is essential that all or nearly all of the carbon dioxide in the breathing space at the end of expiration is eliminated. Should any carbon dioxide remain in the breathing space, it will be re-inhaled with the next breath. For a device with a large dead space, the lowest inhaled carbon dioxide can still be elevated. The lowest carbon dioxide levels have been measured to exceed 0,5%[33]. Repetition of this pattern could result in an increase in the concentration of carbon dioxide being re-inhaled into the lungs. A significant increase could result in hypercarbia, thus stimulating the physiological responses to increased carbon dioxide that have been discussed previously. This recording would be expected to change with RPD type, size of person, and workrate.

Based on respirator carbon dioxide limitations addressed in other standards, it is evident that most requirements are below concentrations known to impair physical or psychological performance, possibly as a built-in margin of error for individuals who might exhibit greater carbon dioxide sensitivity than others. Nevertheless, the differences between maximal allowable concentrations and concentrations known to significantly impair performance suggest that slightly higher levels of carbon dioxide could be permitted without harm for certain respirators and for certain periods of time.

At high workloads, the demand for oxygen increases to meet the metabolic demands of working muscles. This increased $\dot{V}O_2$ is accompanied by increases in heart rate and cardiac output. The increase in oxygen consumption is directly related to workload as shown in Table 3. This table is modified from ISO/TS 16976-1. The first five classes are described in ISO 8996.

**Table 3 — Metabolic rates and oxygen consumption associated with eight levels of work likely to be encountered in a variety of occupations. The carbon dioxide ($CO_2$) values represent the amount of carbon dioxide being exhaled into the atmosphere or respirator breathing space.**

| Class | Activity | Metabolic rate<br>W•m$^{-2}$ | $\dot{V}O_2$<br>l/min<br>(STPD) | $\dot{V}CO_2$<br>l/min<br>(STPD) |
|---|---|---|---|---|
| **1** | Rest | 65 | 0,344 | 0,282 |
| **2** | Low MR | 100 | 0,528 | 0,433 |
| **3** | Moderate MR | 165 | 0,872 | 0,715 |
| **4** | High MR | 230 | 1,215 | 1,090 |
| **5** | Very high MR | 290 | 1,533 | 1,38 |
| **6** | Very, very high MR<br>(2 h) | 400 | 2,114 | >2,144 |
| **7** | Intensive work<br>(15 min) | 475 | 2,510 | >2,510 |
| **8** | Exhaustive work<br>(2 to 5 min) | 600 | 3,171 | >3,171 |
| NOTE 1 The figures in this table are based on a person measuring 1,75 m, weighing 70 kg and with a BSA of 1,84 m$^2$. | | | | |
| NOTE 2 Derived from ISO/TS 16976-1. | | | | |

It is essential that individuals engaging in activities within the range of workloads outlined in Table 3 are able to increase their oxygen uptake to meet the increase in metabolic demand. If that person is wearing an RPD, it is essential that the RPD allows for this increase in oxygen consumption. If the person is wearing an APR in a "normal" atmosphere at sea level, then the partial pressure of oxygen is sufficient to allow the person to increase their $\dot{V}O_2$ to meet an increase in metabolic demand. Should a person be using an APR in an "abnormal" atmosphere (e.g. the partial pressure of oxygen is sub-atmospheric) then the person might be protected from particulates by the filter but might not be able to increase their $\dot{V}O_2$ to meet the metabolic demand because the amount of available oxygen in the atmosphere is too low (hypoxia). In a hyperoxic or hypoxic environment, there is no significant physiological difference between wearing an APR and not wearing an APR at all.

If the person is wearing an SCBA, then as long as the device is functioning correctly, the wearer should be able to increase their $\dot{V}O_2$ regardless of the atmospheric concentration or partial pressure of oxygen. However, since the air supply is limited, an increased $\dot{V}O_2$ will empty the air cylinder sooner than at a lower $\dot{V}O_2$.

Moreover, as the oxygen consumption increases in response to increased workload, so will the metabolically produced carbon dioxide. The increased production of carbon dioxide ($\dot{V}CO_2$) will be exhaled to either the atmosphere or into the breathing space of the RPD. If exhaled into the breathing space of the respirator, there is the possibility that the carbon dioxide will not be completely removed from the RPD breathing space and, therefore, will be re-breathed by the wearer. Re-breathing carbon dioxide will result in hypercarbia and stimulate ventilation. The greater the workload, the greater the chance of re-breathing carbon dioxide and