

# WHY DO HORSES' LUNGS BLEED?

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*"... the lie in the throat as deep as to the lungs" - Shakespeare*

If a racehorse dies on the track from so-called exercise-induced pulmonary hemorrhage (EIPH) and a necropsy is carried out within the hour, both lungs will be seen to be swollen and waterlogged. Instead of being a healthy pink color, both lungs will be dark red, except perhaps for the smallest lobe at the front of each lung. On the surface of the lung will be a hemorrhagic rash. These spots will be distributed in the same characteristic way in both lungs, occurring on those portions that are closest to the horse's tail in the horizontal plane and closest to its spine in the vertical plane (i.e., a tail-top or caudo-dorsal distribution). The distribution of the spots matches the distribution of the dark red discoloration (congestion and hemorrhage) in the depth of the lungs. A distinctive feature of the lung changes is that the lesions are bilaterally symmetrical (i.e., left and right lungs have the same lesions, to about the same degree and in the same places)<sup>1</sup> Both bronchi will be filled with a tenacious, pink foam. This heavily blood-stained foam may also fill the windpipe and even appear at both nostrils. When the lung tissue is sliced, the tail-top regions of both lungs will be black with hemorrhage. Instead of being light, dry and puffy like a well-made soufflé, the tissues will be heavy, wet and solid.

The medical term for a waterlogged lung is *pulmonary edema*. Because the structure of lung tissue is little more than a flimsy honeycomb of delicate air sacs surrounded by equally fragile capillary blood vessels, the edema is accompanied by intense vascular congestion and hemorrhage into the loose tissue that lies in the spaces between the air sacs and the blood vessels (Fig 1). The mechanical forces that cause the edema provide the key to explaining the characteristic tail-top distribution of the hemorrhages (Fig 2). Nevertheless, the fundamental lesion is pulmonary edema rather than pulmonary hemorrhage. It could best be described as hemorrhagic edema.<sup>2</sup>

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<sup>1</sup> The significance of this rather unusual feature of lung pathology will be referred to again later, as it is of importance in explaining the cause of the problem.

<sup>2</sup> Furosemide (Lasix) has long been the diuretic of choice for the management of pulmonary edema in man. At first glance, it seems appropriate that this same drug should have become established as a supposed preventative for the same problem in the horse (even though to this day, most veterinarians do not accept my opinion that the underlying pathology is pulmonary edema). It is unlikely, however, that Lasix (or Salix as it is now called in equine medicine) can have any preventive value and its popularity probably depends on other factors, such as the loss of water reducing the weight of the horse.

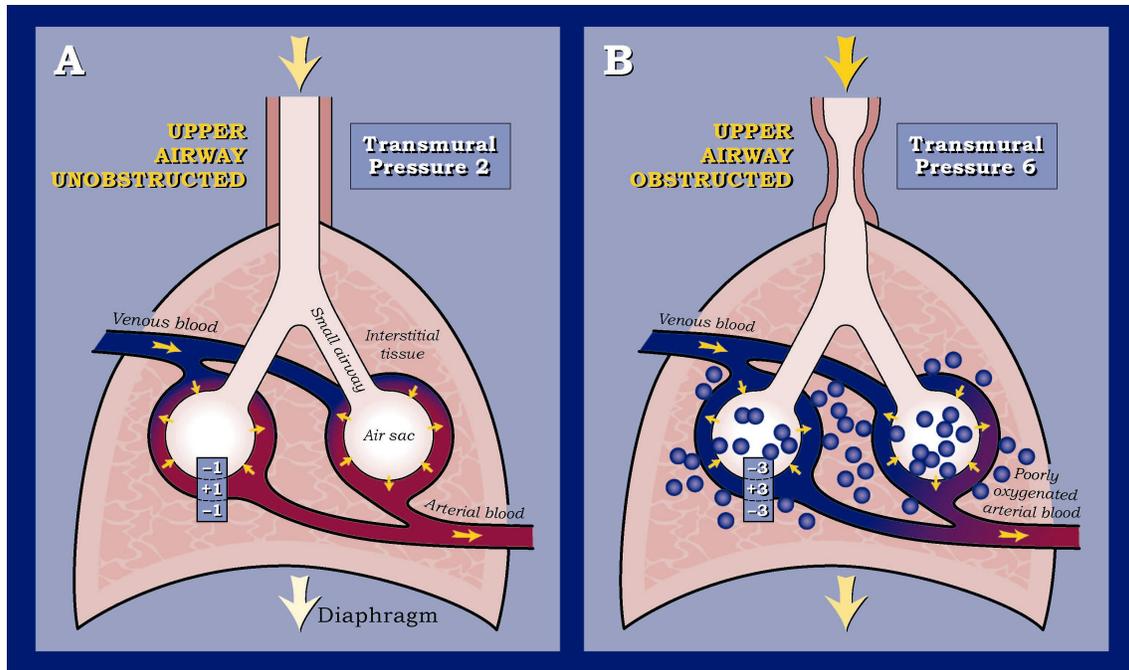


Fig 1. The aerodynamics of lung bleeding. The rounded areas represent two of the thousands of air sacs in the lung. The shaded tubes leading to them represent the small airways of the lung. Venous blood (blue) flows through capillary vessels in intimate contact with ventilated air sacs and becomes arterial blood (red). The large yellow arrows represent the flow of air on inspiration as the diaphragm flattens. The small yellow arrows around the air sacs represent the gas exchange (oxygen and carbon dioxide) that occurs at the delicate membranous interface between capillary blood vessel and air sac (the blood/gas barrier).

- A. Normal inspiration with no obstruction of the upper airway. The negative pressure generated in the air sacs and in the interstitial tissue of the lung is within physiological limits and is represented by the symbolic figure 'minus one'. The pressure of blood in the capillaries is also within physiological limits and is represented by the figure 'plus one'. The difference between the two pressures on each side of the (the transmural pressure) is 2 (the difference between -1 and +1). Only gas exchange takes place at the blood/gas barrier.
- B. Forced inspiration against an obstructed upper airway, caused in this illustration by RLN. The negative pressure in the air sacs and the interstitial tissue of the lung is greatly increased. For the purpose of explaining the principle involved, let us call it 'minus three'. The positive pressure of blood in the capillaries is also increased (+3) because of mechanisms described in the text. The transmural pressure is now 6 (the difference between +3 and -3). Edema fluid (together with some red blood cells) moves from high to low pressure and is drawn into the air sacs and the interstitial spaces from the blood vessels and lymphatics of the lung.

Unfortunately, if the necropsy is delayed several hours, as is usual, the pulmonary edema will have dispersed or been masked by artifactual lung changes that occur after death in any horse, regardless of the cause of death. The ephemeral quality of pulmonary edema may explain why the true nature of what has been called EIPH has not yet been recognized, and why lesions compatible with asphyxia have often been wrongly attributed to *post-mortem* change.

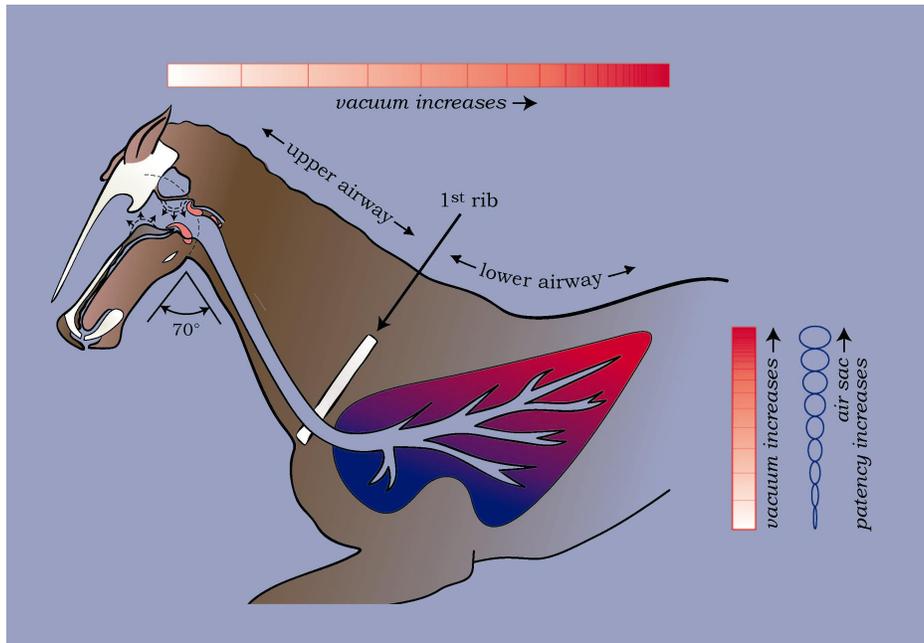


Fig 2. Showing how an upper airway obstruction (caused by poll flexion in this illustration) results in the development of an abnormally high vacuum pressure when suction is applied during inhalation. The aerodynamics of gas flow down tubes is such that the vacuum pressure increases with the distance from the point of obstruction. It follows from this example of an obstruction at the level of the throat that the vacuum pressure in the larynx will be less than the vacuum pressure in the windpipe and that this, in turn, will be less than that generated at the tail end of the lungs. This principle of aerodynamics explains why it is that when a lung 'bleeds', the greatest intensity of lesions (red areas) occurs at the tail end of the lung. The lung is suspended from the spine and is a highly elastic organ. Because of this, it behaves like a slinky and the air sacs at the top of the lung tend to be larger and more patent than those at the bottom of the lung. The larger air sacs at the top of the lung are more exposed to the abnormally high vacuum pressure on inhalation than the smaller air sacs at the bottom of the lung. This explains why the 'bleeding' lesions are more intense at the top of the lung. Together, these two principles explain the top-tail distribution of the lesions. Finally, both lungs are exposed to the same forces and this explains why the lesions are the same in both lungs (bilaterally symmetrical).

The name *exercise-induced pulmonary hemorrhage (EIPH)* was first proposed over 20 years ago. At the time, it was appropriate as being purely descriptive of a problem, the cause of which was unknown. With the new evidence on causation that has come to light since then, I feel that a purely descriptive name is no longer appropriate. Instead, I propose *asphyxia-induced pulmonary edema (AIPE)*. My reasons are as follows:

- The bronchial and sometimes nasal discharge that we have called a *hemorrhage* is not, in fact, blood, but heavily blood-stained edema fluid. It does not, for example, have the ability to clot. Similarly, the hemorrhage that occurs in the tissues of the lung is only secondary to the primary problem, which is edema
- The word *exercise* in the name is not appropriate, because although pulmonary edema occurs during exercise (galloping in the racehorse and traction in the draft horse) it is not unique to exercise. Under certain circumstances (specifically

suffocation), the same pulmonary edema, with its rather characteristically distributed hemorrhagic rash on the surface and hemorrhagic congestion in the depths of the lung, occurs in the standing or even the recumbent horse. I have seen pulmonary edema occur in horses that have died from accidental asphyxia in the stall, in a horse trailer, on a treadmill, and from spasm of the larynx following general anesthesia<sup>3</sup>. The common factor is not exercise but asphyxia.

- The basic pulmonary pathology is not *hemorrhage* but edema.<sup>4</sup>
- The anatomical location of the primary problem is not the lungs but the upper airway. The *pulmonary* lesions occur only as a sequel to upper airway obstruction. In other words, the lung lesions are secondary to the primary problem, which is upper airway obstruction, or asphyxia.

I support the hypothesis that pulmonary edema will occur in any horse that has to breathe-in (inspire) against the abnormal resistance of a closed or partially closed upper airway. I define the upper airway as being that part of the respiratory tract lying between the nostrils and the windpipe at the level of the first rib. Fortunately, in most horses the degree of asphyxia is partial rather than complete and, accordingly, the clinical picture runs the gamut from no detectable signs at all, to sudden death. In between, the clinical signs of partial asphyxia include varying degrees of exercise intolerance, with or without laryngeal stridor, the rattle of a displaced soft palate and, occasionally, the appearance of blood-like fluid at the nostrils. In those suffocated horses that survive, the signs are usually transient and, apart from a few days during which they may show a catarrhal discharge at both nostrils, most affected horses *appear* to make a complete recovery. Nevertheless, as red blood cells are an irritant to lung tissue, repeated episodes of pulmonary edema are likely to leave a steadily accumulating legacy of low-grade bronchiolitis. It is, I think, a chronic bronchiolitis from this source that has been termed “small airway disease” by those researchers who support the hypothesis that “small airway disease” is the cause of the problem, not - as I believe - its effect.

Upper airway obstruction will result, at each intake of breath, in the development of an abnormally strong vacuum in the air sacs of the lung. As the walls of the air sacs are about fifteen times thinner than a standard sheet of airmail paper, this - in turn - results

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<sup>3</sup> No veterinarian would dream of designing a research protocol which depended on testing the asphyxia hypothesis by deliberately asphyxiating a series of horses to see whether the lesions of ‘bleeding’ could be reproduced experimentally. From time to time, however, even under the best of managements, accidents do happen in which horses asphyxiate. These accidents can be regarded as ‘natural experiments’ and much can be learned from them, as indeed was the case in those accidents mentioned above. That the characteristic lesions of ‘bleeding’ can be reproduced ‘experimentally’ by an episode of asphyxia represents crucial evidence in support of the asphyxia hypothesis. None of the other hypotheses as to the cause of ‘bleeding’ have the benefit of this experimental evidence.

<sup>4</sup> From this it follows that if a racehorse performs badly and on endoscopy no blood-stained edema fluid is detected in the airway, this is not enough to rule-out the possibility that it may - nevertheless - have developed pulmonary edema. The next logical step in evaluation would be chest radiography. Because of the transient nature of pulmonary edema, however, this step would have to be taken with some urgency if the evidence was not to be missed. On the racetrack, such an examination is, of course, out of the question.

in fluid (and, inevitably, many red blood cells) being sucked out of the capillary blood vessels into the interstitial tissues of the lung and small airways; hence the hemorrhagic edema (Fig 1). The tail-top and bilaterally symmetrical distribution of lesions in both lungs is exactly what would be predicted from an upper airway obstruction during inspiration, based on aerodynamics principles and the horse's respiratory anatomy, (Fig 2).

The aerodynamics of the upper airway during inhalation can be likened to fluid flow through a straw. The physical laws that govern fluid flow are the same as for gas flow. If the straw is undamaged and the suction force not excessive, you are able to suck fluid easily out of a glass and the fluid will flow generously, up to a limited maximum speed. But crimp the straw and now try to suck again. The suction pressure needed will be much greater and the flow less. If the straw is severely damaged, the walls of the straw may collapse and block the flow completely.

Now think of your mouth as the horse's lungs, the straw as the upper airway, and the glass of fluid as atmospheric air. When the airway is unobstructed, air will get sucked into the lungs on inspiration with minimum effort and with minimum suction pressure. Under these conditions, a horse uses the least amount of energy to breathe and the lungs are only exposed to suction pressures that the delicate tissues of the lung can withstand without harm. For example, the negative pressure in the air sacs of the lung will not be so low that the difference between it and the positive pressure in the capillaries on the other side of the air/blood barrier is so great as to imperil the effectiveness and viability of the barrier. In other words, the exchange of gases will occur but not the passage of fluid. The difference of pressure on either side of the delicately thin wall that constitutes the air/blood barrier is known as the transmural pressure (Fig 1).

Any obstruction of the airway will reduce the flow of air, increase the work of breathing, and increase the suction pressure applied to the tissues of the lung. The horse will be denied the oxygen it needs, will have to spend more energy in getting less oxygen, and the transmural pressure at the level of the air/blood barrier may well be so high that heavily blood-stained edema fluid gets sucked out of the lungs into the small airways, i.e, the horse 'bleeds'.

If the airway is obstructed beyond a critical point then, when suction is applied on inspiration, the walls of the airway may well collapse entirely and the horse would be totally unable to breathe. Under these conditions, even one forced but futile inspiratory effort (one stride at the gallop) would be sufficient to create a damaging negative pressure in the air sacs and, accordingly, an abnormally high transmural pressure. A copious effusion of edema fluid into the airways would follow.

An example of the above mechanism can be drawn from childhood memories of the red welt that could be produced on one's arm by sucking on it fiercely. As the tissues of the lung are far more delicate than skin and much more vascular, it is not difficult to imagine how such an effect would suck 'blood' out of the lungs. An even more graphic example can be found in the diary of John Evelyn for 7<sup>th</sup> May, 1662: "*I waited on Prince Rupert to our Assembly (in August, 1662 this assembly was granted a charter and named the Royal Society) where we tried several experiments in Mr. Boyle's vacuum. A man thrusting in his arm, upon exhaustion of the air, had his flesh immediately swelled so as the blood was near bursting the veins: he drawing it out we found it all speckled*"

In the last few years, a group of researchers have proposed the hypothesis that 'bleeding' is caused by what they regard as the normal horse's ability to generate an unusually high capillary blood pressure in the lungs at fast exercise. This results, they suggest, in a mechanical rupture of the capillary walls and they have named this explanation the capillary stress hypothesis. They also maintain that 'bleeding' is a normal event in the horse at high speed and, therefore, an acceptable phenomenon in the racehorse.<sup>5</sup> I question the premise and disagree with its corollary. Otherwise our conflicting hypotheses are similar in so far as both support the idea that 'bleeding' is caused by a high transmural pressure at the level of the air/blood barrier (Fig 1). But whereas the capillary stress hypothesis proposes that the high transmural pressure is attributable to an increase of positive pressure on the blood side of the barrier, I believe that it is due to an increase of negative pressure on the air side of the barrier. Furthermore, whereas the capillary stress hypothesis encompasses the idea that 'bleeding' is physiological, I believe it to be pathological. To maintain that it is physiological for air sacs of the lung to be filled with fluid and not air surely amounts to a denial of one of the fundamental principles of mammalian respiratory physiology. Other arguments for refuting the capillary stress hypothesis include the following:

- If capillary wall rupture was the cause, the fluid that flooded the airways would be pure blood and one would expect it to clot.
- If high capillary blood pressure were the mechanism, the distribution of 'hemorrhage' in the lungs would be quite different from the tail-top distribution that is encountered. One would expect the most severe hemorrhage to occur in the region where the barometric pressure was greatest, that is in the bottom of the lung rather than the top.

Though I cannot accept that a normally high blood pressure in the capillaries is

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<sup>5</sup> The conclusion that the pulmonary hemorrhage is actually *caused* by exercise provides a bizarre twist on the original name for the problem (EIPH) that was never intended but seems to have entered here into the sub-conscious thought processes.

sufficient to mechanically tear open the tissues at the air/blood barrier, I can accept a mechanism whereby the capillary blood pressure could be abnormally increased. If this is so, then 'bleeding' could be associated with a transmural pressure rise that is contributed to by increases on both sides of the barrier, i.e., a higher positive pressure on the blood side and a lower negative pressure on the air side. If the upper airway is obstructed on inspiration, as it often is (because of the bit and other factors too), this would have the effect of abnormally increasing the flow of venous blood into the lungs from the right side of the heart, thereby increasing the capillary blood pressure. If the upper airway is obstructed on expiration, this would have the effect of delaying the return of arterial blood from the lungs to the left side of the heart. This too would tend to increase the capillary blood pressure and, in addition, cause a reduction in the rate of lymph drainage from the lungs. Both of these factors would lead to pulmonary edema, venous congestion and a rise in pulmonary capillary pressure.

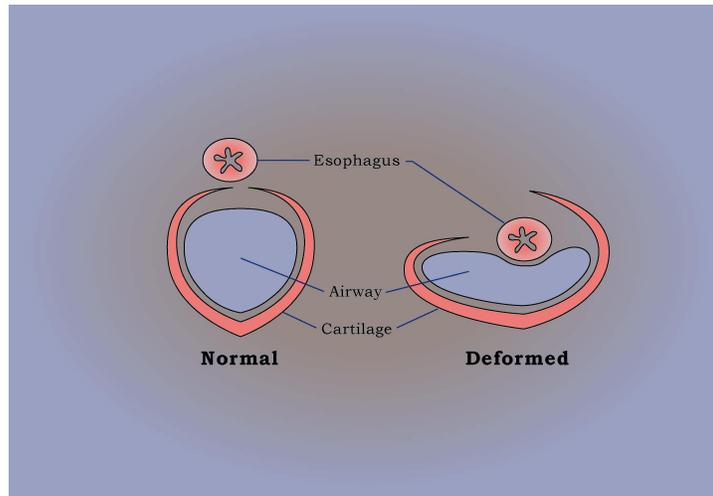
The 'bleeding' problem in horses has, I believe, many similarities to high altitude pulmonary edema (HAPE) in man. Shortage of oxygen (hypoxia) is thought to be the trigger factor in the development of HAPE. Shortage of oxygen in the blood (hypoxemia) happens also to be a recognized and unique feature of fast exercise in the racing Thoroughbred and this may be no coincidence<sup>6</sup>. No other athletic animal is known to exhibit this feature. In addition, because of the abnormally increased negative and positive pressures in the obstructed airway of a galloping horse at each inspiration and expiration (respiratory rate 120/min or greater), the lungs are being subjected to rapid and abnormally large pressure swings (barotrauma).

80% or more of horses in training show evidence of 'bleeding' on endoscopic evidence alone. The prevalence is even higher if one takes into account microscopic evidence from lung washes. Upper airway obstruction in racehorses is quite common enough to explain this alarmingly high prevalence.

- The bit is a common cause of upper airway obstruction
- At racing speeds any position of the head and neck other than maximum extension of the poll constitutes an airway obstruction at the throat. The use of a bit enables the rider to demand such poll flexion rather easily.
- There are many defects of conformation that represent potential sources of upper airway obstruction. Examples include narrow jaws and therefore narrow throats and larynges and deformities of the windpipe (Fig 3).
- Obstruction of the airway is a feature of almost every known disease of the upper airway. RLN occurs, to varying degrees, in over 95% of horses.

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<sup>6</sup> Thoroughbreds are typically thought to develop a shortage of blood oxygen (arterial hypoxemia) during galloping as a result of a gas exchange limitation at the level of the air/blood barrier. It may be, however, that the unexpected shortage of oxygen should be blamed primarily not on this but on upper airway obstruction from the bit, lack of poll extension and other causes.



*Fig 3. Cross sections of a normal and deformed windpipe at the level of mid-neck. As can be seen, the diagrams illustrate the almost circular section of the large windpipe on the left and the flattened section of the small windpipe on the right. Gases pass most easily down circular tubes of wide diameter. But there is an additional factor because, during fast exercise, each of these tubes will behave differently (dynamically). It will be noted that the membranous lining of the normal windpipe is supported around the whole of its circumference by cartilage, so its wall is rigid and well designed to withstand the suction forces of inhalation without collapsing. In stark contrast, the deformed windpipe has an unsupported area along its top flat surface, into which the gullet (esophagus) has dropped. When a suction force is applied to this tube, it will tend to collapse and further obstruct the airway. Gases pass most easily down tubes with rigid walls rather than flaccid ones. It is widely thought that tracheal deformities are rare and confined mostly to ponies but, as the result of surveys that I have carried out at abattoirs, I have to report that deformities such as the one depicted above are common in horses. Many a racehorse is handicapped by a defect of this sort.*

In the live horse, RLN can be diagnosed and graded by scoping the airway at rest or, better still, at exercise on a treadmill. In the dead horse it can be diagnosed by an examination of the laryngeal muscles and nerves, as it was - for example - in the death of the horse, MR. NICKERSON, in the Breeders Cup Sprint of 1990. The reason that RLN is not more often diagnosed at necropsy is that the necessary examinations are not being carried out. If the laryngeal muscles and nerves were routinely examined, the evidence would be found. Unfortunately, such examinations are not yet a standard part of a necropsy protocol and so the evidence is being lost, even in those programs in which all fatalities on the racetrack are necropsied.

Though there is ample proof that 'bleeding' affects performance when a horse dies on the racetrack, it is not easy to measure its effect when the horse survives. Nevertheless, the occasional deaths are alone sufficient to emphasize that 'bleeding' is not normal. It is true to say that, within the racing Thoroughbred population, 'bleeding' is statistically common and might therefore be considered to be the norm, or average *statistically*. But it is not true to say that it is normal *physiologically* for a horse in the wild

to 'bleed' when galloping. The air sacs of the lung, in every mammal, should contain air not fluid. It is reasonable to assume that any athlete (whether human or equine) that 'bleeds' is less likely to be a successful performer than one with a patent airway and a healthy pair of lungs.

'Bleeding', has been described by supporters of the capillary stress hypothesis as an "unavoidable consequence of competitive training and racing" and, from this, it has been implied that 'bleeding' is a hazard that the racing industry and the betting public need to accept as a condition of racing. It has also been suggested that 'bleeding' is normal and inevitable. Given the current rules of racing (i.e., the fact that stewards of racing mandate the use of a bit) and the genetic make-up of the inbred Thoroughbred (responsible for hereditary deformities and diseases of the airway), I reluctantly accept that episodes of asphyxia in racehorses could never be entirely eliminated. Nevertheless, I neither accept the 'normality' nor the inevitability of the situation and believe that, with care, the 'bleeding' episodes could be reduced in frequency and severity. Once it is recognized that the cause of 'bleeding' is asphyxia followed by pulmonary edema, and that this is abnormal, an important step will have been taken towards a more successful management of the problem.

Racehorses could, for example, be permitted to race in a crossover design of bitless bridle. Such horses would breathe more freely and would be less likely to develop dorsal displacement of the soft palate and other problems caused by the bit, such as epiglottal entrapment. Breeders and buyers could focus more on selecting horses with fewer conformational defects of the respiratory system, and less severe degrees of recurrent laryngeal neuropathy. Owners, trainers, and jockeys could adopt management strategies to minimize the occurrence of asphyxia for those horses with known problems. For example, they could enter the horse for shorter races and lower grades of race; provide longer recovery periods between races; let the horse run its own race (no rating); and advise the jockey to avoid flexing the horse's poll. While congenital deformities and diseases of the upper airway are difficult or impossible to alleviate, poll flexion does lend itself to correction. An adjustment of expectations is also part of the management process, i.e. expect less of one's horse if the weather is hot and humid, or windy, or if the going is heavy. Anything that increases the amount of physical work that a racehorse has to accomplish (such as added weight) will render 'bleeding' more likely.

In the above discussion of the cause of 'bleeding', three hypotheses have been mentioned. These have been referred to as the *asphyxia*, the *capillary stress* and the *small airway disease* hypotheses. The first two have been discussed at some length and I have put forward my reasons for backing the asphyxia hypothesis over the capillary stress hypothesis. The small airway disease hypothesis has not yet been given much attention. I will conclude by explaining why I think this hypothesis can be ruled-out. A hypothesis cannot be proved, no matter how much supportive evidence is accumulated, but it can be disproved. Sir Karl Popper, a famous philosopher of science

(and politics), made this point with an example. The hypothesis that “*all swans are white*” can never be proved by identifying vast numbers of white swans but it can be disproved by identifying one black swan.

So, how can we disprove the hypothesis that “*all instances of ‘bleeding’ are caused by small airway disease*”? What is wrong with this theory? Let us first consider the proposition that the hypothetical small airway disease is a virus disease of one sort or another. In the first instance, ‘bleeding’ is too prevalent and too persistent in the same horse over a period of years for us to be able to say that ‘bleeding’ is both started and perpetuated by respiratory virus infections of the lung. A viral pneumonia is essentially an acute and relatively short-lived infection. Furthermore, horses when first experiencing ‘bleeding’ do not have any of the symptoms of a virus infection. They do not have a persistent fever, they do not cough frequently and neither do they have a catarrhal nasal discharge. In addition, ‘bleeding’ does not occur in epidemics that coincide with epidemics of viral respiratory disease.

Whatever management problem or disease causes ‘bleeding’ it has to be an extremely common one, because we know that ‘bleeding’ occurs in 80% or more of racehorses. If ‘bleeding’ is not caused by viral infections of the lung, then the only other known disease of the horse’s chest that might conceivably be sufficiently common to be considered as the culprit is chronic allergic bronchiolitis (“*heaves*”). This disease is also known as chronic obstructive pulmonary disease (COPD). More recently COPD has been considered to be the later stages of something that carries the rather unhelpful name of ‘small airway disease’ and its unintentionally appropriate acronym SAD. It might be noted that this is a name that simply describes the anatomical location of some pathology but without describing the pathology or hinting at its cause. If chronic allergic bronchiolitis were the cause, one would not expect to encounter ‘bleeding’ in those countries where this disease is rare, for example, New Zealand and Australia. Such is not the case. Even though the environmental conditions in these countries are conducive to healthier horses’ lungs than in Europe and North America (horses are out in the fresh air most of the time), ‘bleeding’ still occurs. ‘Bleeding’ occurs even when horses are never stabled. Furthermore, dust-free stable management does not reduce the prevalence of ‘bleeding’. Similarly, none of the medications that are designed to alleviate chronic lung disease and which are widely used in North America in the hope of curing ‘bleeding’ are effective. Most ‘bleeders’ do not have and do not later develop, a persistent cough and the exaggerated flank movement (the so-called *heave line*) which are characteristic of horses with more advanced chronic bronchiolitis.

At necropsy, the nature and distribution of the lesions found in ‘bleeders’ are not typical of the distribution of any known infectious or allergic disease. In fact quite the opposite. Viral and bacterial pneumonias tend to occur in the bottom of the lung rather than the top and, as far as their side distribution is concerned, infections are patchy in their

distribution and asymmetrical. They certainly do not exhibit the quite remarkable degree of bilateral symmetry that is so characteristic of the 'bleeder' lesions. The tail-top regions of the lung are, as was pointed out nearly 20 years ago by the pathologist James Rooney , "at very low risk for pneumonia, bronchitis and flu, etc.,." In contrast to all these contradictions, there is no better way for the bilateral symmetry of the 'bleeder' lesions to be produced than by an obstruction of the upper airway. The tail-top distribution in each lung also lends itself easily to explanations based on the principles of aerodynamics and the anatomy of the lung (see the caption to Fig 1). The bilateral symmetry and the tail-top distribution can both be reproduced in this way by 'natural experiments' as already noted.

The above evidence provides plenty of disproof of the *small airway disease* hypothesis. As the *capillary stress* hypothesis has also been disproved, this leaves the *asphyxia* hypothesis as the only survivor. I have tried to disprove this hypothesis myself and so have my colleagues but without success. By the agreed guidelines of science, any valid (testable) hypothesis that cannot be disproved earns its right to credence at the present state of knowledge, i.e., until such time as new evidence to refute it becomes available. The asphyxia hypothesis is a good hypothesis in that it is testable and highly vulnerable. All that anyone has to do to disprove it is to show that 'bleeding' occurs in the absence of upper airway obstruction.

I conclude that the appropriate name for 'bleeding' is asphyxia-induced pulmonary edema.