CHAPTER 8

THE LUNG AT HIGH ALTITUDE: BETWEEN PHYSIOLOGY AND PATHOLOGY

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Abstract: The lungs play a pivotal role in adaptation to high altitude. The increase in ventilation and the rise in pulmonary artery pressure are the first features of lung response to hypoxic exposure. At high altitude the lungs can also be affected by high-altitude pulmonary oedema, a severe form of acute mountain sickness. In healthy subjects the ascent to high altitude is also associated with alterations in lung function, which have been in part interpreted as an effect of extra vascular lung fluid accumulation. The patterns of respiratory function changes at high altitude are discussed, taking into account the body fluid movement and the increase in endothelial permeability induced by hypoxic exposure. As the problem of “respiratory” patients at high altitude is very important, a short summary of the guidelines for altitude exposure of asthmatic and COPD patients is reported at the end of the chapter.

Keywords: pulmonary artery pressure; high altitude; physiology.

Introduction

The lung is the first interface between environmental hypoxia and the metabolic machinery of the body, so that any factor impairing the ventilation and the lung mechanics can condition the physical performance. This is particularly true at high altitude, where the movement of large quantities of air is required, especially during exercise, and the total body function is impaired by arterial oxygen desaturation [54,55]. At altitude, many factors can affect the ability of the lung to successfully carry out its tasks, despite the facilitation induced by ongoing ventilatory acclimatization, which is secondary to carotid body sensitivity to hypoxia: as for instance, an overall greater effort for breathing,
pulmonary vasoconstriction with increased pulmonary artery pressure, slightly decreased strength of respiratory muscles [17, 25], ventilation/perfusion heterogeneity and oxygen diffusion limitation, essentially tied to the lower driving pressure for oxygen from the air to the blood and the shorter time for equilibration of oxygen at the alveolar-endothelial membrane site, due to the more rapid transit time [63]. Furthermore, at high altitude the lungs can also be affected by one of the two severe forms of acute mountain sickness (AMS), i.e., high altitude pulmonary oedema (HAPE), which is a well-known life-threatening complication of high altitude exposure. This illness usually develops within the first 2–5 days after acute exposure to altitudes above 2500–3000 m, but is more frequent after a rapid ascent (>300 m/day) to an altitude above 4000 m and an overnight stay. Altitude, speed and mode of ascent and above all individual susceptibility, are the most important determinants for the occurrence of HAPE [3]. An excessive rise in pulmonary artery pressure preceding oedema formation is the crucial pathophysiological factor; decreased fluid clearance from the alveoli may also contribute to such non-cardiogenic pulmonary oedema, as well as the stress failure of pulmonary capillaries [62]. In subjects suffering from this severe and acute illness, the respiratory function and oxygenation are severely impaired [28].

Even in healthy subjects, the ascent to high altitude is associated with alterations in lung function, which have been in part interpreted as an effect of extravascular lung fluid accumulation. The mechanisms of these changes and whether they reflect early stages of high-altitude pulmonary oedema (HAPE) have been recently debated. In other words: is the HAPE the end stage of interstitial lung fluid accumulation in the same way as high-altitude cerebral oedema (HACE) is considered the end stage of AMS?

First of all, we think that the extravascular lung fluid accumulation should be seen in the wider “scenario” of the bodies fluid movement during high altitude exposure, which is a quite complex issue.

Body Fluid Movement

It is well known that exposure to high altitude is associated with changes in body fluid compartments. In particular, subjects can face both dehydration and peripheral oedema while spirometric changes, suggestive of extravascular lung fluid accumulation, can appear [59]. Dehydration can be caused frequently by the very low absolute humidity at high and very high altitudes. This is mainly due to the fact that water loss through sweating is increased during exercise, as well as insensible water loss induced by ventilation. Increased ventilation is a common adaptation feature of high