15th January 2013

BITTED MOUTHS CAUSE WATERLOGGED &'BLEEDING' LUNGS:

Racehorses need management, not medication

Robert Cook[1] FRCVS, PhD

The causes of events are always more important than the events themselves

\- Cicero

ABSTRACT

Prompted by the USA debate over Salix medication, the author - who has been researching problems of the horse's head, neck and chest since 1958 - offers his opinion on the cause of 'bleeding' in Part I and gives his recommendations for prevention in Part II. The article is freely available to all stakeholders in the USA. As the prevention of 'bleeding' has nothing to do with Salix medication, the information is also relevant to racing worldwide and to all other disciplines.

'Bleeding' is only one sign of a larger problem, waterlogging (edema) of the lung; this, in turn, is caused by asphyxia. The most common cause of asphyxia is elevation and dorsal displacement of the soft palate and this, in turn, is caused by the bit. By breaking the lip seal, a bit dissipates what should be a negative pressure in the oral cavity and oral part of the throat. As a result, the soft palate in a running horse no longer clings to the root of the tongue. A bit is also contraindicated on other physiological grounds, as it generates 'eating' responses which are in conflict with the 'exercising' responses required for running. Finally, the bit is contra-indicated on account of its negative impact on the welfare and safety of both horse and rider. Through fear and pain, the bit triggers a monstrous number of unwanted behaviors and, in addition to pulmonary edema, causes 40 or more diseases. The end result is reduced performance. A bitted racehorse is a handicapped racehorse.

In happy contrast, the crossunder bitless bridle provides better communication and is painless, safer, and compatible with the physiology of exercise. It has no side-effects, causes no disease and does not interfere with performance. It is recommended that racing administrators update the rules of racing to allow this more humane option, which offers a major advance in the welfare of horse and rider. It is predicted that such a step will significantly reduce the prevalence of 'bleeding' and catastrophic accidents and do much for the image of racing worldwide.

[1] Professor of Surgery Emeritus, Cummings School of Veterinary Medicine, Tufts University, Grafton, MA
Chairman, BitlessBridle Inc.,
T: (443) 282 0472 E:drcook@bitlessbridle.com W: www.bitlessbridle.com

PART I: THE PROBLEM

INTRODUCTION

Since 1970, much has been written about the diagnosis and prevalence of 'bleeding' in the racehorse.[2] On both these aspects of the problem there is a wide measure of agreement among researchers. By comparison, little progress has been made on the question of cause. There is no consensus among researchers and most equine practitioners reluctantly accept that the jury is still out. Yet no disease can be treated until its cause is known. 'Bleeding' is a symptom, not a disease. Salix medication aims to 'treat' a symptom, which is the wrong approach. A weed cannot be eliminated by pruning; it must be dug up by the root. Race day Salix medication is an example of ineffectual pruning. The USA is the only country that permits it.

To be precise, Salix medication is a misguided effort at prevention, not treatment. In my opinion, the basic pathology of the disease loosely referred to as 'bleeding' is pulmonary edema caused by obstruction of the upper airway (asphyxia), i.e., obstruction at any point between the nostril and the entrance to the chest (Fig.1) (5, 6, 7, 13). Another name for pulmonary edema is waterlogging of the lung. Horses either die from it or recover spontaneously. In non-fatal cases there is no need for treatment. Most episodes, serious though they are during a race, are only temporary. The edema quickly disperses when the horse stops running.[3] Similarly, unless postmortem examinations are carried out within minutes of a sudden death, evidence of pulmonary edema will have disappeared or been masked by massive pulmonary congestion.(5) The word 'edema' may not even be mentioned in the pathologist's report.

The ephemeral nature of pulmonary edema may explain why the name of this potentially fatal disease has failed to be registered in our collective thinking about the problem. Instead, attention has been focused on the supposed 'bleeding.' When a problem is called exercise-induced pulmonary hemorrhage (EIPH) and defined as 'the presence of red blood cells (or their products) in the respiratory tract after exercise,' this only names one sign of pulmonary edema (16). EIPH was a purely descriptive name given in the 1970s when the problem's high prevalence

[2] For convenience, the text refers to the Thoroughbred racehorse and flat racing but all that is written is also relevant to the harness horse. In fact, because of the harness horse's multiple bits and the enormous pressure that a driver can place on them, soft palate problems in the harness horse are even more common than in flat racing.

[3] Repeated episodes of pulmonary edema will, nevertheless, lead eventually to the establishment of a low grade inflammation of the lungs which may require treatment. This condition is variously referred to as inflammatory airway disease or small airway disease.

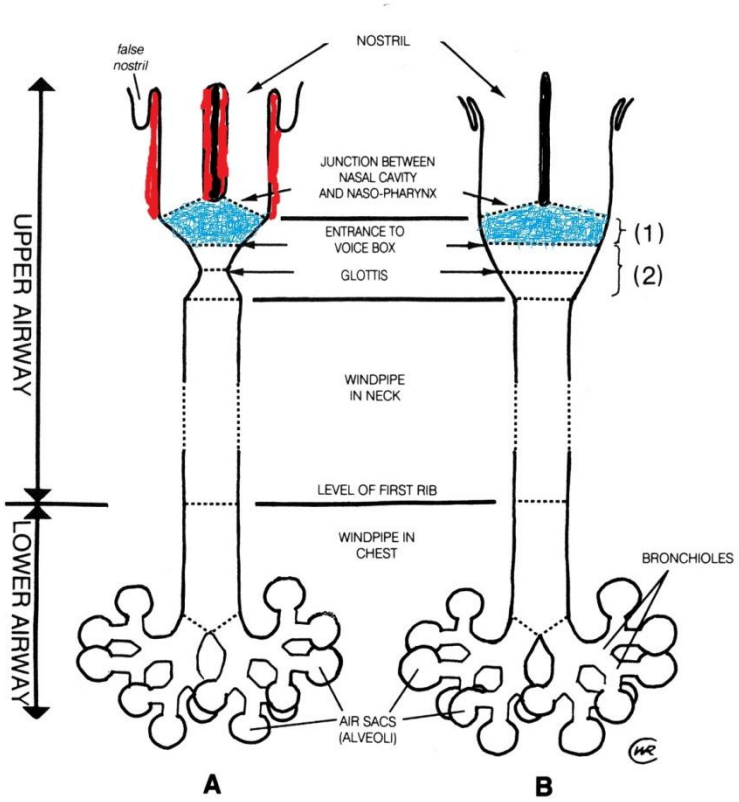

Fig.1. Bird's eye view of the respiratory tract of the horse at rest (A) and at exercise (B) showing its basic anatomy and the way the conformation of the nostrils, throat and voice box changes when a feral horse runs in order to permit unobstructed airflow. The nasopharynx or nasal part of the throat (blue) is most vulnerable to obstruction in the domestic horse because of the presence of the bit. Note how the flared nostril renders the false nostril a potential space only and funnels air into the nasal cavity. The red areas represent erectile tissue in the resting horse that shrinks at exercise to enlarge the airway.

was first recognized. At that time, little thought had been given to its cause and so a cause-based name was not possible. Unfortunately, the descriptive name persists and now acts as a barrier to understanding.

'Bleeding' is only one sign of pulmonary edema and a relatively late-developing sign at that. By 'late' in this context, I mean a few seconds later than the onset of asphyxia and the start of waterlogging. In the absence of oxygen, mammals die very quickly. From a complete obstruction of its airway, a galloping horse - breathing at the rate of two breaths a second - would probably die in less than 10 seconds (20 breaths). In the same time period, a partial obstruction could be enough to cause a horse to stumble, fall and break a leg. A horse that was alive and well in the starting gate can be down and dead before racing 300 yards. Pulmonary edema, when severe, kills quickly. Fortunately, though catastrophic accidents occur, most episodes of pulmonary edema are not fatal. Well over 90% of racehorses are

only partially asphyxiated. They develop some degree of pulmonary edema ('bleed') in every race yet live to run another day.

Pathological changes, devastating to a horse's ability to breathe (and run), have occurred in the lung well before blood-tinged edema fluid begins to fill the air sacs and long before this is seen at the nostrils. The early evidence of pulmonary edema in a racehorse is expressed by signs of poor performance, premature fatigue and reduced speed. This common triad of symptoms, which may or may not be recognized, stems from the horse having difficulty in breathing. Extrapolating from the symptoms of pulmonary edema in man, a horse may also be experiencing chest pains and the frightening sensation of drowning. As the small air sacs of its lung get flooded with edema fluid, oxygen exchange is impaired. Later signs include falls, breakdowns, fractures and sudden death (12, 17). The appearance of blood at the nostrils is only the tip of the iceberg. Based on endoscopic evidence of blood in the respiratory tract after racing, we know that virtually every racehorse develops pulmonary edema. Blood biochemistry confirms that racehorses develop hypoxia (a shortage of oxygen). If this evidence does not lead us to acknowledge why this occurs in the sport of racing and act accordingly we fail in our humanitarian duties.

All researchers agree that the disease results in red blood cells moving from the blood vessels of the lung into its interstitial tissue and air sacs. Strictly speaking, the fluid that fills the air sacs is not pure blood so it is not a true hemorrhage. It is edema fluid, heavily tinged with red blood cells. The term 'capillary stress' is used to describe the phenomenon but does not explain what causes the stress. Use of the wrong word can lead us astray in our thinking. To get a grasp on the problem of 'bleeding' we must use the right words. For the rest of the article, therefore, I will avoid using the term 'bleeding' and refer instead to pulmonary edema. It's a bit of a mouthful but it will remind us of the true nature of a disease caused by the horse's mouth being full of a bit.

Two major schools of thought exist on cause (9, 10, 11, and 18). One school avers that pulmonary edema is caused by too much positive pressure in the blood vessels. Let's call this school the 'pushers.' The other school favors the idea that it is caused by too much negative pressure in the air sacs. We'll call this school, which is the one to which I belong, the 'suckers.' In simple terms, it boils down to a debate between too much 'push' or too much 'suck.' The 'pushers' cite abnormally high blood pressure in the Thoroughbred as the cause and regard this as an attribute (even a virtue) of the elite racehorse. Some members of this school even accept that 'bleeding' is 'normal' for the Thoroughbred and, therefore, unavoidable. This overlooks the fact that plough horses used to bleed (develop pulmonary edema), that dressage horses still do and that a horse of any breed which gets cast in the stable can die of pulmonary edema without ever having been exercised. The

‘suckers’ cite asphyxia as the cause of pulmonary edema, i.e., the abnormally high negative air pressure that a running horse develops in its lungs during attempts to breathe-in against an airway obstruction. The suck hypothesis, unlike the push hypothesis, is consistent with the known facts about the disease. It also leads to an achievable solution. AVOID ASPHYXIATING A HORSE. Athletes need all the air they can get and we should ensure that this vital yet least expensive fuel is readily available at all times, not just for racehorses but for all horses.

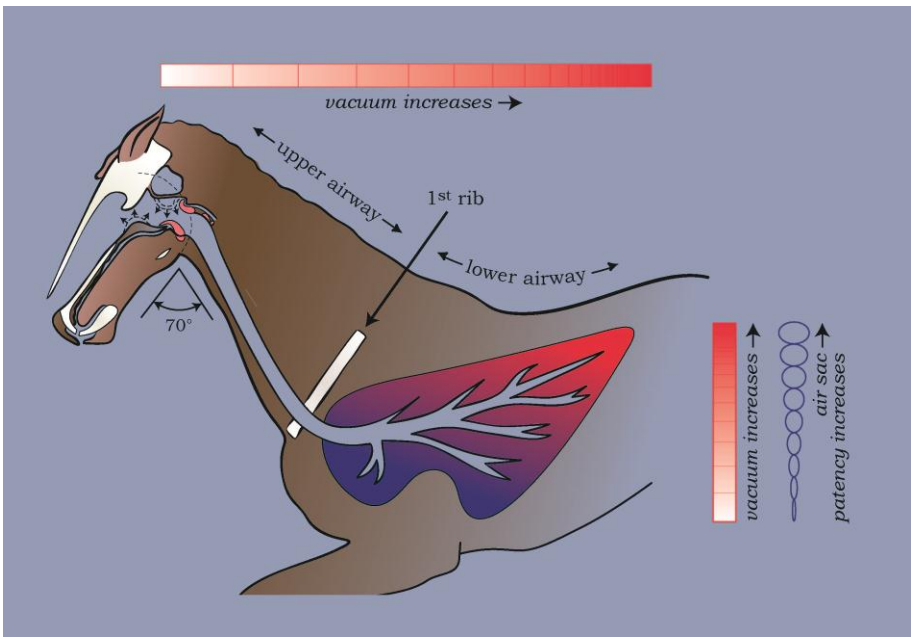

Fig. 2: Showing how, in this case, poll flexion causes an obstruction of the throat. It explains why obstruction of any sort causes the ensuing pulmonary edema (red areas) to occur along the topside and tail-end of the lung.

One thing is certain, Salix medication on race-day is not the answer as it fails to remove the cause. In the 1970s, Dr. Alex Harthill, an equine practitioner in Kentucky, made a perceptive guess at the nature of the problem when he likened it to pulmonary edema in man. On this basis, he applied as a possible preventive in the horse something that was used as a treatment for the same problem in man, i.e. furosemide, a diuretic. His ‘treatment’ became popular with trainers, not because it prevented bleeding (it didn’t) but because it seemed to improve performance, perhaps by reducing body weight. But it does not follow that something which *might* alleviate a symptom, once it appears, will prevent the symptom occurring in the first place. In the horse, Salix is not a ‘vaccine’ for bleeding. There was also another gap in the logic. If pulmonary edema was the basic pathology of bleeding, the question still needed to be asked. ‘What causes the pulmonary edema?’

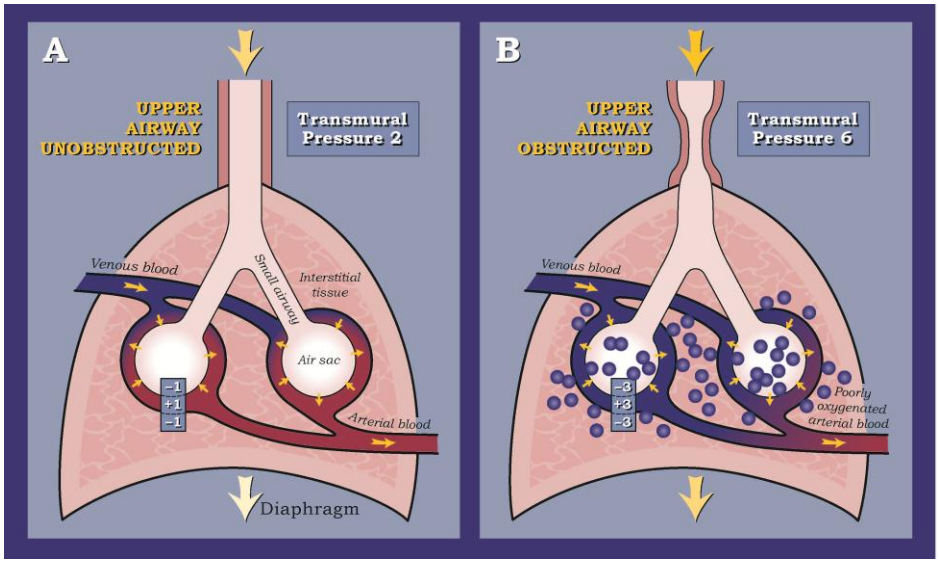

Fig. 3: Showing why pulmonary edema occurs when the upper airway is obstructed. Each diagram shows a pair of lungs represented by one symbolic air sac. Each air sac is surrounded by its capillary blood vessels and supported by a matrix of interstitial tissue.

A: Airway unobstructed. The flattening diaphragm sucks air into the lung with minimal resistance to airflow and the negative pressure of inspiration in the air sac and the interstitial tissue is represented by the symbolic figure of minus 1. Blood pressure in the capillaries is also relatively low at plus 1. The pressure gradient between blood vessel and air sac is small - say 2, an imaginary figure for the purpose of the example. This is enough to allow normal gas exchange to take place, i.e., for oxygen to pass from air to blood and carbon dioxide to pass in the opposite direction, but not enough to cause leakage of fluid and red blood cells.

B. Airway obstructed. The pressure gradient at the air/blood barrier is high – say 6. Red blood cells and plasma are sucked out of the capillaries into the interstitial tissue and the air sacs. The lung becomes waterlogged (edematous), the work of breathing is hugely increased, gas exchange is impeded, venous flow of blood to the lungs increases, the heart is put under strain, and performance is reduced.

Nearly 40 years ago, I hypothesized that what was then referred to as epistaxis or 'broken blood vessels' was originating from the lungs, not the nose (2). I was proved right about the location of the bleeding, which was the focus of the article. But, as an aside in the same article, I surmised that bleeding may be a sequel to some primary disease of the lung. Many colleagues followed me down this same path and some still support the idea but it was the wrong path. In the face of new evidence I subsequently changed my thinking (5). At each stage, the anatomical location for my explanation of cause has shifted back from the lungs, first to the voice box and then the throat. Finally, I have come to recognize that the real culprit is not even in the respiratory tract but lies at the very start of the digestive tract. The cause has been hiding in plain sight all along; it's the bit (19, 20, 21, 28, 30, 33, 52, and 57).

For many years I have been of the opinion that the root cause of pulmonary edema was any upper airway obstruction (5). Initially, I surmised that the most common cause of this asphyxia was recurrent laryngeal neuropathy (laryngeal paralysis or paresis). I still believe that this is a cause but now recognize that the bit is the major cause.[4] From early days, I was also interested in the phenomenon of dorsal displacement of the soft palate but was unable explain its cause. (1, 4, 19, 28, 30, 35, 57) Since 1998, I have come to recognize that the bit is also the major cause of instability, elevation and dorsal displacement of the soft palate and that these are all different ways in which an obstruction in the throat can come about, with or without bit-induced flexion at the poll (33).

Bit-induced poll flexion is easy enough to understand as a cause of asphyxia. Racehorses are rated and prevented from extending their head and neck as a running horse was evolved to do (Fig. 7). No human athlete has to suffer such a counter-productive handicap. But the reader may ask, 'How can the bit cause soft palate problems?' Retraction of the tip of the tongue to avoid the bit and poll flexion provides a partial answer only. It was not until last year that the complete answer suddenly dawned on me. Following-up on a hunch, a literature search on the physiology of swallowing in mammals confirmed my suspicion that, at times other than when eating, the oral cavity of the feral horse is under negative pressure. It is this vacuum, I believe, that prevents the long soft palate of the horse from floating up at exercise and blocking the airway in the throat (Fig. 6). A bit breaks the lip seal, permits air to enter the oral cavity and the digestive part of the throat and this, in turn causes instability, elevation and dorsal displacement of the soft palate (Cook 2012 Unpublished material).

Something else has been overlooked. Dorsal displacement of the soft palate (DDSP) is a normal and necessary feature of the swallowing reflex (Fig. 4). If it failed to occur when eating and drinking, food and water would reflux down the nose and/or invade the lungs and the horse would die of inhalation pneumonia. The presence of one or more metal rods in a horse's mouth stimulates inappropriate digestive system reflexes that should only occur in a relaxed, peaceful, eating-or drinking horse, i.e., tongue movement, salivation, chewing, DDSP and swallowing. Such reflexes are contraindicated and counter-productive in any ridden or driven horse. They are anathema to a racehorse. Athletic performance requires the diametrically opposed set of 'flight-and-fight reflexes, i.e., stimulation of respiratory, cardiovascular and musculo-skeletal system reflexes. So quite apart from the pain that a metal foreign body in the mouth causes when pressure is applied (itself hostile

[4] This is not the place to explain why I think it possible that the bit might one day be indicted as being partly responsible for causing recurrent laryngeal neuropathy but a footnote to this effect is appropriate.

to maximum performance), a bit generates the wrong set of responses in the throat. The throat of any mammal is a switch plate mechanism that has to be capable of serving two functions, rapid breathing or swallowing (Fig. 5). But it cannot do both at the same time. Every parent warns a child not to eat while running.

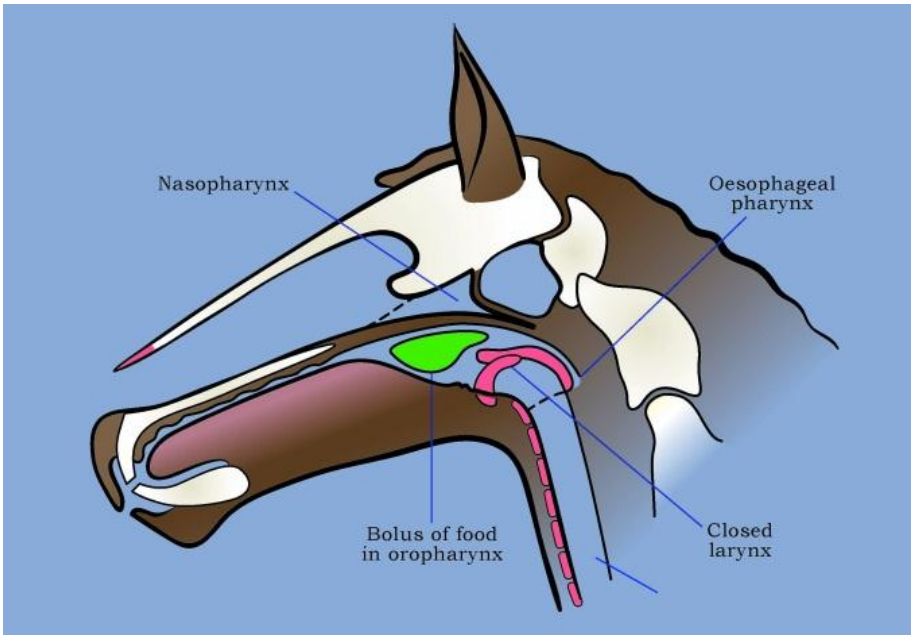

Fig. 4: Showing the changes in the throat when a horse swallows dry food or water. Dorsal displacement of the soft palate (DDSP) is a normal part of swallowing and only abnormal if it occurs during rapid breathing.

Because it incorporates a causal mechanism, negative pressure pulmonary edema (NPPE) is a better name for 'bleeding' than EIPH (13). The disease in the horse is analogous to NPPE in man. An Internet search for NPPE will demonstrate the similarities and further explain the pathophysiological mechanisms involved.

Just like skin but infinitely more delicate, the lining membrane of the lungs' air sacs are exposed to the atmosphere. The 'skin' of the horse's lung becomes bruised for the same reason that human skin bruises with the suck of a hickey. Asphyxia increases the force of suction pressure applied to the 'skin' of the lung when a horse breathes-in. But whereas a hickey is not life-threatening, a waterlogged lung can be. To bit a racehorse is akin to waterboarding a terrorist. A toxic combination of asphyxia, pain and fear can lead to fatigue, falls, fractures and fatalities. A bitted horse that is healthy in the starting gate can be 'drown'd dead' before it reaches the quarter pole. A jockey, ejected from the saddle to meet a hail of metal-tipped hooves, shares a similar risk of sudden death.

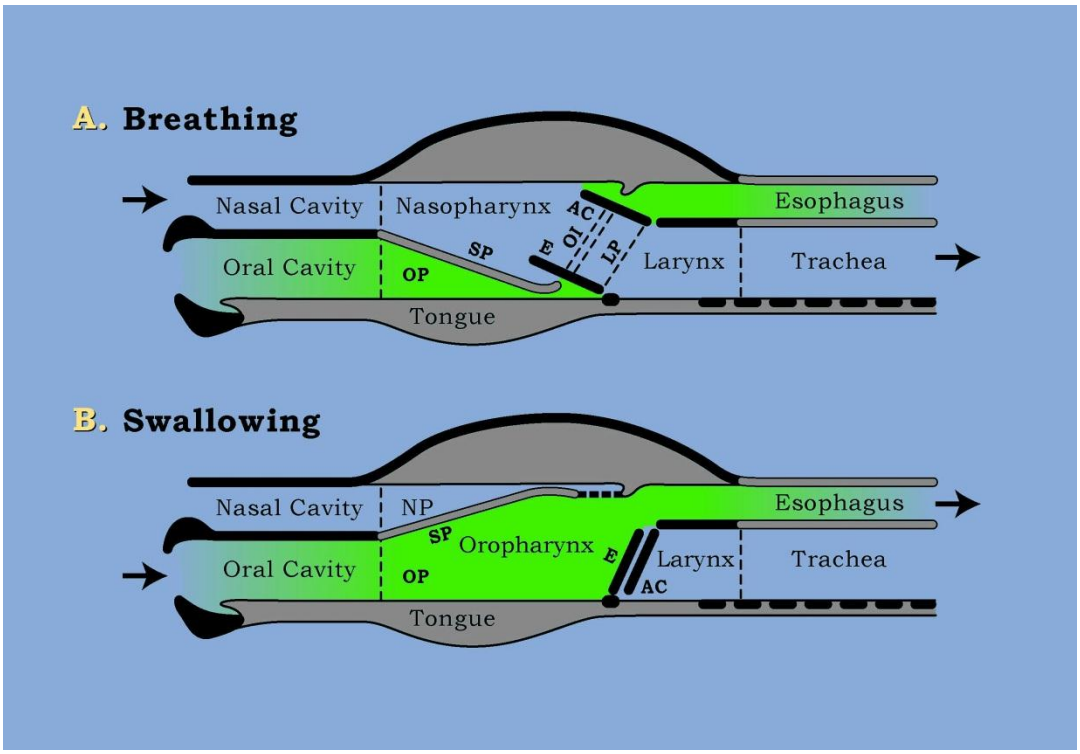

Fig. 5: *Showing the manner in which the soft palate and the cartilages of the voice box act as switch plates to enable the throat to serve two functions, at different times, i.e., rapid breathing (A) or swallowing (B). When running, the soft palate lies in contact with the root of the tongue, as in Fig.6b. For the purpose of this diagram, air is shown in the oral cavity but perhaps this liberty can be excused on the grounds of simplifying this complex dual function. Lowering of the soft palate enlarges the nasal part of the throat for breathing and elevation enlarges the oral part of the throat for swallowing. Similarly the 'flapper' cartilages when raised, open the voice box and close the entrance to the gullet. When dropped, they open the gullet and close the voice box. When swallowing, the soft palate is elevated and comes unbuttoned from the voice box, forming a seal against the roof of the throat to prevent reflux of food or water into the nasal cavity. The oral part of the throat is now expanded at the expense of the nasal part of the throat. The 'flapper cartilages fold down to close the entrance to the windpipe and prevent food or water from inundating the lungs. Finally, the epiglottis swings back to 'double-lock' the voice box. For rapid breathing, the soft palate must be in the lowered position and clinging tightly to the root of an <u>immobile</u> tongue.*

Key: AC – arytenoid cartilages (the 'flappers'); E = epiglottis; LP = laryngopharynx (a food channel permitting simultaneous grazing and quiet breathing); NP = nasopharynx (nasal part of throat); OI = ostium intrapharyngium (a 'button-hole in the soft palate, into which the voice box should firmly fit at all times except during swallowing and coughing); OP = oropharynx (oral part of throat); SP = soft palate. Black = bone or cartilage. Brown = soft tissue. Note how the least supported section of the airway is the throat. It has to be mobile to carry out its dual function, so relies on mechanisms other than walls of bone or cartilage to maintain its rigidity during rapid breathing, e.g., the oral vacuum and longitudinal stretching by poll extension.

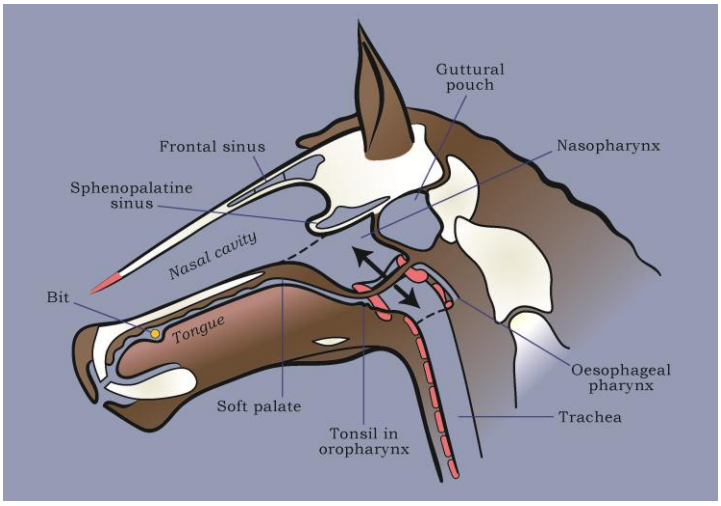

Fig. 6a: Showing the basic anatomy of the upper airway in a bitted horse at rest. Note that the lips are not 'set' and, even though the mouth is not gaping, some air is present in the mouth, the digestive part of the throat (oropharynx) and, perhaps, the first section of the gullet (oesophageal pharynx). Air will also be present in the voice box part of the throat (laryngo-pharynx) though cannot be illustrated in this diagram (see Fig. 5). The double-ended arrow indicates airflow between the nasal part of the throat (nasopharynx) and the voice box. The front cartilages of the voice box (the epiglottis and the 'flapper' cartilages) serve in the manner of a grommet to engage the voice box in the soft palate 'button-hole.'

Key: White = bone; pink = cartilage

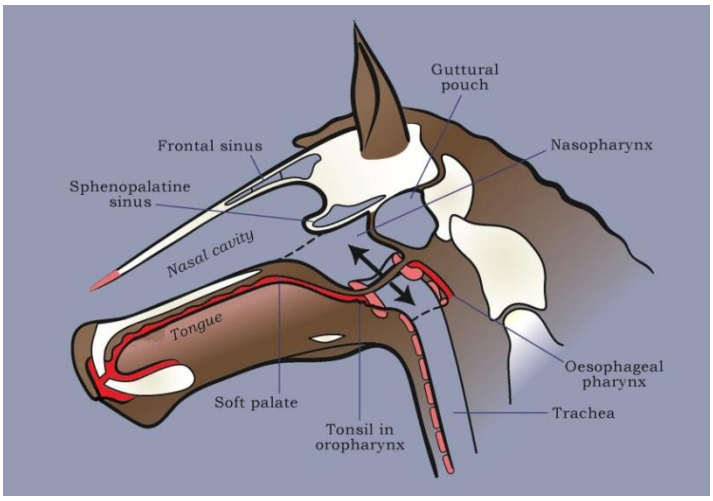

Fig 6b: *Diagram as above but without a bit in place. The lips are 'set' (closed). A vacuum is present in the red regions and will also be present in the laryngo-pharynx. The vacuum adds stability to the respiratory part of the throat (nasopharynx) in a galloping horse taking two breaths every second. For the purpose of illustration, the red area is shown as an actual space but, of course, a vacuum obliterates the space in practice and 'glues' the soft palate to the root of the tongue. A vacuum in the laryngopharynx will also render more secure and airtight, the seal between the soft palate 'button-hole' and the voice box, preventing a rush of air on expiration from escaping into the oral part of the throat (oropharynx).*

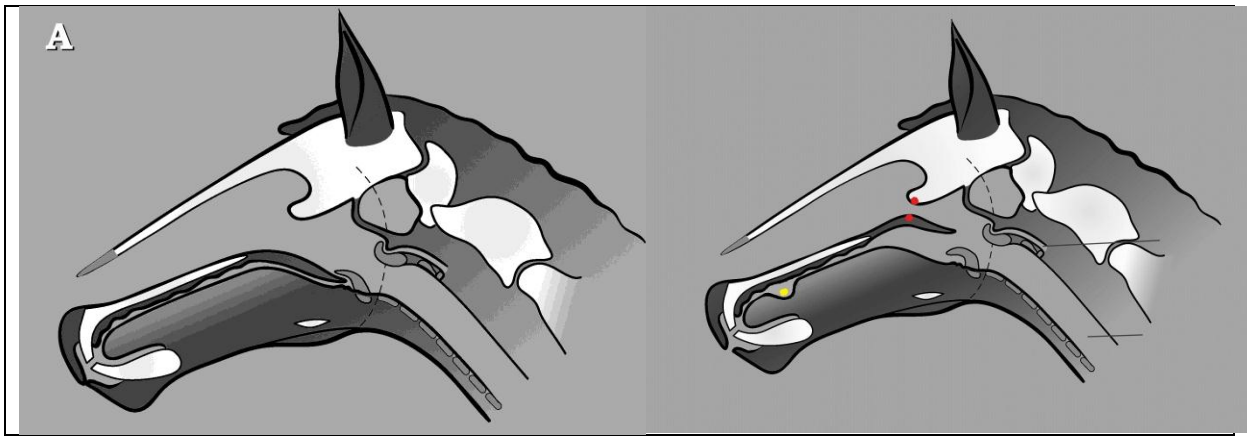

Fig. 7: The diagram on the left shows a bit-free horse with 'set' lips and, let us suppose, an intact oral vacuum. The poll is well extended and the throat is not obstructed. On the right, the lips are parted because of the bit and the oral vacuum has been dissipated. The soft palate is dorsally displaced and the throat airway is severely obstructed at the point marked by the red spots. The horizontally disposed apertures that connect the nasal cavity with the nasal part of the throat (the two posterior 'nostrils' or nares) have been almost completely blocked by the elevated soft palate acting like a flapper valve. With this degree of asphyxia, a galloping horse would only have to try and breathe in twice (something that takes one second) before its lungs would be seriously damaged by massive barometric bruising (barotrauma). Acute pulmonary edema would develop in both lungs.The laws of aerodynamics are such that a 50% reduction in the diameter of a tube increases the resistance to flow by a factor of 16. In this example, the degree of reduction in the diameter of the posterior nostrils is closer to 75%.

References and further reading are appended to Part II

PART II: THE SOLUTION

Bit-induced loss of the oral vacuum is now proposed as a unifying hypothesis for the cause of NPPE ('bleeding') in the horse and many other all too familiar problems in the racehorse that, until now, have been considered to be of unknown cause. These include instability, elevation and dorsal displacement of the soft palate, epiglottal entrapment (33, 48) and dynamic collapse of the walls of the respiratory tract during inspiration. A collapse or 'sucking-in' of the airway tube can occur at a number of different places along the tube, wherever its wall is unsupported by bone or cartilage. Many a horse will experience collapse at more than one section. Caving-in can occur at the junction of nasal cavity and throat (the posterior nostrils), in the throat itself, at the entrance to the voice box, and throughout the windpipe. All of these different obstructions can be attributed, predominantly, to the bit.

The laws of aerodynamics relating to gas flow along tubes explain that negative pressure in the lung of a horse on inspiration will increase as distance from the obstruction increases (5, 14). Consistent with this law, any asphyxia causes the most severe edema at the tail end of a horse's lung and explains its symmetrical distribution in both lungs. In bulleted form, the evidence on which future action over medication might be taken in the U.S.A. and over the rules of racing in all countries is as follows:

- Upper airway obstruction (asphyxia) is the cause of pulmonary edema
- The problem is akin to negative pressure pulmonary edema in man.
- The bit causes asphyxia by breaking the lip seal and eliminating what should be a vacuum in the horse's mouth
- Elevation &/or dorsal displacement of the soft palate in the throat is the primary anatomical location of the asphyxia.
- Other bit-induced causes of asphyxia include poll flexion, tongue and jaw movement
- Obstruction in the throat leads to a cascade of further airway collapse in the voice box and windpipe
- The abnormally high negative airway pressure generated by asphyxia during inspiration will be greatest at the tail-end of the lung, hence the distribution of the edema.

- Left-sided recurrent laryngeal neuropathy ('roaring' from a partially paralyzed 'flapper') is a less common cause of asphyxia but by no means rare.[5]
- 'Bleeding' is a sign of asphyxia
- Salix prevents neither asphyxia nor pulmonary edema.
- The bit does not control the horse. On the contrary, it hurts and frightens horses and is the most common cause of loss of control, together with hundreds of other unwanted behaviors and another 40 or more diseases (24, 46, 47, 52)
- The crossunder bitless bridle is painless and provides a safer, more humane, and more effective rein-aid with no side-effects (33). It enables a rider to communicate without triggering soft palate problems and other cascade-effect causes of asphyxia. It does not generate abnormal negative pressure in the lung and will not cause pulmonary edema.

A plan for resolving the race-day Salix issue in the U.S.A., for providing a major advance in the welfare and safety of horses and jockeys worldwide, and for improving the public image of racing could be introduced in either two or three stages, depending on the country:

1. Medication
2. Education
3. Administration

Stage 1: MEDICATION ISSUES (USA racing):

As Salix does not remove the cause of pulmonary edema, its use on race-days cannot be justified. Salix medication should be banned with all speed. Such a step will:

- Improve U.S. racing's public relations
- Restore the confidence of the gambling public in U.S. racing
- Restore the confidence of overseas buyers in USA-raced bloodstock.

A crisis is dangerous but also an opportunity.

[5] Horses with a complete paralysis of the left-side of the voice box (laryngeal hemiplegia) will probably have been withdrawn from training before they reach the racetrack

Stage 2: EDUCATION (Worldwide racing):

This stage will take a little time, as all stakeholders will need to become familiar with the pressing need for an update in equitation management, i.e., the introduction of a change in the rules of racing to allow use of the crossunder bitless bridle. It is not suggested that the bit should be banned (even though such a reform can be justified on welfare and safety grounds) as this step would be fiercely resisted and would endlessly delay the introduction of a less controversial bitless option. Once a bitless option is offered, the bit will fall out of favor and fade away in the passage of time, as bitless horses will simply perform better. At some stage in the distant future, it will be possible for administrators, without raising a hue and cry, to quietly strike the Bronze Age bit from the rules.

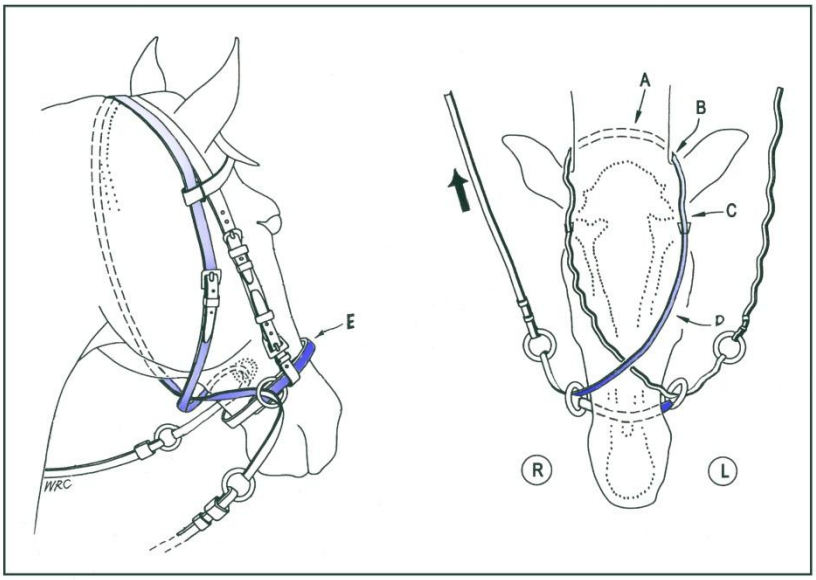

Fig. 8: Two diagrams showing the crossunder bitless bridle. The one on the right is a worm's eye view. The bridle has a figure-of-eight configuration with two loops, one across the bridge of the nose and the other over the poll. Pressure and release on one rein (thick arrow) provides a painless rein-aid for steering, nudging the opposite side of the head. Pressure and release on both reins signals for slow and stop by hugging the whole of the head. Strap pressure on skin (as opposed to steel on bone) is well distributed over a large surface area and is painless. Such as it is, the greatest pressure occurs over the bridge of the nose, with less pressure under the chin, even less up the cheek and least pressure at the poll, as indicated by the attenuation of the blue shading.

Fig 9: Correct fitting of the crossunder bitless bridle.

Part of the education process will have a practical component. Trainers should be encouraged to use the crossunder bitless (CB) bridle for training purposes on at least some days of the week. This will allow exercise riders and jockeys to gain confidence in the new approach and demonstrate to their own satisfaction, the many benefits that accrue for both horse and rider. Until the rules of racing are updated, a bit will still have to be used on race-days but trainers will recognize that some of the benefits of bitless training will carry over to racing. For example, horses will have a better attitude to racing; their mouths will be less sore; and their lungs less bruised.

In 2005, a limited trial for this purpose was conducted by nine trainers in the U.K. A copy of the feedback I received and sent to the Veterinary Committee of the British Horseracing Authority is available online (36, 37, and 51)

Owners and trainers should lobby racing administrators to draw their attention to the disadvantages of the bit and the advantages of a safer and more effective rein-aid. To train an athlete, the last thing you want to do is to be economical with the oxygen. Trainers can point out that it makes no sense to deprive a racehorse of something for which a racehorse has such a huge need. Mandating a piece of equipment that puts a crimp on a horse's supply of oxygen could render racing organizations liable for catastrophic accidents.

Stewards and administrators of racing should be encouraged to witness bitless training gallops and recognize the welfare and safety benefits for horses and riders.

When a sufficient number of trainers have trained their horses bitless, administrators of racetracks could write races for bitless horses and monitor the results. Controlled experiments to test the validity of the CB bridle under racing conditions can never be staged but these 'natural experiments' would serve as tests of concept.

Racing organizations could fund researchers to independently repeat and explore the documented condemnation of the bit that I have published over the last 15 years. Researchers could, for example:

a) Carry out the tests I have described in my currently unpublished article submitted to the peer-reviewed journal Equine Veterinary Education. I would be glad to make this article available for the purpose. The radiographic and endoscopic tests are designed as ways in which my hypothesis on the cause of pulmonary edema (loss of the oral vacuum) could conceivably be refuted. If the tests refute my hypothesis, it is 'back to the drawing board' and we start again on a different search for the cause of pulmonary edema.[6] But failure to refute would justify acceptance of the hypothesis at the present state of knowledge and provide a basis for action, i.e., a rule update to permit the CB bridle.

b) Repeat the same or similar experiments to the one I carried out in 2008 in which I compared the performance of the same four horses, first when bitted and then – a few minutes later - when ridden for the very first time in the CB bridle (55). Independently scored by a dressage judge, the average score was 37 bitted and 64 bitless (a 75% improvement in the first 4 minutes of being bitless)

c) Take a group of horses and give them a standardized gallop test, with and without bits, using overground endoscopy to observe what differences occur in the incidence and severity of soft palate instability, elevation and displacement.

Stage 3: ADMINISTRATIVE UPDATE OF THE RULES OF RACING:

The long-entrenched belief that the bit controls a horse is a myth. In reality, the bit is the most common cause of complete loss of control. Reasons for permitting the CB bridle as an option to the bit can be summarized as follows:

- Elimination of pain, respiratory/digestive conflict, mental stress and fear
- A major reduction in soft palate problems
- Fewer accidents (stumbling, falling, rearing, bucking, bolting, etc.,)[7]
- Performance improvement

[6] It is worth noting that refutation of the oral vacuum hypothesis would not refute the body of evidence that indicts the bit on other physiological, welfare and safety grounds.

[7] In 15 years of field testing, on horses of all ages and temperaments, ridden by riders of all ages and experience from children upwards, and under a wide variety of conditions worldwide, not one accident has been reported to me as being caused by the CB bridle

- Economic benefits: Fewer lost days on account of sore mouths, bridle lameness, sore shins, unsatisfactory and delayed response to training; reduced wastage.
- Bringing the rules of racing into line with current evidence on welfare and safety

The hope is that in order to reduce the prevalence of pulmonary edema and catastrophic accidents, racing's administrators worldwide will, one by one, be persuaded by the evidence to allow bitless racing. It will take only one administration to lead the way and claim the distinction of being the first to introduce a rule update to significantly advance the welfare and safety of horse and rider. Whichever administration is far-sighted enough to do this will also have the opportunity to monitor its 'natural experiment' and publish results.

In this article, I have focused on the evidence for change derived from the problem of bit-induced pulmonary edema. Since 1988, I have promulgated the hypothesis that asphyxia is the cause of pulmonary edema (5). Since 1998, with the benefit of additional evidence, I have been more specific and made the claim that the primary airway obstruction occurs in the throat, is associated with soft palate elevation, and is caused predominantly by the bit destroying the oral vacuum at exercise. Significantly, in the last 25 years – though there has been plenty of time - neither the original hypothesis nor its more specific version has been refuted. On the other hand, I have published my refutations of competing hypotheses and these have not been disputed by their advocates. By the rules of the game of science, a hypothesis that is refutable (i.e., is a proper scientific statement) yet – after a reasonable period of time - has not been refuted, deserves to be accepted 'at the present state of knowledge' and regarded as a legitimate platform for action. If administrators decline to act, they could be required to show on what grounds they can defend a rule of racing that mandates the exclusive use of a bit, a device that is often in clear breach of their own state, federal or country's animal welfare guidelines and laws.

In the last 15 years, field-testing of the CB bridle in non-racing disciplines has already condemned the bit on many counts. So even if the evidence on the cause of pulmonary edema in racing was not considered sufficient grounds for action, there is still an abundance of compelling evidence that the bit is unnecessary, unsafe, inhumane and carries an immense burden of dangerous side-effects. Field-testing in non-racing disciplines has shown the CB bridle, compared to the bit, to be a safer, more effective rein-aid with no side-effects. Quite apart from the role that the bit plays in the cause of pulmonary edema (predominantly in racing but not exclusively), it is also irrefutably known to cause hundreds of behavioral problems and at least

forty other diseases (45, 47, 52). This is not the place to list these 'non-bleeding' side-effects of the bit, but as sore mouths are such a common problem in racehorses, it is appropriate to mention just one (38, 39). The ages 2 to 4 are the critical years for a flat racehorse's career, coinciding with the period during which their permanent dentition are erupting. This is reason enough to avoid compounding a horse's discomfort with a metal rod that lies, for example, over the long root of the erupting canine in a stallion. If unwanted welfare-and-safety hazards, are not enough to persuade racing's administrators to allow a more effective, safer and humane rein-aid, it can be added that the bit is a notorious 'spoiler' of performance and hugely escalates the expense of training.

The evidence in support of allowing the CB bridle for racing is overwhelming, whether this is derived from the occurrence of pulmonary edema or from other diseases and unwanted behaviors. If the CB bridle was allowed in racing, there will be an immediate improvement in the horse's quality of life and rider's welfare. I predict too that a significant reduction in the prevalence of pulmonary edema and catastrophic accidents will be recognized in the passage of time.

SUMMARY OPINION:

Race-day Salix should be banned. However, this will do nothing to prevent pulmonary edema and the prevalence of catastrophic accidents will not be reduced. Prevention of any disease requires removal of its cause. The bit is the primary cause of asphyxia and this in turn causes pulmonary edema. The crossunder bitless bridle provides a humane and practical solution to many problems, including 'bleeding.'

REFERENCES AND FURTHER READING

Items are listed chronologically and online links appended

1. Cook, W.R. (1963): "The problem of tongue swallowing". *Veterinary Handbook*, London, England: Stud and Stable
2. Cook, W.R. (1974): Epistaxis in the racehorse. *Equine Veterinary Journal*, 5:45-58
3. Cook, W.R. (1976): "Broken blood vessels in the racehorse: A veterinary research report". *British Racehorse* 38:80-83
4. Cook, W.R. (1981): Some observations on form and function of the equine upper airway in health and disease, Part I: The pharynx. *Proceedings of the 27th Annual Convention of the American Association of*

*Equine Practitioner*s, p. 355-392, Nov.-Dec

5. Cook, W.R., Williams, R.M., Kirker-Head, C.A. and Verbridge, D.J. (1988): Upper airway obstruction (partial asphyxia) as the possible cause of exercise induced pulmonary hemorrhage in the horse: an hypothesis. *Journal of Equine Veterinary Science*, 8:11-26
6. Cook, W.R. (1988): Hypotheses on exercise-induced pulmonary hemorrhage in horses. *Journal of the American Veterinary Medical Association*, 193:8-10 (Letter to editor)
7. Cook, W.R. (1989): "*Specifications for Speed in the Racehorse; the Airflow Factors*". Russell Meerdink, Inc., Menasha, WI
8. Cook, W.R. (1990): "Ban Lasix and find cause of bleeding." *Thoroughbred Times*, 6:18
9. Cook, W.R. (1992): Etiology of exercise-induced pulmonary hemorrhage in the horse. *Journal American Veterinary Medical Association*, October (letter to the editor).
10. Cook, W.R. (1992): Questions theory on cause of exercise-induced pulmonary hemorrhage. *Journal American Veterinary Medical Association*, 201:1661-1662
11. Cook, W.R. (1993): Exercise-induced pulmonary hemorrhage in the horse is caused by upper airway obstruction. *Irish Veterinary Journal*, 46:160-161
12. Cook, W.R. (1993): "Don't abandon the facts." *Thoroughbred Times* 9: #14 April 2
13. Cook, W.R (1996): "Another view on bleeding; asphyxia is suggested as the cause of pulmonary edema" *Thoroughbred Times*, p52, November 9
14. Verbridge, D.J., Cook, W.R., and Kirker-Head, C.A. (1996): Measurement and comparison of cervical tracheal pressures in two exercising horses. *The Equine Athlete* 9, 16-19
15. Cook, W.R. (1998): Use of the Bit in Horses. *Veterinary Record* February to November, Letters to the Editor. http://www.bitlessbridle.com/Use_of_the_Bit_in_Horses-Veterinary_Record.pdf
16. Cook, W.R. (1997): EIPH or AIPE? *Equine Athlete* March/April, p23 http://www.bitlessbridle.com/EIPH_or_AIPE.pdf
17. Cook, W.R. (1998): Breakdowns, bleeding and bits. *Thoroughbred Times.* 5 Sept, p 21 http://www.bitlessbridle.com/Article-2.pdf
18. Cook, W. R. (1999): Why do Horses' Lungs Bleed? http://www.bitlessbridle.com/Why_do_horses_lungs_bleed.pdf
19. Cook, W.R. (1999): "Asphyxia as the cause of bleeding and the bit as the cause of soft palate displacement" Guest Commentary *Thoroughbred Times,* November 27, pp18-19 http://www.bitlessbridle.com/asphyxia.pdf
20. Cook, W.R. (1999): Pathophysiology of Bit Control in the Horse. *Journal Equine Veterinary Science* 19: 196-204 http://www.bitlessbridle.com/pathophysiology.pdf

21. Cook, W.R. (1999): The ear, the nose, and the lie in the throat. In *"Guardians of the Horse: Past, Present and Future."* Ed: Rossdale, P.D., Greet, T.R.C., Harris, P.A., Green, R.E., and Hall, S... British Equine Veterinary Association and Romney Publications, pp 175-182. http://www.bitlessbridle.com/The_Ear_Nose_Lie_in_Throat.pdf

22. Cook,W.R (2000); A note on the head bob of the galloping horse at liberty and the harmful effect of the bit on the head bob http://www.bitlessbridle.com/Article-3.pdf

23. Cook, W.R. (2001): "The penalties of the bit and the benefits of bitlessness" *Pferdegesunde*

24. Jahiel, J (2001): What is this new Bitless Bridle? *HorseCity.com* http://www.bitlessbridle.com/Article-5.pdf

25. Peters, R (2001): Who Needs Bits? *Natural Horse, 3, 44-47* http://www.bitlessbridle.com/Who_Needs_Bits.pdf

26. Cook,W.R (2001): On Talking Horses; Barefoot and Bit-free *Natural Horse,* 3,19 http://www.bitlessbridle.com/ON_TALKING_HORSES.pdf

27. Cook, W.R. (2002) The Horse's Bit: A Bronze age anachronism and cause of many an idiopathic problem. http://www.bitlessbridle.com/Article-8.pdf

28. Cook, W.R. (2002): Bit-induced asphyxia in the horse: Elevation and dorsal displacement of the soft palate at exercise. *Journal of Equine Veterinary Science,* 22, 7-14 http://www.bitlessbridle.com/Article-6.pdf

29. Cook, W.R. (2002): "No bit is best." *Thoroughbred Times,* October 19, p 18 http://www.bitlessbridle.com/NO_BIT_IS_BEST.pdf

30. Cook,W.R (2002): Not Snoring but Suffocating: Bit-induced Asphyxia. http://www.bitlessbridle.com/NOT_SNORING_BUT_SUFFOCATING.pdf

31. Cook,W.R (2002): On Mouth Irons, 'Hoof Cramps,' and the Dawn of the Metal-Free Horse. *Natural Horse,* 4, 37-40

http://www.bitlessbridle.com/mouth_irons_and_hoof_cramps.pdf

32. Cook,W.R (2002): The effect of the Bit on the Behavior of the Horse http://www.bitlessbridle.com/Article-8.pdf

33. Cook, W.R. and Strasser, H (2003): "*Metal in the Mouth: The Abusive Effects of Bitted Bridles."* Sabine Kells, Qualicum Beach, BC Canada

34. Cook, W. R (2003): Bit-induced pain: A cause of fear, flight, fight and facial neuralgia in the horse. *Pferdeheilkunde* 19,1 Jan/Feb. English with German summary http://www.bitlessbridle.com/2003%20Pferdeheilkunde.pdf

35. Cook, W.R. (2005): Treatment for dorsal displacement of the soft palate in horses. *Veterinary Record,* December 3, 157, 752 http://www.bitlessbridle.com/Aetiology_of_DDSP.pdf

36. Cook, W. R. (2005) : Rational for allowing the crossunder bitless bridle for

racing http://www.bitlessbridle.com/JC_RACING_presentation.pdf

37. Cook, W. R (2005): Risk & Safety Factors in Flat & Steeplechase Racing Associated With All Bitted Bridles, Compared With the Predicted Risk of Using the Crossover Bitless Bridle. http://www.bitlessbridle.com/BB_SAFETY_FOR_RACING.pdf
38. Cook,W.R (2005): Sore Mouths in Horses http://www.bitlessbridle.com/SORE_MOUTHS.pdf
39. Cook,W.R (2005): Teeth, Bones and Brushing. *Veterinary Record. 156, 751* http://www.bitlessbridle.com/Vet_Rec_Teeth_bones_brushing.pdf
40. Cook,W.R (2005): Horsemanship: Art or Science? *Western Farm, Ranch and Dairy* Fall/Winter edition 6, p43 http://www.bitlessbridle.com/What_makes_a_fast_horse_fast.pdf
41. Cook,W.R (2005): What makes a fast horse fast? A small and complex answer to a large and simple question. http://www.bitlessbridle.com/What_makes_a_fast_horse_fast.pdf
42. Van Daalen, T (2006): Use of a Bit = Abuse of a Horse. *Bit (The Netherlands)* 131, pp16-19, April. http://www.bitlessbridle.com/Van_Daalen_BIT_journal_Use_and_Abuse.pdf
43. Cook,W.R (2006): Tradition and the Status Quo or Science and Advance? A welfare reform currently denied. *Veterinary Times* Nov/Dec http://www.bitlessbridle.com/Tradition%20or%20Science.pdf
44. Cook,W.R (2006): The Evolution of Bitless Equitation. *http://www.bitlessbridle.com/Evolution_of_bitless_equitation.pdf*
45. Cook, W.R. (2007): "Bit-Induced Fear part 1: From Human hand to Horse's Head. *Veterinary Times,* Volume 37, No. 12, April 9, pp 18-23. http://www.bitlessbridle.com/dbID/210.html
46. Cook, W.R. (2007): "Bit-Induced Fear part 2: Bits and Diseases they can Cause. *Veterinary Times,* Volume 37, No. 14, April 30, pp 22-25.
47. Cook, W.R. (2007): "Bit-Induced Fear part 3: Use of the Bit and bad Behaviour. *Veterinary Times,* Volume 37, No. 19, May 28, pp 36-38.18
48. Cook,W.R (2007): Epiglottal entrapment http://www.bitlessbridle.com/dbID/396.html
49. Cook,W.R (2007): Why is a Bit an Impediment to a Horse? http://www.bitlessbridle.com/dbID/302.html
50. Cook, W.R. (2008): "Metal, Myth and Equine Misery." Animal Welfare Science, Ethics and Law Veterinary Association Journal, January http://www.bitlessbridle.com/dbID/380.htmlook
51. Cook, W. R. (2008) : Report on a six-month trial of the crossunder bitless bridle (CBB) for flat race and steeplechase training in the UK http://www.bitlessbridle.com/HRAreport.pdf

52. Cook, W. R. (2009): Prevention of Accidents to Riders caused by Tack: Feel it, log it, fix it." Published online at www.bitlessbridle.com http://www.bitlessbridle.com/dbID/419.html
53. Cook,W.R (2009): On the Bit: A misleading and mischievous phrase http://www.bitlessbridle.com/on_the_bit.pdf
54. Cook, W.R. (2009): The Crossunder Bitless Bridle's Bibliography: Evidence submitted to the USEF Dressage Committee in September, 2009 in support of a rule change proposal to permit the crossunder bitless bridle for dressage. http://www.bitlessbridle.com/CBBbibliography.pdf
55. Cook, W.R. and Mills, D.S (2010): "Preliminary Study of Jointed Snaffle vs. Crossunder bitless bridles in four horses." *Equine Veterinary Journal.* Vol. 41, 827-830 http://www.bitlessbridle.com/CHAexperiment.pdf
56. Cook, W.R. (2011): "Damage by the bit to the equine interdental space and second lower premolar." *Equine Veterinary Education, 23, 355-360* http://www.bitlessbridle.com/DamageByTheBit.pdf
57. Cook, W.R. (2011): "What Causes Soft Palate Problems and Bleeding in Racehorses? The answer is on the tip of your horse's tongue." "Horses For Life, Volume 62 http://www.bitlessbridle.com/SoftPalateAndBleeding.pdf
58. Bergstein, Stan (2011): "Furosemide could be outmoded by equipment." Daily Racing Form, 6/7/2011
59. Cook,W.R (2011): The Pain-Free Harness Horse: Promoting Performance by Demoting the Bit http://www.bitlessbridle.com/PainFreeHarnessHorse.pdf
60. Cook,W.R (2011): New Tool Benefit Denied by an Old Rule: Rider/Horse safety trumped by tradition http://www.bitlessbridle.com/NewToolOldRule.pdf