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REVIEW

A journey between high altitude hypoxia and critical patient hypoxia: What can it teach us about compression and the management of critical disease?☆

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KEYWORDS
Hypobaric hypoxia;
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Mitochondria

Abstract High altitude sickness (hypobaric hypoxia) is a form of cellular hypoxia similar to that suffered by critically ill patients.

The study of mountaineers exposed to extreme hypoxia offers the advantage of involving a relatively homogeneous and healthy population compared to those typically found in Intensive Care Units (ICUs), which are heterogeneous and generally less healthy.

Knowledge of altitude physiology and pathology allows us to understanding how hypoxia affects critical patients.

Comparable changes in mitochondrial biogenesis between both groups may reflect similar adaptive responses and suggest therapeutic interventions based on the protection or stimulation of such mitochondrial biogenesis.

Predominance of the homozygous insertion (II) allele of the angiotensin-converting enzyme gene is present in both individuals who perform successful ascensions without oxygen above 8000 m and in critical patients who overcome certain disease conditions.
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PALABRAS CLAVE
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Aclimatación a la altitud;
Enfermo crítico;
Factor inducible por la hipoxia;
Mitocondria

Un viaje entre la hipoxia de la gran altitud y la hipoxia del enfermo crítico: ¿qué puede enseñarnos en la compresión y manejo de las enfermedades críticas?

Resumen La hipoxia de la altitud (hipoxia hipobárica) no deja de ser una hipoxia celular similar a la que presentan los enfermos críticos. Estudiar a los alpinistas expuestos a la hipoxia extrema ofrece la ventaja de que es una población relativamente homogénea y sana, en contraste con la población heterogénea y generalmente menos saludable que suele observarse en las Unidades de Cuidados Críticos. El conocimiento de la fisiología y la enfermedad de la altitud abren caminos

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para comprender en qué medida afecta la hipoxia a los pacientes críticos. Los cambios comparables en la biogénesis mitocondrial entre ambos grupos pueden reflejar respuestas adaptativas similares y sugieren intervenciones terapéuticas basadas en la protección o estimulación de la biogénesis mitocondrial.

El predominio del alelo homocigótico de inserción (II) de la enzima de conversión de la angiotensina está presente tanto en las ascensiones exitosas sin oxígeno por encima de los 8.000 m como en la supervivencia de algunas enfermedades de los enfermos críticos.
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Introduction

Humans have been able to climb mountains to a height of over 8000 meters without the need for supplementary oxygen. The first ''eight thousand'' to be conquered was the Annapurna (8091 m) in the year 1950: this was achieved by breathing atmospheric air, and there had been no plans to use supplementary oxygen. The summit of Mount Everest (8848 m) was reached two years later, though this time with the help of supplementary oxygen, and in 1978 the peak of this highest of all mountains was reached breathing only environmental air. The climbers Reinhold Messner and Peter Habeler proved wrong the physiological theories predicting that humans could not reach the top of this mythical mountain breathing only atmospheric air. These theories, shared by both physiologists and high altitude mountaineers, were fundamented upon the principle that a maximum inspired oxygen partial pressure of 43 mmHg precluded human tolerance of hypoxia.[1]

An expedition led by West, the American Medical Research Expedition to Everest (AMREE), which reached the summit of Mount Everest in 1981 without oxygen, marked a turning point in this physiological challenge.[2] Important respiratory physiologists and leading mountain climbers, with Hackett and Milledge, participated in the expedition.[3] In a recent article, West recalled this investigation into human tolerance of extreme hypoxia.[4] In the year 2007, a British expedition known as the Caudwell Xtreme Everest Expedition (CXEE) further contributed new knowledge in this field.[5]

These studies, which have helped to improve our knowledge of high altitude physiology and disease, have also opened ways to understand how hypoxia affects critically ill patients and in general all individuals exposed to hypoxemia and cellular hypoxia.[6] It is no exaggeration to affirm that the hypoxemia-adapting mechanisms induced by high altitude play a role similar to that of the hypoxia response observed in critical patients, and that knowledge acquired in mountain medicine may be transferred to the critical care setting.

The mechanisms underlying adaptation to hypoxia are still little known, and investigation in this field among critical patients is difficult. Mountaineers exposed to extreme hypoxia offer the advantage of constituting a relatively homogeneous and healthy study population, in contrast to the heterogeneous and generally much less healthy individuals seen in the Intensive Care Unit (ICU). The main research question of the present review is: What can the healthy mountain climber model of hypoxia teach us about critical patient management?

Hypobaric hypoxia and tissue hypoxia

Hypobaric hypoxia (HH) refers to high altitude hypoxia, i.e., a lowering of atmospheric or barometric pressure (BP). The oxygen partial pressure in atmospheric air (PO_2) remains constant above 11,000 m, and the gas is always in a proportion of 21%. At sea level, BP is 760 mmHg and PO_2 is 159.2 mmHg. However, as we rise in altitude, BP decreases, and consequently although oxygen remains present in the same proportion as at sea level, PO_2 also decreases. In other words, increasing altitude is associated to lesser BP, lower PO_2 and, consequently, lower inspired oxygen pressure. Considering that BP is about 405 mmHg at the top of Mont Blanc (4810 m), the PO_2 is 84 mmHg. Likewise, at the summit of Mount Everest, the theoretical BP is 236.3 mmHg, with a PO_2 of 49.5 mmHg (Table 1 and Fig. 1).

The consequence of HH is tissue hypoxia. In turn, high altitude cellular hypoxia is the same as that observed in critical patients when oxygen delivery to the tissues is impaired as a result or cause of well known disease processes.

In most body tissues the primary mechanism producing energy in the form of adenosine triphosphate (ATP) is oxidative phosphorylation, which takes place in the internal membrane of the mitochondrion. Under conditions of hypoxia, energy homeostasis and metabolic adaptation face the challenge of maintaining cell functions and energy.[7]

Many physicians were surprised to discover that mountain climbers at the top of Mont Blanc have a partial oxygen pressure in arterial blood (PaO_2) of about 40 mmHg, versus about 25 mmHg at the summit of Mount Everest, and that they moreover perform very strenuous exercise with these PaO_2 values.[2,8]

Two expeditions have been able to estimate alveolar gas at the summit of Mount Everest, along with the corresponding PaO_2 values (Table 2). The first expedition was the AMREE led by West.[3] In this expedition, the data were estimated from samples of venous blood and expired alveolar gas up to an altitude of 8050 m, while only alveolar gas samples were collected at the summit. The next expedition was the CXEE.[5,8] In contrast to the previous expedition, on this occasion supplementary oxygen was used above 7100 m,

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