Acute mountain sickness

Acute mountain sickness (AMS) occurs due to hypoxic hypoxia, which occurs when climbing to a high altitude.

Symptoms

In addition to the other symptoms typical of hypoxia, there are also very serious problems in the form of pulmonary edema and cerebral edema.

Pulmonary edema

It arises primarily from people who have been subjected to strenuous physical activities after climbing to a high altitude. According to a valid theory, the swelling is caused by the fact that some pulmonary arteries do not have enough muscle to contract during hypoxia. The pressure then damages the walls of the capillaries, causing the alveoli to be filled with fluid and blood cells.
Symptoms of pulmonary edema are extreme tiredness, inability to breathe, blue or grey lips, possibly nails (cyanosis), gargling or bubbling breathing, coughing, tightness or pressure in the breasts. Sputum, when it is expectorized, is foamy, pinkish, liquid.

Cerebral edema

It occurs as a result of brain arteriol dilation, which occurs as a result of an attempt to supply the brain with less oxygenated blood. This increases the pressure in the capillaries, which then push more fluid into the surrounding tissue.
Swelling of the brain is manifested by impaired thinking, loss of coordination, lethargy, confusion, marked changes in behaviour and staggering gait (i.e. drunkenness-like conditions).

Other symptoms

Symptoms of acute mountain sickness also include vomiting and frequent urination, which can lead to dehydration as the feeling of thirst is lost.
Symptoms diminish when alkalosis is prevented by the administration of acetazolamide or when cerebral edema is reduced by large doses of glucocorticoids. But the basic aid to the afflicted AMS is to **descend to lower altitudes** as soon as possible after symptoms appear.

Acclimatisation

Acclimatisation to high-altitude conditions takes place within **4-5 days** of climbing the mountains and involves the interplay of several compensatory factors.

Ventilation and balance of pH

Immediately after climbing into a high-altitude environment, the body reacts by hyperventilating with up to 5 times more ventilation than normal. This causes some increase in partial oxygen pressure in the arteries, but most importantly a greater elimination of CO_2, which induces respiratory alkalosis. Increased pH of body fluids inhibits respiratory center chemoreceptors in the brainstem, so initially it dampens the stimulant effect of hypoxia and the ventilation response to hypoxia paradoxically decreases. This is counteracted by active transport of H+ to the cerebrospinal fluid (and possibly the development of lactic acidosis in the cerebrospinal fluid), thereby reducing the pH around the chemoreceptors and increasing the ventilation response, which then grows steadily until **day 4** after exit. After that, the hyperventilation decreases (but only in trained highlanders does the ventilation reach its original pre-output levels) as the effect of increased erythrocytes sets in.

Influence od erythropoiesis

Erythropoietin, a hormone that stimulates the production of new erythrocytes, does become secerpted at an increased rate from the first day after output, but decreases somewhat under the influence of hyperventilation. During acclimatisation, there are also changes in the tissues. Respiratory alkalosis shifts the Hb dissociation curve to the left. In contrast, erythrocytes produce 2.3-DPG in hypoxia and this shifts the dissociation curve to the right. The result is a small shift of the curve to the right, reducing the affinity of Hb to O_2, making O_2 easier for tissues to reach. However, this effect is not very large and becomes less relevant when there is an increase in erythrocytes. Furthermore, mitochondria and myoglobin are multiplying.

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