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Correspondence

An endoscopic test for bit-induced nasopharyngeal asphyxia as a cause of exercise-induced pulmonary haemorrhage in the horse

In their article on sudden death in racehorses, Lyle *et al*. [1] expressed the hope that their study would stimulate hypothesis-led investigations into possible causes.

Currently, there is no consensus on the first cause of exercise-induced pulmonary haemorrhage (EIPH). But the 2 leading mechanistic hypotheses are only at odds over the flimsiest of barriers – the pulmonary air/blood barrier. The majority opinion is that 'bleeding' occurs because of abnormally high capillary pressure on the blood side of the barrier. Let us call this the blood pressure hypothesis. The minority opinion is that it occurs because of abnormally low negative pressure on the air side of the barrier – the air pressure hypothesis.

The majority maintain that high pulmonary pressure is an inherent characteristic of the Thoroughbred. In other words, that the first cause of EIPH is the Thoroughbred itself. But EIPH is not confined to the racing Thoroughbred. It also occurs in the racing Standardbred, Arabian and Quarter Horse. It is not even confined to racing as it also occurs in the hyperflexed dressage horse, the draft horse with a paralysed larynx and a horse of any breed that gets cast in its stall with its head twisted. Long before I contracted a conflict of interest, my colleagues and I at Tufts concluded that asphyxia was a possible cause of EIPH [2].

The 'blood-pressure-in-the-racing-Thoroughbred' hypothesis does not lend itself to testing nor to a solution by removal of the supposed first cause. Regrettably, it gives credence to the sad idea that 'bleeding' is incurable or even physiological and needs to be 'managed' with medication. Because the blood pressure hypothesis is invulnerable to refutation, I conclude that it is not a scientific hypothesis.

In contrast, the air pressure hypothesis is highly vulnerable and eminently refutable. In 1988, we listed a number of ways in which asphyxia could occur, naming recurrent laryngeal neuropathy as the most likely candidate. Since then, I have realised that though this is prevalent enough to match the prevalence of EIPH, the severity of the neuropathy in many cases is insufficient to entirely explain the problem. In the last 15 years, I have come to recognise that the ubiquitous bit is a much stronger candidate for causing asphyxia and that it brings this about by triggering instability and dorsal displacement of the soft palate [3–7].

At liberty, the running horse has a closed mouth, sealed lips and an immobile tongue and jaw. I now have evidence that its oral cavity and oropharynx are under negative pressure (Cook 2012, unpublished material). By breaking the lip seal, I believe that the bit triggers a cascade of problems from lip to lung. In anatomical, if not physiological, order these are: 1) Loss of the oral vacuum, 2) dynamic collapse of the nasopharynx (soft palate instability and dorsal displacement) , 3) obstruction of the choanae , 4) gaping of the pharyngeal orifices to the guttural pouch, 5) dynamic collapse of the larynx, 6) dynamic collapse of the dorsal membrane of the trachea with, over time, permanent distortion of the tracheal cartilages and 7) EIPH. Shakespeare's phrase is apt, *'The lie in the throat as deep as to the lungs.'* Allen and Franklin [8] report endoscopic observations, during moments of soft palate instability, consistent with loss of the oral vacuum, i.e. a flattened epiglottis and convexity in the most caudal section of the soft palate.

Further light on the effect of the bit is shed by Hong Kong statistics for 2004/2005 [9]. During the training and racing of 1358 Thoroughbreds the prevalence of 'blood at one or both nostrils' was 5.74%. During swimming, when 1155 of these same horses, on 150,000 occasions, were (presumably) wearing nothing but a halter, there was no 'epistaxis'.

Exercise-induced pulmonary haemorrhage is not exclusively dependent on exercise and neither is it a true haemorrhage. So-called 'epistaxis' is not blood but oedema fluid coloured with red blood corpuscles. A more precise and scientifically useful name would be negative pressure pulmonary oedema (NPPE). An internet search reveals that this relatively uncommon but life-threatening emergency in man is analogous to the bizarrely common and potentially fatal EIPH in the racehorse, a subset of NPPE in the horse.

The literature on NPPE in man provides an explanation for the abnormally high pulmonary pressure in the horse. In a review of NPPE, Deepika *et al*. [10] state that the primary mechanism is upper airway obstruction. This generates a markedly negative intrapleural pressure transmitted to the pulmonary interstitium, an increased venous return to the right side of the heart and a rise of pulmonary capillary pressure.

The NPPE evidence in man blends the 2 competing hypotheses for EIPH in the racehorse, citing air pressure as the causal factor and blood pressure as a secondary effect. I submit that the same blend applies to the horse.

The technology of overground nasopharyngoscopy provides a way of putting this to the test. The null hypothesis could be tested that if a horse was first ridden in a bitted bridle there would be no improvement in the patency of the choanae and nasopharynx as judged by endoscopy when the same horse was ridden again under similar conditions in a bitless bridle. If improvement occurred the null hypothesis would be refuted and the air pressure hypothesis supported.

An addition to the standard endoscopy protocol will be necessary in order to evaluate the patency of (at least) one choana and the rostral two-thirds of the nasopharynx. To evaluate these critical regions of the airway, a step that is currently omitted, the distal tip of the endoscope must be placed at the caudal end of the nasal cavity. By positioning the endoscope in the caudal half of the nasopharynx only, information on dorsal displacement of the soft palate and laryngeal problems is gathered but some information on rostral palatal elevation will be overlooked and choanal stenosis, a potentially catastrophic ball valve obstruction due to the Bernoulli effect, can never be documented.

Science advances either by refutation of hypotheses or by the failure of determined efforts to refute them. Science does not advance anything like as convincingly by simply adding evidence in support of a hypothesis. As the air pressure hypothesis has not been refuted in 25 years it seems to have survivor fitness but this could be because it has never been tested. But if the air pressure hypothesis survived attempts to refute it with overground endoscopy evidence this would assert its claim to be acknowledged and point to the bit as the major cause of NPPE in the horse. Further research into the effect of the bit on the horse is needed to ratify or refute the bit as the cause of NPPE.

Author's declaration of interests

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