Hypothesis Article

A hypothetical, aetiological relationship between the horse's bit, nasopharyngeal asphyxia and negative pressure pulmonary oedema

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Summary

Tentative explanations for the aetiology of soft palate instability and dorsal displacement at exercise are based on the proposition that it is caused by one or more, as yet unidentified, neuromuscular or inflammatory diseases. As judged by reviews, none of the treatments based on these assumptions are entirely satisfactory. Most researchers agree that their cause is unknown. In 1999, Cook proposed that they were caused by the bit breaking what should be an airtight lip seal. The present article describes a development of this hypothesis – loss of oral compartment subatmospheric pressure. The feral horse is an obligate nose-breather and runs with a closed mouth and no air in the oral cavity and oropharynx. Evacuation of air from these compartments is proposed as the primary mechanism, strengthened by the cohesive force of surface tension, whereby the soft palate resists the elevating force of negative pressure on inspiration. It is suggested that subatmospheric pressure in the oral compartment is generated by a single swallow before running. Poll extension supplements the rigidity of the nasopharyngeal tube and reinforces the *ostium intrapharyngium* seal. In the domesticated horse, the lip seal is broken by a bit and bit-induced poll flexion, jaw and tongue movement often breaks the ostium seal. Oral atmospheric pressure is proposed as the mechanism for nasopharyngeal asphyxia. It is considered that negative pressure pulmonary oedema – currently known as exercise-induced pulmonary haemorrhage and conventionally thought to be a separate problem – is one of its clinical signs. It is suggested that the bit is the most common cause of nasopharyngeal asphyxia and that this, in turn, results in a cascade of dynamic obstructions in the larynx and cervical trachea, leading ultimately to pulmonary oedema and 'bleeding.' It is concluded that these are sequelae to rules in many disciplines that mandate bit usage.

Introduction

Palatal dysfunction is cited as the most common equine upper airway problem (Allen *et al.* 2012). It is a familiar problem in racehorses but also occurs in sport horses (Erck-Westergren 2011). The literature reveals a cart-before-horse situation. Removal of cause is a prerequisite for treatment, yet publications recommending treatment concede that the cause is unknown.

Since 1998, countless horses have been switched overnight from bitted to bitless bridles. Fifteen years of 'natural experiments' have demonstrated behavioural improvement and disease regression after bit removal (Cook 1999, 2000, 2002, 2003, 2005, 2011, 2013; Cook and Strasser 2003; Cook and Mills 2010). A bit is a foreign body in the oral cavity, causing pain and fear. It triggers digestive system responses rather than the respiratory and cardiovascular system responses required for exercise.

The objective of this article is to explore the evidence for the proposition that there is a causal link between the bit, nasopharyngeal asphyxia and negative pressure pulmonary oedema ('bleeding'). This biomechanical hypothesis is evaluated by examining 2 subsidiary hypotheses: 1) that oral compartment pressure in the horse at liberty when running is subatmospheric; and 2) that oral compartment pressure in the bitted horse when exercised is atmospheric and causes nasopharyngeal asphyxia.

The term 'nasopharyngeal asphyxia' includes soft palate dysfunction, instability, elevation and dorsal displacement. The term 'oral compartment' describes the conjoined oral cavity and oropharynx.

Materials and methods

The hypotheses are founded on the evidence of physics, anatomy, physiology and 'one-medicine'.

Results

In support of Hypothesis 1

1. When air is eliminated between two objects, they are held together by atmospheric pressure. This is the principle of the suction cup, which, it is proposed, applies to the horse's throat when running – the 'two objects' being root of tongue and soft palate. The cohesive force of surface tension would reinforce the adhesion. Nasopharyngeal asphyxia does not occur in the horse at liberty. Even though nasopharyngeal pressure when running will be slightly below atmospheric on inspiration and slightly above on expiration, the pressure difference between the oropharyngeal and nasopharyngeal compartments is not sufficient to bring about their communication.
2. The horse is an obligate nose-breather and its physiology when running seems geared towards excluding air from the oral compartment. The feral horse runs with sealed lips, a closed mouth and no slobbering. The pharynx is presumed to be in its respiratory configuration (soft palate apposed to root of tongue). The oral compartment is sealed caudally at the soft palate's elastic 'buttonhole,' the *ostium intrapharyngium* (Cook 1981). The ostium fits

snugly around the abducted larynx and the seal is further secured by extension of the poll applying longitudinal tension on the nasopharyngeal tube.

3. As deglutition in man and other mammals evacuates air from the oral compartment (Engelke *et al.* 2011) it is probable that the same occurs in the horse. This would explain why swallowing seems to be a precursor to running.
4. Pharyngeal radiography shows that the oral compartment contains no air.
5. 100% patency of the nasopharynx when running would be favoured by zero patency of the oropharynx, a closed mouth and immobile tongue.
6. The *tensor palati* muscle is the only soft palate depressor. Even if this acts normally during exercise in the bitted horse (which it may not because of *levator palati* action being triggered by the bit), it acts on the rostral third of the palate only. There has to be some other mechanism for depressing the caudal two-thirds.
7. Oral subatmospheric pressure is documented in mammals. Studies of suckling in monkeys and pigs showed that liquid moved out of the nipple as the intraoral space was expanded by tongue movement and jaw opening (German *et al.* 1992). The same authors advanced the hypothesis *'that all mammals use a negative pressure suction for acquisition. . .'*
8. To drink, a horse depends on subatmospheric pressure in its oral compartment. With head down and lips immersed, a small midline orifice opens in the midline. Retraction of the tongue enlarges the oral cavity, creates a vacuum and draws-up water. At a critical intake, deglutition occurs; subatmospheric pressure is re-established; and the cycle continues. The ability to generate an oral subatmospheric pressure by swallowing would also facilitate airflow when running.
9. Oral subatmospheric pressure in the running horse would make ergonomic sense. Evolutionary selection would favour such a parsimonious mechanism for maintaining patency of the nasopharyngeal airway.
10. Anaerobic bacteria are a significant part of the oral flora in mammals. This supports the proposition that, for much of the time, the mammalian oral cavity is devoid of air.
11. Consider what might happen if, when running, oropharyngeal pressure was atmospheric. With air above and below the soft palate, rapid oscillating airflow would set it fluttering like a blanket in a high wind. This is, of course, what often happens in a bitted horse.

In support of Hypothesis 2

1. Even one bit breaks the lip seal as does a tongue-tie, whether by pin-hole puncture or frank gaping of the mouth. Oropharyngeal atmospheric pressure would elevate the soft palate during rapid inspiration, as would nasopharyngeal subatmospheric pressure.
2. The bit contacts the apex of the tongue, its most sensitive part, triggering its retraction. As the tongue is a muscular hydrostat, if the apex is retracted and the body compressed by a closed jaw, its root will expand, elevate the soft palate and break the ostium seal (**Figs 1** and **2**).
3. An open mouth releases the body of the tongue from compression and allows the horse greater freedom to evade the bit by moving its tongue. Because tongue and larynx are both anchored to the hyoid apparatus, movement of the tongue moves the larynx and this would tend to disrupt the ostium seal.
4. Bit-induced poll flexion when running (itself physiologically contraindicated) by eliminating longitudinal tension on the nasopharyngeal tube, would weaken the ostium seal and allow air into the oropharynx (**Fig 3**).
5. A bitted horse swallows at intervals when running. As deglutition interrupts breathing this is pathophysiological. Deglutition may be necessary to prevent inhalation of bit-induced saliva or it may simply be an attempt to maintain the airway by restoring oral subatmospheric pressure.
6. For racehorse 'wind' examinations, I used to instruct riders to bring their horse to rest in front of me as quickly as possible after the gallop. This enabled me to hear persisting respiratory noise and palpate the larynx for fremitus. I noted that horses invariably swallowed once on coming to rest. Many a horse stopped making an inspiratory noise the moment it swallowed. I now interpret this swallow as the horse's need to maximise its airway by restoring oral subatmospheric pressure.
7. The sphenopalatine branch of maxillary nerve V supplies sensory innervation to the soft palate. As my experience tells me that referred pain from the bit is a trigger for the facial pain of trigeminal neuralgia and a common cause of the headshaking syndrome (Cook 2003), it could also refer pain to the soft palate. If so, gag reflexes when running could be yet another mechanism for disturbing the ostium seal.
8. Epiglottal entrapment, seldom seen in nonracehorses, is characterised by freely mobile oropharyngeal mucosa forming a hood on the dorsal surface of the epiglottis (**Fig 4**). In my opinion, this occurs because an open ostium seal exposes the oropharynx to the negative pressure of inspiration. As a result, oropharyngeal mucosa gets dragged into the airway. I consider epiglottal entrapment to be evidence of repeated loss of oral subatmospheric pressure.
9. Racing, the discipline in which nasopharyngeal asphyxia is most common, is also the discipline in which respiratory rates are highest, inspiratory negative pressure greatest and nasopharyngeal dynamic collapse most likely.
10. Within racing, nasopharyngeal asphyxia is most common in harness horses. Whereas in flat racing one bit is more usual, harness horses commonly have two. Furthermore, with the driver's mechanical advantage of long reins and a firm seat, harness horses are subjected to even more bit pressure than Thoroughbreds. The lip seal is jeopardised by an open mouth and the ostium seal by poll flexion.
11. When swallowing, soft palate elevation opens the pharyngeal orifice of the auditory tubes and maintains atmospheric pressure in the middle ear. If palatal elevation opened the pharyngeal orifices when running, the cartilages would act as a bicuspid valve, further obstructing the nasopharynx on inspiration (**Fig 10**).
12. A cascade of aerodynamic problems will affect all sections of the upper airway caudal to a primary obstruction. With soft palate elevation, obstruction will start at the choanae (**Fig 5**). Once elevated, even a little, the Venturi effect will further increase inspiratory negative pressure, which, in turn will exacerbate the elevation. The soft palate will tend to close the choanae, acting like a

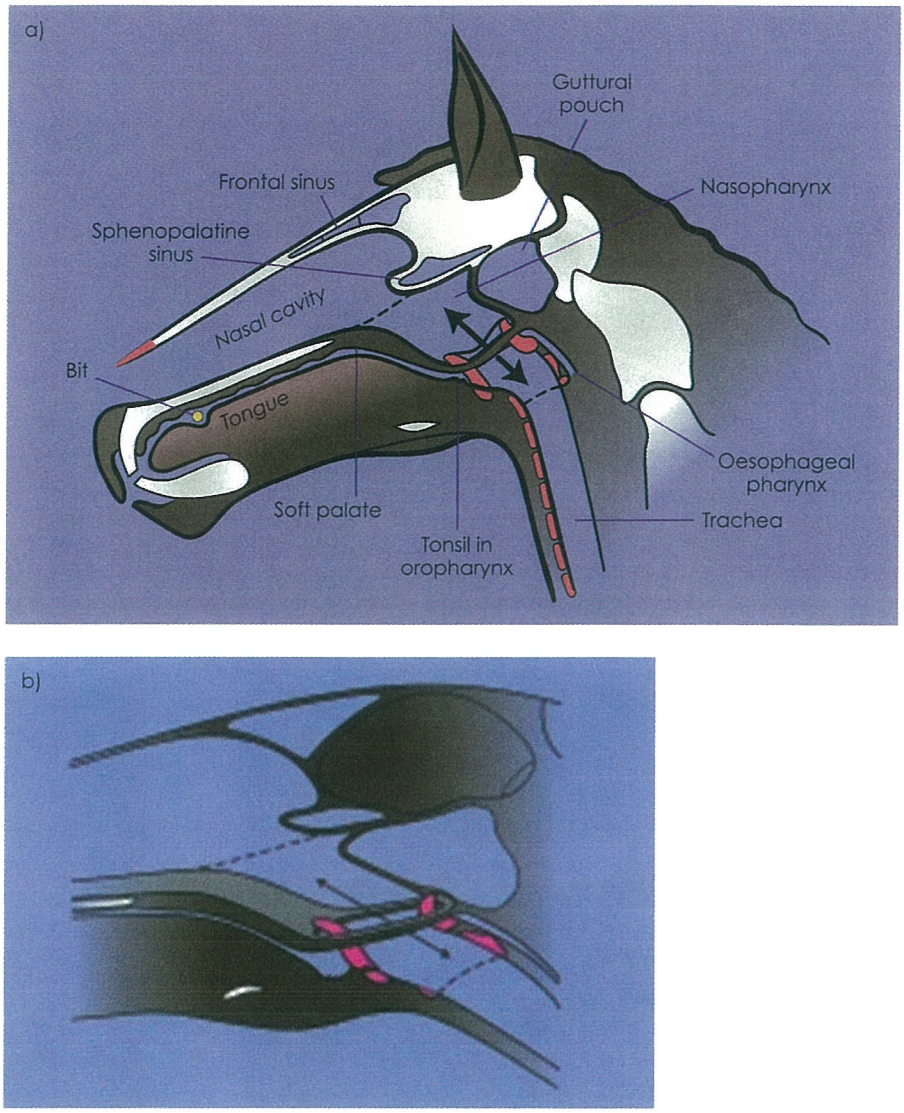

Fig 1: a) Pharyngeal anatomy: when the lip seal is broken by the bit, air will be present in the oral compartment, laryngopharynx (not shown) and oesophageal pharynx. The broken line indicates the position of the choanae. The most rostral portions of the choanae are obstructed by a slight soft palate elevation. b) Perspective view of the soft palate's ostium intrapharyngium a moment prior to dorsal displacement, with air in the oropharynx.

flapper valve. Caudally, unsupported sections of the airway can be expected to undergo dynamic collapse. Affected structures will include the roof of the nasopharynx and its floor (the soft palate), aryepiglottic folds, epiglottis, arytenoid cartilages (especially on the left in horses with recurrent laryngeal neuropathy), vocal cords and the dorsal membrane of the cervical trachea. Terminally, both lungs will incur barotrauma.

13. Repeated dynamic collapse of the dorsal membrane of the trachea explains, in my opinion, the permanent deformity of the tracheal cartilages that I have seen so frequently at *post mortem* examination (**Fig 6**). These changes, like epiglottal entrapment, provide tangible evidence of repeated episodes, when running, of oral atmospheric pressure and nasopharyngeal asphyxia. The progressively more severe deformity of individual rings along the course of the cervical trachea is, I consider, a physical manifestation of the laws of gas flow along tubes. The force of negative pressure on inspiration increases with distance from the source of obstruction (Cook *et al*. 1988). This would explain why the cervical trachea is most severely deformed at the thoracic inlet and why pulmonary bleeding is both bilaterally symmetrical and caudally distributed.
14. A horse exhibiting clinical signs consistent with nasopharyngeal asphyxia when exercised in a bitted bridle is less likely to exhibit these signs in a bitless bridle (Cook 2013). For example, I examined a 2-year-old Thoroughbred that had made an inspiratory noise since first schooled as a yearling and found it to have no significant recurrent laryngeal neuropathy. However, inspiratory stridor occurred at exercise in a snaffle bridle and laryngeal fremitus was palpable. Minutes later, when the same exercise test was repeated with the same rider but with a bitless bridle, there was neither inspiratory stridor nor laryngeal fremitus.
15. An ability to refute competing hypotheses for the aetiology of nasopharyngeal asphyxia adds support as follows:
    (a) *Neuromuscular disease of the pharynx*: Horses that exhibit nasopharyngeal asphyxia do not typically exhibit dysphagia. As deglutition is a neurologically demanding manoeuvre, I maintain that normal swallowing indicates normal neurology.

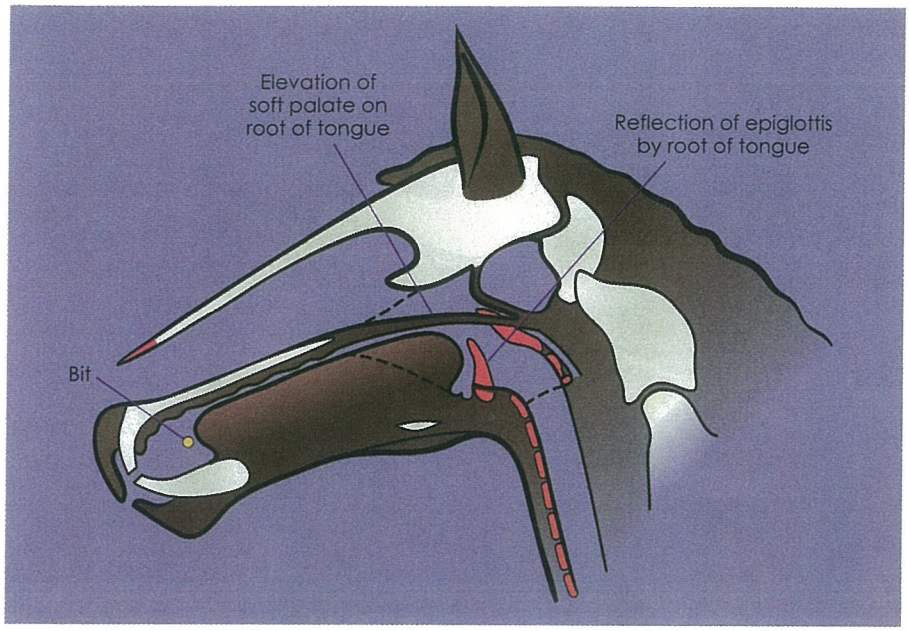

Fig 2: Effect of bit-induced tongue retraction. Caudal bulging of the root of the tongue elevates the soft palate, obstructs the choanae and partially closes the aditus laryngis.

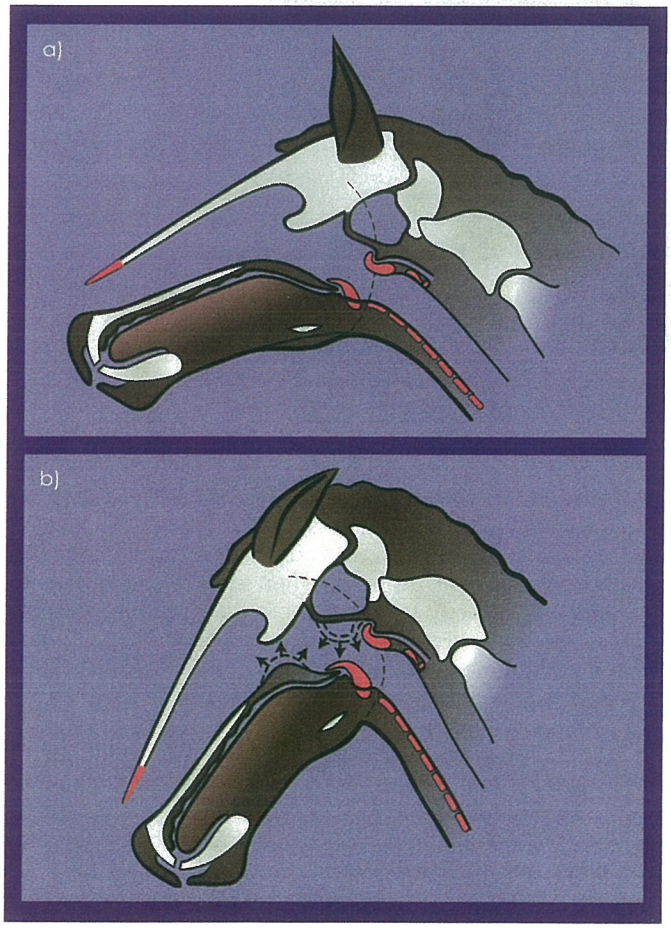

Fig 3: Effect of poll extension (a) and flexion (b). Extension increases nasopharyngeal patency. Flexion decreases it and leads to dynamic collapse on inspiration (arrows).

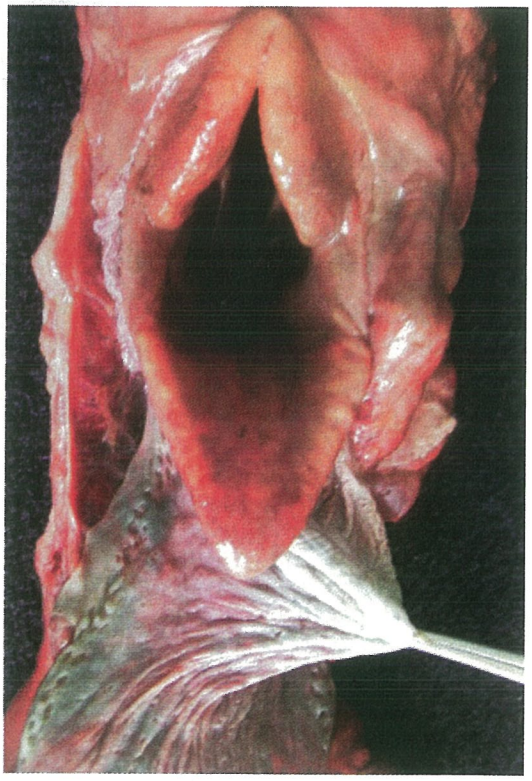

Fig 4: Freely mobile oropharyngeal mucosa. When the ostium intrapharyngium seal is broken it is this tissue that gets sucked over the dorsal face of the epiglottis, constituting epiglottal entrapment.

(b) *Response to treatment*: After a systematic review of the efficacy of 23 interventions for nasopharyngeal asphyxia (predominantly surgical), the authors concluded that *'it is currently not possible to determine which procedure is the most appropriate* (Allen *et al*. 2012).' Although this is not a refutation of

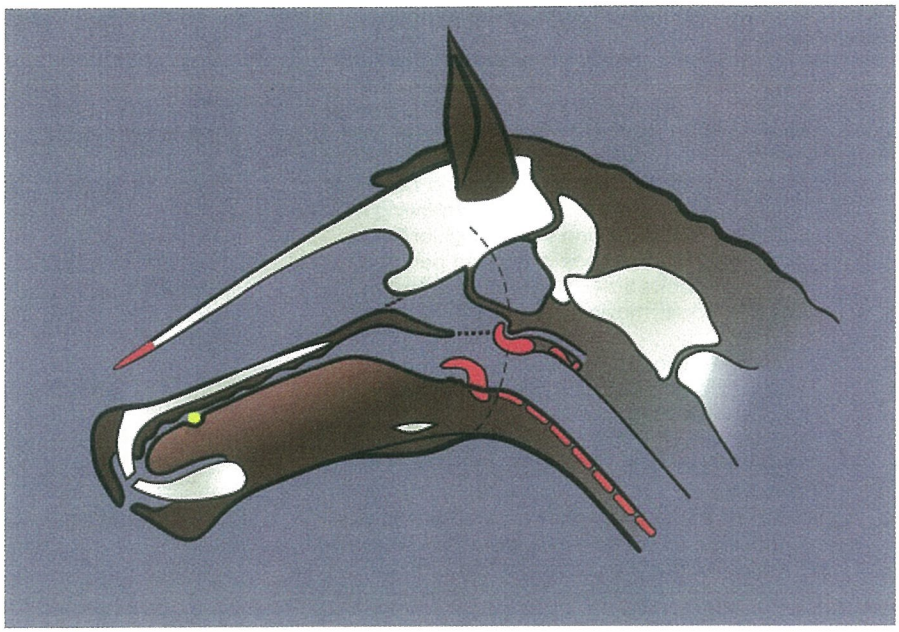

Fig 5: Dorsal displacement of the soft palate in a bitted horse. The rostral half of the soft palate is almost completely blocking the choanae. The nasopharyngeal tube, instead of being smoothly continuous with adjoining sections of the upper airway has developed a narrow orifice at both ends (broken lines). A complete blockage of the choanae could occur during exercise, as it does during deglutition. Neither partial nor complete blockage have been observed during endoscopy studies published to date, as – with the endoscope's distal end in the nasopharynx – choanal viewing is impossible. A different endoscopy protocol is needed to collect choanal data.

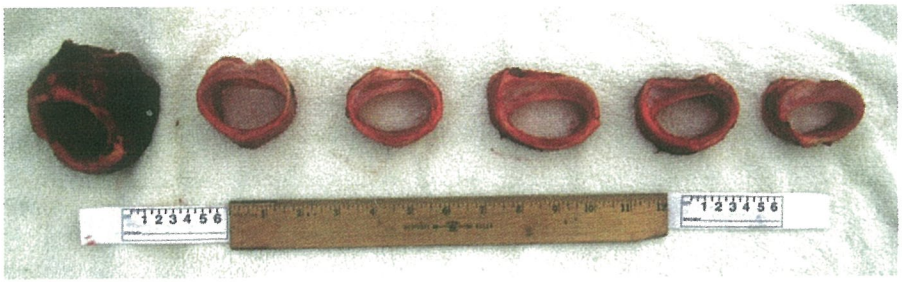

Fig 6: Progressive restriction of the tracheal lumen. From left to right, the cricoid cartilage is followed by cross-sections through cervical tracheal rings 1, 9, 18, 27 and 36, of a racehorse. Separation of the dorsal membrane from each tracheal ring constitutes an obstruction in itself but renders the lumen liable to additional (dynamic) collapse during rapid inspiration. In a normal cervical trachea, the free ends of the tracheal rings are described in anatomy texts as overlapping but this was not so for any ring in this specimen.

the tentative aetiologies on which these interventions were based, neither is it an endorsement of any one.

(c) *Pharyngeal lymphoid hyperplasia and inflammatory airway disease*: These have been associated with nasopharyngeal asphyxia (Erck-Westergren 2011) but a cause and effect relationship has not been established. Pharyngeal lymphoid hyperplasia is normal in young horses. Inflammatory airway disease could, in my opinion, be the effect (not cause) of repeated episodes of nasopharyngeal asphyxia and its sequel, pulmonary bleeding.

16. Allen and Franklin (2012) report morphological evidence during soft palate instability prior to dorsal displacement of the soft palate (DDSP) consistent with oral compartment atmospheric pressure, i.e. a flattened epiglottis and convexity in the most caudal section of the soft palate.
17. Kelly *et al*. (2013) reported that from 57 Thoroughbred yearlings examined with dynamic endoscopy when lunged, 18 (33%) exhibited DDSP. As the lunge rein was attached to the bit (P.G. Kelly, personal communication 2013), it is my opinion that this high prevalence of nasopharyngeal asphyxia in an otherwise healthy population can be most simply attributed to atmospheric pressure in the oral compartment.
18. Arguments that refute competing hypotheses for pulmonary bleeding support *Hypothesis 2* (Cook 2014).

Hypotheses are more rigorously tested by attempts to falsify them than by mustering evidence in their support. I invite colleagues to refute *Hypothesis 1* or 2 by showing that the results of the following tests do not agree with my predictions.

Tests to refute Hypothesis 1

See **Table 1**.

Tests to refute Hypothesis 2

For *Tests 1–7*, see **Table 2**. For *Tests 8–10* see below.

Test 8. Observe horses at ridden exercise, both fast and slow, in a variety of bits. If lips are constantly sealed, this would refute *Hypothesis 2*.

Test 9. High-speed treadmill studies have demonstrated that DDSP occurs in bitless horses. This might appear to refute the implication in *Hypothesis 2* that the bit is the major cause of nasopharyngeal asphyxia during 'normal' (ridden) exercise. However, endoscopy studies are not studies of normal exercise

TABLE 1: Tests for attempting refutation of *Hypothesis 1*: that oral compartment pressure in the horse at liberty when running is subatmospheric

No.	Test	Question	Prediction
1	Open a horse's mouth for a moment and allow it to close	Does a swallow follow?	Yes
2	Exercise an unbridled horse in a round pen or on a lunge line so that evidence of swallowing may be observed by watching the left jugular furrow.	At any pace, does the horse swallow?	No
3	Allow a horse to eat hay in a round pen and, before a bolus is swallowed, require him to run.	Before he starts to run, does he close his mouth and swallow?	Yes
4	Observe horses moving at liberty, at all paces	Are lips sealed and dry, and jaws and tongues immobile?	Yes
5	A 3-stage radiography study:		
i	Take a lateral view radiograph of the pharynx in an unsedated horse at rest	Is there air in the oropharynx?	No
ii	Apply a mouth gag and take a second radiograph	Is there air in the oropharynx	Yes
iii	Remove gag, wait for swallow and take a third radiograph	Is there air in the oropharynx	No
6	Place an in-dwelling catheter in the mouth and, when the lip seal is once again intact and the horse has swallowed, use a 50 ml syringe to inject measured incremental volumes of air into the oral compartment. Observe its effect using fluoroscopy.	Does the horse eventually swallow?	Yes
7	Using fluoroscopy, inject a small volume of air into the oral compartment as above then manually extend and flex the poll	Does head position influence the location of the pocket of air?	Yes

TABLE 2: Tests for attempting refutation of *Hypothesis 2*: that oral compartment pressure in the bitted horse when exercised is atmospheric and causes nasopharyngeal asphyxia

No.	Test	Question	Prediction
1	During the pharyngeal fluoroscopy *Test 6* (**Table 1**)	Does palatal instability occur?	Yes
2	Using pharyngeal fluoroscopy with a horse in a snaffle bridle, explore the effect of applying different degrees of rein pressure	Does palatal instability correlate with increased rein pressure?	Yes
3	Listen to those horses that, during training exercise in a bitted bridle, regularly develop signs consistent with nasopharyngeal asphyxia (inspiratory stridor), yet on the basis of prior examination reveal no obvious cause. Repeat the same exercise and listening test after removing the bit.	Does inspiratory stridor continue? (Note: Stridor caused by a significant degree of recurrent laryngeal neuropathy may result in the persistence of a less noisy stridor. Recurrent laryngeal neuropathy as the cause can be ruled-out on the basis of laryngeal palpation and laryngoscopy)	No
4	Persuade a jurisdiction to allow a bitless bridle for racing	Does the prevalence of nasopharyngeal asphyxia and 'bleeding' decline?	Yes
5	Using overground endoscopy to observe the choana and rostral nasopharynx, stage the same exercise test on horses with and without a bit, ridden/driven by the same rider/driver, over the same terrain, under similar environmental conditions	Is the degree of palatal instability less when bitless?	Yes
6	Using overground choanal endoscopy as above, compare the prevalence and degree of palatal instability in two groups of sport horses, one bitted and one bitless.	Is the degree of palatal instability less when bitless?	Yes
7	Using overground choanal endoscopy, compare the patency of the nasopharynx in dressage horses during a warm-up routine, first with their nasal bone vertical to the ground and then in the hyperflexed (Rollkur) position.	Is patency of the nasopharynx better when the nasal bone is vertical to the ground?	Yes

any more than are bitted studies. An inherent feature of both treadmill and overground studies is that the data collection method, endoscopy, itself introduces an artefact. An endoscope interferes with airflow by obstructing the nostril, ventral meatus, choana and nasopharynx (**Figs 7–9**). Even if an endoscope of 10 mm external diameter occupied only half the cross-sectional area of the choana it would increase resistance to flow by a factor of 16. For example, a catheter of 6 mm, external diameter, decreased airflow through the nostril by 17.7% and increased the work of breathing (Art *et al.* 1990).

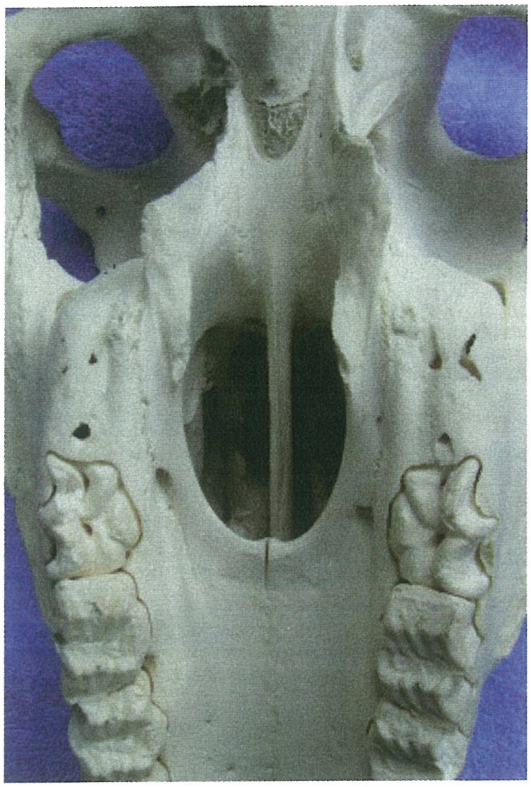

Fig 7: Choanal anatomy: skeletal boundaries.

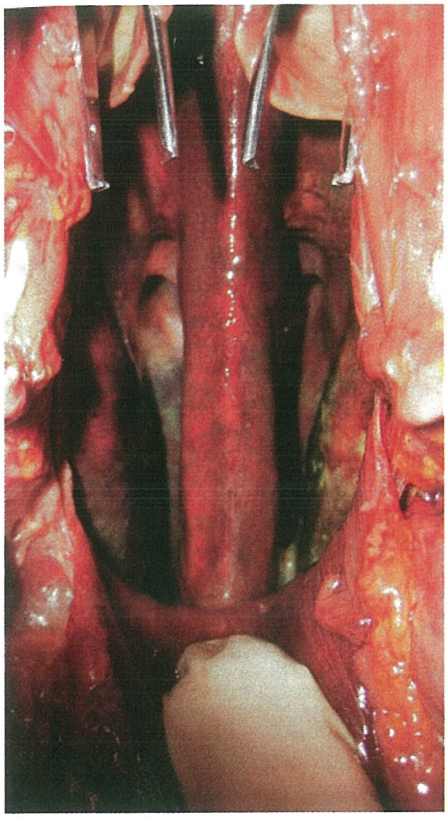

Fig 8: Choanal anatomy: mucosal boundaries; forceps protrude from each pharyngeal orifice.

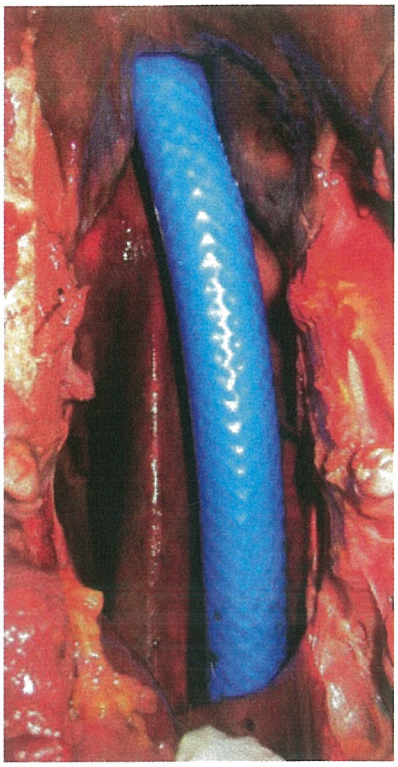

Fig 9: An intranasal tube of 20 mm external diameter occludes a Thoroughbred's choana. The distal end of the tube (top) is lodged in the pharyngeal recess.

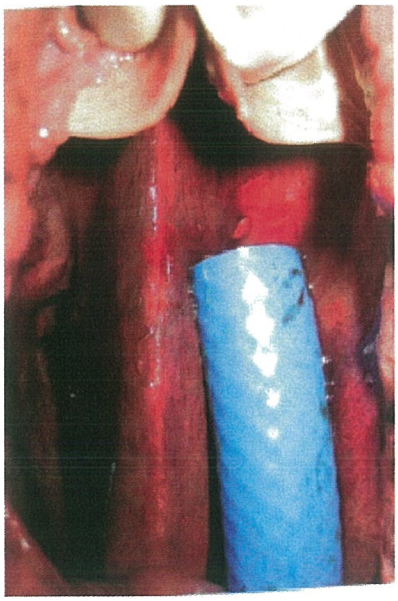

Fig 10: View of the choana as in Figure 9 but with the intranasal tube partly withdrawn. Mild digital pressure is being applied, in a dorsal direction, on the ventral edge of the medial cartilages of the guttural pouch pharyngeal orifices, causing them to buckle open and meet in the midline like a bicuspid valve. As elevation of the soft palate opens the pharyngeal orifices during deglutition, I contend that dorsal displacement of the soft palate when running is likely to have the same effect. Gaping of the medial cartilages would further obstruct the nasopharynx.

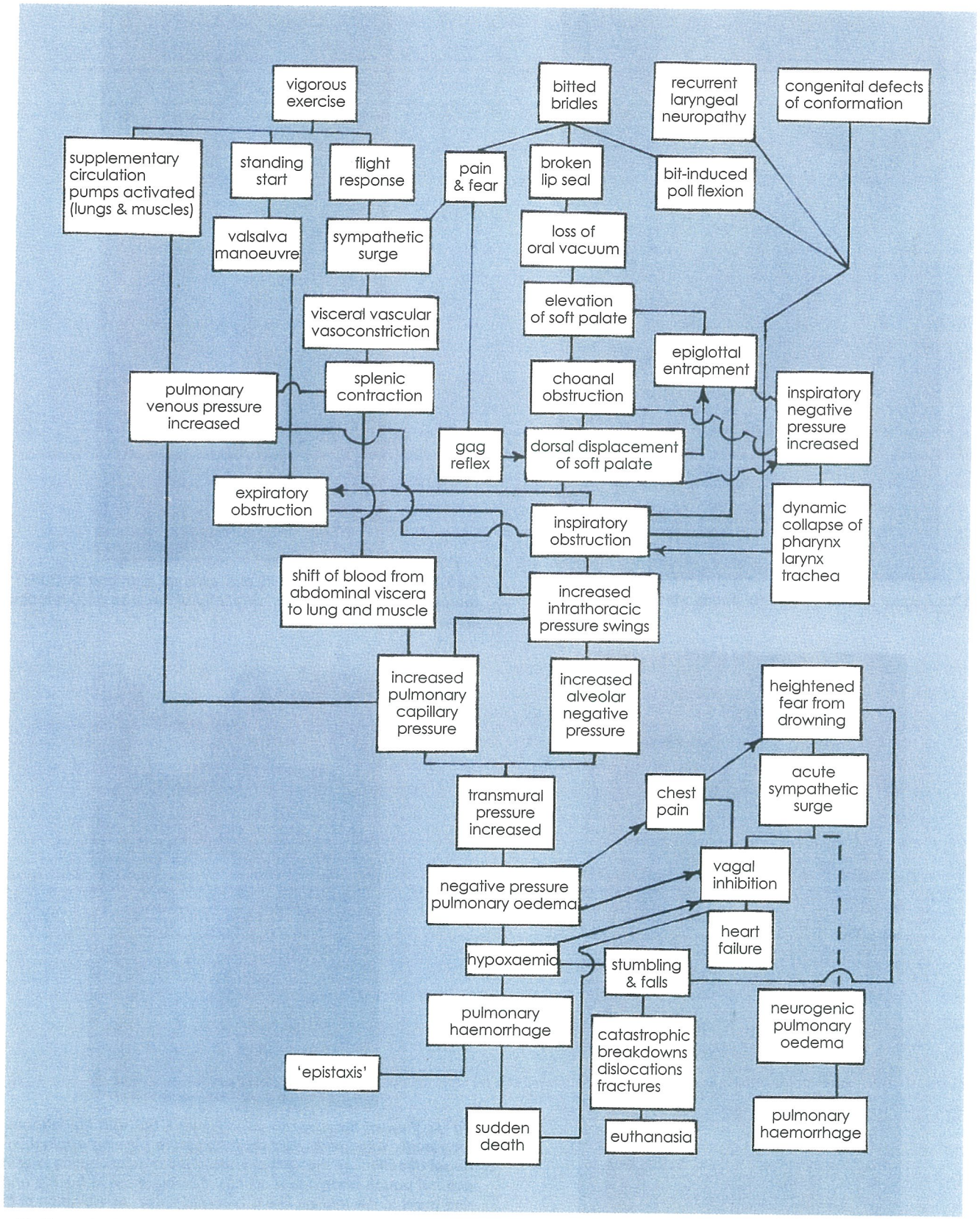

Fig 11: Flow chart summarising what I believe to be the relationships between the bit, nasopharyngeal asphyxia, negative pressure pulmonary oedema and sudden death. At top right of the chart, 'congenital deformities of conformation' include stenosis or atresia of the choanae, cleft soft palate and pharyngeal arch defects. All these are rare and affected horses are seldom put into training. At bottom right of the chart, a broken line to a disease in man called neurogenic pulmonary oedema indicates a possible connection that has yet to be studied in the horse. A specific term for heart failure caused by pulmonary disease is cor pulmonale.

The distal end of an endoscope in the nasopharynx must also interfere with airflow from the patent nostril. Its movement could trigger gag reflexes. These impediments would generate turbulence, increase nasopharyngeal negative pressure on inspiration and open the ostium seal. In the absence of a bit, an endoscope can still cause DDSP.

Test 10. With overground endoscopy in a bitted bridle, the lip seal will also be broken. A feature of all the overground endoscopy studies and many of the treadmill studies published to date is that the horses were bitted. With such a protocol, I submit that iatrogenic episodes of nasopharyngeal asphyxia can be expected, there being foreign bodies in both mouth and nose.

Conclusion

Currently, the hypothesis that the bit causes pulmonary oedema is not refuted. A flow chart summarises what I believe to be the consequences of bit usage (**Fig 11**).

In my opinion, nasopharyngeal asphyxia is the cause of exercise-induced pulmonary haemorrhage (EIPH), which is analogous to a rare but life-threatening disease in man known as negative pressure pulmonary oedema (NPPE) (Bhaskar and Fraser 2011; Cook 2014). NPPE in man is caused by upper airway obstruction and is, I submit, a model for exercise-induced pulmonary haemorrhage. It would advance the understanding and prevention of this serious problem in the horse if it too was known as NPPE.

Discussion

I propose that the bit is the predominant cause of nasopharyngeal asphyxia and that NPPE is one of its signs. Contributory factors include variations in usage, respiratory rates, rein tension, poll flexion and a horse's pain threshold.

Potential relevance: Unless the hypothesis can be refuted a cause will have been suggested (the bit) and a basis for treatment indicated (its removal) for nasopharyngeal asphyxia and NPPE. As sequelae to these common diseases include hypoxaemia, exhaustion, breakdowns, falls, fractures and sudden death, the article is relevant to:

- the welfare and safety of horse and rider/driver
- the public image of racing
- the furosemide debate
- poor performance

Medical and surgical interventions do not remove the cause of nasopharyngeal asphyxia. Until at least one racing jurisdiction permits the use of a bitless bridle, removal of the bit cannot be tested under racing conditions but it can be and has been tested during training.

Author's declaration of interests

W.R. Cook is Chairman and major shareholder of Bitless Bridle Inc. and owns a patent on the crossunder bitless bridle.

Source of funding

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