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PHYSIOLOGY OF BREATHING AND THE CONSEQUENCES OF UNSCIENTIFIC EXPOSURE TO ALTITUDE UNDER EXERTION CONDITIONS

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Abstract

This study aims to highlight the physiological aspects in basal conditions and the related changes that occur under effort conditions. The research is based on the study of specialized materials in the field of physiology and medicine, in an attempt to develop a study that captures these significant changes that occur in the human body during intense physical exercises. Also, this study captures the major changes that occur in altitude conditions, specific to athletes participating in training camps in such geographical areas; the risks of unscientific exposure to altitude; the clinical picture of altitude syndrome and the main ways to recover an athlete that suffers from altitude syndrome.

Keywords: effort, physiology, respiration, altitude, recovery

1. Introduction

The human body is a living organism, and different scientific disciplines research it multilaterally, like any living object. Human physiology deals with the study of interpersonal strategies from an individual and group viewpoint. It also aims at the interaction between the body and the environment, including important functions of the body, such as digestion, locomotion, reproduction or respiration.

2. Materials and methods

The present paper aims to highlight the main physiological changes of the respiratory system during the transition from basal conditions to effort conditions, as

well as understanding the mechanisms through which this transition is made, consulting scientific articles and medical books.To carry out this review study, the analysis of specialized studies was used as the main research method.

The hypothesis of the study is the following: if the main physiological changes that occur in the respiratory system are known, an overload of this system can be avoided. Also, a good understanding of the main compensatory mechanisms of hypoxia can influence the body's adaptation process to altitude and effort in altitude conditions.

3. Results and discussions

3.1. Anatomical components of the respiratory system

The human anatomy is extremely well defined, each component having an essential role from a mechanical and functional point of view. It has a well-individualized structure with respect to the morphology of the respiratory system, broken into elements and systems with distinct functions. The components of the respiratory system are the airways and the respiratory organs themselves.

3.2. The respiratory process

The respiratory process includes four essential mechanisms: pulmonary ventilations, respiratory gas exchange, the transportation of oxygen and carbon dioxide and the metabolic oxygen consumption at the cellular level.

Pulmonary ventilation is the movement of air in two directions, between the external environment and the alveoli. Inspiration is an active process performed by the contraction of the diaphragm muscle and intercostal muscles, a process in which, during the effort, including the contraction of the sternocleidomastiodine and scalene muscles. Exhalation is a passive process, which in effort becomes active by the contraction of the internal intercostal muscles and the right abdominals. (Cole, 2004).

The pressure of the fluid between the space formed between the visceral and the parietal pleura is called pleural pressure. It is generally negative. Under basal conditions, at the beginning of inspiration, pleural pressure measures - 5cmH_2O, and towards the end of inspiration it reaches -7.5 cm H_2O, representing the increase of

lung volume by 0.5l. On expiration, the order of events is reversed. Alveolar pressure is the air pressure in the alveoli of the lungs. When not inhaled or exhaled, the value of the pressure in the alveolar air is considered to be equivalent to the value of the pressure in the atmosphere, ie 0cm H_2O. In order for air to enter the lungs, under basal conditions, the alveolar pressure drops to -1cm H_2O, allowing 0.5 l of air to enter the alveoli. Exhalation, in the 2-3 seconds of the process, is achieved by increasing the pressure to + 1cm H_2O and removing a volume of 0.5 l of air from the alveoli. (Daniels and Orgeig, 2003)

Respiratory gas exchange is actually the diffusion of oxygen from the alveoli into the pulmonary blood, after the alveoli have been ventilated with fresh air and is an extremely important step, because in this way the supply of O_2 is ensured and the excess CO_2 is eliminated. Gases are basic molecules that travel easily in comparison to each other, which is called diffusion, if they are absorbed in such liquids by particular gases or gases. Kinetic energy from continuously moving molecules is the source of energy for the creation of the diffusion process. Diffusion happens in various directions: from a higher concentration to a lower concentration, and in the direction of the concentration gradient, or vice versa, from the lower concentration to the higher concentration gradient.

The air contains about 79% Nitrogen and 21% O_2, so the diffusion rate of each of these gases is directly proportional to the pressure determined by each gas, being called the partial pressure of that gas. This total air pressure, of 760 mmHg, encountered at sea level, is in fact the sum of the partial pressure of Nitrogen: 600 mmHg and O_2: 160 mmHg. (Feldman, Mitchell and Nattie, 2003) The partial pressure of each gas in the air, forces the solubility of that gas in the blood of the alveolar capillaries. Thus, the diffusion of gases through the respiratory membrane is represented by the unit of the respiratory system, consisting of: respiratory bronchiole, alveolar ducts, alveolar sacs and pulmonary alveoli.

The respiratory membrane has a surfactant on the surface, a substance responsible for reducing the surface tension of alveolar fluid. (Haverty, Kenney and Hodgson, 1988) It also comprises of the alveolar epithelium, the epithelium basement membrane, the interstitial gap between the alveolar epithelium and the capillary

membrane, the epithelium basement membrane, which merges from place to place, and the capillary endothelium membrane. The average diameter of the pulmonary capillaries is about 5 microns, and because the size of the erythrocytes is larger, about 7.5 microns, when it is crossed, the alveolo-capillary membrane is deformed. The ability of the respiratory membrane to ensure gas exchange between the alveoli and the pulmonary blood is expressed quantitatively in the form of diffusion capacity. Basically, it refers to the volume of gas that will diffuse through the respiratory membrane every minute, when the partial pressure gradient of the gas is equal to -1cmHG. (Cole, 2004)

Table 1. O_2 and CO_2 difusion through respiratory membrane (Patel and Cooper, 2018)

O_2 diffusion capacity under basal conditions	**20 ml/min/mmHg or 230ml O_2 difused each minute through respiratory membrane**
O_2 diffusion capacity under stress conditions	65ml/min/mmHg (3 times bigger than during rest) or 748 ml O_2 difused each minute through respiratory membrane
CO_2 diffusion capacity under basal conditions	400-500 ml/min/mmHg
CO_2 diffusion capacity under stress conditions	1200-1300 ml/min/mmHg

The increase of the diffusion capacity is facilitated by the following processes:

- opening a large number of capillaries
- Overexpression of open capillaries increases the surface area
- Optimizing the relationship between alveolar ventilation and capillary perfusion
- Increased respiratory membrane capacity (Hochachka et al., 2002)

Regarding the oxygen and carbon dioxide transport, it is known that oxygen joins the network after diffusing into the pulmonary blood and must be transferred to all tissues of the body. There are two sources of oxygen circulating in the blood: bound to hemoglobin, thus forming oxyhemoglobin in procent of 97%, or physically dissolved in plasma, the 3% remanining. The factors that influence oxygen binding are:

- pO_2 (O_2 partial pressure)
- pCO_2 (CO_2 partial pressure)
- pH
- 2-3 diphosphoglycerol (2-3 DPG) (Howard, 2001)

The Oxy-Hb dissociation curve is sigmoid. Although the affinity for oxygen is initially low, there are changes in the structure of Hb that are transmitted successively, so the affinity for the last molecule of O_2 increases 300 times.

Table 2. pO_2 and pH modifications: Bohr effect (Patel and Cooper, 2018)

pO_2 increases	**O_2-Hb binding increases**
pO_2 decreases	favors the binding of O_2 to Hb
pH increases	binding O_2 - Hb increass
pH decreases	favors the release of O_2 from Hb

The dissociation curve of OxiHb reveals the progressive increase of the percentage of OxiHb in parallel with the increase of blood p O_2, representing in fact, the degree of saturation of Hb. In the blood that leaves the lungs and enters the systemic arteries, the pO_2 value is approximately 95mmHg, from the dissociation curve it results that the degree of saturation of the systemic arterial blood is on average 97%; in contrast, in normal venous blood, pO_2 has a value of about 40 mmHg and Hb saturation is 75%.

The maximum amount of O_2 that can be combined with Hb is easily calculated because for every 100ml of blood there are 15 g Hb and every 1g of Hb can carry a

maximum of 1.34 ml O_2. Therefore, by multiplying the 15g of Hb by 1.34 ml of O_2, 20.1 ml of O_2 is obtained; which means that the 15 g of Hb from the 100 ml of blood, carries on average 20 l of O_2. Typically, the saturation of the blood with O_2 is 97%, according to calculations it is reduced from 20ml O_2 to 19.4 ml O_2 for every 100ml of blood. (Jordan, 2001).

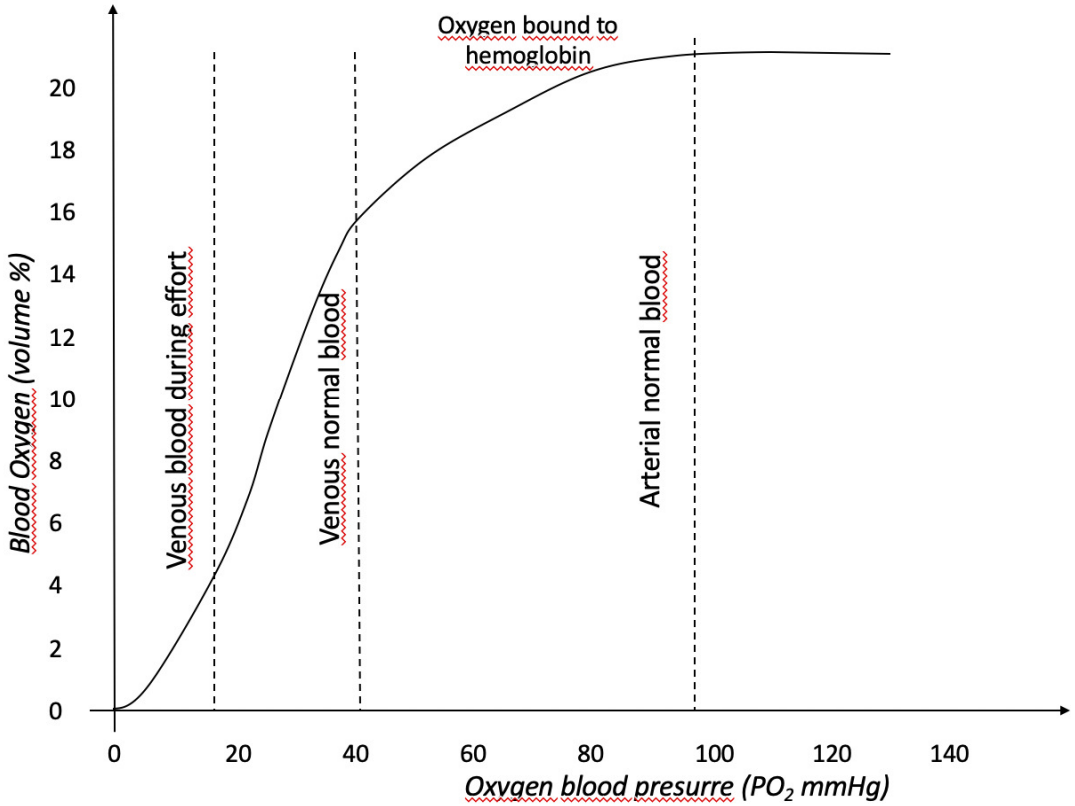

Figure 1. The O_2 relathionship with Hb (West, 2004)

Under exertion conditions, muscle cells have an increased rate of O_2 utilization, and with particularly intense and long-lasting exertion, this will cause pO_2 to decrease in intestinal fluid, from 40 mmHg to values of only 15mmHg. Thus, under such conditions, only an amount of 4.4 ml of O_2 remains bound to Hb for every 100 ml of blood. However, under exertion, cardiac output increases up to 6-7 times, resulting in an increase in the amount of O_2 transported in each volume of blood, about 3 times. This results in a 20-fold increase in tissue O_2 transport during exertion. (Lai-Fook, 2004).

Table 3. Physiological parameters modifications (West, 2004)

	Basal conditions	**Stress conditions**
Coefficient of O_2 use	25%	75-85%
15g Hb	maximum 20 ml of O_2/100ml blood	Grows 20 times
or		
1g Hb	1,34 ml O_2	
Oxygen pressure	40 mmHg	15 mmHg
Cardiac input	5-6 l/minute	Grows 6-7 times

Physically dissolved oxygen in plasma water, at the normal value of arterial pCO_2 of 95 mmHg, 0.29 ml of oxygen is dissolved in 100 ml of blood. When the pO_2 in the blood decreases to the normal value of 40 mmHg in the tissue capillaries, the amount of dissolved O_2 decreases to 0.12 ml. Thus, an amount of 0.17 ml of O_2 is normally transported in tissues dissolved in a volume of 100 ml of arterial blood. During intense exercise, when hemoglobin releases 3 times more O_2 into tissues, the relative amount of oxygen transported in dissolved form decreases to 1.5% of the total amount of oxygen. (Lee, 2000)

The metabolic oxygen consumption at the cellular level is a complex process. If O_2's partial pressure is minimal, so intracellular biochemical reactions typically take place since the cellular enzyme systems are designed in such a way that when pO_2 reaches 1 mmHg, the supply of oxygen is no longer a limiting factor. Thus, the main limiting factor becomes adenosine diphosphate (ADP). As long as intracellular pO_2 remains greater than 1mmHg, control over the rate of oxygen consumption rests with ADP. (Mason, Greene and Voelker, 1998) When cells produce energy, they convert adenosine triphosphate (ATP) to ADP. Thus, increasing the concentration of ADP causes an increase in metabolic oxygen consumption. In turn, oxygen combines with nutrients at the cellular level and thus provides energy for the conversion of ADP to

ATP. Physiologically, the rate of conversion of ATP to ADP controls the rate of cellular oxygen consumption. (McConnell and Romer, 2004) The following energy sources are required to perform respiratory labor:3-5% of total body energy in basal conditions. During exertion it increases 50 times, and the only limiting factor of exertion is the inability to supply energy for respiratory labor. (Miller, 2004)

3.3. Relathionship with cardiac output

During intense exercise, pulmonary blood flow increases 4-7 times. The lungs take over this extra flow in 3 ways:

- By increasing and sometimes even tripling the number of functional capillaries
- By dilating all capillaries and increasing blood flow at least twice through capillaries
- By increasing the pressure in pulmonary arteries. (Richardson, 2003)

3.4. The effects of low oxygen pressure on the body

Atmospheric pressure measures different values depending on altitude. Due to these pressure drops, causes can occur in varying degrees of hypoxia, because as the atmospheric pressure decreases, the partial pressure of oxygen also decreases proportionally and it remains 21% lower than atmospheric pressure. Thus, PO2 at sea level is 159 mmHg, while at an altitude of 15000 m it is only 18mmHg. Carbon dioxide and water vapor lower alveolar oxygen because, regardless of altitude, the ozone is permanently excreted from the lung blood, and the water on the respiratory surfaces often evaporates and thereby lowers the concentration of O2. As long as the body temperature remains at normal temperatures, irrespective of altitude, the water vapor pressure in the wells remains 47mmHg.

Table 4. Physiological parameters at altitude (West, 2004)

Altitude	**0 (m)**	**3000 (m)**
Atmospheric pressure (mmHg)	760	523
PO_2 from inspired air (mmHg)	159	110
PCO_2 from alveoli (mmHg)	40	36
PO_2 from alveoli (mmHg)	104	67
Saturație O_2 (%)	97	90

Starting with the altitude of 1500m, the O_2 pressure decreases and as well, the pulmonary ventilation increases, so it results that the pressure of carbon dioxide in the arterial blood will decrease, leading to the hyperventilation process. Thus, when more air reaches the alveoli, the better oxygenated will be the blood. In response to tissue hypoxia, the kidneys secrete a hormone called erythropoietin, which stimulatesc the production of red blood cells and Hb by the bone narrow. (Tsai, Johnson and Intaglietta, 2003)

Table 5. Hb concentration at altitude (West, 2004)

Altitude	**pO_2 (mmHg)**	**SO_2 (%)**	**C_{Hb} (g/100 ml)**
2500m	74	94	19.8
Sea level	100	97	15

3.5. Altitude syndrome

Oxygen deficiency can lead to hypoxia and this can have immediate consequences to an unacclimatized athlete.The main symptoms are described in the image below:

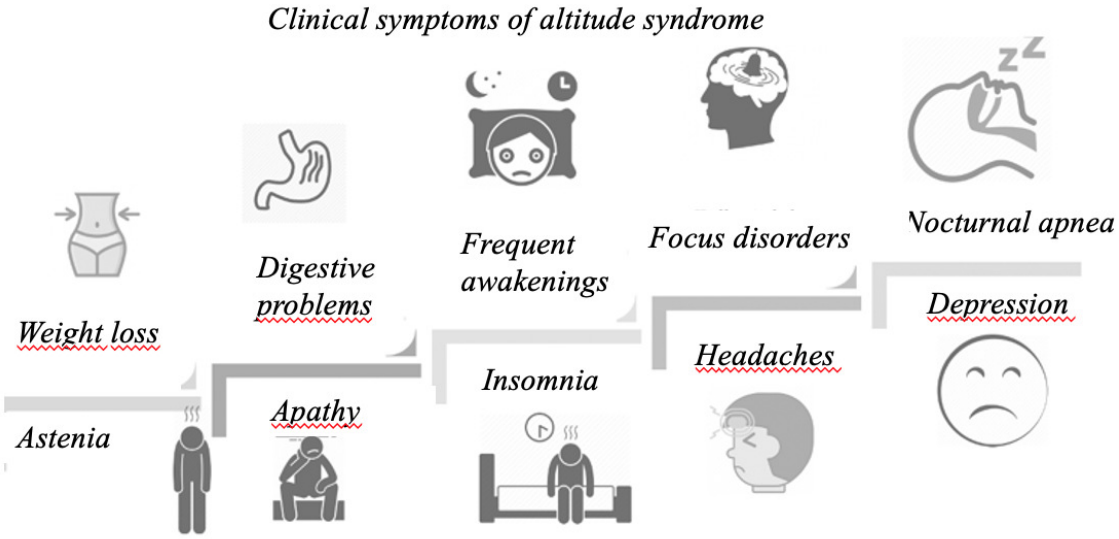

Figure 2. Clinical symptoms of altitude syndrome

Under high stress conditions, the possibility of unintended exposure to altitude contributes to reduced oxygen saturation that induces hypoxia and, in turn, hypoxia may cause respiratory and metabolic acidosis. The pathological consequences consist in the installation of respiratory acidosis and the fact that the diffusion in the capillary alveolar membrane will be slightly altered.

Hypoxemia is classified into 3 degrees, depending on its intensity:

- Light pO_2 = 95-60 mmHg
- Moderate pO_2 = 60-45 mmHg
- Severe pO_2 = 60-45 mmHg

Metabolic acidosis corresponds to disorders of the metabolosm due to the accumulation of acids (H +) and excessive loss of bases (H^-). A well-known example is lactic acidosis due to intense muscular effort. Among the effects of metabolic acidosis, the pH is visibly altered to become acidic and lactic acid buildup occurs. (Patel and Cooper, 2018)

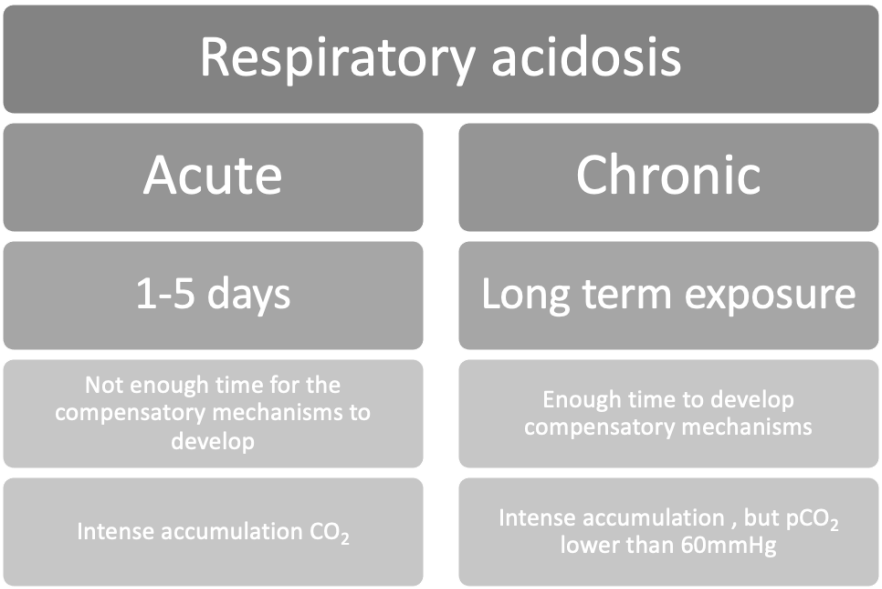

Figure 3. Respiratory acidosis

3.6. Adaptation to altitude

The production of red blood cells is directly determined by hypoxia. After several weeks of exposure to hypoxia, the hematocrit slowly rises from a normal value of 40-45% to an average value of 60%. Also, Hb increases from 15 g/dl of blood to values reaching 20 g/dl. Also, blood volume increases about 20-30%, and if we multiply this increase by the increased concentration of blood Hb, we will actually have a total increase in Hb with values of more than 50%. Diffusion of O2 through the lung membrane has a value of 21ml/mm/Hg in resting conditions, and during physical effort it increases 3 times. Also, this diffusion capacity increases even at altitude, being caused by the increase in lung air volume that increases the alveolo-capillary exchange surface.

Also, the increase in pulmonary arterial pressure causes the forced entry of blood into as many pulmonary capillaries as possible, especially in the upper lung areas. Cardiac output increases by up to 30% immediately after ascent to high altitudes, but after a period of several weeks it returns to normal rates with the increase in blood hematocrit. The increase in the number of capillaries in the systemic circulation from non-pulmonary tissues is another form of adaptation to altitude, being a process called angiogenesis. (West, 2004)

The best and simplest ways for recovery are simple. The first measure to be taken as soon as possible is to change the environment and return to a normal altitude. Specialized medical monitoring is recommended to track vital functions and exercise parameters. Biochemical blood analysis is essential at this stage and is repetitive until optimal values are reached. O_2 therapy is a quick fix to correct possible hypoxia.

4. Conclusions

For performance athletes, it is absolutely necessary that the technical team contains medical specialists who are aware of the latest drugs and basic principles of sports nutrition.

The specific sport must be avoided, with the practice of other complementary, recreational sports, to reduce the mental stress on the athlete. The use of medical techniques at least twice in a training macrocycle for the correct monitoring of physiological parameters.

Strict monitoring, control and optimization of sports performance will be done through invasive and non invasive techniques.

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