All diseases have a cause and no logical treatment or prevention for any disease can be developed until the cause is known. The aim of all medicine should be removal of the cause. One way of testing the validity of a theory as to the cause is to see whether the disease disappears when a suspected cause is removed.

Dr Joseph O'Dea (Thoroughbred Times, October 30, 1999) has performed a valuable service by emphasizing three points.

- The cause of bleeding is known
- The cause is upper airway obstruction (partial asphyxia)
- Lasix does nothing to remove the cause and, not surprisingly, is ineffective as a preventive measure

Dr James Rooney, in 1970, was the first to put forward the theory that bleeding was caused by asphyxia. In 1974, I published evidence supporting the fact that the blood originated from the lung and not, as had previously been assumed, from the nasal cavity. But for many years, not much credence was given to the asphyxia theory. In 1988, I published evidence, including experimental evidence, in support of the asphyxia theory, together with evidence to refute the alternative theories. Since then, I have written 20 more articles supporting various aspects of this same theory and have given my reasons for naming the problem asphyxia-induced pulmonary edema (AIPE), rather than exercise-induced pulmonary hemorrhage (EIPH).

Most of my research colleagues still feel that asphyxia is only one of many competing theories. The theory they currently favor is that bleeding is normal (physiological) and an inherent part of racing. But, unlike the asphyxia theory, their theory fails to explain many aspects of bleeding, including its occurrence in draft horses during slow exercise and in horses that are not even doing any exercise but are accidentally suffocated. I agree, with regret, that bleeding is an inherent part of racing as presently practiced, but I disagree, most emphatically, with the idea that it is normal and unavoidable. The difference of opinion is important. If one accepts the rather surprising contention that bleeding is normal, then presumably prevention is not called for, as there is neither need nor scope for such a step. Conversely, if bleeding is abnormal, this does at least lend itself to the hope of prevention, IF the cause can be removed.

In view of the above, I found it gratifying to read in O'Dea's article that "most observant racetrack practitioners recognize that bleeding is most frequently caused by… obstruction … in the upper air passages …." Figures 1 and 2 explain how, in my opinion, asphyxia causes bleeding.

O'Dea notes that the next step is to define the specific causes of upper airway obstruction, in order to devise a more logical approach to prevention. The purpose of this article is to respond to this challenge and, in doing so, to draw horsemen's attention to a recently discovered cause of asphyxia which has been staring us all in the face for 6000 years.

Some racehorses bleed more than others but it turns out that they all bleed to some extent. Therefore, for the asphyxia theory to hold-up under scrutiny, the collective causes of asphyxia have to be equally universal. Upsetting though it may be, it is true that racehorses are being asphyxiated on a daily basis. The list includes:
1. **Poll flexion**: Anything less than complete extension of the poll at the gallop constitutes an upper airway obstruction. The obstruction occurs at the level of the throat (Figs 1 and 3b). During a race, and particularly during training exercise, racehorses are frequently galloped with their heads in a position of partial (Fig 3b) or even complete poll flexion (Fig 1). The bit method of control is actually dependent on poll flexion. It follows that, by definition, all horses wearing bits have to undergo varying degrees of suffocation from time to time. And in case a reader should feel that transient suffocation is surely acceptable, let me add that if a horse is breathing at galloping speed, when stride and breathing rate is over two a second, it may take as little as three obstructed breaths to damage the lungs and initiate bleeding.

![Diagram](image)

**Fig 1.** Showing how the asphyxia theory of bleeding explains how and why the hemorrhages (black dots) are distributed along the top side and tail end of the lungs. If the upper airway is obstructed (in this particular example by poll flexion), an abnormally powerful vacuum develops in the airway on inspiration. For aerodynamic reasons, the vacuum gets more intense as the distance from the site of obstruction increases; hence the tail end distribution. As lung tissue is rather elastic and the lungs are suspended from the spine, they act in the manner of a slinky. The air sacs at the top of the lung are always more dilated than those at the bottom. Because they are more open, they are more affected by the abnormal vacuum pressure; hence the top side distribution.
Fig 2. Showing the pressure changes that occur in the lung on inspiration, with and without obstruction of the upper airway.

2A: Upper airway unobstructed. In the absence of great resistance, the diaphragm draws air into the lung easily and with minimal effort. The vacuum pressure in the air sac is normal (-1) and so is the blood pressure in the capillaries (+1). The differential or transmural pressure across the exquisitely thin membrane that divides air from blood (the air/blood barrier) is therefore 2 (these are fictitious figures for the purpose of explanation). This difference is not enough to suck fluid and red blood cells out of the lungs and into the air sacs and so normal gas exchange takes place.

2B: Upper airway obstructed. Because of the high resistance to airflow, the diaphragm has had to work harder and make more effort to draw air into the lung and the vacuum pressure in the air sac is now abnormal (-3). Fluid begins to be sucked from the capillaries into the interstitial tissues of the lung (pulmonary edema) and this, in turn, causes the blood pressure to rise (+3). The transmural pressure is now 6, which is large enough to cause heavily blood-stained fluid to be sucked across the blood/air barrier from high to low pressure. Like the interstitial tissues, the air sacs also become water-logged (edematosus) and normal gas exchange is prevented. The horse becomes short of oxygen (hypoxic).
Fig 3. Diagrams showing an unobstructed upper airway (A) with the poll extended and the soft palate in the correct position for rapid breathing. Airflow at the gallop will be relatively smooth and peaceful. Compare this with the obstructed upper airway (B), in which the poll is partially flexed and the soft palate is dorsally displaced. The airway at the throat (nasopharynx) is now seriously reduced in overall diameter, especially at the tail end of the soft palate, where tremendous turbulence of airflow is generated in a galloping horse breathing at a rate of not less than two breaths per second.

Key: The black dot in the mouth indicates the position of the bit; GP = guttural pouch

1. **Recurrent Laryngeal Neuropathy (RLN):** This inherited disease of the voice box, throttle or larynx, also known as laryngeal paralysis, laryngeal hemiplegia, laryngeal hemiparesis, 'lazy flapper', 'roaring' and a number of other stable names, is extremely common. The degree of neuropathy varies from slight to complete paralysis but, once again, all Thoroughbreds have some degree of this disease. If severe enough, it causes - at exercise - a bottleneck in the airway at the level of the throttle (Fig 4).

2. **Soft Palate problems:** Owners, trainers, jockeys and veterinarians all know that horses 'flip their palates', 'swallow their tongues', or suffer dorsal displacement of the soft palate (Fig 3). Even the dreaded acronym (DDSP) is only too familiar. The problem causes horse to choke-up (asphyxiate). The cause of this problem has until now been a mystery but read on. In the absence of a known cause, the unsatisfactory 'prevention' has been a tongue-tie; an item that has almost become a standard piece of tack.

3. **Defects of conformation:** The dictates of fashion in the nineteenth century resulted in horses being bred with dainty muzzles, so that they could 'drink out of a pint pot'. Fortunately, the fashion no longer holds sway. Nevertheless, some Thoroughbreds are
still born with relatively narrow heads, narrow jaws, small nostrils, constrictions at the junction of nasal cavity and throat, narrow throats and throttles. Less appreciated is the fact that many are born with, or develop, deformed windpipes. A tall horse with any one of these defects will be at a greater disadvantage than a short horse, because of its logarithmically greater need for oxygen.

4. **The penalty of a rider:** The horse is the only high speed runner that is expected to perform with a weight on and a constricting band around its chest. The weight of a rider on the rib cage of the horse constitutes a handicap to inspiration. First, the added weight increases the demand for oxygen and probably pushes the respiratory system beyond the peak capacity that nature provides. Secondly, the weight must inevitably increase the actual work of breathing, as the horse has to expand its chest on inspiration, in opposition to an abnormal downward force. This must lead to the need for the horse to develop a greater suction force than normal, in order to inspire. Similarly, a girth hinders expansion of the chest on inspiration and will add to this problem. Expansion of the chest is also reduced because the weight of a rider interferes with an energy saving device known as the head bob, as explained in the next section. But before moving on, a recap of this section is appropriate. By requiring a horse to carry a person, we increase the horse’s demand for oxygen and at the same time decrease the availability of oxygen and increase its ‘price’. Man makes the horse work harder (spend more of its available energy) to obtain less oxygen than it needs. And from the total store of energy, the work of breathing will always take precedence over the work of galloping.

5. **Bit traction:** A horse that leans on the bit or has a jockey that hangs on its mouth, develops a rigidity of its neck that reduces the length of its stride (and therefore speed) and also interferes with the natural pendulum swing of its head and neck (the head bob, see Fig 5). The natural rise of the head, in a horse at liberty, facilitates inspiration as it raises the front end of the body and slides the abdominal contents towards the tail, flattening the diaphragm and saving on the energy needed to breathe in. Under conditions of domestication, bit traction and the riders weight combine to reduce the natural rise of the head and neck, making inspiration more difficult and more dependent on an abnormally high suction force on inspiration, the primary cause of bleeding. Another effect of bit traction is to cause a galloping horse to open its mouth, which again is abnormal and can lead to airway obstruction (Fig 10)

6. **The bit as a foreign body in the mouth:** The bit is a potent cause of asphyxia. This statement may shock many but it is so. The trouble is that the bit has been in use for 6000 years and we have grown so accustomed to this piece of Bronze Age technology that we have not asked ourselves what it really does to a horse or whether there is an acceptable alternative. It turns out that a bit in the mouth of a racehorse (or any other horse) is contraindicated, counterproductive, and potentially cruel. To place a bit in the mouth of a horse that is about to exercise, is equivalent to placing a muzzle on a horse that is about to eat. A bit is neither conducive to the welfare of the horse, nor productive with regard to the goals of the rider. It is also redundant and archaic. Fortunately, a more acceptable method of control has recently been introduced, which benefits both horse and rider.

To understand the contradictions of the bit method of control, it is necessary to understand something about the horse’s form and function (anatomy and physiology). Start by examining Figure 8. As soon as we place a bit in a horse’s mouth we are telling the horse to THINK EAT. But now we set the horse in motion and we tell the horse to THINK EXERCISE. We are expecting the horse to do something that nature never intended, to eat and exercise simultaneously. How would readers like to take part in competitive athletics with a bunch of keys in their mouth? A horse can graze or it can gallop but it should not be expected to do both at the same time. Eating requires digestive system reflexes and neurological relaxation; whereas exercise requires energetic musculoskeletal system responses, backed up with tremendous cardiovascular responses. Eating requires peace and quiet; whereas exercising requires fight and flight responses. Eating requires salivation, swallowing, and movement of the lips jaw and tongue;
whereas exercising requires a dry mouth, freedom from the need to swallow and a quiet tongue. These and other comparisons are tabulated in Figure 6.

The throat of a horse has to be capable of serving two functions, breathing or swallowing. But it can only do either of these one at a time. Figure 7 illustrates the switching that has to take place to carry out each function. As can be seen, elevation of the soft palate and so-called displacement is a normal part of swallowing. But it is grossly abnormal when it occurs during rapid breathing.

The presence of a bit confuses a horse neurologically. At any one time, its brain is never completely sure which function to carry out. For example the bit triggers tongue movement. Just as we do when we place a pencil between our teeth, the horse searches for (plays with) the bit with the tip of its tongue. This results in a retraction of the tip of the tongue. When the tip is retracted, the root of the tongue has to go somewhere and tends to hump up in the back of the throat. Because the soft palate lies on top of and in contact with the root of the tongue (Fig 9), movement of the root causes elevation of the soft palate and this, in turn, results in a narrowing of the nasopharyngeal airway. Elevation by itself is enough to cause a galloping horse to suffer partial asphyxiatiion and to become 'thick in its wind'. Not infrequently, movement of the tongue or an attempt to swallow leads to soft palate displacement and now follows an episode of 'choking-up' and transient suffocation. Tying down the tongue cannot be relied upon to prevent this sequence of events. As the tongue only moves because of the bit, which is also responsible for inappropriate salivation and the need to swallow, the logical 'treatment' is to remove the bit. And this can now be done, thanks to the recent introduction of a new design of bitless bridle that provides safer and more effective control than a bit, even for racehorses.

If anyone should doubt the turmoil in the throat caused by the bit, they have only to scope a horse, either at rest or on the treadmill, with a bit in its mouth. They will see that the slightest movement of the tongue triggers massive upheavals of the soft palate and an alarming degree of airway obstruction. We have overlooked this phenomenon in the past because it has been our habit to remove the bit before we scope a horse. Consciously, or unconsciously, we do this because we know that if we leave the bit in the mouth, movement of the soft palate prevents us from even seeing the larynx, let alone making an evaluation of its health.

A great deal more could be said about the advantages of dispensing with the bit. But, as I am the director of a company that distributes the bitless bridle, more will begin to sound like an advertisement. Instead, I will close by listing the practical steps that, in my opinion, a trainer might take to alleviate asphyxia and reduce the prevalence of bleeding and soft palate displacement.

Preventive measures

- Remove the bit, the tongue tie, and the dropped noseband and use the bitless bridle for training
- Demonstrate, for the benefit of the Stewards, the increased safety and welfare advantages of the bitless bridle and request their permission for its use in races
- Avoid excessive poll flexion (rating) during training exercise and races
- If a bit has to be used, encourage the use of a loose rein or, better still, hang a bit from the bitless bridle in such a way as to comply with current regulations yet without putting so much painful pressure on the mouth
- Encourage the head bob
- Make full use of the lightest exercise riders and jockeys, and those with the best hands
- For horses that are known to be severe bleeders, choose short distance races; avoid racing in intense heat and humidity; and provide generous intervals between races.
Lungs that have become edematous during exercise and irritated by the presence of free blood cells after exercise, need plenty of time to recover from the chronic pneumonitis which is a sequel to each episode of bleeding. The mucoid nasal discharge that is often seen for several days after racing is, I believe, a sign of this pneumonitis. Rest, fresh air and dust free stable management is indicated.

- Search for and treat any diseases of the upper airway that are treatable
- If you have the choice, select your horses with care, avoiding in particular, defects and diseases of the upper airway, and very tall horses
- If you do not have the choice, at least have your existing horses surveyed by your veterinarian for the problems mentioned above, in order that you can better manage their schedules
- Do what you can to avoid the need for an overtight girth. Consider the use of a breast harness.
- Forego Lasix medication but reduce bulk feeding prior to a race and limit access to water on the day of racing. Naturally, the same precautions are needed on those days when the horse is to be breezed.

CAPTIONS

Fig 1. Showing how the asphyxia theory of bleeding explains how and why the hemorrhages (black dots) are distributed along the top side and tail end of the lungs. If the upper airway is obstructed (in this particular example by poll flexion), an abnormally powerful vacuum develops in the airway on inspiration. For aerodynamic reasons, the vacuum gets more intense as the distance from the site of obstruction increases; hence the tail end distribution. As lung tissue is rather elastic and the lungs are suspended from the spine, they act in the manner of a slinky. The air sacs at the top of the lung are always more dilated than those at the bottom. Because they are more open, they are more affected by the abnormal vacuum pressure; hence the top side distribution.

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2B: Upper airway obstructed. Because of the high resistance to airflow, the diaphragm has had to work harder and make more effort to draw air into the lung and the vacuum pressure in the air sac is now abnormal (-3). Fluid begins to be sucked from the capillaries into the interstitial tissues of the lung (pulmonary edema) and this, in turn, causes the blood pressure to rise (+3). The transmural pressure is now 6, which is large enough to cause heavily blood-stained fluid to be sucked across the blood/air barrier from high to low pressure. Like the interstitial tissues, the air sacs also become water-logged (edematous) and normal gas exchange is prevented. The horse becomes short of oxygen (hypoxic).

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Fig 4. Two endoscopic views of a larynx in a galloping horse.

A: A healthy larynx that is fully open on both sides. The glottal cartilages ('flappers') are retracted into an almost horizontal position and the passageway for air to pass to and fro through the narrowest part of the larynx, the glottis (black area), is optimally wide. Resistance to the passage of air will be minimal and the suction pressure needed to accomplish inspiration will be moderate.

B: The larynx of a horse with one-sided paralysis (laryngeal hemiplegia). The left flapper has collapsed to the midline position and the glottis is half the area that it should be. The resistance to inspiratory airflow will have increased by a factor of four or more. Accordingly, the inspiratory suction pressure needed for the horse to get air needs to be similarly increased.

Fig 5. Showing the energy saving nature of the head bob in one complete stride of a horse galloping at liberty (no rider). The natural swing of the head and neck pendulum facilitates locomotion as well as respiration but we will focus here on the respiratory aspect only. As the fore legs take weight, the head falls and stretches the strong elastic ligament (the ligamentum nuchae) that runs from the back of the skull to the withers. The thoracolumbar spine tips forward and the guts press up against the diaphragm (open arrow). This mechanically assists expiration without the expenditure of muscular energy. As the hind legs take weight and the recoil of the elastic ligament raises the head, the thoracolumbar spine tips in the opposite direction and the guts now slop back towards the tail. This flattens the diaphragm and mechanically assists inspiration (open arrow). This 'visceral piston', as it is called, is interfered with as the result of a riders weight on the rib cage and by bit traction, both of which reduce the head bob and increases the work of breathing.

Fig 6. Tabulation of the different conditions that the throat has to meet in order to carry out each of its major functions, eating or exercising

Fig 7. Showing the switches which take place in the throat to enable a horse to either exercise or eat (breathe or swallow).

Key: BH = button hole (through which the larynx fits like a grommet when the horse is breathing); E = epiglottis; GC = glottal or arytenoid cartilages (the 'flappers'); NP = nasopharynx (throat); OP = oropharynx (throat); SP = soft palate; VB = voice box or throttle (larynx)

A: Breathing: The soft palate is lowered, the larynx open and buttoned into the soft palate, and the gullet closed
B: Swallowing: The soft palate is dorsally displaced (unbuttoned from the larynx), the gullet open and the larynx closed.

Fig 8: Showing the manner in which the tongue and larynx are joined together anatomically and are both suspended from the skull by the scaffolding of the hyoid bones. Because of this, when the tongue moves, the larynx also moves. If, because of the bit, the tongue moves during galloping, the larynx shifts about like a ship in a storm and this, in turn, interferes with the free passage of air from nose to lung.

Fig 9. Showing what happens when a horse 'swallows its tongue'. If the horse evade the bit and draws the tip of its tongue behind the bit, the root of the tongue bulges backward and upward, causing dorsal displacement of the palate (an unbuttoning of the larynx from the soft palate); a folding back of the epiglottis over the mouth of the larynx; a disastrous interference with the flow of air; and a transient episode of suffocation ('choking-up'). The broken line indicates where the root of the tongue should lie.

Fig 10. If traction on the bit causes a horse to open its mouth during galloping, air enters the mouth, from where it may easily rise into the oropharynx (throat). Once this happens, there now being air above and below the soft palate, the soft palate will blow about like a wet sheet in a gale and the horse will make a gurgling noise. Dorsal displacement of the soft palate may well follow.

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