

## CORRESPONDENCE

# Pathogenesis of high-altitude pulmonary oedema

To the Editor:

We have read with great interest the paper by WEST *et al.* [1], regarding the stress failure mechanism of pulmonary capillaries in the pathogenesis of high-altitude pulmonary oedema (HAPE), and would like to add some considerations. HACKETT *et al.* [2] described a high incidence of HAPE in subjects without the right pulmonary artery. Recently, we have reported a case of unilateral, left sided HAPE in a subject with a right pulmonary artery hypoplasia [3], that also underlines the importance of a restricted pulmonary vascular bed in the development of HAPE. Other reports of HAPE have been described in association with a congenital [4, 5] or acquired [6] pulmonary artery occlusion. In these subjects, HAPE usually occurs at moderate altitude and is sometimes recurrent [5, 6]. All these observations confirm the HAPE susceptibility in subjects with pulmonary artery abnormalities and are consistent with the pathogenetic mechanism proposed by WEST and MATHIEU-COSTELLO [7].

In people without anatomical defects of the pulmonary vasculature the most likely hypothesis is that proposed by HULTGREN [8]: an uneven pulmonary vasoconstriction, that leads to high pressure in the non-vasoconstricted pulmonary vessels. This hypothesis is supported by the radiological appearance of a patchy pulmonary oedema and by the increased and uneven pulmonary perfusion in HAPE susceptible subjects after hypoxic breathing [9]. Although not yet proven in humans, stress failure of pulmonary capillaries appears to be the best model to explain the pathogenesis of HAPE.

## References

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