

## Aetiology and Pathogenesis of Acute High Altitude Sickness

Analysis from the new scientific perspective of the Lung as a mechanically active autonomic organ, under Vagus Sympathetic complementary command.

### Abstract

***Low air mass per volume unit at high altitude is the cause of acute mountain sickness.***

The *autonomic cyclic mechanics of the Lung* start by contraction of the muscles of the lobar bronchi and those of the lobular bronchioles, which while *decreasing their capacities, proportionally increase the molecular mass per volume unit of the inspired air*, thus increasing their molecular expansive forces.

These dynamics lead to displacement of the contained air in the sense towards lower resistance, to then expand the pulmonary structures in balanced forces, once muscular relaxation begins. Finally, the tiny intra-alveolar air masses *distend the alveolar wall in balance with capillary blood pressure*.

These processes are accomplished by two kinds of dynamic cycles

1. Lobar cycles, for air renovation, under Vagus Nerve command.
2. Lobular cycles, for gas exchange with the blood, under Sympathetic Nerve command.

If the atmospheric *mass of air per volume unit* were lower to that demanded by the adaptation limits of the human specie, its molecular expansive force would be insufficient to achieve pulmonary dynamic expansion, with all that implies, as described above.

Therefore, *the expansive molecular forces* of the alveolar air mass would be insufficient to expand the alveoli with the required tension to enable the right balance of blood and air pressure for balance for selective diffusion of Oxygen. The consequence would be pulmonary arterial blood hypertension, hypoxemia and pulmonary oedema, as is the case in Acute Mountain Sickness.

Simultaneous data and graphs of Resultants of these dynamics, discovered by the Author in 1978, in the pleural space of experimental dogs, are analyzed. Related Author's published works are: "Fisiodinámica del Hombre en el Mundo" "The New Theory of Respiratory Dynamics" "Integración Dinámico Funcional del Organismo Viviente". The Aetiology and Pathogenesis of Acute Mountain Sickness

## Purpose

This work, carried out during the year 2000, obeys to my aim of applying the scientific basis of the Pulmonary autonomic mechanics, developed by me during more than twenty two years, to understand the Acute Mechanical failure of the Lung dynamics at High Altitude, known as Acute High Altitude Sickness, with the hope of contributing with its prevention and effective speedy treatment avoiding complications.

## Introductory Remarks

Rigorous interpretation of this Discovery, which represents a historical landmark, led me to define two different kinds of cyclic activity: One that comprises the whole cycle, coincident with the breathing cycles and the other, a series of minor cycles coincident with cardiac cycles, both clinically determined.

This complex *Resultant* is generated by *cyclic variation of forces transmitted to the pulmonary surface*, which origin and development merit to be analysed.

Hence, applying the Newton's *Inertial Principle*  $F = m \cdot a$  (force equals mass per acceleration) it is compulsory to determine the source of the forces, the identity of the mass, and the space limiting its acceleration, as well as the causes of the periods of those cyclic displacements.

## The Force

The main working forces concurrent to the performance of the Respiratory Cycles have two complementary sources, leading to complementary objectives:

### Sources of the forces

1. Universal Dynamics, mediated by the Atmosphere of the Earth.
2. The living Organism.

We can identify, by abstraction, the origin of some partial forces and their objectives.

### 1. Atmospheric force's origin, relative to altitude

Altitude means distance from sea level, in essence, distance from the centre of the Earth, from which its gravitational force is exerted. This force, applied on the Atmosphere, determines its length, relative to the weight of its column of air, the value of which at sea level is 76 cm Hg.

In other words: the *mass* of a column of atmospheric air at sea level is 76 cm Hg and, from it, approximately 21% corresponds to Oxygen, with a partial pressure of about 15,96 cm Hg. And 79% corresponds to Nitrogen, with a partial pressure of 60,4 cm Hg.

Now, this pressure corresponds to the *lower level of human adaptation* in our natural habitat. If the pressure is increased for any cause, it will be increased the Resultant force needed for the diffusion of gases, firstly Oxygen, surpassing the capacity of fixation by the haemoglobin of the blood and the dissolved Oxygen will increase.

If distension of the alveolar structure increases farther on, Nitrogen would also diffuse, with the danger of gaseous emboli.

The mass of atmospheric air and with its proportional masses of Oxygen and Nitrogen decrease during ascent from sea level, because the air column at each upper level diminishes. One must add to this, temperature and humidity, among other factors, with the consequences above mentioned. These elemental concepts need no discussion.

### 2. Organic forces Origin

1. Contraction-relaxation of the airways smooth muscles.

2. Expansive molecular force of the gasses pressurised in each sector of the bronchial and bronchiolar trees.

3. Capillary blood heat transfer to the alveolar air mass.

4. Reflex contraction-relaxation of the *Diaphragmatic Belt* of striated muscles.

5. Contraction-relaxation of the right ventricle.

The pulmonary bronchial and bronchiolar structures are well known, as also are their muscular fibres distribution as *geodesic networks and*

*sphincters*, whose contraction generates the necessary energy to decrease the diameters and length of those passages, *with three main consequent effects*.

1. Proportional reduction in their capacities.

2. Simultaneous proportional widening of the pleural lumen.

3. Momentarily closure of the airways, by segments.

### Physiological objectives of the *lobar bronchi* muscular contraction

1. *To increase pressure in the lobar bronchi contained air*, in accordance with the Boyle Mariotte Principle  $V.P = C$  (Volume per Pressure is a Constant.). *This pressure increase means accumulation of potential energy*, as expansive molecular forces.

2. *To generate Pulmo-diaphragmatic (Vago-Phrenic) mechanical reflex* to incorporate the

working effects of the Diaphragmatic Belt. That is to say, to evoke Diaphragm contraction, followed by automatic somatic-somatic reflexes (Diaphragm-intercostals and abdominal muscles<sup>2</sup>).

3. *To increase the pleural lumen*, in order to decrease resistance to pulmonary gases expansion and arterial blood circulation

### Physiological objectives of the *lobar bronchi* muscular relaxation

1. *Displacement of the previously pressurised gases* towards their destination on the pulmonary periphery, by own molecular expansion, *expanding the Lung*, to accomplish immediate and mediate ends.

*Decrease resistance for Intra pulmonary gas expansion*, with

2. *abdominal press effect towards the low*

*abdomen*.

3. *Abdominal muscles contraction (Diaphragmatic Belt)*, with *press effect towards the thorax*.

4. *Intercostals muscles contraction for thorax expansion (Diaphragmatic Belt)*, with *flood-gate effect*, decreasing resistance to *abdominal blood stream towards the Thorax*.

### The mass.

The intrapulmonary working *factor mass* is the *mass of air contained in each sector of the air passages*, bronchial and bronchiolar, directly renovated from the right and left main bronchi and

the Trachea, where the *air formerly inspired* is pre-acclimatised, that is to say, the *mass per volume unit* of the atmospheric air is pre-adapted, along the extra pulmonary airways, to physical conditions

physiologically demanded, between limits genetically programmed for use and final adaptation in the intra-pulmonary airways, while also accomplishes simultaneous successive and integrated physiological dynamic effects, for a final pulmonary result at the alveolar level, enabling eupnoea.

Acceleration =  $e/t$  (space divided by time)

The *space* is anatomically defined by the length of one file of bronchi and bronchioles up to their alveoli, while the *time* is physiologically determined by the period of the concerned cycles, which change in relation with organic demands, under control of the Vagus and Sympathetic nerves.

### Physiological Objectives of the *Lobular Bronchiole's* Muscles contraction

Dynamics of the lobular bronchioles is similar, in general sense, to the lobar bronchi, with refer-

ences relative to the magnitude of factors as well as local effects and objectives.

## Acute High Altitude Sickness

### Introduction

To understand the Aetiology and pathogenesis of AHAS it is necessary to know the normal dynamics of the Lung in the habitat of natural adaptation of the species, as synthesised in the introductory Remarks.

Living species and individuals are mechanical structures genetically designed for dynamical integration with the Atmosphere, at the very moment of birth, and adaptation to different levels of altitude, *within their own limits*, relative to the mass of air per volume unit.

It is well known that the respiratory function of healthy individuals, in *their local habitat*, disposes of a wide *adaptation range* to organic demands, relative to *physical activity*.

A wider concept of Habitat is *Natural Habitat*

*of Species and Individuals Adaptation*, which is genetically determined and infers the concept of Altitude above sea level, this relative to the distance from the centre of the Earth, as determinant of its *Gravitation Force* and, with it, atmospheric pressure at different geographical levels, which is relative to the *mass of air per volume unit*.

### Aetiology

We are now starting focussing the Aetiology of Acute High Altitude Sickness, which is relative to the functional binomial Man-Earth of which the factor Man is the same and, only one element of the factor Earth has changed: the distance from sea level and its consequent effect on the mass per volume unit of atmospheric air, *which I pointed out as the Cause of the sickness*.

Low air mass per volume unit above the upper limit of human natural adaptation is the cause of the acute failure of the Lung dynamics known as Acute High Altitude Sickness

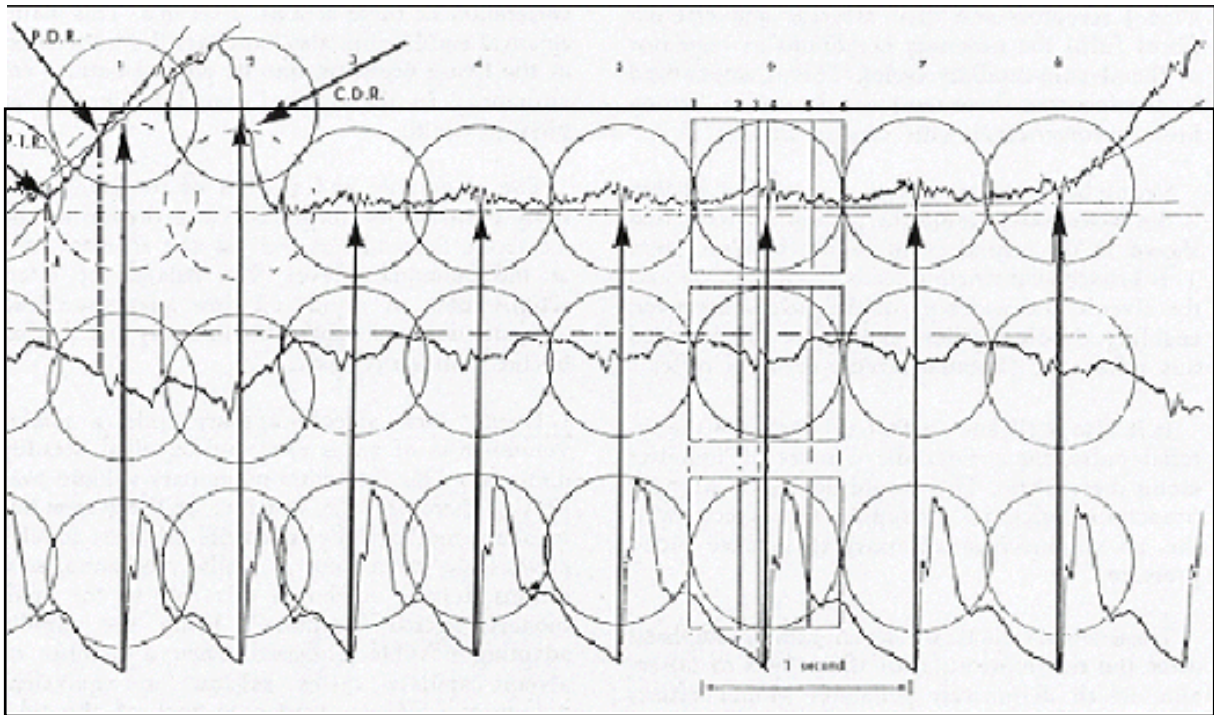


Fig 1. Simultaneous graphs of three main parameters from top to bottoms: 1. Respiratory Pulse: 2. Intra-pleural sub-atmospheric pressure abdominal aortic pulses. The circles defines each lobular alveolar-capillary cycle and the correspondences with the others parameters. The arrows mark the coincident inflexion of capillary and arterial pulses (Graph taken from my book “The New Theory of Respiratory Dynamics”<sup>2</sup>)

## Pathogenesis

Acute High Altitude Sickness is --well known as the sum of signs and symptoms observed in otherwise normal individuals when displaced at high altitude. Therefore, one needs to know how does the *binomial Man-mass of air* at sea level works, for gas exchange with the blood.

The **Physical factor Man** is represented in this moment of my analysis by the joint of the *lobular units* of each and all the lobes in their ensemble, since the lobules receive the masses of air previously adapted along the airways and are the dynamic-functional units for development of the specific cycle leading to gas exchange with the blood, while *the alveoli are surface units for Oxygen diffusion, under effect of the expansive molecular forces of the tiny masses of gases displaced up to them.*

## Mechanical Actions and Effects

1. *Lobular Floodgate* actions and effects in the pleural space.

Retraction of the lobular structure, as a consequence of its bronchioles muscular contraction, widens the pleural lumen to decrease resistance to pulmonary arteriolar blood circulation towards the alveolar capillaries. Floodgate action and effect.

2. *Lobular Press* action by molecular expansion of the air mass displaced up to the alveoli, distend the alveolar-capillary membrane, leading to:

2.1 Oxygen selective diffusion

2.2 Press effect to displace the oxygenated blood towards the pulmonary veins.

Therefore, decrease in alveolar membrane distension generates two well-known signs characterising the beginning of this sickness.

1. *Hypoxemia* because insufficient distension of

the alveolar membrane, distorting Oxygen diffusion

2. *Pulmonary arterial hypertension*, because low alveolar-capillary press action, causing partial retention of pulmonary arteriolar circulation.

## Analytical Review of some clinical Data and Research

On the basis of the above general explanation, I consider it useful to discuss now important clinical and anatomopathologic observations carried out by different authors under various conditions and/or stages of this sickness, but whose interpretations, based on the traditional theory that considers the Lung a passive organ, has been unable to define neither the cause nor the pathogenesis not even the precise rational prevention and treatment, all of which constitutes the purpose of this work.

Hurtado first demonstrated that pulmonary arterial hypertension in humans; this is a sign in patients with this sickness<sup>6</sup>.

Hurtado et al observe that Oxygen partially prevents the rise in pulmonary arterial pressure in exercise.

Arias Stella et al<sup>7</sup>, found that excessive muscularity of distal muscular arteries and arterioles is a prominent feature of the pulmonary arterial bed of native highlanders.

Pulmonary hypertension and hypoxia are the two most characteristic signs of the Acute High Altitude Sickness because they are the two immediate consequences of the mechanical unbalance generated at the alveolar-capillary level, by means of the two mechanical effects described above:

1. *Increased Floodgate action* by which augments the pulmonary artery blood towards the alveolar-capillary units.

2. *Decreased Press action* because low expansive molecular force exerted by the alveolar air mass, which prevents its two consequences:

2.1 Oxygen diffusion, causing hypoxia and displacement of the Oxygenated blood causing retrograde retention of the pulmonary arterial blood, with hypertension.

The observation that acute administration of Oxygen caused little or no fall in pulmonary vascular resistance at rest and that Oxygen partially prevents the rise in pulmonary arterial pressure in exercise<sup>6</sup> are also consistent with our mechanical interpretation since the problem is not lack of Oxygen or hypoxia but lack of enough total air mass. If acute administration of Oxygen prevents rise in pulmonary arterial pressure is because the patient was partially-adapted to that altitude and the additional Oxygen supply the extra-demand because exercise, as in any patient suffering from respiratory insufficiency.

The excessive muscularity of the named arterial vessels is an expected anatomic-pathological consequence, because excessive repeated muscular contraction to overcome the functional obstruction due to lack of alveolar capillary press action and effect.

Peñaloza, Arias Stella et al<sup>8</sup>, observe that pulmonary arterial pressure during childhood is higher at altitude than at sea level.

This observation obeys to the same mechanical cause as exposed above.

Grover et al<sup>9</sup> and Sime et al<sup>1</sup> state that “prolonged removal of the hypoxic stimulus by descent to sea level result in the fall of pulmonary arterial pressure and pulmonary vascular resistance to normal values for lowlanders.

The observations are correct, but the interpretation is wrong. The correct interpretation must be: descent to sea level result in the fall of pulmonary arterial hypertension and pulmonary resistance to the arterial flow because the lung dynamics has recuperated after restitution of a mass of air per volume unit between the limits of natural adaptation.

Reaves and Grover<sup>11</sup> observe that in newborns breathing ambient air, the fall in pulmonary arterial pressure during the first three days of life is less marked than at sea level and Peñalosa et al<sup>12</sup>. Arias-Stella et al<sup>13</sup> observe that throughout childhood pulmonary arterial pressure is higher at altitude than at sea level.

Important observations. Their interpretations are similar to those explained above.

Kronenberg et al<sup>14</sup>, observe that arterial pressure in individuals coming from low lands increased after a few minutes of exposure to altitudes, reaching a plateau after 12 to 24 hours, being reversible by Oxygen administration.

This observations are of theoretical and practical importance since they refers to a relative period in which the strength for adaptation is near its limit and Oxygen administration supply a little amount of the factor mass.

Hurtado and others<sup>15-16</sup> found that systolic peripheral blood pressure was lower in practically all subjects studied.

This very important observation is compatible with re-distribution of the blood because of failure of mechanical factors developed by the thoracic-abdominal muscular and costal belt, as described by me elsewhere.

In synthesis, all these observations, as discussed here, are coherent with expectations derived from my discoveries and their interpretations of pulmonary mechanics, pulmo-cardiac dynamic integration, as well as with the integration and balance of our organisms with the Atmosphere of the Earth.

This pathology is generally interpreted as caused by hypoxia, which is consequent with the prevailing traditional theory of Respiration that considers the lung a mechanically passive organ and, Oxygen, as the gas useful for respiration, since this is the gas necessary for organic metabolism.

## Discussion.

The Living being, is a *complex integrated structural and functional organization*, for maintenance of created life in its natural habitat of adaptation, mainly relative to altitude.

To achieve this purpose disposes of necessary parameters for balance of actions, reactions and functional integrations, in the dynamic balance characterising Life and Health, made objective as eupnoea<sup>3</sup>

Let us now to analyse the posed problem, from the new scientific perspective of the Lung as a mechanically autonomic active organ working under Vagus Sympathetic complementary command.

I discovered the autonomic. *Resultant of cyclic pressure variations of the pulmonary mechanics*, in its pleural surface (Respiratory Pulse) then, I analysed and interpreted its two main cyclic components:

One that embraces the whole cycle knew as "Respiratory", which is only relative to air renovation from the Atmosphere, being generated by lobar bronchial mechanics under Vagus control.

The other, riding on the first: the *lobular-alveolar-capillary* pulses or respiratory pulses strictus sense (pulses for gas exchange with the blood), which are synchronous with the cardiac pulses<sup>2</sup>.

Forces for air *impulsion and auto-impulsion* into the alveoli must balance the alveolar-capillary blood pressure. The *Sympathetic System* coordinates this complex physiological integration.

I have fully described elsewhere the functional

*alveolar-capillary units*, which represent the *pulmo-cardiac functional integration* as a necessary condition. Consequently, obeying Sympathetic Adrenergic control, on a vagal dynamic basis.

### Let us now to discuss the following:

What does the concept *Altitude* mean, from the scientific physic-mathematical analysis and, how does this affect the physiology of living beings, humans among them?

Altitude means distance from sea level, in essence, distance from the centre of the Earth, from which its gravitational force is exerted. This force, applied on the Atmosphere, determines its length, relative to the weight of its column of air, the value of which at sea level is 76 cm Hg.

In other words: the mass of a column of atmospheric air at sea level is 76 cm Hg and, from it, approximately 21% corresponds to Oxygen, with a partial pressure of about 15,96 cm Hg. And 79% corresponds to Nitrogen, with a partial pressure of 60,4 cm Hg.

Now, this pressure corresponds to the *lower level of adaptation* in our natural habitat. If the pressure increases for any cause, it will increase the Resultant force needed for the diffusion of gases, firstly Oxygen, surpassing the capacity of fixation by the haemoglobin of the blood and the dissolved Oxygen will increase.

If distension of the alveolar structure increases farther on, Nitrogen would also diffuse, with the danger of gaseous emboli.

On the contrary, the mass of total air and the proportional masses of Oxygen and Nitrogen decrease during ascent from sea level, since the air column diminishes. One must add to this, temperature and humidity, among other factors, with the consequences above mentioned. These elemental concepts need no discussion.

### Matter of discussion

The Living being, as a *complex integrated structural and functional organization*, for maintenance of created life in its natural habitat of adaptation, disposes of necessary parameters for balance of actions, reactions and functional integrations, in the dynamic balance characterising Life and Health, objectified as eupnoea<sup>3</sup>.

We can pose the following questions.

1. How does Oxygen arrive or it is carried up to the alveoli and, in which physical conditions, all of which subject to possible mathematical interpretation?
2. What is the reason for those physical and physiological conditions?

In the light of my discoveries, analysis and interpretations, the prevailing theory of respiratory mechanics does not give and can not give a single rigorous answer since cyclic transportation of air masses obey pulmonary dynamic phenomena on one side and physiological demands on the other.

I have fully demonstrated, for over twenty-two years, not only that the *Lung is an autonomous mechanically active organ*, but also I have interpreted this activity on a Physic mathematical basis, for physiological application.

Atmospheric Air Transportation throughout the air passages.

Any **Volume** of inspired air, at any altitude has a correlative **mass**. **Volume-mass** that must be displaced throughout the airways in conditions of **pressure, humidity and temperature** physiologically determined, during a time relative to the integrated simultaneous rhythms, generated by their autonomous innervations, which also determine the contracting force of the airways muscular fibres, generating proportional variations in their respective capacities and also simultaneous closure, due to sphincters action (sphincterlike of Miller).

This mechanics is the essence for air transportation, since according to Newton's Inertial Law, I repeat, any displaced mass is a result of a force applied on it, which exerts an acceleration expressed by the equation  $F = m \cdot a$ , from which the equations  $m = F/a$  and  $a = F/m$ . Are deduced.

The **space** for displacement of the air mass does not change, since this is the length of the named airways.

Consequently, the variables are:

1. Volume-mass of air, relative to pulmonary capacity and altitude above sea level.
2. Forces generated by cyclic muscular contraction on the pulmonary structures, to vary their own capacities and, therefore, their contained **air mass per volume unit**, accumulating potential forces to be used during the following phase of muscular relaxation.
3. Rhythms of nerve discharges for integration of the complex cyclic dynamics.

Coordination of these dynamic factors are necessary for simultaneous displacement of the variable masses per volume unit, towards intermediate and final destinations, in required physical (physiological) conditions; to enable selective diffusion of Oxygen in the required amount, to satisfy the demand of load by the haemoglobin of the red cells simultaneously circulating throughout the alveolar capillaries.

The previous paragraphs contain the utmost factors of the problem and pose the need for an explanation about the how's and why's of the variables and their conditions, with physic mathematical perspectives.

The *smooth muscular networks of the airways*, correlative to the local and integrated functional tasks of bronchi and bronchioles, included the fi-

bres disposed as sphincters, on contracting in response to Vagal and Sympathetic nerves discharges, decrease the capacities of their corresponding airways, also closing them momentarily. Consequently, this muscular contraction proportionally pressurise the contained air masses, increasing their mass per volume unit (Boyle-Mariotte Law) also accumulating expansive potential forces to be used during the relaxation period.

When muscular relaxation starts, the expansive force of the gas molecules of the air, previously pressurised, displace themselves in the sense of lower resistance, that is to say towards the destinations programmed in the organic design, immediate and mediate, the alveoli being their final pulmonary destination.

The *mass of inspired air* is simultaneously displaced throughout the different sectors, bronchi, bronchioles, during simultaneous and successive steps.

I have defined four periodical sectors: <sup>4</sup>.

1. Nasopharynx.
2. Larynx-trachea-bronchial.
3. Lobar bronchi.
4. Lobular bronchioles and alveoli.

I shall avoid here the two first sectors, explained apart, and will only refer to the third and fourth, which are closely related to specific pulmonary structures and constitute the first dynamic step of the posed problem.

**Inspiration:** air renovation throughout the airways.

*Relaxation of the lobar bronchial muscles generate a sucking force against the air masses supplied by the right and left major bronchi.* Those masses of air shall fill the capacities left empty by the former displacement of the air masses retained for acclimatisation during the former cycle. This process is simultaneously followed by displacing the air now

filling the upper airways and finally the air from the Atmosphere. This latter is the only objective act defining Inspiration in the traditional sense<sup>5</sup>.

*The next muscular contraction*, due to the Vagus nerve discharge will decrease the bronchial capacity thus pressurising the intra-bronchial mass of air. This fact is followed by muscles relaxation, opening ways for the air mass displacement towards the pulmonary periphery, in order to supply the quote of air demanded by each and all the lobules.

*Simultaneously, the same muscular contraction, when retracts pulls from the parietal pleuras*, widening the pleural lumen, creating potential space for pulmonary gases expansion with to effects flood gate and press action to balance fluids circulation.

*The lobular units repeat the lobar process, but under Sympathetic nerve discharges*, with their own intensity and rhythm, thus, widens the pleural lumen to open ways to arteriolar blood circulation-floodgate action- and simultaneous pulmonary expansion. The lobular bronchiole's muscular relaxation sucks small masses of air supplied by the next lobar bronchi, then pressurises them by muscular contraction. This process leads to circulation towards the capillaries and the air displacement towards the alveoli.

Simultaneously, the lobular structure retracts to open ways for arteriolar blood circulation towards the capillaries. The molecules of gases shall expand, distending the alveolar capillaries membrane in balance with blood pressure, thus enabling selective diffusion of Oxygen in the amount programmed in the structural design, under Central Nervous System control. This expansion also works as a press to displace the oxygenated blood towards the pulmonary veins.

It is obvious that if the mass per volume unit of the inspired air is inferior to that required by the dynamics in progress, the effects produced by the actions and reactions derived from the muscular increased contracting force, will become proportionally increased and, even with the maximal effort of compensation facing the final balance, Oxygen diffusion will not suffice, and the anoxic effect will occur. Besides, parallel *mechanical effects generating signs and symptoms characterising Mountain Sickness* will also be produced, these latter mechanical effects generally ignored until now.

*It is clear that the anoxia in the tissues is a consequence of the primary mechanical problem generated by insufficient mass of inspired air.*

I must emphasise the fact that the posed general problem is Insufficient air "mass", in relation to that required by the pulmonary functional structure, which is designed to generate and develop the mechanical process needed to achieve the functions enabling Life and Health in the natural environment of adaptation of the species and individuals.

The air mass per volume unit supplied at higher altitudes to that of the species adaptation limits results insufficient to distend the pulmonary structures and further along, the alveolar-capillary membranes, generating organic dynamic unbalance.

The functional insufficiency becomes evident once the potential capacity of action of the pulmonary structure is exhausted. It is necessary to insist that the maximal nervous discharges enhance maximal muscular contractions, able to decrease the bronchial capacity up to the limit allowed by the structure of cartilages, which guarantee the permanent filling by "residual air" as a condition for homogeneous cyclic air circulation.

Let's now think in another simultaneously important problem. The pulmonary blood vessels are distributed in parallel with the airways and, elastic and collagen fibres surround both kinds of passages, making them co-participant in common mechanical phenomena.

## Prevention.

It is necessary to know the maximal altitude of physiological adaptation of the Human Living Being's vital structures and, to make aware that if that limit is surpassed, individuals must be provided with chambers or tents of total pressurised air, relative to the adaptation level of the group or individual.

Administration of pure Oxygen has been widely used in prevention and even in therapy, as result of the fact that the autonomic pulmonary mechanics and its direct relationship with the gaseous mass of air are still unknown. As a consequence, the results have not often been satisfactory and, when they look like being, it would be interesting to analyse the conditions of the individuals as well as all the conditions implicit in the supplied measures.

## Conclusions

Living Beings in the environment of their natural integration and adaptation, constitute an Organic-Physical Natural-Unit genetically designed to accomplish basic dynamic functions as an Autonomic Balanced Mechanical System of Fluids, for which the inspired atmospheric

mass of air per volume unit, at the human adaptation limit is the physic-natural factor for integration and balance, when satisfying the organic demand for its basic physiological performance, accomplishing the equation of the Inertial principle  $F = m \cdot a$ .

Life in health is the natural consequence of integration and balance of two primary factors:

1. The Organism supposed healthy

2. The Atmosphere of the Earth, between the geographic limits of adaptation of the specie, which supplies the air mass per volume unit that corresponds to the organic genetic design. Acute High Altitude Sickness is the joint of signs and symptoms derived from organic dynamic insufficiency due to a mass of air supply inferior to that required for the organic dynamic balance.

The Living Being is a functional structure designed to maintain the created life in its natural habitat.

The natural habitat of the humans is the surface of the Earth and its Atmosphere, between limits genetically established.

## The Lung is the organ responsible for this dynamic integration.

The pulmonary lobules are the structures to accomplish the final step of the necessary balance, at alveolar capillary level.

The two factors integrate dynamically at the very moment of berth, direct and primarily by the pulmonary functional structure, in its thoracic ensemble. The factor of integration and balance of the pulmonary dynamics and Universal dynamics is the air mass per volume unit, which is relative to the altitude also relative to the Earth force of gravity.

The tiny masses of air distributed among the alveoli exert expansive forces to distend the alveolar-capillary membranes, with two objectives.

1. To widen the pores of diffusion to open way to the Oxygen molecule and not to that of Nitrogen.

2. To simultaneously perform press action to displace the oxygenated blood towards the pulmonary veins.

Scarce air mass determines compensatory in-

crease in the bronchiolar muscles contraction up to the potential limit, after which hypoxemia and pulmonary hypertension with alveolar infiltration and oedema starts to manifest.

If the problem persists, the organic dynamics farther than the pulmonary limits, starts to manifest as pulmonary and cerebral oedema.

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