Do Phosphodiesterase-5 inhibitors have a role in the prophylaxis or treatment of High Altitude Pulmonary Oedema?

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Introduction

High Altitude Pulmonary Oedema (HAPE) is a non-cardiogenic pulmonary oedema associated with altitude, characterised by the triad of excessive pulmonary artery hypertension (PAH), high protein permeability in pulmonary capillaries, and normal left heart function.[1–3] Originally described by Houston in 1960, it remains a leading cause of preventable death at altitude, with an estimated mortality of 44% if untreated, and 4–11% if treated with descent and oxygen.[4–6] Whilst incidence is generally low – 0.01%, 0.2% and 2% at high, very high, and extreme altitudes respectively – it increases dramatically with faster ascents.[7] With the rise in tourism and the associated rapid ascent profiles, it would suggest that the incidence of HAPE is likely to increase worldwide.

It is now accepted that the "stress failure" model of pulmonary oedema described by West *et al.* is most likely to represent the pathogenesis of HAPE in-vivo.[8,9]

Methodology

Results

Discussion

Recommendations and Conclusion

References

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