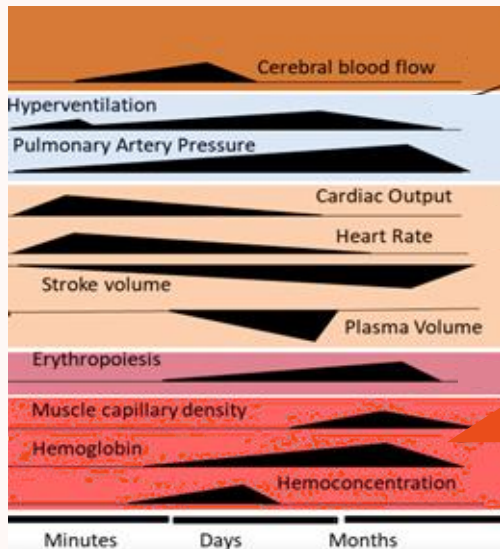


Exploring The Pathophysiology Of High-Altitude Sickness

Background

High-Altitude Illness (HAI) is a collective term for Acute Mountain Sickness (AMS), High-Altitude Cerebral Oedema (HACE) and High-Altitude Pulmonary Oedema (HAPE) due to failure to acclimatise at elevations above 2,500m¹.

The risk of AMS begins at altitudes >2,500m, (at which >140 million live, and 40 million people visit). HAPE typically occurs >3,000m and HACE >4,000m².



Pathophysiology

High-altitude hypobaric hypoxia induces whole body physiological acclimatisation responses which attempt to restore the blood oxygen content as ppO₂ decreases².

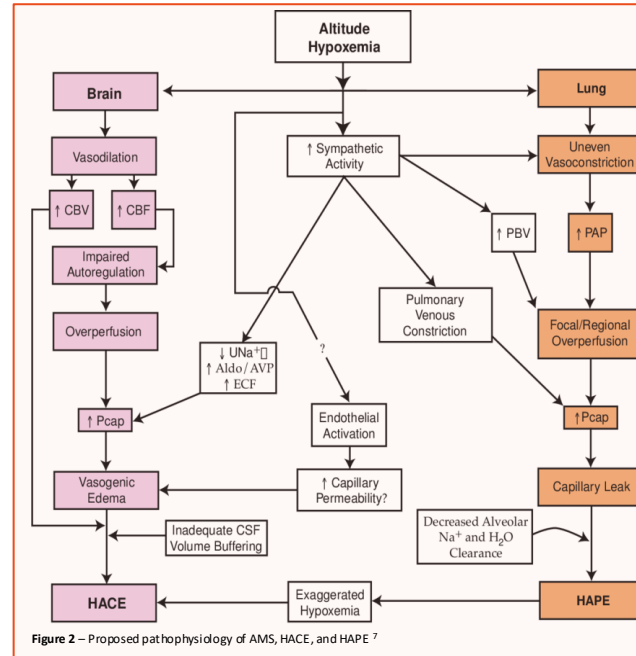
Acclimatisation is a complex and not yet fully understood mechanism, however theories exist⁵.

HACE and AMS represent a "pathophysiological continuum"⁴. Cerebral hypoxia stimulates vasodilation, increasing CBF and CBV. This impairs autoregulation, leading to overperfusion, which contributes to vasogenic oedema⁵.

The exact cause of HAPE is unknown. It is thought that pulmonary hypoxia causes uneven pulmonary vasoconstriction, forcing fluid out and damaging the endothelial barrier, resulting in oedema. Increased pulmonary vessel resistance leads to pulmonary hypertension, compounded by physical exertion. Additional SNS activation and catecholamine release leads to endothelial damage, transferring fluid into alveoli⁶.

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Conclusion

This review could be further developed by additional research as the pathophysiology of HAI is complex and not yet fully understood. This could lead to reduced incidence of HAI and subsequent mortality through improved diagnosis, prevention, and treatment.

