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General Articles

Exercise-induced pulmonary haemorrhage (EIPH) in horses results from locomotory impact induced trauma - a novel, unifying concept

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Summary

Exercise-induced pulmonary haemorrhage (EIPH) in horses, although of major welfare and economic importance worldwide, is of uncertain cause. It is accepted that the dorsocaudal region of the lung is particularly prone to the condition, but present theories of causation cannot satisfactorily explain the mechanism or pattern of occurrence. We propose that EIPH results from locomotory impact induced trauma; the mechanism being similar to that producing lung tissue damage following thoracic impact injury.

In impact injury, the localised impulsive load on the chest wall is transmitted by pressure waves through the lung at a slower speed than in the chest wall. The waves are subsequently reflected from the distal chest wall and other structures, producing a complex pattern of wave motion; waves travelling from regions of large cross-section to narrower ones are amplified in magnitude, consequently these regions can experience very high local stresses. Compression/dilation and shear waves are produced within the parenchyma and the latter particularly have been implicated as the cause of parenchymal damage and rupture with oedema and haemorrhage. This form of soft tissue damage has been shown to occur at remarkably low loads with an impact velocity greater than about 11 m/s and pressure exceeding approximately 14 kPa.

In the horse, the lung is subjected to comparable levels of locomotory derived impulsive force during moderate to high speed exercise and this is the basis of the mechanism causing EIPH. During locomotion, the force following ground-strike of the front legs is transmitted, with some attenuation, through the forelimbs to the scapulae. The anatomical arrangement of the scapula, coupled with the direction of the force at the shoulder (scapulo humeral joint) produces an impulsive force on the rib cage, approximately just below mid height of the frontal aspect of the chest approximately over the fourth rib. As a result, pressure waves are transmitted through the lung parenchyma towards the dorsal and caudal regions; these waves are subsequently reflected at the distal chest wall, spine and diaphragm causing a complex pattern of wave interaction. The observed locations of EIPH are at the sites where wave intensity is expected to be greatest due to changes in cross section and reflection. Based on available information, it is estimated that impulsive forces of more than 100 kPa, lasting approximately 10 ms, would be applied to the chest wall by each scapula in a 500 kg horse when galloping; this level of force would be sufficient to cause oedema and haemorrhage as observed in impact induced injury.

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Introduction

Bleeding in the lung of racehorses during racing (exercise-induced pulmonary haemorrhage: EIPH) has been recognised for some time (Cook 1974; Pascoe *et al.* 1981; Whitwell and Greet 1984). Originally, it was considered that EIPH was a phenomenon particularly associated with Thoroughbreds and commonly thought to impair performance. It is now well documented to occur almost universally in racehorses (Mason *et al.* 1983; Clarke 1985) and frequently in other classes of horse undertaking a wide range of intense competitive exercise (Clarke 1985); more recently, it has been clearly documented in the racing camel (Akbar *et al.* 1994) and, possibly, in greyhounds (King and Raskin 1991). However, there is still considerable debate as to the basic cause of the phenomenon in horses (Pascoe 1997).

Exercise-induced pulmonary haemorrhage is associated with damage to the lung alveolar epithelium, the pulmonary, and possibly bronchial, capillary networks, and the leakage of blood into the interstitial and alveolar airspaces. It is now well accepted to be microscopically focal, but generally symmetrically apparent in the dorso-caudal region of the diaphragmatic lobes, extending cranially along the dorsal surface (Mason *et al.* 1983; Pascoe 1997). The bilateral symmetry and distribution of lesions suggest that the process develops essentially laterally to adjacent affected pulmonary segments (i.e. at a constant caudal level), rather than cranially in a diffuse manner (O'Callaghan *et al.* 1987).

Here, a coherent explanation is introduced of the underlying