Controversy: exercise-induced pulmonary haemorrhage in the horse

S.W. White,[1] G. Parsons,[2] G. Mactaggart,[1] D. McLeod,[1] M. Hamut,[1] D.B.F. Cottee[1] and A.W. Quail,[1]
[1]Cardiovascular Research Programme, Hunter Medical Research Institute, and Discipline of Human Physiology, School of Biomedical Sciences, Faculty of Health, University of Newcastle, Callaghan, NSW 2308, Australia and [2]Pulmonary and Critical Care Medicine, University of California Davis, Davis, CA 95616, USA.

Exercise-induced pulmonary haemorrhage (EIPH) is of concern world-wide due to high numbers of thoroughbred horses which bleed. Once detected, horses are banned from racing due to potential collapse on the track and the risk to jockeys and other starters. In Australia the initial ban is 3 months; another bleed incurs a life-time ban. The cost to the racing industry of this rejection is spectacular. In Australia, the issue is the subject of Government Reports. The financial input estimate to the industry is US$ 6.24B. In 2006 it was estimated that approximately 17,280 horses bled with a potential life-time ban and output cost of US$ 70.4M. However, the postulated mechanisms and solutions are dependent on experimentally testable postulates, which are highly controversial and expensive to test. EIPH occurs in any working horse, and in other species at high work levels, including Olympic man.

Current theories (West, 2003) focus on extreme pressures on the heart and pulmonary blood vessels during, and at the end of an event. Limited data suggests elevation of mean aortic pressure to 240 mmHg, mean pulmonary artery pressure to 120 mmHg, an estimated pulmonary capillary pressure of 70 mm Hg, and left atrial pressure to 70 mm Hg. These pressures may rupture the ‘thin but strong’ alveolar membrane made of epithelium, collagen IV, and endothelium. By contrast, Manohar (1992) argues that the bronchial capillary bed may rupture.

We favour the hypothesis that the bronchial capillary bed is more vulnerable to the physical forces than the alveolar circulation, because, i) if the intravascular pressures are as documented, it is likely the systemic bronchial capillary is subject to higher pressures than the pulmonary capillary; ii) the protection of pulmonary capillary pressure afforded by vasoconstriction evoked by excitation-resetting of CNS vagal activity during exercise (Quail *et al.*, 2007), may be withdrawn by thermoregulatory inhibition; iii) the high pressure in the bronchial circulation is also beyond any autoregulatory control of resistance vessels. Capillary pressures will thus rise due to high distending pressures invoked by the high systemic (upstream) mean pressure and high pulmonary (downstream) mean pressure, irrespective of drainage site. It is inescapable that bronchial capillary pressures must exceed pulmonary capillary pressure. These vessels lack the ‘thin but strong’ structure of the alveolar membrane, and will thus constitute the primary site of EIPH.

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Manohar M. (1992) *American Journal of Veterinary Research*, **53:** 925-9.
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